

Mary K. Mulcahey, John W. McNeil II,
and Matthew T. Provencher

Epidemiology

The shoulder is the most commonly dislocating joint in the human body, often occurring in the anterior direction. Stability of the shoulder joint is provided by three primary mechanisms [1]: (1) concavity compression, (2) compression of the humeral head onto the glenoid by the rotator cuff muscles, and (3) glenohumeral ligaments and capsule. With regard to instability, however, the more relevant anatomy includes the static and dynamic stabilizers of the glenohumeral joint. The static stabilizers consist of the bony anatomy, glenoid labrum, capsular ligaments, and the rotator interval, whereas the rotator cuff and the scapular stabilizers comprise the dynamic stabilizers. The glenoid labrum contributes to shoulder stability in several ways. It serves as an attachment point for the capsule and the surrounding ligamentous structures, effectively anchoring them to the glenoid. The labrum also contributes to the concavity-compression mechanism by increasing the concavity of the glenoid [2]. Glenohumeral dislocation often results in soft tissue (e.g., labral tear, capsular stretching) or bony injuries (e.g., glenoid or humeral head bone loss) and is therefore frequently associated with persistent deficits of shoulder function and a high risk of subsequent instability episodes in young, active patients [3–10].

Recurrent instability can be atraumatic or may occur following a traumatic event and is frequently classified as a subse-

quent dislocation, subluxation event, or persistent apprehension [11, 12]. Patients with recurrent instability may present with chronic pain (<6 months) in abduction and external rotation as their only symptom [13]. In addition, the unstable painful shoulder (UPS) was described by Boileau et al. [14] in 2011 as an indication of unrecognized anteroinferior instability that causes persistent pain in young athletes. These patients have anatomic lesions suggestive of instability; however, there is often no history of recurrent instability episodes [14].

Recent studies report that the rates of recurrent anterior instability following arthroscopic stabilization procedures range from 4 to 18 % [15–20] versus 0–7 % in open stabilization procedures [16, 18], although there remains considerable debate about the optimal treatment strategy. Several risk factors contribute to the rate of recurrent anterior instability following operative stabilization. The most commonly reported risk factors include age of the patient (<age 30=higher risk), capsular stretching, generalized ligamentous laxity, number of anchors used, and participation in contact sports [15, 20–26]. Glenoid or humeral head bone loss has also been identified as a risk factor for recurrent instability [15, 20, 21, 23, 24, 27]. A study by Boileau et al. in 2006 [15] elucidated several factors associated with recurrent instability following arthroscopic Bankart repair. Greater than 25 % loss of the glenoid surface, a large Hill-Sachs lesion, a stretched inferior glenohumeral ligament, and anterior hyperlaxity were all significantly related to failure. This study concluded that patients had a 75 % recurrence rate in the presence of a stretched inferior glenohumeral ligament, anterior hyperlaxity, or a glenoid compression fracture involving more than 25 % of the glenoid surface [15].

In 2007, the instability severity index score (ISIS) was proposed as a means of identifying risk factors associated with recurrent instability [21]. This study identified six risk factors for recurrent instability including age less than 20 years at the time of stabilization; participation in competitive sports, contact sports, or any athletics requiring persistent overhead activity; shoulder hyperlaxity; Hill-Sachs lesion visible on a plain anteroposterior (AP) radiograph with the arm in external

M.K. Mulcahey, MD (✉)
San Diego Arthroscopy and Sports Medicine,
6719 Alvarado Road, Suite 200, San Diego 92120, CA, USA
e-mail: marykmulcahey@gmail.com

J.W. McNeil II, BA
Department of Orthopaedics, Naval Medical Center, San Diego,
34800 Bob Wilson Drive, Suite 112, San Diego 92134, CA, USA
e-mail: john.mcneil.ctr@med.navy.mil

M.T. Provencher, MD, MC, USN
Department of Orthopaedics,
Sports Medicine Service, Massachusetts General Hospital,
175 Cambridge Street, Boston 02114-2723, MA, USA
e-mail: mattprovencher@gmail.com

rotation; or loss of the normal sclerosis on the inferior border of the glenoid on a plain AP radiograph. Patients with a score greater than 6 points had a recurrence rate exceeding 70 %, which led the authors to recommend arthroscopic anterior stabilization only for patients with a score of 6 or less. For those patients with a score of greater than 6 points, an open surgical procedure (i.e., Latarjet) was recommended because of the unacceptably high rate of recurrence [21].

Trauma is a significant risk factor associated with recurrent shoulder instability following stabilization [28–30]. This factor is especially relevant in contact athletes, with the highest rates of recurrence being reported for men’s football, wrestling, and hockey [31]. Contact athletes suffer a much higher rate of failure of stabilization procedures compared to the general population [28, 30, 32]. Cho et al. [28] reported a recurrence rate of 28.6 % in collision athletes versus only 6.7 % in non-collision athletes following arthroscopic stabilization for anterior shoulder instability.

Recurrent instability may occur in the setting of major trauma after the initial repair or may result from minimal force. The etiology commonly involves a soft tissue or bone tissue, and sometimes both. The most commonly reported factors contributing to failure are diagnostic and technical failures, capsular or labral insufficiency, and glenoid or humeral head bone loss, or both [15, 23, 24, 26, 28, 32, 34, 35].

Pathophysiology

It is important to understand the pathology that is commonly associated with anterior glenohumeral instability and to appropriately address these lesions when they are diagnosed.

Lesions of the Glenoid Labrum and Ligamentous Attachments

Bankart Lesion

Injury to the glenoid labrum and associated ligamentous attachments commonly occur following anterior shoulder dislocation. Avulsion of the anterior labroligamentous structures from the anteroinferior glenoid rim is known as the Bankart lesion (Fig. 16.1). This is often considered the “essential lesion” of anterior shoulder instability, with 90 % of all anterior shoulder dislocations having associated Bankart lesions [36]. The labrum and attached ligaments are often found anterior to the glenoid rim. The inferior and middle glenohumeral ligaments are therefore unable to perform their stabilizing functions at end range of motion. Additionally, the labrum no longer serves to stabilize or deepen the glenoid socket. The force required to translate the humeral head anteriorly decreases by 50 % in the absence of the glenoid labrum [37].

Anterior Labroligamentous Periosteal Sleeve Avulsion (ALPSA)

This lesion was initially described by Neviaser in 1993 [38]. The labroligamentous complex heals on the medial aspect of the glenoid neck (Fig. 16.2); however, recurrent instability is possible given the incompetence of the anterior inferior glenohumeral ligament (IGHL). ALPSA lesions are not commonly associated with first-time anterior dislocations, rather “time-dependent” and “recurrence-dependent” etiologies have been proposed [39]. In 2007, Yiannakopoulos et al. compared intra-articular lesions present in acute and chronic shoulder instability and found that almost ALPSA lesions were found in shoulders with chronic instability [40].

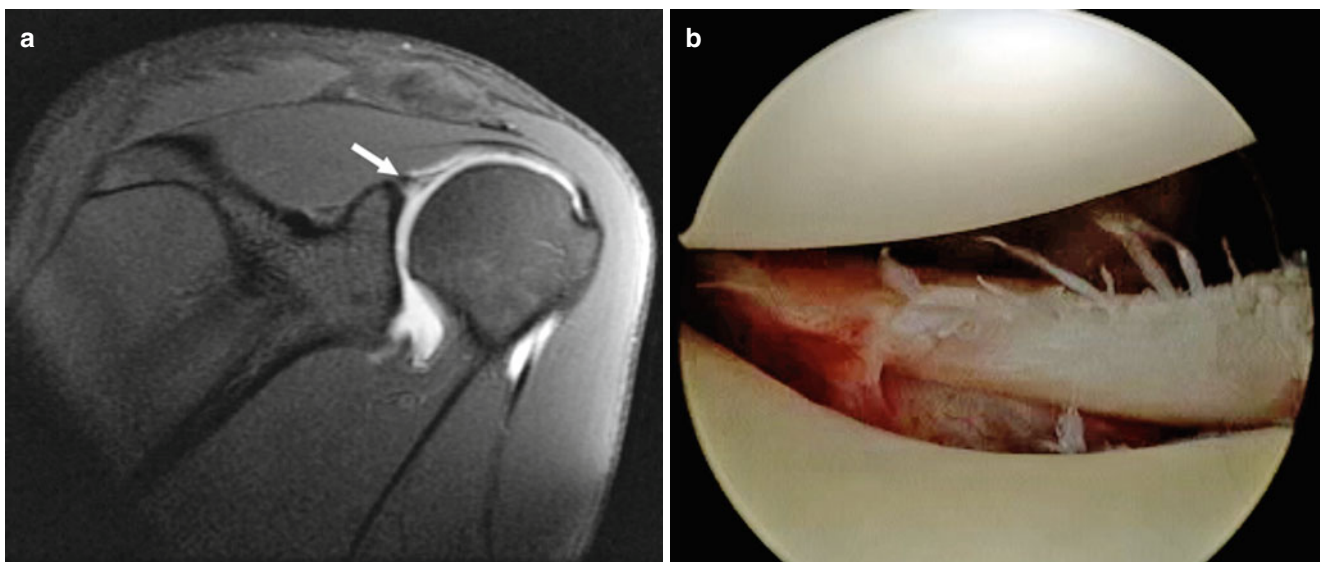


Fig. 16.1 Coronal MRI view (a) and arthroscopic image, *white arrow* shows superior extent of anterior labral tear, which extends inferiorly and torn off anterior glenoid (b) depicting a Bankart lesion

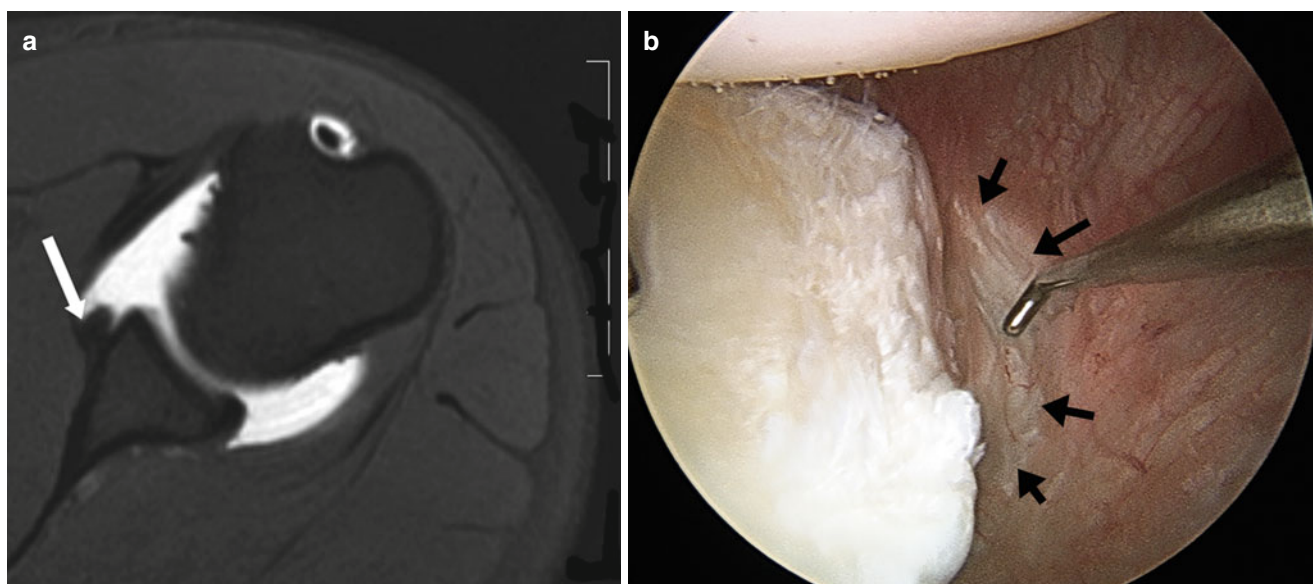


Fig. 16.2 Axial oblique MRA view, *white arrow* demonstrates ALPSA labral tear (a) and arthroscopic image (b) depicting an ALPSA lesion (*black arrows*)

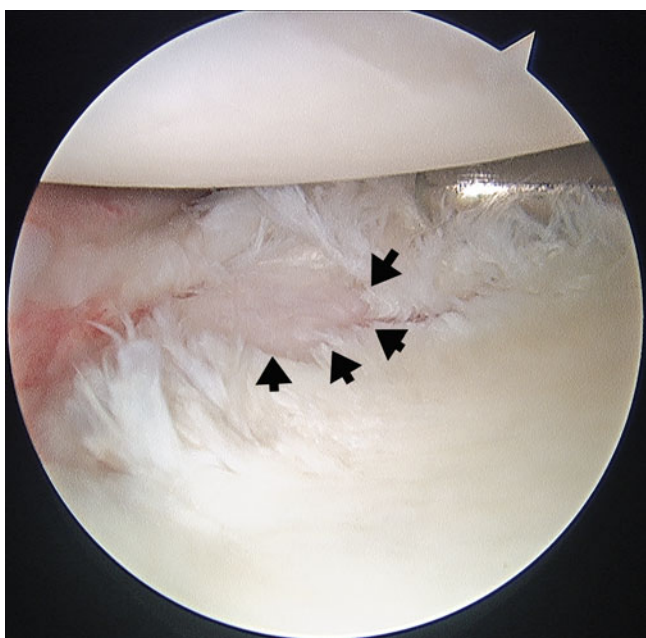


Fig. 16.3 Arthroscopic image demonstrating a GLAD lesion (*black arrows*)

Glenolabral Articular Disruption (GLAD)

Neviaser also described the GLAD lesion in 1993 [41]. This lesion consists of a shear injury to the articular cartilage on the anteroinferior aspect of the glenoid and the attached glenoid labrum (Fig. 16.3) and commonly occurs with forced adduction to an abducted, externally rotated arm.

Bony Bankart Lesion

The bony Bankart lesion occurs when an anterior glenohumeral dislocation of the humeral head causes a fracture of

the anteroinferior portion of the glenoid rim (Fig. 16.4). Although the bony architecture of the glenoid is small, it serves a critical function in maintaining the stability of the glenohumeral joint [42]. Even a small fracture of the anterior glenoid allows the humeral head to easily subluxate anteriorly.

Humeral Avulsion of Glenohumeral Ligament (HAGL)

Humeral detachment of the glenohumeral ligaments (Fig. 16.5) was first noted by Bach et al. in 1988 [43]. A classic biomechanical study of the inferior glenohumeral ligament found that in 25 % of specimens, the ligaments were avulsed from the humerus [44]. The term “HAGL” was coined by Wolf et al. in 1995, in a study in which they reported a 1–9 % incidence of this lesion following anterior shoulder dislocation [45].

Isolated capsular injury is rare following anterior shoulder dislocation, accounting for only between 0 and 11 % of injury patterns. Capsular injuries, which are more often seen in recurrent instability, commonly occur in association with other pathologies [46].

Superior Labral Anterior and Posterior (SLAP) Tears

SLAP tears are not considered a primary lesion in anterior instability; however, this injury often occurs in patients following glenohumeral dislocation. Hintermann et al. identified a 7 % incidence of SLAP tears in a series of 212 patients treated arthroscopically for anterior shoulder instability [36]. Persistence of a SLAP tear may complicate the overall recurrence after instability repair.

Fig. 16.4 Coronal (a) and sagittal (b) 3D CT images demonstrating a bony Bankart lesion

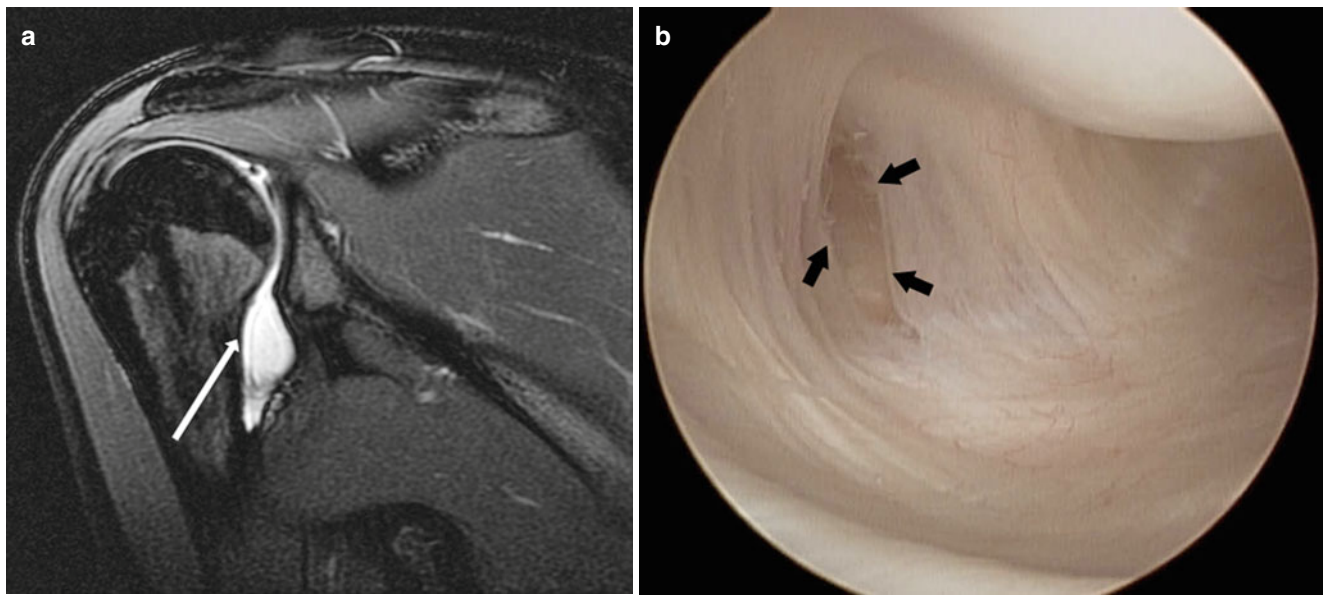
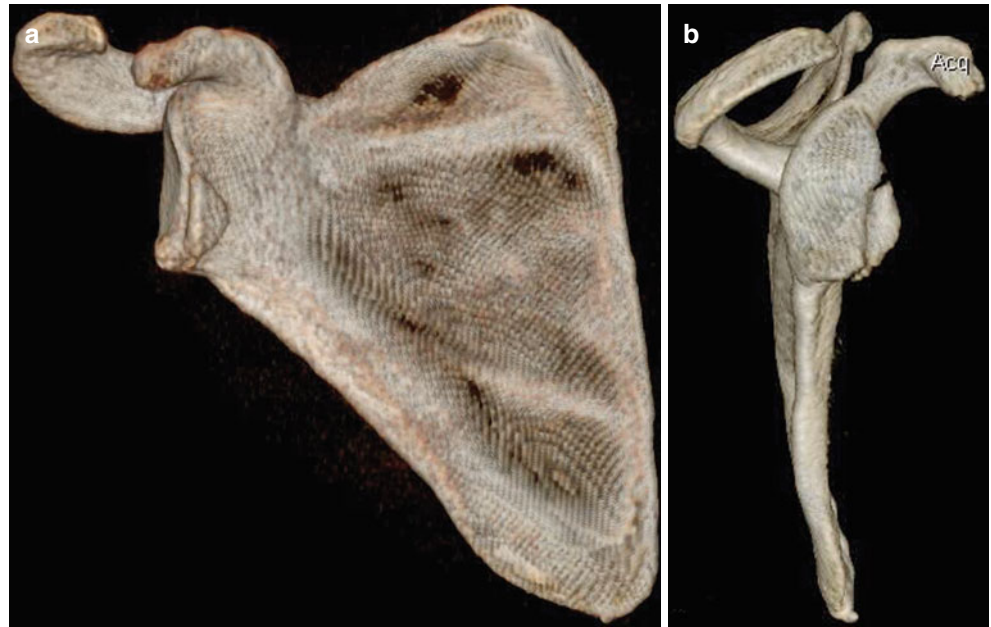


Fig. 16.5 Coronal MRA (a) and arthroscopic image, white arrow demonstrates HAGL tear (b) demonstrating a HAGL lesion (black arrows)

Bony Humeral Lesions

Hill-Sachs Lesion

The Hill-Sachs lesion is a compression fracture of the posterolateral aspect of the humeral head that occurs as a result of impaction with the more dense anteroinferior glenoid during anterior dislocation of the glenohumeral joint (Fig. 16.6a).

Burkhart and De Beer [23] initially defined the Hill-Sachs lesion as “engaging” when the humeral head defect engages

the rim of the glenoid while the shoulder is in a position of abduction and external rotation (Fig. 16.6b). Several studies support an association between an engaging Hill-Sachs lesion and anterior glenoid bone loss in some patients with recurrent anterior shoulder instability [23, 26, 47–49].

Hill-Sachs lesions are associated with 40–90 % of anterior shoulder instability events [40, 50–53], and the incidence may approach 100 % in patients with recurrent anterior instability [53]. Hill-Sachs lesions most commonly occur in association with anterior capsuloligamentous

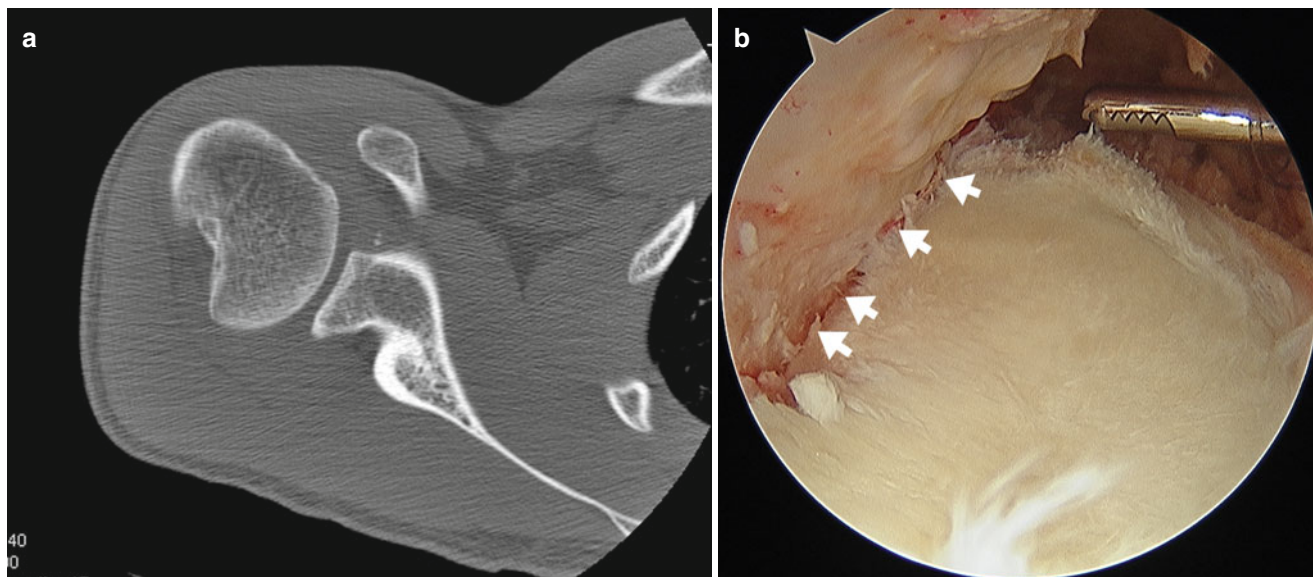


Fig. 16.6 CT image of a Hill-Sachs lesion (a) and arthroscopic view of the Hill-Sachs lesion that has easy “engagement” with the glenoid (b) shown by *white arrows* marking anterior glenoid and humeral head engagement over anterior rim

avulsion (i.e., Bankart lesion) [51] but may also be seen in association with anterior glenohumeral ligamentous pathology and glenoid bone loss (i.e., bony Bankart lesion) [48, 54]. Numerous classification and grading systems exist for Hill-Sachs lesions [26, 47, 50, 55, 56], although none of them have been deemed optimal for directing successful management. The most important factors to determine if a Hill-Sachs lesion is clinically significant are its size and whether it is engaging [23]. Lesions involving <20 % of the humeral head articular surface are rarely of clinical significance, while lesions >40 % of the articular surface are almost always clinically significant and are implicated as an underlying cause of recurrent instability [47, 57]. Management of midsize lesions (20–40 % of humeral head articular surface) is challenging.

Other factors to take into account when determining a management plan for Hill-Sachs lesions include the extent of concomitant glenoid bone loss, the extent of engagement with the glenoid, and the location and orientation of the lesion [49]. In midsize Hill-Sachs lesions, the injury is a bipolar problem with associated glenoid bone loss worsening the humeral-side defect and increasing the risk of instability. Yamamoto et al. [58] described the Hill-Sachs lesion based on the location and size of the humeral head defect and on the amount of glenoid bone loss. Using a cadaveric model, they determined that the distance from the contact area between the glenoid and the humeral head to the medial margin of the footprint was 84 % of the glenoid width. The authors concluded that a Hill-Sachs lesion outside of this glenoid track was at high risk for engagement and,

consequently, recurrent instability [58]. Based on this model, large amounts of glenoid bone loss increase the significance of even small Hill-Sachs lesions [49].

History

Shoulder instability is defined as the inability to maintain the humeral head centered on the glenoid. When evaluating a patient with suspected shoulder instability, it is critical to obtain an accurate history. The patient should be asked to describe the position of the shoulder at the time of the initial dislocation as well as the mechanism of injury. It is also important to determine the frequency of dislocation episodes and the functional disruption that is caused by the instability [59]. A thorough history should also include the necessity for medically assisted reduction versus self-reduction, activity level (including contact versus noncontact sports), amount of time that has passed since the initial dislocation, and any treatment provided to the patient [60].

The provocative anterior instability position (typically with the shoulder abducted and externally rotated) as well as the amount of trauma required for instability to occur have significant implications for overall management. Dislocation with simple daily activities such as reaching overhead suggests different diagnoses (e.g., multidirectional instability and glenoid hypoplasia) than instability episodes that occur in the setting of more significant trauma [60].

Patients will often describe feelings of pain in extremes of motion or a sense of impending instability. The patient may

also report a history of subluxation or dislocation with the shoulder in certain positions (most often abduction and external rotation and with overhead activities). Although these are the most common symptoms, many patients will complain of numbness, transient sharp pain, or weakness as their only symptom of instability [60].

Clinical Examination

A thorough physical exam is paramount to making the correct diagnosis and for determining the appropriate treatment plan. It should be noted that instability of the glenohumeral joint is a clinical diagnosis that is confirmed with a careful history and examination. Furthermore, the direction, type, and classification of shoulder instability as well as operative plan are based mainly on supporting features from the history and physical examination. Evaluating a patient for laxity versus instability is imperative. It is important to remember that the presence of shoulder laxity does not necessarily signify associated instability. Instability is a perception experienced by patients during a dislocation or subluxation event. Laxity, on the other hand, is a normal finding of the glenohumeral joint, given the minimum oblique translation of the humeral head on the glenoid that is required for normal shoulder function [1, 61]. Shoulder laxity and instability are assessed by performing translation testing for laxity (anterior, posterior, and inferior sulcus) and symptomatic directional instability, which is an important indicator of shoulder instability [60]. External rotation with the arm at the side will often decrease the sulcus sign; failure to do so indicates a pathologic rotator interval [62, 63].

Most patients with shoulder instability will have normal range of motion, neurovascular exam, and strength of both the shoulder girdle and periscapular muscles [60]. Initially, the patient should be asked to demonstrate the position of the shoulder at the time of injury and the mechanism of injury. Examine the contralateral shoulder first to demonstrate the typical positions of instability as well as the specific physical exam tests to be performed, so the patient is able to anticipate what will happen during examination of the affected shoulder. There are three key components to the clinical exam: (1) anterior/posterior apprehension tests, (2) examining the glenoid concavity (e.g., load-and-shift test), and (3) assessing the muscles that compress the humeral head against the glenoid [59].

The anterior apprehension test is performed by placing the arm in abduction, extension, and external rotation. Conducting the posterior apprehension test involves placing the arm in adduction, midflexion, and internal rotation. Pain alone is insufficient evidence for instability. More revealing

is confirmation from the patient that this position elicits the sensation that he/she has when she shoulder is ready to dislocate [59].

The status of the glenoid concavity can be assessed by having the seated patient relax and place the forearm on the thigh. Anterior and posterior humeral head translation is then evaluated as an indication of overall joint laxity. The humeral head is then pressed into the glenoid cavity while anterior followed by posterior translation is attempted (the load-and-shift test). Unrestricted translation of the humeral head while it is being pressed into the glenoid cavity suggests a deficiency of the glenoid lip in that direction [59]. It is imperative to note the point at which the humeral head begins to dislocate and engage on the glenoid. The presence of a significant engaging Hill-Sachs lesion or associated bone loss may be indicated by dislocation or engagement of the humeral head on the glenoid with the arm at the side, in 30° of external rotation [23] or in lesser degrees of abduction (45°) and external rotation. Shoulder instability in the midranges of abduction/external rotation is a common symptom in patients with engaging Hill-Sachs lesions [60] or in patients with glenoid bone loss.

Assessing the muscles that compress the humeral head into the glenoid includes evaluation of the isometric strength of the subscapularis, supraspinatus, and infraspinatus [59].

Imaging

Following a traumatic shoulder dislocation, plain radiographs should be obtained including true anteroposterior, axillary lateral, and scapular Y views. In patients with a history of recurrent anterior shoulder instability, or if there is suspicion for a bone defect, specialized views are indicated including apical oblique (Fig. 16.7a) [64, 65], West Point view (Fig. 16.7b) [66], or Didiee [67] views. For further evaluation of humeral head defects, including the Hill-Sachs lesion, the Stryker Notch view (Fig. 16.7c) [67] and a true anteroposterior in internal rotation should be obtained (Table 16.1) [68, 69].

Occasionally, surgeons may wish to obtain additional information regarding capsular and labral tissues, the bone, the rotator cuff, or the neurologic status of muscles. In these cases, further tests including magnetic resonance imaging (MRI), computed tomography (CT), electromyography, or diagnostic arthroscopy may be warranted [59]. Magnetic resonance arthrography (MRA), which involves injection of gadolinium into the glenohumeral joint, provides additional detail than that obtained with standard MRI. MRA is preferred by many surgeons over MRI as a diagnostic study for labral tears. The coronal oblique view

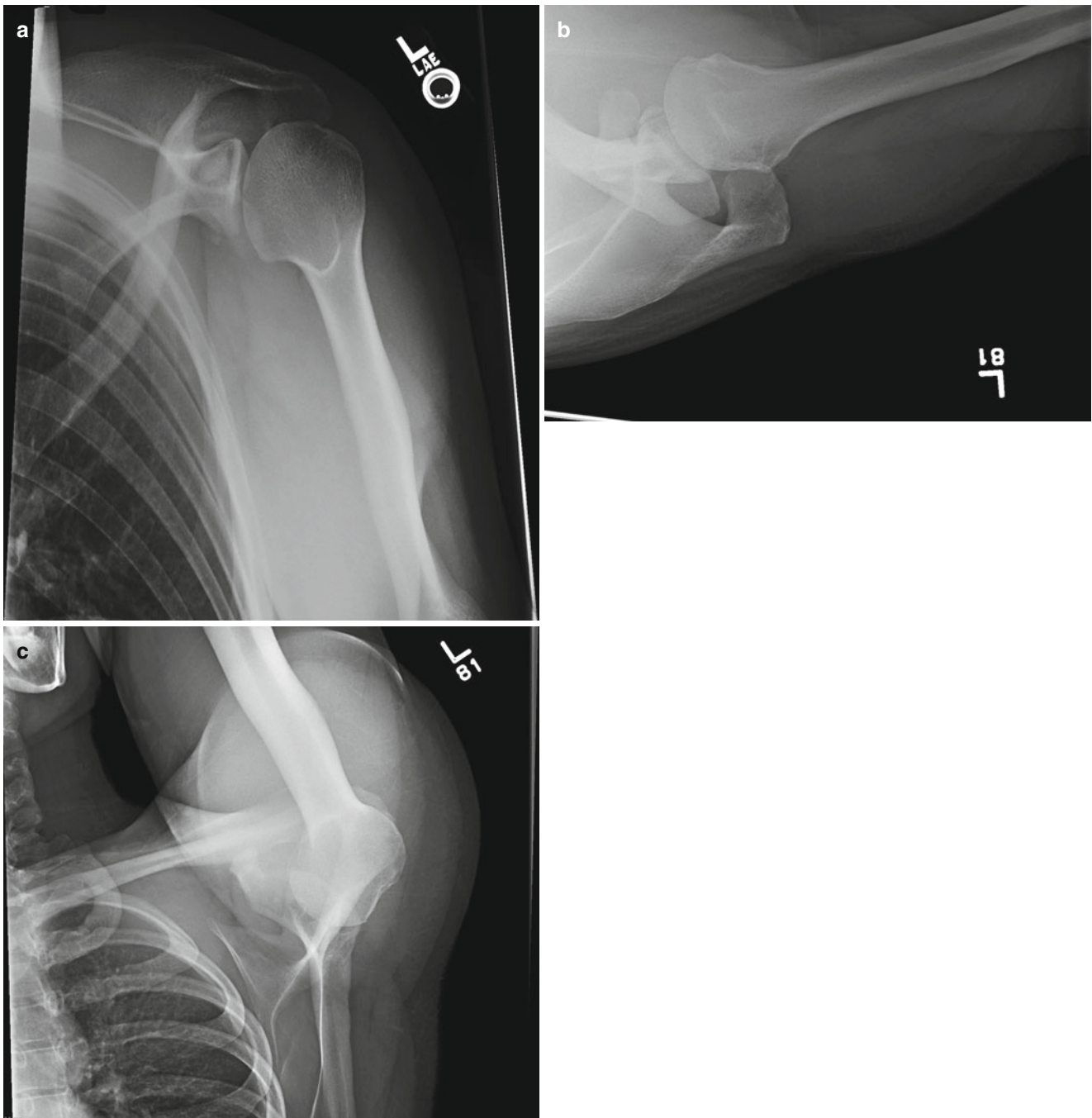


Fig. 16.7 Plain radiographs demonstrating a true AP (a), West Point view (b), and Stryker Notch view (c) of the shoulder

is the best view to identify SLAP tears, while the axial oblique view demonstrates anterior and posterior labral tears. CT is indicated to rule out bone deficiency (glenoid and humeral head). A 3D CT can also be ordered to more accurately identify and quantify bony deficiency. Additionally, the humeral head can be subtracted from the 3D CT, providing the best means of quantifying glenoid bone loss (Fig. 16.8) [46].

Treatment: Indications and Contraindications

Traumatic anterior dislocation of the glenohumeral joint is a very common injury and is associated with a high risk of recurrent instability episodes in young active patients. Numerous factors must be taken into account when deciding

Table 16.1 Specialized radiographic views used to evaluate patients with recurrent shoulder instability

Specialized views	Position of patient	Used to assess	Demonstrates
Apical oblique [68, 69]	Supine. Involved arm is flexed at the elbow and hand is placed across the chest. Injured shoulder is placed in the 45° posterior oblique position. Central beam is angled 45° caudad	Shoulder injuries	Glenoid rim fractures, Hill-Sachs lesions, humeral head subluxation, soft tissue calcification
West Point view [68, 69]	Prone. Involved shoulder on a pad raised 8 cm from tabletop. Head and neck are turned away from involved side. The cassette is placed at superior aspect of shoulder. X-ray beam is centered on the axilla with 25° downward angulation from the horizontal and 25° medial angulation from the midline	Lesions of the anteroinferior glenoid rim	Soft tissue calcification adjacent to anterior or anteroinferior rim of the glenoid or fracture of the glenoid rim
Didiee view [68, 69]	Prone. The cassette is placed under the shoulder. Arm parallel to the table top with a 7.5-cm pad under the elbow. Dorsum of hand on the hip with the thumb directed upward. Beam angled 45° lateromedially and is aimed at the humeral head	Anteroinferior margin of the glenoid	Hill-Sachs lesion
Stryker Notch view [68, 69]	Supine. Involved shoulder is raised vertically and the palm is placed behind the head. The elbow points toward the ceiling and the humerus is perpendicular to the table. The cassette is placed on the tabletop, directly below the shoulder	Posterolateral portion of the humeral head	Compression in posterolateral portion of the humeral head (i.e., Hill-Sachs lesion)

**Fig. 16.8** 3D CT with humeral head subtraction demonstrating attrition of the glenoid

on operative versus nonoperative management of anterior shoulder instability including etiology (e.g., traumatic versus atraumatic or generalized ligamentous laxity), age, frequency of instability episodes, activity level, and associated pathology (e.g., glenoid or humeral head bone loss, rotator cuff tear, SLAP tear) [70].

Nonoperative management of shoulder instability in active patients less than 30 years old results in a recurrence rate of 17–96 % versus 4–22 % in patients treated with arthroscopic stabilization procedures [71, 72]. This data supports early arthroscopic repair following first-time dislocation for young, active patients or those involved in overhead sports, although not all patients are ideal surgical candidates and the algorithm for optimal treatment continues to evolve.

Decision-Making Algorithm

Management of primary anterior shoulder dislocation continues to be an issue of considerable debate. The group at highest risk of recurrent dislocation has been defined as 18–30-year-old athletes participating in collision or overhead sports who sustain a dominant-side shoulder injury [73]. Numerous studies demonstrate age and sex to be two of the most important factors in determining the risk of recurrent instability [5–8, 10, 74]. Recurrence rates following first-time dislocation range from 17 to 96 % [73] and the risk of recurrence has been noted to be highest within the first 2 years following the initial dislocation [10].

The pathology associated with acute shoulder dislocation is significant and includes an 87 % incidence of Bankart lesions with 64 % also suffering a Hill-Sachs lesion and an 18 % incidence of both capsular tearing and rotator cuff injuries [75]. Multiple dislocations can lead to progressive intra-articular pathology including worsening bone loss (humeral head and anterior glenoid), capsular attenuation, damage to the rotator cuff, and superior labrum and biceps anchor injuries [73]. Quality of life is also an important consideration when determining whether to perform early surgical intervention. Studies by Kirkley et al. [76] and Robinson et al. [10] both suggest that recurrent shoulder instability negatively impacts a patient's quality of life by preventing return to preinjury level of play. Based on these data, it is reasonable to recommend surgical stabilization after first-time dislocation in young male athletes participating in collision or overhead sports. Open Bankart repair was originally considered the gold standard in the management of young athletes with shoulder instability; however, arthroscopic technique and surgeon experience have improved to such an extent that results of arthroscopic repair are equal to those of the open repair technique [77].

Patients with traumatic, recurrent anterior instability may also be candidates for arthroscopic stabilization; however, careful patient selection is imperative to maximize results. A thorough history and physical exam should be used to confirm anteroinferior laxity and adequate bone stock (glenoid and humeral head) to support arthroscopic repair. Advanced imaging including CT or 3D CT may also be warranted to visualize and quantify any bony deficiency. Patients with anteroinferior instability and no significant bone loss are candidates for arthroscopic repair; however, patients with glenoid bone loss >20 %, Hill-Sachs lesions >25–30 %, or engaging Hill-Sachs lesions may warrant an open procedure (i.e., Latarjet). Soft tissue injuries (e.g., HAGL lesion) may also require open repair [78].

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Shoulder arthroscopy can be performed with general anesthesia, inter-scalene block, or a combination of the two depending on the preference of the surgical team and patient. Patients can be placed in either the lateral decubitus or beach-chair position. The beach-chair position has the advantage of providing easy access to the glenohumeral joint, ability to see the anterosuperior, inferior, and anterior aspects of the joint, and ease of conversion to an open procedure if necessary. Patient positioning is based on surgeon preference; however, for cases of shoulder instability, the

authors prefer to place patients in the lateral decubitus position since it allows easy access to the entire glenoid, labrum, and capsule. With longitudinal and direct balanced suspension of the arm, this position allows for greater distraction of the glenohumeral joint and hence increased space for passing instruments during the repair. A limitation of lateral decubitus positioning is the difficulty of obtaining precise rotational control during instability repair. Appropriate tensioning of the capsule and inferior glenohumeral ligament is especially challenging in the lateral decubitus position and may result in stiffness and decreased external rotation post-operatively [60].

The examination under anesthesia (EUA) is a critical component of the procedure. It can provide information about the direction and extent of translation and may alter operative planning with regard to how much capsular plication to perform [60]. A patient's pain on physical exam in the office setting may lead to underestimation of the pathology or degree of instability of the shoulder. The range of motion should be assessed in elevation, external rotation with the arm adducted, and external and internal rotation with the arm abducted to 90°. Examining the shoulder for stability is performed by applying anterior, posterior, and inferior force while moving the arm throughout a range of abduction and rotation [79].

Portals

Proper portal placement is essential to perform an accurate diagnostic arthroscopy, appropriate soft tissue mobilization, and accurate placement of anchors. It is important to take sufficient time to mark out the location of the portal sites. Begin by clearly delineating the bone outlines of the acromion, distal clavicle, and coracoid with a surgical skin marker. Take care to mark out the inferior surfaces of the bone landmarks because portal entry points are measured from these surfaces [79].

After positioning the patient, standard posterior and anterosuperior portals are created and a thorough diagnostic arthroscopy is performed. If a Bankart lesion is identified, an additional mid-glenoid portal can be established at the 3 o'clock position on the glenoid using an 18-gauge needle to aid with localization slightly superior to the subscapularis tendon. Labral pathology at the 4–6 o'clock position can be difficult to address through these standard portals. Establishing a 7 o'clock portal approximately 2–3 cm lateral and 1 cm inferior to the posterior portal provides excellent access to the inferior aspect of the glenoid and may be used for percutaneous placement of anchors on the posterior and inferior aspects of the glenoid [60] (Fig. 16.9).

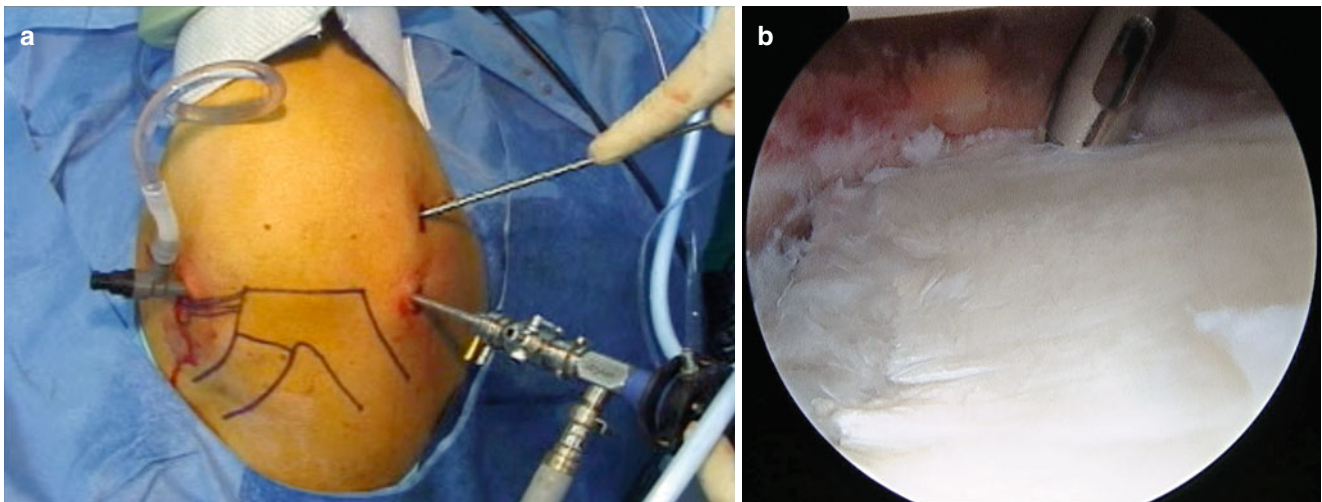


Fig. 16.9 Location of posterolateral portal in relation to standard anterior and posterior portals (a); arthroscopic view of instrumentation introduced through posterolateral portal (b)

The pathology of an anterior labral tear is best viewed from the anterosuperior portal. Visualization from this portal decreases the risk of the surgeon missing ALPSA lesions and allows one to more easily evaluate anteroinferior glenoid bone loss and the extent of the labral tear posteriorly [60]. With the arthroscope in the anterosuperior portal, it also affords excellent visibility while the surgeon works through the mid-glenoid portal, ensuring complete preparation of the tear and repair.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Diagnostic evaluation of the glenohumeral joint should be performed systematically in order to avoid overlooking any pathology. It is important to assess for pathology commonly associated with a Bankart lesion including a HAGL lesion, ALPSA lesion, and SLAP tears. Establish the standard posterior portal. After entering the joint, identify the biceps-labrum complex and rotate the camera to center the glenoid on the monitor screen. Inspect the rotator interval and superior glenohumeral ligament. The rotator interval may be widened or lax in patients with glenohumeral instability [33, 79].

Establish the anterior portal using the outside-in technique, with the goal of placing the portal in the center of the rotator interval. Evaluate the biceps tendon and pull the extra-articular portion of the tendon into the joint using a probe to inspect it for tearing or inflammation. Inspect the biceps-labrum complex using a probe inserted through the anterior cannula. Abduct and externally rotate the shoulder

to determine whether the superior labrum elevates off the glenoid [79].

While maintaining the arthroscope in the posterior portal, evaluate the subscapularis recess and the superior border of the subscapularis tendon. Direct the arthroscope inferiorly to inspect the anterior labrum and the middle glenohumeral ligament. Evaluate the attachment of the anterior labrum to the glenoid using a probe passed through the anterior cannula. Fraying, tearing, or separation of the labrum from the glenoid may indicate instability [79]. With the arthroscope directed at the 5 o'clock position, inspect the inferior glenohumeral ligament. Assess both tension and insertion strength, using a probe.

Determine whether a “drive-through sign” is present inferiorly. This sign refers to the easy passage of the arthroscope between the humeral head and the glenoid, with the camera positioned at 6 o'clock. Recall that the drive-through sign indicates only glenohumeral laxity, not necessarily gross instability [79].

To view the posterior labrum from the posterior cannula, withdraw the arthroscope until it sits slightly anterior to the posterior capsule. Rotate the arthroscope until it points at the 6 o'clock position. Evaluate the posterior labrum for fraying, tears, or separation of the labrum. Continue inferiorly, to visualize the posterior-inferior glenohumeral ligament. Internally rotate the arm and note the tensioning of the ligament [79].

For thorough assessment of the rotator cuff tendons, direct the arthroscope superiorly. Abduct and externally rotate the shoulder until the anterior aspect of the supraspinatus tendon can be visualized. Inspect the cuff insertion from anterior to posterior and assess the insertion of the

supraspinatus on the humeral head. No exposed bone should be visible between the articular surface of the humeral head and the insertion of the supraspinatus tendon. Partial articular surface tears are present when some bone is exposed between the remaining intact supraspinatus tendon and the articular surface [79].

After evaluating the posterior insertion of the rotator cuff, direct the arthroscope inferiorly and externally rotate the shoulder. This allows visualization of the posterolateral humeral head and evaluation for the presence of a Hill-Sachs lesion [79]. Evaluate the humeral head and glenoid for signs of osteoarthritis.

Place the arthroscope in the anterior portal and reinspect the posterior labrum, capsule, and posterior rotator cuff. Position the shoulder in abduction and external rotation, to assess for internal impingement between the posterior-superior labrum and the posterior rotator cuff and capsule. Note the normal pear shape of the glenoid, which can be observed from this perspective. Loss of the normal inferior glenoid widening signifies bone loss in the anteroinferior glenoid and may be present in patients with glenohumeral instability [79].

Step-by-Step Procedure [78] (Figs. 16.10 and 16.11, Box 16.1)

- Examination of shoulder under anesthesia.
- Position patient (beach chair vs. lateral); we prefer lateral decubitus to allow ease of access to the entire 360° of the labrum.
- Mark bony landmarks (acromion, distal clavicle, and coracoid) on the skin.
- Place the posterior portal in line with the glenoid, which is nearly parallel to the lateral aspect of the acromion. Make this portal 2 cm distal and directly in line with the lateral edge of the acromion.
- Perform thorough diagnostic arthroscopy. Identify all pathology and formulate a plan.
- Place the anterosuperior portal high in the rotator interval, immediately posterior or just anterior to the biceps tendon first using an 18-gauge needle. Make the portal incision just anterior to the anterior edge of the acromion. Insert a switching stick.
- Create the anterior mid-glenoid portal, which is just above the subscapularis tendon. An 18-gauge needle is inserted from outside-in starting next to the coracoid and entering the joint immediately superior to the subscapularis tendon. The two anterior portals should be placed as widely apart as possible. An 8.25 mm cannula is inserted into the mid-glenoid portal and will serve as the primary working portal.
 - Ensure that the angle of approach coming into the joint will allow the appropriate angle for drilling and placement of anchors.
 - Evaluate ability to manipulate instrumentation and shuttle sutures inferiorly.
- The arthroscope is placed in the anterosuperior portal and will remain there for the duration of the case (switched over the switching stick).
- Adequately mobilize the anterior-inferior capsulolabral complex. Visualization of the subscapularis muscle fibers medial to the capsule-labral complex indicates adequate release of the capsulolabrum.
 - A bump can be placed in the axilla to lateralize the humeral head and improve visualization, versus a lateral translation strap that is well padded.
- After the labrum and capsular attachments to the glenoid are adequately released, roughen the glenoid with a burr or bone rasp to encourage soft tissue healing.
- Place the first anchor.
 - This may be done either from anterior or from a posterolateral percutaneous portal (7 o'clock portal – see manuscript for description).
 - The first anchor is placed near 6 o'clock position from either the posterolateral portal (percutaneous) or from the anterior mid-glenoid portal (via 8.25 mm cannula).
 - Ensure that the drill guide is well seated on the glenoid with a gentle mallet tap just prior to drilling to prevent slipping.
 - Insert the anchor per manufacturer recommendations and then begin capsulolabral repair from inferior to superior.
- Anterior-inferior capsulolabral repair.
 - Place a shuttling suture or passing device instrument at the most inferior location. This will be used to shuttle the non-absorbable suture from the most inferior anchor.
 - Place the initial suture anchor at the 6 o'clock position, 1–2 mm onto the articular surface of the glenoid and 5–10 mm cephalad to the shuttle suture to appropriately shift the tissue superiorly and retention the IGHL.
 - A combined stitch can be used to tension both the capsule and repair the labrum by passing a curved suture passer through the capsule 5–10 mm lateral to the labrum, exiting the capsule, reentering deep to the labrum, and emerging just lateral to the articular margin.
 - Repeat the process of shuttle/suture anchor placement until normal anatomy has been restored.
- A total of 3 anchors are utilized for a typical anterior instability repair, but more may be required if the tear

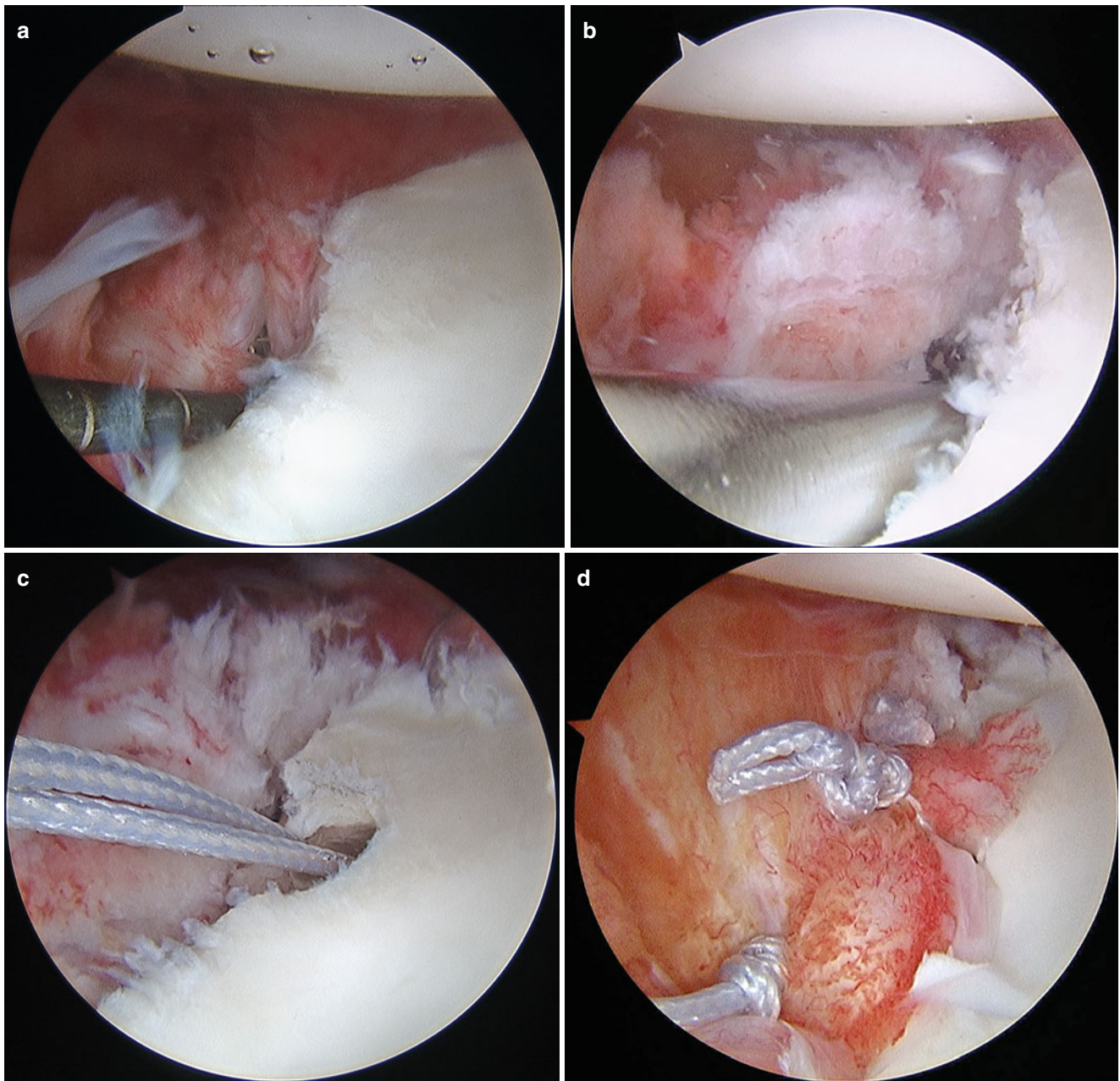


Fig. 16.10 Arthroscopic identification of an ALPSA lesion (a), preparation of the glenoid (b), and repair of the lesion (c, d)

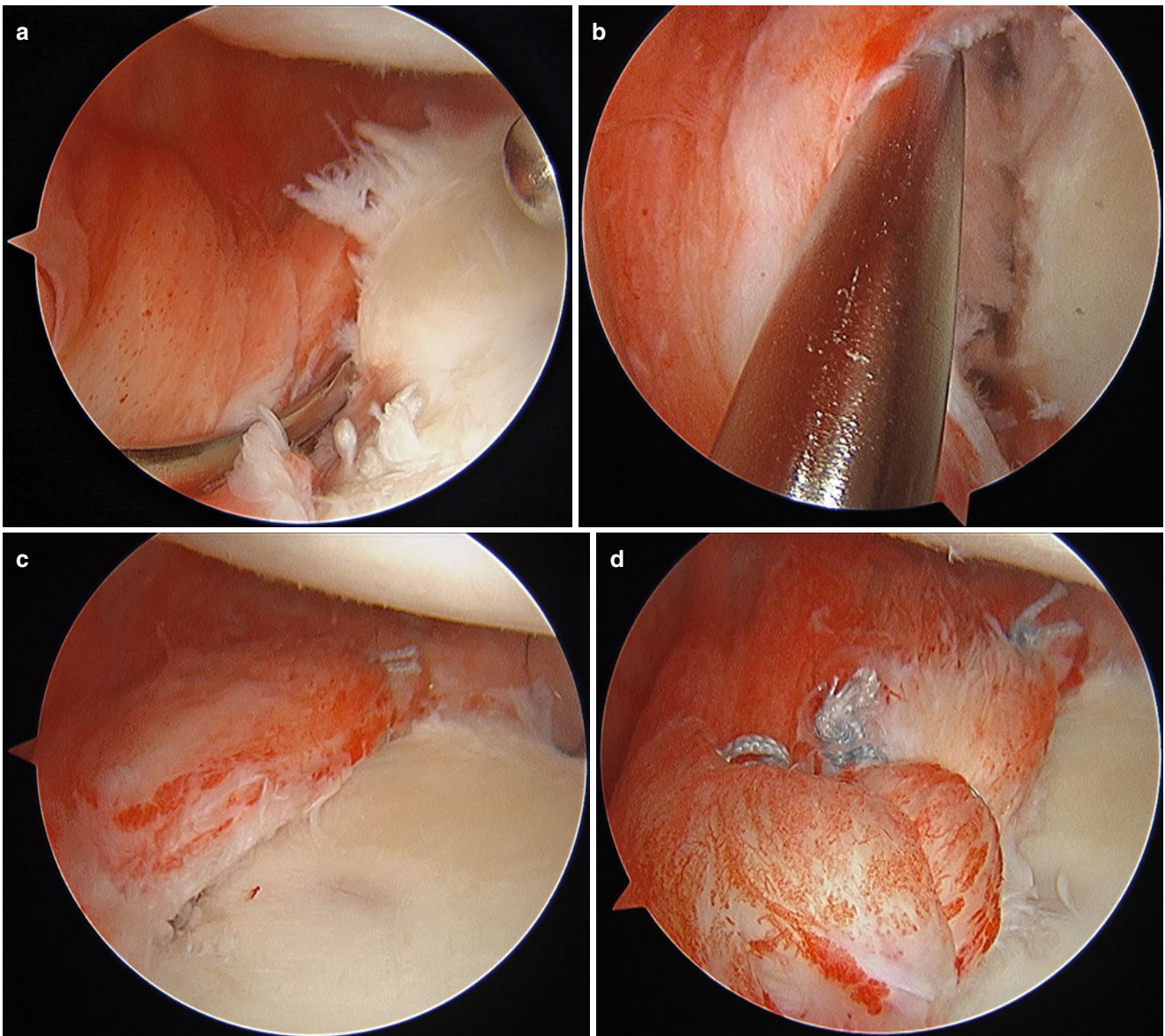


Fig. 16.11 A 26-year-old male with anterior labral tear 2 months out from initial injury with recurrent instability, demonstrating arthroscopic repair steps. Initial tear as viewed from the anterior superior portal (**a**);

preparing the anterior glenoid with an elevator and 3.0-mm bone cutting shaver (**b**); first anchor at 6 o'clock inserted from the posterolateral (7 o'clock) portal (**c**); final repair construct with 3 total anchors (**d**)

extends posteriorly. The anchors are typically placed at the 5:30–6:00, 4:30, and 3–3:30 position, with 6 o'clock being the most inferior. Care is taken not to overtighten the labral tissue near the rotator interval as this may constrict the mobility of the capsule and labrum, thereby limiting external rotation at the side.

- The arthroscope may be moved to the posterior portal prior to placement of the last anchor if it is too “crowded” to place the final superior anchor (usually about the 3 o'clock position) with the scope in the anterosuperior portal.
- The arthroscopic wounds are closed in standard fashion, and the wounds are covered with a dry sterile dressing, followed by placement of the affected arm in a padded abduction sling.
- The patients participate in an arthroscopic instability therapy protocol with a sling for approximately 5–6 weeks, starting with strengthening of the scapular stabilizers and passive motion of the shoulder with flexion to 90–120°, abduction to 45, but avoiding abduction and external rotation combined for about 4–5 weeks. Progressive ROM is started at 4–6 weeks, and then a gradual strengthening program including more scapular stabilizing and strengthening exercises. After ROM is restored and the patient has good scapular control, they are allowed to return to sport-specific training and then full activities around 5–6 months.

Box 16.1: Tips and Tricks

- In the lateral decubitus position, the posterior portal is made in line with the lateral edge of the acromion and 1 cm inferior to the posterior tip. This allows for a slightly downward trajectory from the posterior portal, thereby facilitating instrumentation during the case.
- The anterosuperior portal is positioned high in the rotator interval. Following the diagnostic arthroscopy, the arthroscope can be transferred to this portal for excellent visualization of the anterior glenoid.
- The mid-glenoid portal is also created in the rotator interval, slightly proximal to the subscapularis tendon. It is important to provide at least a 2- to 3-cm skin bridge between the anterosuperior and mid-glenoid portals in order to avoid crowding intra-articularly while performing the case.

- A posterolateral portal (7 o'clock position) may be created to allow for percutaneous anchor placement. Additionally, a small cannula may be inserted to facilitate glenoid anchor placement and repair of the labrum inferiorly.
- The axillary nerve is most vulnerable at the 6 o'clock position (12.5–15 mm from the glenoid), which increases with abduction.

Postoperative Care

Preoperative patient counselling in conjunction with intraoperative findings and exact surgical procedure help determine the appropriate postoperative rehabilitation program. Several factors are important to take into account including the type of pathology, direction of instability, quality of the tissue at the time of repair, and any other associated injuries (e.g., rotator cuff tear, biceps tendon tear). The authors recommend an abduction sling for the majority of instability repairs, as it maintains the shoulder in a neutral to slightly externally rotated position.

Physical therapy often begins 7–10 days following most routine instability repairs. Gradual progression with passive and active-assisted range of motion will occur over the first 4 weeks (forward elevation [FE] to 130°; external rotation [ER] to 30°). Between 4 and 6 weeks, these ranges increase to FE 130–180° and ER 30–60°. The subsequent weeks focus on progressing active range of motion with resistive strengthening being incorporated at 8–12 weeks and return to full sports and normal activities at 4–6 months in most cases [60].

Literature Review

Numerous studies have examined arthroscopic versus open repair of recurrent glenohumeral instability. Tables 16.2 and 16.3 summarize the results of the literature over the past 5 years. The rate of recurrent instability following arthroscopic treatment ranges from 2 to 18 % (Table 16.2), while that for open management ranges from 0 to 9 % (Table 16.2). These studies support the dramatic improvement in arthroscopic management of this complex problem and demonstrate that the success rate of arthroscopic treatment essentially equals that of open management, with the added benefit of decreased morbidity. Regardless of the technique, the overall goal of instability surgery is to restore anatomic alignment of the labrum with the glenoid.

Table 16.2 Summary of literature published between 2007 and 2012 on the results of primary arthroscopic treatment of recurrent glenohumeral instability

Author	Year	Study type	Procedure	Sample size	Follow-up (months)	Primary outcome variable	Primary outcome scores	Physical exam	Recurrences no.	Factors associated with recurrence
Ahmed et al. [80]	2012	Retrospective review	Bankart repair and capsular shift	302	60	Recurrent instability	WOSI DASH	Not done	40 (13.2 %)	Severity of glenoid bone loss (≥ 25 %), engaging Hill-Sachs lesion, age at surgery (<age 20)
Zaffagnini et al. [81]	2012	Retrospective review	Modified Caspari	49	13.7 \pm 2.2 years	Redislocation	Rowe UCLA Constant	Not done	6 (12.5 %)	Six patients suffered redislocations; however, the mechanism is not described. Specific factors associated with recurrence were not described
Boileau et al. [82]	2012	Case series	Remplissage and Bankart repair	47	24	Recurrent instability	Rowe Walch-Duplay Constant-Murley Subjective Shoulder Value (SSV)	\downarrow ER	1 (2.1 %)	Redislocation associated with a fall while playing basketball. Specific factors associated with recurrence were not described
Netto et al. [83]	2012	Randomized controlled	Bankart repair	17	37.5 (20–56)	Recurrent instability	DASH	No \downarrow ROM	2 (12 %)	Both patients suffered redislocation and required surgical intervention with an open technique. Specific factors associated with recurrence were not described
Mahirogullari et al. [84]	2010	Prospective randomized	Bankart repair	34	26.6 (12–51)	Recurrent instability	Rowe VAS	\downarrow ER	2 (5.9 %)	Redislocation following a fall while playing football and while descending stairs. Specific factors associated with recurrence were not described
Voos et al. [20]	2010	Case series	Bankart repair	83	33 (24–49)	Recurrent instability	ASES L' Insalata VAS	No loss of ER	13 (18 %)	<age 25, ligamentous laxity, Hill-Sachs lesion >250 mm ³
Porcellini et al. [85]	2009	Prospective cohort study	Bankart repair	385	36	Redislocation	None	Not done	31 (8.1 %)	Male sex, age at first dislocation (<age 22), time from first dislocation until surgery (>6 months after dislocation)
Robinson et al. [86]	2008	Randomized controlled	Bankart repair	42	24	Recurrent instability	WOSI DASH	No \downarrow ROM	3 (7 %)	All three patients suffered a redislocation. The mechanism of injury was not described, nor were specific factors associated with recurrence

Table 16.3 Summary of literature published between 2007 and 2012 on the results of primary open treatment of recurrent glenohumeral instability

Author	Year	Study type	Procedure	Sample size	Follow-up (months)	Primary outcome variable	Primary outcome scores	Physical exam	Recurrences no.	Factors associated with recurrence
Netto et al. [83]	2012	Randomized controlled	Open Bankart repair	25	37.5 (20–56)	Recurrent instability	DASH	No ↓ ROM	0 (0%)	Not described
Zaffagnini et al. [81]	2012	Retrospective review	Open capsular shift and Bankart repair	33	15.7 ± 2.2 years	Redislocation	Rowe UCLA Constant	Not done	3 (9%)	Three patients suffered redislocations; however, the specific factors associated with recurrence were not described
Emami et al. [87]	2011	Retrospective review	Bristow-Latarjet	30	2–8 years (mean 5 year)	Recurrent instability	Rowe	↓ ER	0 (0%)	Not described
Mahirogullari et al. [84]	2010	Prospective randomized	Bankart repair	30	26.1 (12–52)	Recurrent instability	Rowe VAS	↓ ER	1 (3.3%)	Redislocation following a fall 12 months post-op. The specific factors associated with recurrence were not described
Rahme et al. [88]	2010	Prospective cohort	Bankart repair	68	63 (27–110)	Recurrent instability	Rowe	↓ ER	4 (5.9%)	One patient experienced a single redislocation during sleep, while the other 3 patients suffered multiple redislocations during sports activities. The specific factors associated with recurrence were not described
Ogawa et al. [89]	2010	Retrospective review	Open Bankart repair	167	8.7 years (mean 5–20 year)	Recurrent instability OA	Rowe	↑ ROM	8 (4.8%)	One patient suffered a redislocation, while the other 7 experienced subluxation events. The specific factors associated with recurrence were not described
Uchiyama et al. [90]	2009	Case series	Open modified inferior capsular shift	50	61 (24–172)	Recurrent instability	Rowe UCLA	↓ ER	3 (5.8%)	One patient suffered a redislocation 24 months post-op. The other 2 patients experienced a single subluxation event during judo practice (at 3 and 4 months post-op). The specific factors associated with recurrence were not described
Pagnani [91]	2008	Case series	Open Bankart and capsular shift	103	24 (24–74)	Recurrent instability	ASES	↓ ER	2 (2%)	One patient suffered a redislocation and another patient experienced a subluxation. The specific factors associated with recurrence were not described

Summary

Successful management of patients with anterior shoulder instability is challenging and depends largely on the accurate diagnosis and treatment of the underlying pathology. Awareness of the pathoanatomy contributing to recurrent anterior glenohumeral instability is paramount in order to appropriately manage this complex problem. A thorough understanding of the principles of anterior instability repair combined with the pearls provided should allow for the comprehensive approach to patients with anterior shoulder instability and ultimately lead to improved patient outcomes.

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