NCBI Bookshelf. A service of the National Library of Medicine, National Institutes of Health.

StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2019 Jan-.

# Anterior Shoulder Instability

**Authors**

Matthew Varacallo[1]; Scott D. Mair[2].

**Affiliations**

[1] Department of Orthopaedic Surgery, University of Kentucky School of Medicine
[2] Dept. of Orthopaedic Surgery, UK

Last Update: February 12, 2019.

## Introduction

Glenohumeral instability encompasses both dislocation and subluxation events, and instability events commonly affect the general population. Approximately 1% to 2% of the general population will experience a glenohumeral dislocation in their lifetime.[1] The young, active, athletic population is particularly susceptible to shoulder instability events. Over 95% of shoulder instability events occur in the anterior direction.[2][3]

Over the last few decades, the knowledge regarding the risk factors, diagnosis, nonoperative and surgical management, and rehabilitation principles has continued to improve, permitting clinicians to provide optimal management of shoulder instability patients. A comprehensive history, clinical exam, diagnostic imaging studies, and accurate characterization of each patient's instability pattern will help enable healthcare providers to properly treat and manage each patient appropriately.

## Etiology

Historically, clinicians used the acronyms *TUBS* and *AMBRI* to classify shoulder instability into two broad, etiologic categories:

- Traumatic, Unilateral, Bankart lesion, Surgery (TUBS)
- Atraumatic, Multidirectional, Bilateral, Rehabilitation, Inferior capsular shift (AMBRI)

However, while these acronyms were traditionally helpful to aid providers in the identification and treatment of patients presenting with acute or recurrent instability, these categories do not help the clinician differentiate instability from soft tissue hyperlaxity. As our knowledge regarding the etiology and underlying pathophysiology at clinical presentation evolved over the last few decades, clinicians began to recognize the need to differentiate possible complex shoulder instability patterns that can present in the setting of recurrent and repetitive microtrauma (often low-energy) from acute macrotrauma (often high-energy). Thus, it is more helpful to classify based on[4]

- Uni- or multi-directional instability
- Traumatic or atraumatic
    - Traumatic cases are often unidirectional with an associated capsulolabral injury
    - Atraumatic cases are often multidirectional with associated hyperlaxity
- Presence or absence of accompanying soft tissue hyperlaxity
    - Causes of soft tissue hyperlaxity, including patulous capsular laxity, can be congenital, or secondary to repeated microtrauma, major trauma, multiple instability events, and recurrences, or a combination of all of these factors

## Epidemiology

There are few reports in the literature regarding the estimated incidence rate of shoulder dislocations among the general population. The general consensus in the literature cites an incidence rate of traumatic unidirectional dislocations ranging from 1% to 2% in the general population.

One study from the 1980s reported that over a 10-year period the adjusted incidence of initial traumatic shoulder dislocations was 8.2 per 100000 person-years. The incidence rate seen in males was greater than that seen in females.[5] A subsequent study from the 1980s reported that in Denmark the incidence rate of shoulder dislocations was about 17 dislocations per 100000 person-years.[6] A 1995 study from Sweden reported an incidence of 24 dislocations per 100000 person-years.[7] A population-based cohort study of nearly 17000 patients in the United Kingdom investigated over 20 years (1995 to 2015) found[8]:

- 72% of shoulder dislocations occurred in men
- The highest incidence rate (80.5 per 100000 person-years) occurred in male patients, aged 16 to 20 years
- The highest incidence rate in female patients occurred in women aged 61 to 70 years
- The overall incidence rate in male patients was 40.4 per 100000 person-years

A 2013 study out of Ontario, Canada similarly found that young male patients had the highest incidence of shoulder dislocation events, in addition to the highest risk of recurrent dislocation events.[9]

A 2010 study reported from United States census data analyzed the epidemiologic trends for emergency department presentations secondary to shoulder dislocations from 2002 through 2006. The authors noted an overall incidence rate of 23.9 per 100000 person-years, with the male incidence rate being about 2.6-fold that of the female incidence rate.[10]

**Athletic population considerations**

The young athletic population has garnered significant attention in the literature secondary to the multitude of risk factors predisposing these patients to primary and recurrent dislocation events.

A 2007 study by Owens et al. prospectively collected all traumatic shoulder instability events at the United States Military Academy over a 12-month period. The study followed over 4000 students during this period, and the authors found that the probability of a student sustaining at least one shoulder instability event during the academic year to be 2.8%.[11] The study encompassed subluxation and dislocation events. Other pertinent findings included:

- Greater than 85% of all instability events occurred in males
- 85% of instability events were subluxation episodes, while the remaining 15% were dislocation events

A 2014 study reported the prevalence of shoulder dislocation during one season of rugby was about 15%. The authors noted that in rugby players entering the current season with a history of a previous shoulder dislocation managed non-operatively. In this subset of athletes, about 55% sustained a shoulder dislocation during the season.[12] Other collision sports such as football have demonstrated incidence rates up to 0.51 per 1,000 athlete-exposures.[13]

A 2018 epidemiologic study comparing high school and collegiate athletes across nine different sports demonstrated that collegiate athletes are at higher risk of shoulder dislocation compared to their high school counterparts.[14] The overall incidence rate was 2.04 per 100000 athlete-exposures; an exposure defined as one athlete participating in one practice or game/competition.

*Multicenter Orthopaedic Outcomes Network (MOON)*

The MOON instability cohort published a 2018 study reporting the descriptive epidemiology of patients undergoing shoulder stabilization procedures across a multicenter consortium[15]:

- 82% (709 of 863 total patients) were male
- Anterior dislocations made up the majority of cases; about three-fourths in both male and female patient subgroups
- Football and basketball were the most common sport-related presentations
- Anterior labral tears were seen in two-thirds of case presentations, while 41% had a Hill-Sachs lesion noted on pre-operative MRI

*Adolescent and pediatric patient considerations*

A recent Level II prognostic cohort study reported results from approximately 2,000 adolescent and pediatric patients ages 10 to 16 years presenting to an emergency department and required closed shoulder reduction. Results showed that the incidence of dislocation was highest among males aged 16 years (164.4 episodes per 100000 person-years), while the incidence was lowest among patients aged 10 to 12 years. Moreover, the re-dislocation rate was highest in 14 to 16-year-old patients (37% to 42% re-dislocation rate) and the lowest re-dislocation rates (0% to 25%) were in patients aged 10 to 13 years old.[16]

## Pathophysiology

**Anatomy**

The glenohumeral joint is a complex, mobile, multiaxial, ball-and-socket articulation that allows coordinated motion in the frontal, transverse, and sagittal planes. The latter allows for 360 degrees of circumduction. The relatively shallow glenoid fossa articulates with the much larger humeral head to allow for a wide range of physiologic motion. In addition, the joint capsule is relatively lax. Shoulder movements occur secondary to the dynamic and coordinated articulations at four distinct joints:

- Sternoclavicular
- Acromioclavicular
- Glenohumeral
- Scapulothoracic

*Static and dynamic stabilizers*

The static stabilizers include the glenohumeral articulation, the labrum, the glenohumeral ligaments, rotator cuff interval structures, and the negative intra-articular pressure. The dynamic stabilizers consist of the rotator cuff muscles, the deltoid, and the scapular and periscapular stabilizers.

**Shoulder instability cascade**

The pathophysiology and pathologic cascade for shoulder instability acute episodes, recurrence, and long-term implications. There is significant variability in the amount of shoulder translation in asymptomatic, normal subjects. However, shoulder instability is a pathologic process that results in an excessive translation of the humeral head on the glenoid that ultimately results in pain, weakness, and shoulder dysfunction.

There have been several anatomic risk factors that have been identified as potential contributors to developing shoulder instability. For example, increasing degrees of glenoid version away from neutral in addition to an inferior inclination of the glenoid vault is associated with anterior shoulder instability.[17]

To accurately diagnose and classify shoulder instability, it is critical to distinguish joint laxity from true instability. The latter characteristically demonstrates the presence of symptoms in association with abnormal laxity. The pathologic soft tissue laxity can be present either from repetitive microtrauma, high-energy acute trauma (e.g., traumatic dislocations), or it can be present congenitally. Shoulder instability implies some degree of disruption to the static

and/or dynamic stabilizers of the glenohumeral joint, including the osseous structures, capsule, labrum, and glenohumeral ligaments. In regards to the latter, the anterior and posterior bands of the inferior glenohumeral ligament (IGHL) are the most critical ligamentous structures in the maintenance of shoulder stability.

**Unidirectional instability**

Unidirectional instability can present following acute trauma or following one or multiple low-energy instability events. The latter may be present in the setting of unidirectional instability with or without hyperlaxity. When there is associated soft tissue hyperlaxity, an accompanying capsulolabral lesion is less likely. In effect, the unidirectional instability is often attributable to the patulous capsular tissue that predisposes the shoulder to recurrent instability events. However, the same unidirectional instability scenario in a patient without soft tissue hyperlaxity most commonly will present with an associated capsulolabral injury.

Following a traumatic, primary shoulder dislocation, there are a significant number of patients that will demonstrate at least a mild degree of either chondral or osteochondral lesions.[4][18] Bony defects can be on the humeral side, glenoid side, or a combination of both. It has been previously established that bony defects on either side can significantly contribute to the risk of recurrent instability events.[19]

*Humeral sided defects*

The Hill-Sachs lesion is an impaction injury to the posterosuperolateral humeral head that occurs in association with anterior instability events. Described in 1940 by Hill and Sachs,[20] the authors highlighted the so-called line of condensation that was apparent on the posterosuperolateral humeral head and best viewed on the internal rotation shoulder radiograph. The Hill-Sachs lesion occurs when the humeral head dislocates anterioinferiorly, and the aforementioned area of the humeral head abuts the anterior glenoid rim and upon impact creates a compression fracture on the humeral head. The true incidence of Hill-Sachs lesions remains elusive although studies have cited its association with anterior shoulder instability events to range from 40% to almost 100% of cases, with the highest rates seen in cases of recurrent anterior instability.[21][22][23][24]

The impact of recurrent instability on the Hill-Sachs lesion becomes problematic as the abnormal anterior soft tissue laxity predisposes the Hill-Sachs lesion to repeatedly abut against the harder cortical bone at the anterior glenoid.[25] In addition, Hill-Sachs lesions can be engaging or non-engaging. Engaging lesions have been previously described by Palmer and Widén,[26] and later by Burkhart and De Beer.[27] These occur when the Hill-Sachs lesion's long axis is parallel to the long axis of the anterior glenoid rim, causing the lesion to engage with the rim during defect engages the rim of the glenoid while the shoulder is in any combination of positions. The authors previously described the athletic position of the shoulder (i.e., 90 degrees of abduction and from 0 to 135 degrees of external rotation). A non-engaging lesion will not engage with the anterior glenoid rim when the shoulder is in an abducted and externally rotated position. The analogous lesion seen in association with posterior shoulder instability is a humeral head defect located at the anterosuperomedial region and is referred to as the reverse Hill-Sachs lesion.

*Glenoid sided defects*

The prevalence of glenoid bone loss is also not consistently reported in the literature although most authors agree that in the setting of recurrent instability there will inevitably be some degree of glenoid bone loss.[28] Some studies have cited up to 22% of patients will present with some degree of glenoid deficiency after an initial dislocation event,[24] increasing to 25% to 90% of patients presenting with recurrent instability,[21][28][29][30][31] and in 90% of patients presenting status post failed primary stabilization procedures.[27]

The nature of glenoid defects is best appreciated on three-dimensional CT scans, having the ability to differentiate between a true bony Bankart lesion from the more erosive changes that can be seen representing an attritional bone loss state. The former is considered a true fractured fragment containing a portion of the anterior glenoid rim. The latter often appears rounded off with blunted edges on imaging, indicative of a more chronic, erosive process secondary to recurrent instability or, in the acute setting, a low-energy compression fracture. The description of the location of these defects often references relative to a clock face created by drawing a circle around the glenoid. Saito et al. reported that the most common location of glenoid defects in the setting of anterior instability to be between 2:30 and 4:20.[32]

The pathologic mechanism of glenoid bone defects is likely multifactorial. In general, blunted osseous glenoid defects can present in the setting of either:

- Acute, low-energy, compression fracture
- Chronic/recurrent, low-energy, attritional deficiency and erosive changes over time

Chronic attritional glenoid erosive changes can develop over time following a high-energy, index dislocation event that suffers an accompanying glenoid injury initially. The injury is either managed nonoperatively or ultimately fails surgical fixation to ultimately result in subsequent instability events with progressive attritional glenoid erosion over time.

Glenoid rim fractures and acute avulsion injuries often result secondary to high-energy impact collisions. Moreover, traumatic dislocations yielding associated glenoid injuries can produce large, anterior glenoid rim fracture fragment(s), or smaller glenoid rim avulsion injuries. Burkhart and De Beer advocated that presence of an acute glenoid rim fracture suggests a significant axial load impacted the glenoid rim at the time of dislocation. The authors compared shoulder dislocation events seen in South African rugby players compared to American football players. The former had about a 10% incidence rate of associated glenoid rim fractures, while American football players in this particular study had a 0% association rate of glenoid rim fractures.[27] The authors speculated that the discrepancy was attributable to the fact that during rugby, a greater axial load force of impact is transmitted through the glenoid from the humerus to generate the characteristic anterior glenoid rim fracture. In contrast, the shoulder dislocation mechanism seen during football is more rotational in nature, resulting in a relatively smaller anterior glenoid rim avulsion injury as the inferior glenohumeral ligament pulls the fragment off during the anterior dislocation.[30]

**Multidirectional instability**

Since the initial description of multidirectional shoulder instability (MDI), our knowledge regarding the pathologic findings and objective criteria for diagnosing MDI remain debated. Part of the clinical ambiguity is attributable to the lack of a precise definition for MDI. Neer and Foster initially described MDI as anterior and posterior instability associated with involuntary inferior subluxation or dislocation.[33] Other authors have defined MDI as instability in at least two to three directions.[34][35] The estimated prevalence of MDI also remains elusive given the previously mentioned ambiguity.

The same principles applied earlier for unidirectional shoulder instability classifications are also applicable to the MDI setting. Anterior and posterior capsulolabral injuries will often accompany MDI without soft tissue hyperlaxity. MDI patients with soft tissue hyperlaxity will often present with instability in two or more directions and display signs of generalized hyperlaxity on physical exam. Patients more commonly report relatively frequent, recurrent low-energy subluxation events.

**Long-term implications**

*Altered shoulder girdle biomechanics*

Glenoid bone loss decreases the available articular arc and generates glenoid-humeral mismatch. The former generates a smaller articular arc length and decreased surface area by which the glenoid cavity can resist transmitted axial forces from the proximal humerus. Thus, failing to address glenoid bone loss even at the time of surgical fixation will result in significant shear forces imparted to a repaired capsulolabral interface.[27]

Alterations in the static and dynamic scapular movements present in the setting of glenohumeral instability. Scapular dyskinesia is reportedly present in up to 80% of instability patients.[36] Biomechanical studies analyzing the at-risk scapular posturing leading to pathologic anterior tensile loads and shear forces include excessive:

- Anterior tilting
- Internal rotation

- Protraction

In addition, scapular dyskinesia predisposing to anterior shoulder instability also includes decreased upward rotation of the scapula during scapulothoracic movements. The end-result of altered scapular posturing yields excessive tensile loads and shear forces experienced by the anterior band of the inferior glenohumeral ligament (aIGHL).

*Recurrent instability*

Recurrent instability can develop in the setting of any of the above mentioned underlying pathophysiologic mechanisms. Furthermore, recurrent instability can present following nonoperative and operative management. Several studies have indicated that almost 90% of contact athletes and up to two-thirds of non-contact athletes with significant glenoid bone loss ultimately fail surgical stabilization secondary to associated Bankart lesion or abnormal glenoid morphology affecting overall glenohumeral joint biomechanics.[27]

*Dislocation arthropathy*

In 1982, Neer first described a subset of patients with a history of shoulder instability events (or required surgical stabilization procedures) that developed glenohumeral arthritis at long-term follow-up.[37] The condition was officially termed dislocation arthropathy, and in 1983 Samilson and Prieto reported that these arthritic changes could follow even a single dislocation event.[38] Although the role of recurrent dislocations is unclear, the pathophysiology underlying the development of dislocation arthropathy is likely related to acute changes that occur during a primary traumatic instability event, followed by long-term arthritic predisposition secondary to altered glenohumeral joint biomechanics and progressive, attritional, erosive osseous changes that occur secondary to the recurrent instability events.

By definition, dislocation arthropathy describes degenerative changes of the shoulder joint. Hovelius and Rahme conducted a prospective study of 257 primary anterior shoulder dislocations in 255 patients. At 25-year follow-up, 27% had mild glenohumeral arthritis, and 34% had moderate to severe arthrosis.[39] Although the role of recurrent dislocations is unclear, the pathophysiology underlying the development of dislocation arthropathy may be related to acute changes during the primary or recurrent traumatic instability events, the ensuing altered glenohumeral joint biomechanics, and/or attritional changes that occur secondary to the recurrent instability.

## Histopathology

Various histologic studies have investigated the proprioceptive characteristics of the soft tissue elements of the glenohumeral joint. Ruffini corpuscles and Pacini corpuscles are distributed throughout the capsuloligamentous complex.[40] The afferent endings are susceptible to injury during instability events, resulting in delayed delivery of proprioceptive signaling that subsequently results in delayed signal transmission and delayed and/or dysfunctional muscle responses, thereby compromising both the static and dynamic soft tissue stabilizing elements supporting the glenohumeral joint.[41] Histopathologic studies comparing biopsied static stabilizing elements during intraoperative arthroscopy were analyzed and compared to macroscopic degree/grade of injury noted during surgery. The authors reported limited variations in cell density, matrix swelling, and collagen fiber disruptions.[42]

## History and Physical

Clinicians evaluating patients with either acute or chronic shoulder instability should obtain a comprehensive history. Providers should document any detailed elements regarding the index injury for both first time versus chronic presentation dislocators. In addition, other elements often include the following:

- First-time dislocators[43]:
    - Patients presenting after a single acute event typically report a recent history of high-energy trauma or collision impact causing the dislocation
    - Clinicians should inquire regarding:

        - Degree of trauma (high- or low-energy impact mechanisms)
        - Sports activity and position(s)
        - Discern a true dislocation from a subluxation event
            - Elicit the requirement for on-field or on-site manual reduction; presentation to the emergency department +/- sedation requirements

- Chronic cases[44]:
    - Patients typically present in delayed fashion once the range of motion limitations begin to significantly impact daily activities
    - Clinicians should gather a detailed history for any inciting instability events
        - The initial injury may be overlooked, and the patient subsequently develops chronic instability/recurrence
        - Heightened clinical suspicion is warranted in the setting of:
            - History of seizures or electrical shocks
            - Polytrauma in which the shoulder instability was overlooked or missed
        - Low-energy, recurrent subluxation cases
            - Shoulder instability episodes during sleep may be indicative of more complex instability that may involve significant bone loss
            - Clinicians should elicit for any medical comorbidities or family history of underlying connective tissue disorders or generalized hyperlaxity on the exam

A thorough history also includes sports participation and position(s) (if applicable occupational history and current status of employment, hand dominance, any history of injury/trauma to the shoulder(s) and/or neck, and any relevant surgical history.

**Physical Examination Pearls[45][46][47][48][49][50]**

*C-spine / neck exam*

Co-existing cervical radiculopathy should be ruled out in any situation where neck and/or shoulder pathology is in consideration. Observation of neck posturing, muscular symmetry, palpable tenderness, and active/passive ROM should undergo evaluation. Special tests that are helpful in this regard include the Spurling maneuver, myelopathic testing, reflex testing, and a comprehensive neurovascular exam.

*Shoulder exam*

Examiners should observe and compare bilateral shoulder girdles for any notable asymmetry, scapular posturing, and muscle bulk comparison or any atrophic changes. The skin should be observed for the presence of any previous surgical incisions, lacerations, scars, erythema, or induration.

Chronic instability patients will almost always exhibit at least a mild degree of asymmetry. The deltoid muscle often demonstrates atrophy in chronic dislocators. The findings can be rather subtle, especially in obese patients.

In the setting of chronic anterior instability, the clinician may be able to appreciate a palpable anterior fullness. Upon observation, the posterior shoulder (when viewed from the side of the patient) will be relatively flat relative to the anterior fullness. Chronic anterior and posterior instability patients may also exhibit corresponding posterior and anterior acromial prominences, respectively.

Scapulothoracic motion and scapular winging should also be evaluated during active and passive motion.

After the observational component of the physical examination, the active and passive ROM is documented. This exam finding may be limited in the setting of initial follow-up in the clinic after an acute instability event, or the setting of any complex instability case, especially in cases of glenoid bone loss.

In the setting of chronic anterior instability, the clinician should attempt to assess the current status of the axillary nerve, although chronic dislocators often exhibit excellent deltoid function as well as internal and external rotator strength. Specific testing of the supraspinatus muscle can be difficult when passive ROM is limited. Any evidence of significant muscular weakness may hint at an underlying associated neurologic deficit.

The clinician should perform a detailed sensory examination in all acute and chronic instability patients. In addition to axillary nerve function, motor function of the elbow, wrist, and hand should undergo an assessment to rule out the possibility of a brachial plexus injury associated with the dislocation. Distal pulses should be assessed at the wrist as well.

Older patients and in the setting of suspected concomitant shoulder pathologies (e.g., rotator cuff injuries or biceps tendon pathology), specialized testing for these pathologies should also be a consideration.

**Provocative examination testing/maneuvers[51]:**

**Shoulder instability considerations**

Global tissue laxity should undergo assessment by examining glenohumeral translation and hypermobility at the shoulder joints and other joints in the body if applicable. Hyperlaxity at other joints (e.g., elbow and knee hyperextension) may aid in the clinical diagnosis of underlying MDI-related diagnoses or connective tissue disorders.

**Anterior apprehension test:**

The anterior apprehension test is performed by lying the patient supine on the examination table. The examiner positions the shoulder to 90 degrees of abduction and 90 degrees of external rotation while applying an anteriorly-directed force to the proximal humerus. The test is positive if it reproduces the symptoms of anterior instability.

Apprehension at lower degrees of abduction may suggest glenoid bone loss. Patients may guard the shoulder during the examination, but in most circumstances, the provider can determine if the apprehensive position is reproducible the patient's feelings of anterior shoulder instability.

**Jobe relocation test:**

The Jobe relocation test is utilized with the previous apprehensive testing maneuver. Once the patient reports a subjective feeling of reproduction of the shoulder instability symptoms, the examiner applies a posteriorly directed force while keeping the shoulder in the same apprehensive position. Resolution or improvement of symptoms indicates a positive test result.

**Load-and-shift test:**

The examiner uses one hand to apply an axial load through the elbow to center the humeral head within the glenoid. An anterior and posterior directed force is then applied at 0-, 45-, and 90-degrees of shoulder abduction. Increased translation at increasing degrees of shoulder abduction implies compromise of the IGHL.

Grading of the magnitude of humeral head translation is as follows:

- Grade 1:
    - Increased translation compared with the normal shoulder
- Grade 2
    - Indicates humeral head translation to, but not over, the glenoid rim
- Grade 3

- Indicates translation of the humeral head over the glenoid rim

The load-and-shift test is up to 98% specific, but it has poor sensitivity for detection of unidirectional and multidirectional instability.

**Other exam considerations**

The shoulder also should be examined for associated posterior instability as well as multidirectional instability. In every patient undergoing surgery, examination under anesthesia should take place and the results compared with those of the uninjured contralateral side.

Depending on patient age, the clinician may expect various degrees of associated shoulder pathologies. For example, older patients presenting with acute shoulder instability are susceptible to associated rotator cuff injuries. Pain with weakness against resisted abduction or external rotation may suggest the presence of associated rotator cuff injuries. For example, patients with anterior shoulder instability in end-range positions demonstrate some degree of external rotation weakness.[52] An increased level of passive external rotation with the arm at the side is suggestive of a subscapularis tendon tear, whereas reduced external rotation in 90 degrees of abduction may indicate a medially healed Bankart lesion.[53]

## Evaluation

**Radiographs**

Recommended imaging includes a complete radiographic trauma series, consisting of a true anteroposterior (AP) image of the glenohumeral joint (i.e., the "Grashey" view). The true AP image is taken with the patient rotated between 30 and 45 degrees offset the cassette in the coronal plane. Alternatively, the beam can be rotated while the patient remains neutral in the coronal plane. In addition to AP imaging, an axillary lateral radiograph should be obtained. This view may be difficult to accomplish in the acute traumatic setting. However, the importance of obtaining a complete radiographic series with adequate orthogonal imaging cannot be overemphasized. Unfortunately, it is not uncommon to miss shoulder dislocations, especially posterior dislocations, when an incomplete radiographic evaluation is all that is available.

*Pertinent findings*

The axillary lateral view provides essential information about the position of the humeral head relative to the glenoid. Additionally, the presence and degree of degree of anterior (or posterior) glenoid erosion or glenoid dysplasia, as well as any potential humeral head defects can be appreciated on this view as well. Anterior glenoid rim fractures or glenoid avulsion injuries may also be present. The presence of medial neck scalloping or a possible avulsion injury is visible on AP imaging in the presence of an associated humeral avulsion of glenohumeral ligaments (HAGL).[54]

**Advanced imaging modalities**

Magnetic resonance imaging is the preferred imaging modality to visualize for the presence of associated labral injuries. The addition of intra-articular contrast (i.e., MR arthrography) increases the sensitivity and specificity for the identification of associated injuries. Further, MRI imaging can provide clinicians with an accurate assessment of potential osseous glenohumeral defects or glenohumeral avulsions (off the glenoid or humeral sides).

CT imaging is the preferred imaging modality when attempting to quantify bone loss. Although a plethora of quantification techniques exist, the three-dimensional reconstruction techniques are superior to two-dimensional assessments. The latter relies on the proper sagittal orientation along the long axis of the glenoid to accurately characterize glenoid bone loss.[55]

Three-dimensional en face reconstructions of the glenoid with humeral subtraction have become the gold standard for glenoid assessment given their ability to provide easily reproducible quantification of glenoid morphology.[55][56]

## Treatment / Management

:The initial management of acute shoulder instability depends on a multitude of factors. The acute management always should consist of manual reduction (if necessary) following confirmation of a dislocation event. The exact timeline for the initial period of immobilization remains debated. The acute management (e.g., sideline team physician management) consists of sling immobilization with avoidance of a return to play until completion of a comprehensive assessment.

Owens et al. previously proposed an algorithm to help guide the management of mid-season traumatic anterior shoulder instability in high-level athletes. The authors suggested a set of considerable clinical and sport-specific factors that could serve to guide the management of instability in athletes.[57]

Relative indications for nonsurgical management of traumatic anterior shoulder instability include:

- First-time dislocations
- Osseous defects of the glenoid less than 25%
- Humeral head (i.e., Hill-Sachs) defects less than 25%, non-engaging lesions
- Athlete factors:
    - Desires to return to sport in-season
    - Noncontact athlete
    - Non-overhead/non-throwing athlete
    - Responds to bracing during competition and able to complete sport-specific drills without instability

By contrast, surgical management would be advocated earlier in the management course if the case meets any of the following criteria

- Associated injuries are present:
    - Rotator cuff tear (e.g. >50%)
    - Large bony Bankart lesions; glenoid defects greater than 25%
    - Humeral head lesions (e.g., Hill-Sachs) contributing to recurrent instability (greater than 25%; engaging Hill-Sachs lesion)
    - Proximal humerus fracture requiring surgery
    - Irreducible dislocation
- Other relative surgical indications include, but are not limited to:
    - First-time dislocators and age less than 20 years
    - Overhead, throwing athletes
    - Contact sport athletes
    - Primary soft tissue pathology and:
        - Recurrent instability is noted upon return to play
        - The athlete is unable to tolerate bracing or shoulder restrictions
        - Inability to return to baseline performance level following an initial treatment regimen consisting of brief immobilization, full/painless ROM on exam, PT/strengthening protocol, supervised sport-specific training, and attempted bracing with the return to play

- the player is near the end of the season or in the offseason and requests earlier surgical intervention

A 2014 study by Dickens et al. reported results from a multicenter prospective study of in-season Division I collegiate athletes. The authors reported that despite returning to play at a median of 5 days, 27% of athletes could not complete their season and underwent surgical stabilization.[58]

**Nonoperative management[59]:**

*Physical therapy (PT) and bracing*

- Following brief immobilization and cryotherapy use for pain control during the initial 1 to 2 weeks following an acute instability event, formal PT protocols will vary based on provider-specific preference
- In general, following the initial immobilization period, patients will be weaned from sling use, followed by targeted therapy goals focusing on achieving full active/passive ROM, gradual progression to strengthening exercises that focus on dynamic glenohumeral stabilizers and periscapular stabilizers
- When ROM and strength are comparable to the normal side, sport-specific drills can be initiated and return to play with a brace can be considered
- Rehabilitation performed in a supervised setting may improve results, although time to return to sport varies by the program. In most situations, return to play is given consideration after about 2 to 3 weeks

**Surgical management**

The optimal approach and technique to address anterior shoulder instability remain controversial.

*Open versus Arthroscopic repair techniques*

Traditionally, open Bankart repairs offered good to excellent results with minimal risk of redislocation (about 2%) in the setting of minimal osseous and primarily soft tissue only instability cases.[21] Arthroscopic procedures theoretically offer advantages of decreased loss of motion, decreased morbidity, avoidance of subscapularis tenotomy, and the capacity to address concomitant intra-articular pathology, Studies have shown a faster return of preoperative muscle strength.[60][61][62]

A 2017 study reported the surgical trends in management from 2008 through 2012. Arthroscopic stabilization procedures are being performed at a rate of greater than 90% to manage anterior shoulder instability, with the annual rates increasing each year during the study period.[63]

The benefits to arthroscopy include the ability to assess and address concomitant associated shoulder injuries. Also, Burkhart and De Beer previously emphasized the importance of utilizing the arthroscopic procedure as a dynamic diagnostic tool to help identify osseous lesions “at risk” of compromising the functional glenohumeral stability following the single performance of an isolated capsulolabral soft tissue repair.[27]

*Arthroscopic Bankart Repair*

In the setting of primarily soft tissue-based pathology, repair and relocation of the detached capsulolabral complex to its anatomic location are the goals of the surgical technique. Thus, the surgeon aims to restore the static restraining capability of the anterior band of the IGHL. Various suture anchor devices allow for an anatomic repair of the labrum, and techniques will often include capsular plication to address the pathologically lax capsular structures. This concept becomes even more important in the setting of chronic instability cases.

*Bone loss considerations*

Although the exact percentage of bone loss suggested to trigger a change in surgical fixation technique employed remains debated, more aggressive glenoid augmentation procedures are considered as glenoid bone loss approaches and exceeds 25%. Augmentation procedures include autogenous coracoid transfers (Latarjet technique), tricortical

iliac crest, and various allograft tissue augmentation techniques. The distal tibial allograft is a commonly utilized technique in the setting of advanced glenoid bone loss, or the setting of revision surgery.[55]

In the setting of moderate- to large-sized Hill-Sachs lesions, especially engaging lesions, consideration should be given to performing a remplissage procedure. During the arthroscopic procedure, the arm is brought into increasing degrees of shoulder abduction and external rotation to check for a potentially engaging lesion which could lead to postoperative instability event recurrence.

Burkhart and colleagues in 2014 proposed an algorithmic approach. The treatment paradigm broke shoulder instability cases into four subgroups to help guide surgical technique most appropriate to employ[64]:

- Group 1 shoulder instability patients
    - Glenoid: less than 25% defect
    - Hill-Sachs (if present): On track lesion, or non-engaging lesion
    - Technique: Arthroscopic Bankart repair
- Group 2 shoulder instability patients
    - Glenoid: less than 25% defect
    - Hill-Sachs: Off-track lesion, or engaging lesion
    - Technique: Arthroscopic Bankart repair plus remplissage
- Group 3 shoulder instability patients
    - Glenoid: greater than 25% defect
    - Hill-Sachs: On track lesion, or non-engaging lesion
    - Technique: Latarjet procedure
- Group 4 shoulder instability patients
    - Glenoid: greater than 25% defect
    - Hill-Sachs: Off-track lesion, or engaging lesion
    - Technique: Latarjet procedure with or without a humeral-sided procedure
        - The glenoid is addressed first, and following the glenoid augmentation procedure, the further characterization of the Hill-Sachs lesion
            - If the Hill-Sachs lesion is engaging or off track, then humeral-sided procedures are recommended
                - Humeral bone grafting techniques
                - Remplissage procedure

## Differential Diagnosis

The differential diagnosis for shoulder instability is much more focused compared to conditions involving nonspecific, chronic shoulder pain, or even rotator cuff-based pathologies or shoulder impingement conditions.

Pertinent elements to consider on the differential include:

- Dislocation versus subluxation events

- Traumatic versus atraumatic
- Chronicity
- First-time dislocations versus chronic or subacute recurrence
- Unidirectional instability – seen in association with an inciting event/dislocation (anterior, posterior, inferior)
- Multidirectional instability (MDI)
- Associated labral injuries/pathology

## Prognosis

In general, patients can receive education from their providers regarding the risk of recurrence following nonoperative and operative management. In general, prognosis depends on a multitude of factors.

In 2007, the Instability Severity Index Score (ISIS) was originally described to guide orthopedic surgeons regarding the open versus arthroscopic management of shoulder instability surgical procedures [65]. A 2019 study validated the ISIS as a predictive tool to assess the risk of recurrent dislocation following arthroscopic Bankart repairs: [66]

Risk factors associated with recurrence of instability include:

- Younger age (especially under 20 years of age)
- Male gender
- Underlying joint hyperlaxity or generalized hyperlaxity in other joints
- Level and type of sport participation
    - Higher level athletes participating in contact or overhead sports (football, rugby) have a higher risk of recurrence following both nonoperative and operative management
- History of shoulder instability, prior shoulder instability event recurrence episode(s)
- Osseous lesions
    - Glenoid, humeral, and bipolar lesions in consideration

The ISIS is a 10-point scoring system based on these parameters [64]. Patients should be educated to expect, in general, that fewer of the risk factors mentioned above have been shown to result in the following 5-year overall success rates (i.e., no instability recurrence): [66]

- 94% with 1 to 2 risk factors (ISIS score less than or equal to 3)
- 85% with ISIS score of 4 to 6 points
- 55% with ISIS score of greater than 6 points

## Complications

**Redislocation following surgical fixation**

- The main complication of anterior shoulder stabilization is recurrent instability
- Historically, open shoulder stabilization procedures correlated with a lower rate of recurrence compared to arthroscopic procedures [21][67]:
    - Recurrence rates: 2% to 9% reported in the literature

- Arthroscopic Bankart procedures have demonstrated recurrence rates of 4% up to 67% depending on the complexity range of shoulder instability pathology in each case
    - Burkhart and De Beer (2000) reported the results following arthroscopic Bankart repair [27][63]:
        - Without significant bony defects: 4%
        - With significant bony defects: 67%
            - "Significant" defined as either an engaging Hill-Sachs lesion or an "inverted pear" glenoid lesion (i.e., inferior glenoid margins are narrower than the superior and central glenoid margins)
        - A 2017 multicenter database study (2008-2012) reported no difference in rates of revision surgery following open versus arthroscopic Bankart repair procedures
- Utilizing the previously described ISIS scoring scale for pre-operative, clinical exam, and radiographic shoulder instability parameters, a 2019 study reported the 5-year recurrence free instability rates following arthroscopic Bankart repair[66]:
    - 94% with 1 to 2 risk factors (ISIS score less than or equal to 3)
    - 85% with ISIS score of 4 to 6 points
    - 55% with ISIS score of greater than 6 points

**Nerve injuries[68][69]**

- In general, nerve injuries typically manifest as transient, sensory neurapraxia
- Recent studies have demonstrated a lower postoperative rate of nerve injury following arthroscopic (0.3%) versus open (2.2%) Bankart repairs
    - The axillary nerve (most common nerve injury) is adjacent to the inferior capsule and closest to the inferior glenoid rim at the 6 o'clock position
    - The axillary nerve can be injured when placing sutures at the anteroinferior and inferior positions (the 4:30- and 6-o'clock positions, respectively, in the right shoulder) or when repairing capsular lesions, such as a capsular tear or humeral avulsion of the glenohumeral ligament

**Infection**

- No significant difference appreciated between open versus arthroscopic Bankart repairs
    - 22% rate following arthroscopic Bankart repair

**Implant-related problems[70][71][69][71]:**

- Earlier studies (1993 to 2010) reported up to 30% rate of implant-related complications (loosening or breakage) [71]
    - Studies included older implant devices and instrumentation (e.g., metallic staples, bioabsorbable tacks)
- A 2011 study reported implant-related complication rates much lower compared to these earlier studies
    - suture-anchor only devices
    - 0.3% implant-related complication rate

## Postoperative and Rehabilitation Care

Following surgical stabilization procedures, the patient is kept immobilized in a sling for the initial 4- to 6-week postoperative period depending on the surgeon's preference and degree/complexity of the shoulder instability pattern. Following arthroscopic procedures, it is worth mentioning that the subscapularis repair (from an open stabilization procedure) is avoided.

During sling use, patients should be encouraged to perform elbow/wrist/finger ROM to avoid stiffness. An ideal time to incorporate these movements is while the patient is showering and/or before bed.

Following the initial period of shoulder immobilization, passive and active ROM exercises are initialized based on surgeon preference. Typically, the extreme provocative positions are avoided (i.e., shoulder abduction and external rotation) until about 8 weeks following surgery. A goal of full active ROM by the 8-week postop mark is reasonable in most clinical cases.

Progressive resistance and gradual strengthening often begin during the 10- to 12-week postoperative period.

*Return to sport*

Return to sport-specific drills and return to practice and/or competition will vary based on position, sport, level of performance, and the complexity of the injury. In general, return to sport no earlier than 3 months has been advocated in the literature, with a return to high-impact collision sports (e.g., rugby, football) around the 6-month postoperative mark.

*Return to work*

All patients should understand that the return to work timeline will vary depending on the occupational demands of each individual. A heavy physical workload is a known risk factor for prolonged time lost to return to work. A 2017 study reported a mean time to return to work following arthroscopic Bankart repair of 2 months, with return times as early as 1 to 4 weeks following surgery ranging up to 10 months following surgery.[72]

## Consultations

The mantra for managing acute or chronic shoulder instability events should be: when in doubt, consult an orthopedic or primary care sports medicine specialist for further management. While the majority of shoulder instability injuries eventually require surgical fixation, there are certain situations in which nonoperative management can yield acceptable results. That said, the complexity of the overall assessment and management should include consultation with a surgical specialist with experience in managing a wide range of complex instability patterns to provide the patient with the best chance for an ideal outcome.

## Deterrence and Patient Education

Patients and family members (especially when the patient is an adolescent or pediatric patient) should receive education regarding the natural history of shoulder instability events. Patients under the age of 20 years afflicted with at least one shoulder instability event should obtain a referral to a sports medicine specialist for further care and management.

## Enhancing Healthcare Team Outcomes

Shoulder instability events often occur in the young athletic population, and therefore the optimal management of these injuries will often include healthcare personnel spanning from athletic trainer's acute sideline management, primary care (including sports medicine specialists), and orthopedic surgeons. **Level of evidence: II-III**

## Questions

To access free multiple choice questions on this topic, click here.

## References

1. Gottschalk LJ, Walia P, Patel RM, Kuklis M, Jones MH, Fening SD, Miniaci A. Stability of the Glenohumeral Joint With Combined Humeral Head and Glenoid Defects: A Cadaveric Study. Am J Sports Med. 2016 Apr;44(4):933-40. [PubMed: 26851270]
2. Cofield RH, Kavanagh BF, Frassica FJ. Anterior shoulder instability. Instr Course Lect. 1985;34:210-27. [PubMed: 3833942]
3. Walton J, Paxinos A, Tzannes A, Callanan M, Hayes K, Murrell GA. The unstable shoulder in the adolescent athlete. Am J Sports Med. 2002 Sep-Oct;30(5):758-67. [PubMed: 12239016]
4. Joseph TA, Williams JS, Brems JJ. Laser capsulorrhaphy for multidirectional instability of the shoulder. An outcomes study and proposed classification system. Am J Sports Med. 2003 Jan-Feb;31(1):26-35. [PubMed: 12531753]
5. Simonet WT, Melton LJ, Cofield RH, Ilstrup DM. Incidence of anterior shoulder dislocation in Olmsted County, Minnesota. Clin. Orthop. Relat. Res. 1984 Jun;(186):186-91. [PubMed: 6723141]
6. Krøner K, Lind T, Jensen J. The epidemiology of shoulder dislocations. Arch Orthop Trauma Surg. 1989;108(5):288-90. [PubMed: 2789505]
7. Nordqvist A, Petersson CJ. Incidence and causes of shoulder girdle injuries in an urban population. J Shoulder Elbow Surg. 1995 Mar-Apr;4(2):107-12. [PubMed: 7600160]
8. Shah A, Judge A, Delmestri A, Edwards K, Arden NK, Prieto-Alhambra D, Holt TA, Pinedo-Villanueva RA, Hopewell S, Lamb SE, Rangan A, Carr AJ, Collins GS, Rees JL. Incidence of shoulder dislocations in the UK, 1995-2015: a population-based cohort study. BMJ Open. 2017 Nov 14;7(11):e016112. [PMC free article: PMC5695490] [PubMed: 29138197]
9. Leroux T, Wasserstein D, Veillette C, Khoshbin A, Henry P, Chahal J, Austin P, Mahomed N, Ogilvie-Harris D. Epidemiology of primary anterior shoulder dislocation requiring closed reduction in Ontario, Canada. Am J Sports Med. 2014 Feb;42(2):442-50. [PubMed: 24275862]
10. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency departments in the United States. J Bone Joint Surg Am. 2010 Mar;92(3):542-9. [PubMed: 20194311]
11. Owens BD, Duffey ML, Nelson BJ, DeBerardino TM, Taylor DC, Mountcastle SB. The incidence and characteristics of shoulder instability at the United States Military Academy. Am J Sports Med. 2007 Jul;35(7):1168-73. [PubMed: 17581976]
12. Kawasaki T, Ota C, Urayama S, Maki N, Nagayama M, Kaketa T, Takazawa Y, Kaneko K. Incidence of and risk factors for traumatic anterior shoulder dislocation: an epidemiologic study in high-school rugby players. J Shoulder Elbow Surg. 2014 Nov;23(11):1624-30. [PubMed: 25017313]
13. Gibbs DB, Lynch TS, Nuber ED, Nuber GW. Common Shoulder Injuries in American Football Athletes. Curr Sports Med Rep. 2015 Sep-Oct;14(5):413-9. [PubMed: 26359844]
14. Kraeutler MJ, Currie DW, Kerr ZY, Roos KG, McCarty EC, Comstock RD. Epidemiology of Shoulder Dislocations in High School and Collegiate Athletics in the United States: 2004/2005 Through 2013/2014. Sports Health. 2018 Jan/Feb;10(1):85-91. [PMC free article: PMC5753963] [PubMed: 28556681]
15. Kraeutler MJ, McCarty EC, Belk JW, Wolf BR, Hettrich CM, Ortiz SF, MOON Shoulder Instability Group. Bravman JT, Baumgarten KM, Bishop JY, Bollier MJ, Brophy RH, Carey JL, Carpenter JE, Cox CL, Feeley BT, Grant JA, Jones GL, Kuhn JE, Kelly JD, Ma CB, Marx RG, Miller BS, Sennett BJ, Smith MV, Wright RW, Zhang AL. Descriptive Epidemiology of the MOON Shoulder Instability Cohort. Am J Sports Med. 2018 Apr;46(5):1064-1069. [PubMed: 29505730]
16. Leroux T, Ogilvie-Harris D, Veillette C, Chahal J, Dwyer T, Khoshbin A, Henry P, Mahomed N, Wasserstein D. The epidemiology of primary anterior shoulder dislocations in patients aged 10 to 16 years. Am J Sports Med. 2015 Sep;43(9):2111-7. [PubMed: 26254440]
17. Aygün Ü, Çalik Y, Işik C, Şahin H, Şahin R, Aygün DÖ. The importance of glenoid version in patients with anterior dislocation of the shoulder. J Shoulder Elbow Surg. 2016 Dec;25(12):1930-1936. [PubMed: 27855874]
18. Norlin R. Intraarticular pathology in acute, first-time anterior shoulder dislocation: an arthroscopic study. Arthroscopy. 1993;9(5):546-9. [PubMed: 7818617]
19. Di Giacomo G, de Gasperis N, Scarso P. Bipolar bone defect in the shoulder anterior dislocation. Knee Surg Sports Traumatol Arthrosc. 2016 Feb;24(2):479-88. [PubMed: 26704802]

20. Yates JB, Choudhry MN, Waseem M. Managing Bony Defects of the Shoulder Joint that Occur in Association with Dislocation. Open Orthop J. 2017;11:1245-1257. [PMC free article: PMC5721330] [PubMed: 29290863]
21. Rowe CR, Patel D, Southmayd WW. The Bankart procedure: a long-term end-result study. J Bone Joint Surg Am. 1978 Jan;60(1):1-16. [PubMed: 624747]
22. Calandra JJ, Baker CL, Uribe J. The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. Arthroscopy. 1989;5(4):254-7. [PubMed: 2590322]
23. Saupe N, White LM, Bleakney R, Schweitzer ME, Recht MP, Jost B, Zanetti M. Acute traumatic posterior shoulder dislocation: MR findings. Radiology. 2008 Jul;248(1):185-93. [PubMed: 18458243]
24. 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 May-Jun;25(3):306-11. [PubMed: 9167808]
25. Armitage MS, Faber KJ, Drosdowech DS, Litchfield RB, Athwal GS. Humeral head bone defects: remplissage, allograft, and arthroplasty. Orthop. Clin. North Am. 2010 Jul;41(3):417-25. [PubMed: 20497816]
26. PALMER I, WIDEN A. The bone block method for recurrent dislocation of the shoulder joint. J Bone Joint Surg Br. 1948 Feb;30B(1):53-8. [PubMed: 18864946]
27. 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 Oct;16(7):677-94. [PubMed: 11027751]
28. Sugaya H, Moriishi J, Dohi M, Kon Y, Tsuchiya A. Glenoid rim morphology in recurrent anterior glenohumeral instability. J Bone Joint Surg Am. 2003 May;85-A(5):878-84. [PubMed: 12728039]
29. Lo IK, Parten PM, Burkhart SS. The inverted pear glenoid: an indicator of significant glenoid bone loss. Arthroscopy. 2004 Feb;20(2):169-74. [PubMed: 14760350]
30. Bigliani LU, Newton PM, Steinmann SP, Connor PM, Mcllveen SJ. Glenoid rim lesions associated with recurrent anterior dislocation of the shoulder. Am J Sports Med. 1998 Jan-Feb;26(1):41-5. [PubMed: 9474399]
31. DeBerardino TM, Arciero RA, Taylor DC, Uhorchak JM. Prospective evaluation of arthroscopic stabilization of acute, initial anterior shoulder dislocations in young athletes. Two- to five-year follow-up. Am J Sports Med. 2001 Sep-Oct;29(5):586-92. [PubMed: 11573917]
32. Saito H, Itoi E, Sugaya H, Minagawa H, Yamamoto N, Tuoheti Y. Location of the glenoid defect in shoulders with recurrent anterior dislocation. Am J Sports Med. 2005 Jun;33(6):889-93. [PubMed: 15827355]
33. Neer CS, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder: a preliminary report. 1980. J Bone Joint Surg Am. 2001 Oct;83-A(10):1586. [PubMed: 11679614]
34. Bak K, Spring BJ, Henderson JP. Inferior capsular shift procedure in athletes with multidirectional instability based on isolated capsular and ligamentous redundancy. Am J Sports Med. 2000 Jul-Aug;28(4):466-71. [PubMed: 10921636]
35. Altchek DW, Warren RF, Skyhar MJ, Ortiz G. T-plasty modification of the Bankart procedure for multidirectional instability of the anterior and inferior types. J Bone Joint Surg Am. 1991 Jan;73(1):105-12. [PubMed: 1985978]
36. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. A study using Moiré topographic analysis. Clin. Orthop. Relat. Res. 1992 Dec;(285):191-9. [PubMed: 1446436]
37. Neer CS, Watson KC, Stanton FJ. Recent experience in total shoulder replacement. J Bone Joint Surg Am. 1982 Mar;64(3):319-37. [PubMed: 7061549]
38. Samilson RL, Prieto V. Dislocation arthropathy of the shoulder. J Bone Joint Surg Am. 1983 Apr;65(4):456-60. [PubMed: 6833319]
39. Hovelius L, Rahme H. Primary anterior dislocation of the shoulder: long-term prognosis at the age of 40 years or younger. Knee Surg Sports Traumatol Arthrosc. 2016 Feb;24(2):330-42. [PubMed: 26754859]
40. Vangsness CT, Ennis M, Taylor JG, Atkinson R. Neural anatomy of the glenohumeral ligaments, labrum, and subacromial bursa. Arthroscopy. 1995 Apr;11(2):180-4. [PubMed: 7794430]
41. Jerosch J, Castro WH, Halm H, Drescher H. Does the glenohumeral joint capsule have proprioceptive capability? Knee Surg Sports Traumatol Arthrosc. 1993;1(2):80-4. [PubMed: 8536013]
42. Pauly S, Morawietz L, Krüger D, Strube P, Scheibel M. Histopathologic evaluation of passive stabilizers in

shoulder instability. J Shoulder Elbow Surg. 2013 May;22(5):687-94. [PubMed: 22981356]
43. Galvin JW, Ernat JJ, Waterman BR, Stadecker MJ, Parada SA. The Epidemiology and Natural History of Anterior Shoulder Instability. Curr Rev Musculoskelet Med. 2017 Dec;10(4):411-424. [PMC free article: PMC5685954] [PubMed: 29038951]
44. Sahajpal DT, Zuckerman JD. Chronic glenohumeral dislocation. J Am Acad Orthop Surg. 2008 Jul;16(7):385-98. [PubMed: 18611996]
45. Jeanmonod R, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 26, 2018. Geriatric Cervical Spine Injury. [PubMed: 29262191]
46. Varacallo M, Mair SD. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Oct 27, 2018. Rotator Cuff Syndrome. [PubMed: 30285401]
47. Varacallo M, Mair SD. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Oct 27, 2018. Rotator Cuff Tendonitis. [PubMed: 30335303]
48. Varacallo M, Mair SD. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jan 19, 2019. Proximal Biceps Tendinitis and Tendinopathy. [PubMed: 30422594]
49. Varacallo M, Mair SD. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jan 9, 2019. Biceps Tendon Dislocation and Instability. [PubMed: 30475566]
50. Nickerson M, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jan 19, 2019. Swimmer's Shoulder. [PubMed: 29262079]
51. Tzannes A, Murrell GA. Clinical examination of the unstable shoulder. Sports Med. 2002;32(7):447-57. [PubMed: 12015806]
52. Lee SB, Kim KJ, O'Driscoll SW, Morrey BF, An KN. Dynamic glenohumeral stability provided by the rotator cuff muscles in the mid-range and end-range of motion. A study in cadavera. J Bone Joint Surg Am. 2000 Jun;82(6):849-57. [PubMed: 10859105]
53. Deutsch A, Ramsey ML, Williams GR. Loss of passive external rotation at 90 degrees abduction is predictive of a medially healed Bankart lesion. Arthroscopy. 2006 Jul;22(7):710-5. [PubMed: 16843805]
54. Parameswaran AD, Provencher MT, Bach BR, Verma N, Romeo AA. Humeral avulsion of the glenohumeral ligament: injury pattern and arthroscopic repair techniques. Orthopedics. 2008 Aug;31(8):773-9. [PubMed: 18714772]
55. Tennent DJ, Donohue MA, Posner MA. Bone Loss and Glenohumeral Instability. Sports Med Arthrosc Rev. 2017 Sep;25(3):131-135. [PubMed: 28777215]
56. Barchilon VS, Kotz E, Barchilon Ben-Av M, Glazer E, Nyska M. A simple method for quantitative evaluation of the missing area of the anterior glenoid in anterior instability of the glenohumeral joint. Skeletal Radiol. 2008 Aug;37(8):731-6. [PubMed: 18523766]
57. Owens BD, Dickens JF, Kilcoyne KG, Rue JP. Management of mid-season traumatic anterior shoulder instability in athletes. J Am Acad Orthop Surg. 2012 Aug;20(8):518-26. [PubMed: 22855854]
58. Dickens JF, Owens BD, Cameron KL, Kilcoyne K, Allred CD, Svoboda SJ, Sullivan R, Tokish JM, Peck KY, Rue JP. Return to play and recurrent instability after in-season anterior shoulder instability: a prospective multicenter study. Am J Sports Med. 2014 Dec;42(12):2842-50. [PubMed: 25378207]
59. Buss DD, Lynch GP, Meyer CP, Huber SM, Freehill MQ. Nonoperative management for in-season athletes with anterior shoulder instability. Am J Sports Med. 2004 Sep;32(6):1430-3. [PubMed: 15310567]
60. Rhee YG, Lim CT, Cho NS. Muscle strength after anterior shoulder stabilization: arthroscopic versus open Bankart repair. Am J Sports Med. 2007 Nov;35(11):1859-64. [PubMed: 17664342]
61. Cole BJ, Warner JJ. Arthroscopic versus open Bankart repair for traumatic anterior shoulder instability. Clin Sports Med. 2000 Jan;19(1):19-48. [PubMed: 10652663]
62. Fabbriciani C, Milano G, Demontis A, Fadda S, Ziranu F, Mulas PD. Arthroscopic versus open treatment of Bankart lesion of the shoulder: a prospective randomized study. Arthroscopy. 2004 May;20(5):456-62. [PubMed: 15122134]
63. Bonazza NA, Liu G, Leslie DL, Dhawan A. Trends in Surgical Management of Shoulder Instability. Orthop J Sports Med. 2017 Jun;5(6):2325967117712476. [PMC free article: PMC5495509] [PubMed: 28695137]
64. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from

"engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy. 2014 Jan;30(1):90-8. [PubMed: 24384275]

65. 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 Nov;89(11):1470-7. [PubMed: 17998184]
66. Loppini M, Delle Rose G, Borroni M, Morenghi E, Pitino D, Domínguez Zamora C, Castagna A. Is the Instability Severity Index Score a Valid Tool for Predicting Failure After Primary Arthroscopic Stabilization for Anterior Glenohumeral Instability? Arthroscopy. 2019 Feb;35(2):361-366. [PubMed: 30611589]
67. Gill TJ, Micheli LJ, Gebhard F, Binder C. Bankart repair for anterior instability of the shoulder. Long-term outcome. J Bone Joint Surg Am. 1997 Jun;79(6):850-7. [PubMed: 9199382]
68. Saier T, Plath JE, Waibel S, Minzlaff P, Feucht MJ, Herschbach P, Imhoff AB, Braun S. How Satisfied Are Patients with Arthroscopic Bankart Repair? A 2-Year Follow-up on Quality-of-Life Outcome. Arthroscopy. 2017 Oct;33(10):1777-1785. [PubMed: 28669598]
69. Matsuki K, Sugaya H. Complications after arthroscopic labral repair for shoulder instability. Curr Rev Musculoskelet Med. 2015 Mar;8(1):53-58. [PMC free article: PMC4596184] [PubMed: 25532917]
70. Owens BD, Harrast JJ, Hurwitz SR, Thompson TL, Wolf JM. Surgical trends in Bankart repair: an analysis of data from the American Board of Orthopaedic Surgery certification examination. Am J Sports Med. 2011 Sep;39(9):1865-9. [PubMed: 21628637]
71. Jeong JH, Shin SJ. Arthroscopic removal of proud metallic suture anchors after Bankart repair. Arch Orthop Trauma Surg. 2009 Aug;129(8):1109-15. [PubMed: 19271227]
72. Privitera DM, Bisson LJ, Marzo JM. Minimum 10-year follow-up of arthroscopic intra-articular Bankart repair using bioabsorbable tacks. Am J Sports Med. 2012 Jan;40(1):100-7. [PubMed: 22064607]



Bookshelf ID: NBK538234 PMID: 30855822