Glenohumeral Joint Instability: The Orthopedic Approach

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ABSTRACT

The goal of this article is to present a concise review of the current concepts of shoulder instability. This chapter supplements the radiologic assessment of glenohumeral instability, which follows this section.

KEYWORDS: Shoulder, instability

It is important to define the differences between instability and laxity. Instability of the shoulder is defined as abnormal or symptomatic motion, usually translocation of the humeral head with respect to the glenoid. Laxity, however, describes the passive motion characteristics of the joint. Overhead athletes (swimmers, gymnasts, throwers) may exhibit substantially increased laxity without developing symptomatic instability. A complex interaction between the static and dynamic restraints leads to controlled glenohumeral motion. This chapter does not address instability in the setting of proximal humerus fractures and subsequent arthroplasty.

ETIOLOGY AND MECHANISM OF INJURY

Shoulder dislocations occur across all age ranges but most commonly occur in young males of less than 25 years of age. Anterior dislocations occur most frequently—they represent about 90–95% of all shoulder dislocations. The etiology of shoulder instability is most commonly traumatic: a fall onto the outstretched arm in an abducted and externally rotated position, contact in volleyball or basketball with the arm overhead, or tackling during football or rugby with the arm abducted. These positions put the anterior capsule and labrum under tension and lead to their potential failure. This usually results in a Bankart lesion or disruption of the labrum and anterior–inferior glenohumeral ligament complex from the glenoid as well as an impaction fracture on the postero-lateral aspect of the humeral head (Hill-Sachs or McLauglin lesion).

Posterior dislocations of the shoulder are much less common, accounting for only 3–5% of dislocations, but they are frequently missed. At present, the misdiagnosis rate still approaches 50%.[1] The subacromial dislocation (humeral head posterior to the glenoid and inferior to the acromion) is the most common and is frequently locked. Hawkins et al.[2] reported on 41 cases of locked posterior dislocations that were caused by motor vehicle accidents, electroshock therapy, and surgery. Classically, posterior dislocations are described in the setting of seizure disorders and electroconvulsive therapy or electrical shock. Other mechanisms include falls or axial loading with the arm in an adducted and internally rotated position. Football lineman may sustain injury to these posterior restraints with their blocking techniques with an extended arm. This can cause posterior labral or capsulolabral injuries and can lead to subsequent instability.

Multidirectional instability (MDI) has been classified differently by various authors. This instability

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represents abnormal motion in two or three planes. In general, this represents anterior–inferior and posterior–inferior instability. The incidence of multidirectional instability peaks in the second and third decades of life, with most patients being younger than 35 years old.[3] The children and the preadolescent population tend to make up a majority of the voluntary dislocators, which are composed of two types: muscular and positional. The first group of patients is able to sublux or dislocate the shoulder by selective muscle activation, whereas the others need to place the arm in appropriate position (such as forward flexion, adduction, and internal rotation for posterior instability) to induce the subluxation or dislocation.

NATURAL HISTORY OF RECURRENT INSTABILITY

Although the natural history of the dislocated shoulder depends on a variety of factors, it is the age of the patient at the time of the initial dislocation that is most important. The rate of recurrence has been shown to be inversely related to age. Multiple studies in the literature have demonstrated a high recurrence rate in younger individuals. Hovelius et al.[4] reported a prospective study of 247 primary anterior shoulder dislocations with a 10-year follow-up. In their series, operative treatment was required in 62% of patients younger than age 30 years because of recurrence, versus 9% in the population older than 30 years of age. Patients with two or more dislocations in the first 5 years had a 78% chance of another instability episode.

In addition to redislocation, other associated problems include neurovascular injuries, rotator cuff tears, and fractures of the glenoid or humeral head. Rotator cuff tears have a 30% incidence in patients older than 40 years and an incidence of greater than 80% in patients older than 60 years of age.[1] Sonnabend[5] reported on a series of patients older than 40 years of age with primary anterior dislocations. At 3 weeks postinjury, 13 patients had complaints of weakness or pain. Eleven of these patients were found to have rotator cuff tears. Rowe[6] studied 324 shoulder dislocations with a 10-year follow-up. In patients younger than 20 years of age, he found a 94% recurrence rate. In patients older than 40 years of age, the recurrence risk was 14%.

GLENOHUMERAL STABILITY: STATIC AND DYNAMIC CONSTRAINTS

The glenohumeral joint has a ball-and-socket type of configuration. The humerus has an articular surface area that is nearly three times the size of the glenoid fossa.[8] This limited contact area is responsible for the large amount of translational and rotational motion present in the shoulder. The term "glenohumeral joint stability" refers to the process by which the humeral head remains centered on the glenoid during motion.[9] Glenohumeral stability is influenced by a variety of factors. Adhesion–cohesion, concavity–compression, negative intraarticular pressure, and limited joint volume are combined with the static ligamentous restraints and dynamic muscular control to provide stability within the large range of motion. The glenoid labrum accounts for about 10–20% of the static stability by deepening the glenoid socket 50% as well as providing innervation and proprioception.[9,10] A Bankart lesion is a tear of the anterior–inferior glenoid labrum. A recent study by Taylor DC[11] in AJSM in 1997 revealed a Bankart tear in 97% of 63 patients who had a first-time traumatic shoulder dislocation.

The coracohumeral ligament and localized thickenings of the capsule including the superior glenohumeral ligament, middle glenohumeral ligament, and inferior glenohumeral ligament have been described in many references. These ligaments function by developing tension to limit passive motion. In the midrange of motion, the active contraction of the rotator cuff muscles provides the dynamic stability. The combined effect of the cuff muscles works as a force-couple to keep the humeral head centered on the glenoid. The rotator cuff also works in conjunction with the scapular stabilizers, long head of the biceps, and the deltoid.

With the arm in a neutral position, the coracohumeral and superior glenohumeral ligaments are responsible for limiting inferior translation and external rotation. The middle glenohumeral ligament limits anterior and posterior translation with the arm abducted from 40 to 60 degrees. The anatomy of the inferior glenohumeral ligament complex was defined by O'Brien et al.[12] The anterior and posterior bands work together with the arm positioned in abduction. The anterior band is the primary static restraint to anterior translation with the arm abducted and externally rotated, whereas the posterior band of the inferior glenohumeral ligament is the primary static restraint to posterior translation. The complex works as a sling and develops tension anteriorly or posteriorly as the humerus is rotated.

The two anatomic lesions associated with MDI are deficiency of the rotator interval and a redundant inferior capsular pouch.[3] The rotator interval is defined as the space between the supraspinatus anteriorly and the subscapularis superiorly. The joint capsule normally spans this region to include the superior glenohumeral ligament and the overlying coracohumeral ligament. In MDI, attenuation or thinning of the interval is seen with anatomic and arthroscopic evaluation. As mentioned previously, these ligaments help to limit inferior translation, which is the primary direction involved with MDI.

A voluminous inferior capsular pouch can lead to instability in all three directions. Basic science studies have been performed to evaluate the collagen fibers of unidirectional and MDI patients. Rodeo et al.[13] demonstrated increases in collagen cross-links, collagen fibril diameters, cysteine, and elastin content in unstable shoulders (unidirectional and MDI) compared with stable shoulders in a control group. No significant differences were found between the unidirectional and MDI patients. However, in skin samples, smaller mean collagen fibril diameters were found in the MDI patients compared with the unidirectional instability patients.

CLASSIFICATIONS

Many classification systems have been developed to describe shoulder instability.

Schemes related to direction (anterior, posterior, multidirectional), etiology (traumatic, atraumatic, overuse), degree of instability (subluxation versus dislocation), and duration of symptoms (acute, recurrent, fixed) are described in the literature.

Recently, Gerber and Nyffeler[14] described a classification system that distinguishes among static, dynamic, and voluntary dislocation. The static instabilities (class A) are defined by the absence of classic symptoms of instability and are associated with rotator cuff tears and degenerative joint disease. The diagnosis of Class A instability is radiologic, not clinical. Class A1 instability (static superior migration) is defined as an acromiohumeral distance of less than 7 mm on an AP radiograph. The associated supraspinatus and infraspinatus tears are considered to be irreparable by many authors. Static anterior subluxation (Class A2) is a fixed anterior position of the humeral head on the glenoid. Symptoms of subcoracoid impingement are usually present. Treatment usually requires more than soft tissue procedures such as a Laterjet or coracoid transfer. Static posterior subluxation (Class A3) is a fixed posterior position of the humeral head. To date, most authors have found these to be irreversible. Inferior subluxation (Class A4) is characterized by straight inferior translation.

Dynamic instabilities (Class B) are always initiated by trauma and patients present with the symptoms of instability. Class B1 is defined as a chronic, locked shoulder dislocation. Depending on the direction, a Hill-Sachs or McLauglin compression fracture may be present. Class B2 is unidirectional instability without hyperlaxity, and Class B3 is with hyperlaxity. Class B2 accounts for 60% of patients treated for shoulder instability, versus 30% for B3. Multidirectional instability without and with hyperlaxity describes Class B4 and Class B5. Class B5 accounts for 5% of shoulder instability and is the classically described multidirectional instability patient. These patients rarely have bony lesions and usually have capsular lesions, stretched ligaments, and a patulous capsule. Class B6 is a form of instability that is uni- or multidirectional, with voluntary reduction.

The last group is Class C, and these are the patients who can dislocate their shoulders voluntarily.

In classifying MDI with respect to etiology, congential, acquired, or posttraumatic etiologies have been described.[3] The congential category includes connective tissue disorders such as Marfan's syndrome and Ehlers-Danlos, as well as milder forms of generalized ligamentous laxity. A positive family history can frequently be seen in these patients. The acquired category is usually the result of repetitive stress to the shoulder. Traditionally, MDI was thought of as being purely atraumatic, although this is a misconception. The traumatic event is frequently minor. However, when superimposed on the patient's increased laxity, the resultant laxity becomes more disabling. Because of the variety of these presentations, MDI is often thought of as a continuum.

MDI can be classified with respect to direction. The primary direction is usually inferior and combines an anterior or posterior component. Rarely are all three directions involved. Neer[3] described three groups of patients based on direction: patients with anteroinferior dislocations and posterior subluxations, patients with posteroinferior dislocations and anterior subluxation, and patients with dislocations in all three directions.

HISTORY

There are several key elements to a patient's history that are important. First, age at the initial dislocation has been shown to be a key prognostic factor for future instability. Mechanism and arm position help determine the direction of instability. Patients should be asked what arm positions reproduce their symptoms. This can be a valuable clue to the direction and degree of instability. The total number of episodes of instability needs to be documented. Patients should also be asked whether they can voluntarily dislocate their shoulders. From the treatment perspective, it is valuable to differentiate between the two types of volutary instability: 1) muscular—the subluxation of the glenohumeral joint is due to the selective firing of the periscapular muscles and 2) position—resulting in glenohumeral joint subluxation due to altered position of the arm (forward flexion and adduction results in posterior subluxation).[15] Overhead athletes that experience symptoms during the late cocking phase of throwing typically have anterior instability, whereas symptoms in the follow-through phase are indicative of posterior instability. Inferior instability can be found in patients who complain of discomfort or "traction paresthesias" when carrying objects at their side. A family history should also be obtained to determine whether soft tissue disorders such as Marfan's of Ehlers-Danlos are present.

PHYSICAL EXAM

Examination of the acutely dislocated shoulder is usually very painful. In the anterior dislocation, the head is usually palpable anteriorly, with a sulcus seen below the acromion. The muscles are usually in spasm, and the arm is held in slight abduction and external rotation, with internal rotation often limited. Posterior dislocations may demonstrate little evidence of deformity. The shoulder may appear flattened anteriorly with a prominence seen posteriorly. Forward elevation and external rotation are limited to neutral. The shoulder asymmetry may be best seen from above while standing behind the patient.[1] In the setting of an acute dislocation, documenting the function the axillary nerve can be done by voluntary isometric contraction of the deltoid. Sensory innervation over the lateral deltoid has been shown to be less reliable as an indicator of the axillary nerve.

After the acute symptoms following dislocation have resolved, more specific exam tests can be performed regarding instability. The physical exam should include visual inspection for symmetry and atrophy, palpation, range of motion (ROM), strength, previous incisions, and neurologic status. Before the glenohumeral joint is examined exclusively, the cervical spine and scapulothoracic joint need to be examined. Signs of generalized laxity should be documented including hyperextension of the elbows and metacarpophalangeal joints, genu recurvatum, patellar subluxation, and the ability to hyperabduct the thumb.

The examination of laxity needs to be performed on a completely relaxed patient or someone who has been anesthetized. The degree and direction of laxity are of interest as well as the reproduction of symptoms. The affected shoulder is compared with the unaffected side if the symptoms are not bilateral. As these tests are performed, symptoms may develop when the GH joint is placed in the unstable position. A reproduction of pain and a protective reflex guarding may then be noticed by the examiner.

Anterior–Posterior Translation Test

The anterior–posterior (AP) translation test is performed with the patient sitting (Fig. 1) or lying in the supine position. While the medially placed hand stabilizes the scapula, the other hand grasps the humeral head (patient sitting) or elbow (patient supine) and the humeral head reduced into the center of the glenoid and then translates the humerus anteriorly and then posteriorly. The examiner should reference the translation to the glenoid rim. A grading system has been developed, +1 is to the glenoid rim, +2 is over the rim with spontaneous reduction, and +3 is the result of the humeral head locking out over the rim without spontaneous reduction.

Figure 1 This lateral vantage point of the right shoulder demonstrates the hand position of the examiner for the anterior–posterior (AP) translation test.

Sulcus Sign

The sulcus test is performed with the patient sitting or standing while an inferiorly directed or downward force is applied with the arm at the side in neutral and external rotation (Fig. 2). The lateral acromial surface is visualized and palpated for inferior humeral head subluxation. This examination tests for laxity of the coracohumeral and superior glenohumeral ligament. The test is repeated with the arm in external rotation, and an intact rotator interval should diminish the inferior translation.

Apprehension Test

The apprehension test is performed with the patient seated or supine. If the patient is seated, the examiner places one hand on the scapula, with the long finger on the coracoid, the index finger on the anterior humeral head, and the thumb posterior on the humeral head. The other hand supports the forearm and abducts the arm at 45 and 90 degrees while externally rotating the arm into the cocking position (Fig. 3). The patient is directed to report any sensations of apprehension or pain. If symptoms are reproduced, then a posteriorly directed force should be applied to try and relieve the symptoms (Fig. 4). This is known as the relocation test, which is considered positive if the patient's symptoms are relieved. The combination of these tests in the supine position was described by Jobe and Kvitne.[16]

Figure 2 (A) The sulcus test is performed with the patient sitting in this case. The arm position is neutral at the side. The examiner places one hand at the superior aspect of the articulation, while the other hand grasps the distal aspect of the humerus. (B) An inferiorly directed force is applied to the humerus in the neutral position, while the articulation is assessed for inferior subluxation. This tests for laxity of the coracohumeral and superior glenohumeral ligament.

Jerk Test

The jerk test is performed in the supine or sitting position after the apprehension test. The arm is forward flexed to 90 degrees and internally rotated (Fig. 5). The humerus is loaded in a posterior direction and adducted across the chest. A sudden posterior movement or "jerk" indicates posterior subluxation. When the arm is returned to its original position, a second "jerk" may be felt.

Figure 3 The apprehension test, in this case, is performed with the patient in the sitting position, arm abducted to 90 degrees with external rotation. The examiner's hand applies an anteriorly directed force to the humerus and assesses the articulation for anterior translation of the humeral head with the patient reporting a sense of discomfort or apprehension about an impending dislocation/subluxation.

Anterior and Posterior Load and Shift

The load and shift test was described in 1993 by Silliman and Hawkins.[17] For this test, the patient should be

Figure 4 The relocation test is performed after the apprehension test. If the patient's symptoms are relieved, then the test is reported as positive.

Figure 5 The jerk test is performed in this case with the patient is sitting position and generally follows the apprehension test. The arm is forward flexed to 90 degrees and internally rotated. The humerus is loaded in a posterior direction while being adducted across the chest. A sudden posterior jerk, or subluxation, indicated a positive test.

Figure 6 The O'Brien's active compression test is shown in this image. The patient is asked to forward flex the arm to 90 degrees with the elbow extended. The arm is internally rotated so the thumb points down. The patient is then asked to resist a downward force. The test is positive for possible superior labrum anterior and posterior pathology if the patient experiences pain during this maneuver.

seated with the examiner behind the subject. The scapula is stabilized with one hand while the opposite hand first "loads" the humerus and then translates it anterior and posterior. The amount of translation is noted.

Superior Labrum Anterior and Posterior Tests

Superior labral tears were discussed by Andrews et al.[18] as a source of pain and dysfunction in overhead athletes. The SLAP lesion (superior labrum anterior and posterior) was first described by Snyder et al.[19] in 1990. Snyder's classification system describes the morphology of the tear as well as the extent of involvement of the long head of the biceps. He identified four types of lesions: A type I lesion demonstrates degenerative fraying of the superior labrum; a type II lesion occurs when the superior labrum is detached from the glenoid, leaving at least 5 mm of bone uncovered; a type III lesion has a bucket-handle tear of the superior labrum; and a type IV lesion has a bucket-handle tear that extends into the biceps tendon.

There have been several physical exam tests created to elicit findings in those patients with labral tears. All of these tests are similar variations that try to load or place tension on the biceps and superior labrum. Few of these tests have been independently evaluated, and they are not absolutely diagnostic: The examiner must also rely on the history and the imaging modalities when considering this diagnosis. One of the more commonly used SLAP tests is O'Brien's Active Compression test[20] (Fig. 6). The patient is asked to forward flex the arm to 90 degrees with the elbow extended and the arm adducted 15 degrees. The arm is internally rotated so the thumb points down. The patient then resists a downward force on the arm. Next, the forearm is supinated and the maneuver is repeated. The test is positive if pain is experienced in the first position and reduced with the second. Pain localized to the top of the shoulder or the acromioclavicular joint is diagnostic of acromioclavicular joint abnormality. Pain and clicking experienced in the joint is indicative of labral injury.

Speed's test has been described to elicit symptoms in anterior SLAP lesions. Speed's test is defined as anterior shoulder pain in the region of the biceps when resistance is applied to the forward flexed arm (90 degrees) with the forearm supinated. Tendonitis of the long head of the biceps can also cause pain in a similar manner.

IMAGING

Radiographic findings are common in patients with shoulder instability. Acutely, plain radiographs help confirm the degree and direction of instability as well as bony lesions or fractures. The glenohumeral joint is most reliably imaged with three standard views. Standard views for initial review should include the AP shoulder, axillary lateral, and scapular Y-view. Matsen et al.[8] state that these views should be oriented in the plane of the scapula to provide the critical information about the possible associated bony injuries.

For recurrent shoulder instability, additional radiographs may be helpful. Walch et al.[21] showed osseous lesions of the humerus or glenoid in 152 of 160 shoulders (95%) in patients with chronic anterior instability. A Hill-Sachs lesion was identified in 117 of 160 shoulders (73.1%). The glenoid profile view (West-Point or Bernageau) showed an osseous lesion of the glenoid in 126 of 160 shoulders (78.8%). In those patients with recurrent instability, the loss of the normal anterior triangle without an obvious fracture was called the "cliff sign." A rounding off of the anterior glenoid was called the "blunted angle sign." In Walch et al.[21] series, the author always used the unaffected side for comparison. The Stryker Notch view can help identify Hill-Sachs lesions that may not be seen as well on an AP view in internal rotation.

The anterior, inferior glenoid has been proven in the literature to be of importance with regard stability in multiple studies. The traditional axillary view is used to assess the anterior rim of the glenoid,[1] whereas the West Point view details the anteroinferior glenoid rim.[22] Recently, Itoi et al.[23] quantified the size of the glenoid defect as it relates to stability. They demonstrated that an osseous defect with a width greater than 21% of the glenoid length caused instability after Bankart repair and required bone grafting for acceptable results. Itoi et al.[24] showed the West Point view to be an accurate method for assessing anteroinferior glenoid defects. Their study showed a 21% defect to be 18.6% of the intact glenoid on the West Point view. If the West Point view is of limited value or unable to be obtained because of pain or apprehension, then a computed tomography scan should be used to delineate the bony defects. An arthroscopic assessment has been reported by Burkart[25] and has been described as the inverted pear glenoid. Burkhart and DeBeer describe significant bone loss as 25% or greater of the normal glenoid width. In their series of patients, the rate of failure of arthroscopic repairs was significantly greater (67%) when the inverted pear glenoid was present.

If plain radiographs cannot clearly identify the bony anatomy, then computed tomography can be of great assistance for delineating humeral head defects and glenoid bone loss. Computed tomography can also be used to evaluate humeral and glenoid version. Excessive version can lead to instability in that direction.

Magnetic resonance imaging has been shown to be very sensitive in detailing the intra-articular soft tissues. Magnetic resonance imaging can be ordered with or without intra-articular contrast. Standard magnetic resonance imaging can be useful to identify rotator cuff tears, bone contusions, and impaction fractures. Standard magnetic resonance is less sensitive in evaluating labral abnormalities.[26] The ability to distend the glenohumeral joint as well as the improved soft tissue contrast makes magnetic resonance arthrography the procedure of choice to evaluate the capsular volume and labrum (Fig. 7A, 7B). The SLAP lesion (Fig. 8A, 8B), as described by Synder,[19] and the humeral avulsion of the glenohumeral ligament (HAGL)[27] and bony humeral avulsion of the glenohumeral ligament (BHAGL)[28] lesion are best appreciated with arthrography. The additional sequences in abduction and external rotation position have been shown to increase the sensitivity of visualizing tears in the rotator cuff and the anterior labrum as well.

Rarely, electromyographic and nerve conduction studies are ordered to help delineate among root level, brachial plexus, and peripheral nerve injuries.

MANAGEMENT

The management of acute shoulder dislocations has been discussed in great depth in variety of texts and journals. The acute management includes a thorough history and physical exam with the proper radiographs. After the initial assessment, the shoulder needs to be reduced and immobilized for a short period of time before a rehabilitation program is begun. The duration of immobilization in a sling is debatable, but most authors agree that it should be discontinued by the third week. The exercise therapy includes gentle passive forward flexion and external rotation to 40 degrees. At 6 weeks post-dislocation, rotator cuff strengthening and scapular stabilizing exercises are usually begun. At the 12-week mark, sport-specific exercises and plyometrics are used to help athletes get back into their sport. Burkhead and

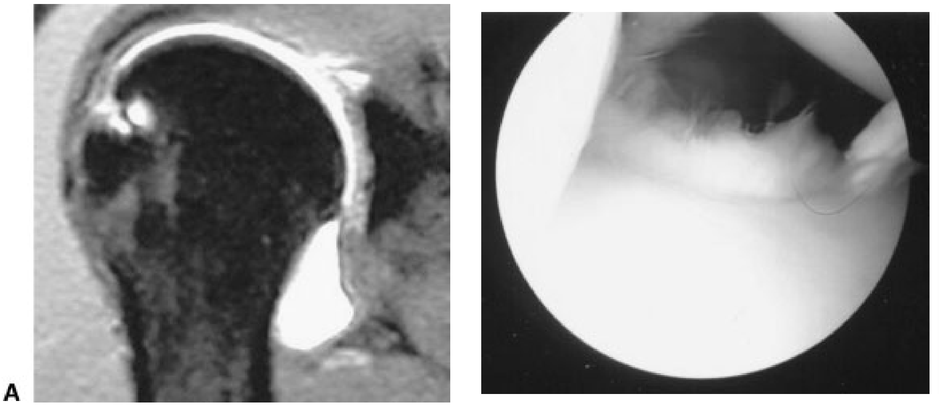
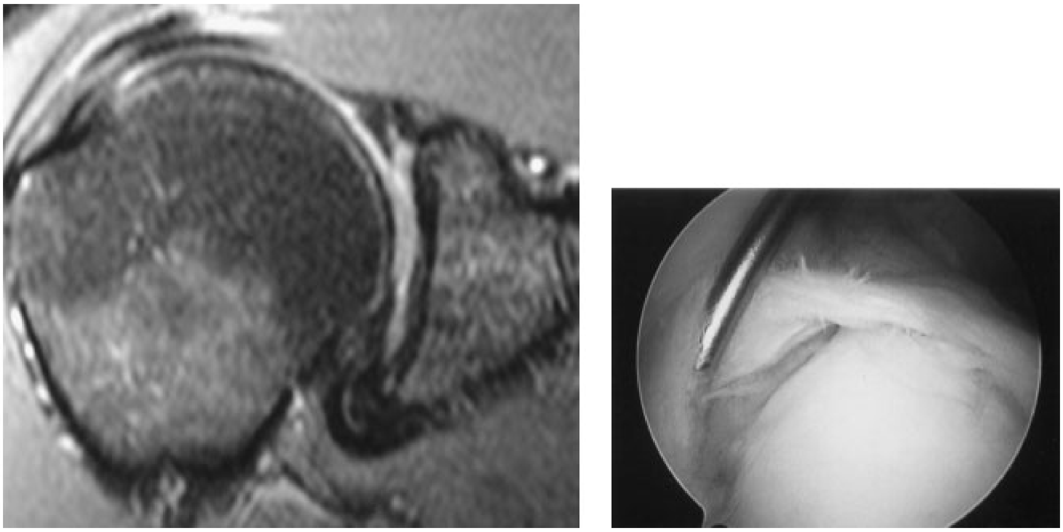


Figure 7 (A) Coronal magnetic resonance arthrogram image shows abnormal contrast collection within the posterior superior labrum consistent with labral tear. (B) Corresponding arthroscopic image shows the torn and frayed appearance of the glenoid labrum.



A B

Figure 8 (A) Standard coronal magnetic resonance image shows abnormal signal within the substance of the superior labrum, consistent with superior labrum anterior and posterior pathology. (B) Corresponding arthroscopic image shows the irregular and frayed appearance of the superior labral tissue, confirming the imaging abnormality.

Rockwood[29] studied the effect of rehabilitation after shoulder dislocation. The rehabilitation included rotator cuff, deltoid, and scapular stabilizers. In the traumatic group, 16% of the 74 shoulders had a good or excellent result. In the atraumatic group, 80% of the 66 patients had a good or excellent result. Their study identifies the importance of etiology with the risk of recurrence and the resulting treatment.

The surgical management of patients after their initial dislocation has become more accepted in recent years. As discussed previously, certain patient populations have a high risk of recurrent shoulder instability. These patients may be best managed with surgery before multiple episodes of recurrent instability have been demonstrated. Chronic instability often leads to impaction fractures and glenoid bone loss, which usually necessitates open procedures. In a prospective study[30] from the U.S. Military Academy at West Point, cadets with acute shoulder dislocations were managed with arthroscopic repair versus nonoperative treatment. This high-demand patient population had a recurrence rate of 14% in the surgical group and 80% in the nonsurgical group. Another study[31] of immediate arthroscopic stabilization after initial dislocation resulted in a rate of redislocation of 15.9% at an average follow-up of 33 months, compared with 47% for exercise treatment.

The role of arthroscopic surgery for shoulder instability continues to evolve. A variety of shoulder diagnoses have been described since the introduction of the arthroscope. Many of these lesions such as the SLAP tears are best treated arthroscopically, but some such as the HAGL and the BHAGL lesions are best treated with open surgery. For the patients with MDI, arthroscopic rotator interval closure as well as anterior and posterior capsular plication methods have been used to stabilize the glenohumeral joint.

Most recent reports of open instability repair are emphasizing anatomic repairs. In patients with significant bone deficiency, most authors would agree that lesions of the glenoid involving 20–25% loss of articular surface or large Hill-Sachs lesions (involving 40% or greater of the humeral head) are best treated with open surgery. For patients with massive Hill-Sachs lesions, often osteochondral allografting or humeral head replacement may be needed to restore the articular anatomy of the humeral head.

Thermal capsulorrhaphy has been used to treat shoulder instability since the late 1990s. This technique uses thermal energy delivered by a holmium:yttrium aluminum garnet (Ho:YAG) laser or radiofrequency (RF) probe. More recently, the RF probes in either the monopolar or bipolar variety have become the standard. The exposure to thermal energy causes the denaturation of collagen fibers with shortening. Collagen shortening begins between 65° and 75°C. As the amount of energy applied increases, so does the shortening. If the collagen is shortened more than 15–20%, the material properties are weakened and the mechanical strength decreases.[32] The surgical indications for thermal capsulorrhaphy remain unclear.[33] The amount of energy applied to the tissue, the length of the application, and the temperature at which to set the probe to result in safe levels of use have not been adequately studied. In the patients who have failed thermal capsulorrhaphy, the revision may be difficult because of capsular necrosis, resulting in marked capsular insufficiency. The other structure at risk is the axillary nerve, which is directly adjacent to the relatively thin, inferior capsule.[34] The enthusiasm for this technique has subsided in the last few years for these reasons.

There have been no randomized, evidence-based human studies to establish the parameters for its use. There have been several studies demonstrating good results with short-term follow-up;[34] however, most studies have used a suturing technique in addition to the use of thermal energy. At present, the use of this technology remains controversial and is used as an adjunct to suture stabilization.

COMPLICATIONS

Surgical complications of glenohumeral instability may be grouped into several categories. Matsen et al.[1] have described these complications in great detail. As with any surgical procedure, complications such as infection, neurovascular injuries, and hematoma formation can occur. Proper surgical technique, knowledge of the surgical approach and anatomy, and hemostasis before closure can minimize many of these complications. The axillary nerve and musculocutaneous nerve are at risk with many of the shoulder procedures, and great care must be taken to identify and protect these nerves while retracting the soft tissues for exposure.

The most frequent complication of both open and arthroscopic repair for instability is recurrent instability; failure rates appear to be related to the surgical technique. Some of the early arthroscopic repairs using the transglenoid sutures and the staple capsulorraphy tended to medialize the repair on the glenoid. This lead to unacceptable failure rates. Open procedures such as the Dutoit staple capsulorraphy also tended to medialize the repair and had recurrence rates of 22%. These operations tended to create an anterior labral periosteal sleeve avulsion lesion, which has been shown to have poor results. With the appropriate technique, arthroscopic repairs have been shown to equal the repair rates of open repairs in patients without significant bone defects. Recurrence rates of 4–7% for arthroscopic Bankart repairs[25,35] have been reported.

The loss of external rotation because of capsular contraction after instability repair can lead to a process called “capsulorraphy arthropathy.” The nonanatomic open repairs (Putti-Platt and Magnuson-Stack) lead to a contracted anterior capsule, causing increased posterior shear stresses on the glenoid. Over time, the articular cartilage degenerates posteriorly, leading to arthrosis and pain.

Subscapularis tendon failure can occur after open instability repairs. The subscapularis muscle may be released or divided during the exposure. On physical exam, a patient may demonstrate weakness in internal rotation by means of the lift-off or belly-press tests or by anterior instability. Meticulous closure of the subscapularis is necessary to prevent this complication. Irreparable tears may need a tendon transfer such as from the pectoralis major.

An additional area of complications relates to the use of implants that are bioabsorbable or metallic. These implants can loosen, migrate, or invoke a granulomatous reaction. This can lead to symptoms requiring reoperation.

The use of thermal energy through laser or radiofrequency devices for the treatment of capsular laxity has brought about its own set of complications. Reports of capsular necrosis, loss of capsular integrity, axillary neuritis, and stiffness following thermal capsulorraphy have been described.[36–38] These complications may be minimized by applying the heat probe in a striped fashion, keeping the temperature settings within the prescribed range, not leaving the probe in one area for too long, and not applying too much pressure to the tip.

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