Shoulder: Instability and Labrum

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The audience for this lecture is clinical and research radiologists, PhDs who are trying to get a better understanding of the anatomic and clinical aspects of this subject, and MRI technologists who perform shoulder MRI.

The fibrous connective tissue glenoid labrum completely rims and deepens the shallow glenoid fossa, increasing the contact area for the humeral head and limiting excessive translation of the joint. The intact labrum also acts as a pressure seal, allowing negative pressure to occur within the shoulder joint during motion, aiding in the dynamic stabilization the joint. In addition, the labrum serves as an anchor for some of the glenohumeral ligaments as well as the long head of the biceps and triceps tendons.

It is helpful to divide the labrum into various regions. This can be accomplished by dividing the labrum anatomically into six segments that include the superior, posterior superior, posterior inferior, inferior, anterior inferior and anterior superior regions. Alternatively, one can use the face of a clock to describe the location of abnormalities. With this method, 12 o'clock is at the top under the coracoid and 6 o'clock is 180 degrees from the top at the inferior labrum. The latter method works when 3 o'clock is considered the mid portion of the anterior labrum and 9 o'clock is at the mid portion of the posterior labrum.

Most labra are 4mm wide and 3mm thick but they can range from 2-14mm. Labra are commonly triangular or round in cross section. Other normal variations in shape include flat, cleaved, notched ror absent labrum. The labrum lies on a fibrocartilage transition to hyaline articular cartilage. Two types of labrochondral junctions are seen. The Type A attachment is associated with an abrupt transition between the labrum and the hyaline cartilage with a free edge margin. The Type B attachment shows a gradual transition between the labrum on all imaging sequences and should not be mistaken for a tear at the base of the labrum. The labrum is normally low signal intensity on all imaging sequences, however it can demonstrate intermediate signal intensity in areas that are 55 degrees to the main magnetic field on shorter TE images, termed the "magic angle phenomenon". This is most common in the posterior superior aspect of the labrum¹.

The labrum is best seen when there is fluid in the joint in the form of an effusion or arthrogram. First time dislocators often have some effusion in the initial few weeks after trauma and an arthrogram is not needed. Otherwise, if the dislocation is more than a few weeks, it is recommended that the patient have a direct MR arthrogram. If that is not available, a high resolution MRI or an indirect (gadolinium intravenously administered) MR arthrogram would be suggested ²⁻⁶. Abduction and external rotation (ABER) imaging can aid in evaluation of the anteroinferior labrum. The ABER sequence obtained during an MR arthrogram improves the

accuracy of interpretation of the labral lesion and can bring out associated articular surface supraspinatus and infraspinatus tears ^{7,8 9}.

If the patient cannot place the shoulder into the ABER position because of instability, the adduction and internal rotation (ADIR) position can be added ¹⁰. This position pushes fluid into the anterior joint space, outlining the anterior interior labrum.

A provocative maneuver can also be performed on the shoulder during MR imaging to better evaluate the posterior labrocapsular structures. This is accomplished with flexion, adduction and internal rotation of the shoulder (FADIR position)¹¹.

Glenohumeral ligaments

The coracohumeral, superior, middle, and inferior glenohumeral ligaments as well as the capsule contribute to shoulder stability. Two ligaments are located at the top of the joint in the rotator interval. These are the *coracohumeral ligament* (CHL) and *superior glenohumeral ligament* (SGHL). The CHL and SGHL ligaments limit inferior translation and external rotation of the adducted shoulder as well as posterior translation of the flexed, adducted and internally rotated shoulder. The CHL originates on the lateral surface of the base of the coracoid process and inserts on the lesser and greater tuberosities, crossing the bicipital groove. The CHL is an extra-articular bursal sided structure that is not seen during arthroscopy. The SGHL originates from the labrum, biceps tendon or in common with the middle glenohumeral ligament in the region of the superior glenoid tubercle. It inserts into the fovea capitis line just superior to the lesser tuberosity of the humerus. It lies parallel to the lateral aspect of the coracoid process and is present in more than 90% of cases ¹². It restrains external rotation below 60 degrees of abduction, and limits inferior subluxation. Variation of the SGHL includes a common origin with the middle glenohumeral ligament and/or biceps tendon. The SGHL can become thickened in patients with an absent middle glenohumeral ligament.

The *middle glenohumeral ligament* (MGHL) has a variable origin from the glenoid, scapula, anteriorsuperior labrum, biceps tendon, inferior glenohumeral ligament (IGHL) or superior glenohumeral ligament ¹³. It merges with the anterior capsule along the subscapularis muscle and tendon, continuing with the subscapularis tendon to the anterior aspect of the proximal humerus just below the attachment of the SGHL on the lesser tuberosity. The MGHL is absent in up to 30% of shoulders. The subscapular recess is more prominent in this situation. The MGHL can show longitudinal splitting or duplication as normal variants.

The *inferior glenohumeral ligament* (IGHL), considered the most important stabilizer of the glenohumeral joint, is a complex that originates at the mid to inferior portion of the anterior glenoid labrum. It drapes for a variable distance from anterior to posterior and inserts on the anatomic neck of the humerus. This ligament is inseparable from the labrum, forming a labroligamentous complex. It is composed of strong collagenous thickenings at its anterior and posterior margins- the anterior and posterior bands, joined by a fibrous thickening of the capsule called the axillary pouch or recess. Variants of the IGHL include high origin above the equator of the glenoid, origination from the MGHL, or a bandlike attachment between the IGHL and SGHL called the periarticular fiber system ¹⁴. The IGHL functions as a sling to support the humeral head and prevents abnormal translation of the humeral head on the glenoid. The IGHL is a major stabilizer of the joint in 90 degrees of abduction and full external rotation, limiting anterior rotation, and is lax when the shoulder is adducted.

Normal variations in the anterior capsulolabral structures

Normal variations of the capsulolabral complex are often seen and can be mistaken for pathologic lesions ¹⁵. These normal variants are seen in the superior and anterior superior aspect of the glenoid.

The normal labral variants in the anterior superior region of the glenoid can be confused with labral tears by those who are not aware of their existence. In distinguishing these normal variants from labral tears, it is helpful to know that isolated tears are uncommon in the anterior superior segment of the labrum. Usually tears in the anterior superior region of the glenoid are associated with SLAP lesions or tears that extend down to the anterior inferior labrum.

A labral variant seen in 11-15% of shoulders in the anterior superior region of the glenoid labrum is the *sublabral foramen or hole*¹⁵. The foramen occurs when the anterior superior portion of the labrum is not attached to the glenoid rim, often in the presence of a small osseous indentation long the anterior border of the glenoid fossa with a pear-shaped glenoid. The foramen can vary from a focal detachment to complete involvement of the anterior superior segment, occasionally dipping below the equator of the glenoid. The foramen has smooth margins without significant displacement (<1-2mm). The adjacent glenohumeral ligaments are also intact. Of interest, the sublabral foramen can provide a pathway for intraosseous bodies to extrude into the subscapular recess¹⁶. The foramen can simulate a paralabral cyst from a labral tear. A high origin of the IGHL may simiulate a sublabral foramen¹⁷. This foramen or hole can be associated with a "cord-like" MGHL in 75% of cases¹⁵. One should not mistake the "cord-like" middle glenohumeral ligament for a labral detachment.

Absence of the anterior superior labrum associated with a rounder and larger "cord-like" MGHL may be congenital, and this has been termed the *Buford complex*. This variant of normal seen in up to 6% of shoulder arthroscopies ^{15,18,19}.

Anterior Instability

Labral lesions associated with anterior instability

Tears of the labrum are common in athletes with instability, especially those in sports that require forceful and repetitive abduction and overhead rotation of the humerus. Tears can also be seen following routine or repetitive trauma, microtrauma, as well as and with ageing and degeneration.

When describing a labral tear, it is best to mention the location(s) of the tear on the glenoid rim, as described in the normal labral anatomy section of this chapter. Morphologic criteria such as absence, fraying, detachment, displacement, fragmentation, or deformity are used to describe labral pathology. Fluid or contrast within the labral substance or at the interface between the labrum and the bone/cartilage substance (if not one of the described normal variants) is also a good indication of a labral tear.

The torn labrum may be unstable with a flap component with an attached piece separated from the main structure or the labrum can have a vertical split, which if complete is called a bucket handle tear. Both of these types of labral tears cause mechanical symptoms such as locking, catching, clicking and popping and are treated with surgical resection or repair.

The anterior inferior labrum is the most frequently affected site of labral pathology related to anterior instability. Injury to the anterior inferior labrum may demonstrate findings mentioned above for routine tears of the labrum but often reach predictable patterns in the form

of a Bankart lesion or it's variants- the ALPSA or Perthes lesion. Some surgeons believe that differentiating these lesions is important for proper management, while others do not. To serve all of the referring physicians, we make a point of differentiating them, or at least describing the distinctions between the Bankart and it's variants. What is important is that these labral tears are associated with inferior glenohumeral ligament complex pathology and that the entire labroligamentous complex is involved in the instability and needs to be treated with attention to the labrum and the anterior band of the inferior glenohumeral ligament.

The most common lesion resulting from an anterior dislocation is the Bankart lesion. This represents a detachment of the anteroinferior labrum and glenohumeral ligament from the glenoid rim. The avulsed labrum is no longer attached to the scapular periosteum. The detached labrum may float upward in the joint, producing a glenoid labrum ovoid mass (GLOM sign). Neviaser described a variant of the Bankart lesion known as the anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesion²⁰. This represents an avulsion of the inferior glenohumeral ligament complex from the anterior inferior glenoid with an intact periosteum. The avulsed anterior inferior labrum displaces medially and rotates inferiorly along the denuded anterior scapular neck. These lesions eventually heal in this medially displaced position, leading to recurrent anterior instability because of persistent incompetence of the inferior glenohumeral ligament labral complex. With time, the detached labrum and glenohumeral ligament become synovialized and enlarge, creating a chronic mass lesion in that location. This lesion has the best surgical outcome when it is dissected from the scapular neck and repaired laterally to the level of the glenoid rim. ALPSA lesions can also involve the inferior labrum near the 6 o'clock location. The third variant in this location is termed the Perthes lesion, named after a German physician who described it in the early 1900's ²¹. This lesion is an avulsion of the anterior inferior labrum and IGHL with an intact scapular periosteum that connects to the IGHL but is stripped from the glenoid. The Perthes lesion is not as easily identified on MR arthrography as the other Bankart type lesions ²², but it is well visualized with the addition of ABER positioning of the shoulder.

Occasionally related to instability, but more often caused by forced adduction injury to the shoulder with the arm in abduction and external rotation, a *glenolabral articular disruption (GLAD)* lesion is a nondisplaced superficial tear of the anterior inferior labrum that is accompanied by fibrillation and erosion of the adjacent articular cartilage $^{23 \ 24}$.

Glenohumeral ligament and capsular lesions associated with anterior instability

Tears of the SGHL have been associated with chronic multidirectional instability and shoulder dislocations. Tears of the SGHL may also be seen in association with tears of the MGHL ¹³.MGHL tears were seen in 58% of shoulders with glenohumeral instability treated arthroscopically in one study ²⁵. Tears of the MGHL present in different patterns and they are best seen on sagittal and axial MR images ¹³. The ligament may be floating in the anterior capsular space, detached from the scapular insertion, foreshortened, thickened and wavy. Another pattern of rupture is the split longitudinal tear, extending down the length of the MGHL without complete disruption of the ligament. This can simulate a normal variant-the bifid MGHL. MGHL tears are frequently associated with superior labral tears and have been categorized as a type VII SLAP lesion ²⁶. The MGHL tear may also be associated with tears of the rotator interval and SLAP V lesions that extend to the anterior inferior labrum ²⁷.

There are several IGHL lesions associated with anterior glenohumeral instability. Approximately 40% of IGHL lesions are seen at the glenoid attachment. At the opposite end, humeral avulsion of the IGHL, termed the *HAGL lesion* may occur. Although the HAGL lesion typically results from a first-time dislocation in persons older than 35 years of age, this is not true of all patients and some do not have a history of prior dislocation. The HAGL lesion is occasionally associated with a tear of the subscapularis tendon and can cause recurrent anterior instability ²⁸. The HAGL lesion is seen on axial MR images as a thick, wavy and irregular ligament that does not attach to the humeral head. On coronal MR images, the normal "U" shape of the anterior band of the IGHL looks like a "J" when the ligament is disrupted ²⁹. It is important to identify the HAGL lesion. An unrecognized HAGL lesion is a cause of failed Bankart repair ^{30,31}. In up to 20% of cases of HAGL, a patient can avulse a bony fragment along with the IGHL from the humeral attachment. This bony HAGL injury is termed a "*BHAGL*" lesion ³².

When the IGHL is avulsed at the humeral and labral attachments, a condition seen in younger patients, it is called a *"floating anterior glenohumeral ligament" (floating AIGHL)* ^{33 34}. The floating AIGHL lesion is often accompanied by a Hill Sachs lesion.

In 35% of cases the inferior glenohumeral ligament can tear in midportion and not at either attachment. This is more common in the posterior aspect of the axillary pouch and is often present after an initial dislocation without an associated labral tear. MR arthrography may aid in detection of such a capsular disruption, although it can sometimes be confused with incidental contrast extravasation.

It has been noted that one can have a "peel back" of the anterior band of the IGHL at the glenoid attachment without tearing the anterior inferior labrum. This is related to the difference in composition of the tissues at the attachment of the IGHL to the labrum and bone, with a weaker labral attachment. This is known as the anterior ligamentous periosteal sleeve avulsion (*ALIPSA*), and it can represent a glenoid-sided failure of the IGHL due to instability, although in some cases, it may be a developmental anomaly.

An avulsion of the IGHL at the glenoid attachment is called a glenoid avulsion of the glenohumeral ligament (*GAGL*) lesion and it has a "J" configuration on coronal images in the opposite direction of a HAGL lesion. GAGL lesions are uncommon. The IGHL may also be lax, which should be treated as an intrasubstance ligament failure.

Osseous lesions associated with anterior instability

Osseous lesions that are seen following anterior instability are not as common as soft tissue lesions but are still quite frequent. These lesions, including the more common Hill Sachs lesion of the humeral head and the Bankart lesion of the anterior inferior glenoid rim, as well as greater tuberosity and superior humeral head fractures are important to recognize and are not always seen on radiographs³⁵.

The *Hill Sachs* lesion manifests as a depression along the posterolateral aspect of the humeral head superiorly in the top 2 cm usually above the level of the coracoid. It is seen in approximately 75% of patients with anterior instability. Although most commonly involving the bone, the lesion may just appear in the humeral head cartilage or may produce bone marrow reactive change without cortical deformation. It is important to quantify the amount of bone loss in the area of the Hill Sachs defect. Large defects that are parallel to the anterior glenoid rim can engage when the shoulder goes from external to internal rotation, resulting in recurrent instability and locking which must be repaired with bone graft.

The *osseous Bankart lesion* may be small or large enough to extend above the glenoid equator. Most osseous Bankart lesions are seen on MRI, but small fractures may be missed. The osseous Bankart fracture is well seen on CT as a fragment of bone associated with irregularity of

the adjacent glenoid rim. The presence of an osseous Bankart fracture is an indication for open repair.

The majority of patients with the diagnosis of anterior instability show some bone loss anteriorly. This is probably multifactorial in etiology. The shape of the glenoid fossa is often like a pear with a narrower superior portion compared to the inferior portion. The normally pear-shaped glenoid rim may become deficient inferiorly following anterior dislocation, producing an *inverted pear* configuration ^{36 37}. In patients with a deficient glenoid, a coracoid bone graft is placed in the defect using a non-arthroscopic open technique to restore the normal articular arc of the glenoid, termed the modified Latarjet procedure ^{38,39}.

Posterior Instability

Posterior labral and capsular tears are much less common than anterior ones, accounting for up to 5% of all cases of shoulder instability ⁴⁰. Posterior instability is more often encountered in association with atraumatic recurrent posterior multidirectional instability, or repetitive microtrauma, but can also be seen with traumatic posterior dislocation, a redundant posterior capsule, osteochondral lesions, posterior labroligamentous tears, as well as in the setting of electric shock or seizures, which produce adduction, flexion, and internal rotation of the glenohumeral joint. The structures that are important for posterior glenoid rim, labrum, and capsule, as well as the rotator cuff and rotator interval. The subscapularis muscle counterbalances posterior subluxation and the rotator interval capsule aiding in resistance to posterior humeral translation, and therefore abnormalities of these regions will also be associated with posterior instability ⁴¹ 42.

Labral lesions associated with posterior instability

Posterior instability is associated with lesions of the posterior labrum. A particular type of posterior labral tear is the posterior labrocapsular periosteal sleeve avulsion injury termed the *POLPSA* lesion ^{43 44}. This lesion is an avulsion of the attachment of the posterior capsule and the periosteum, resulting in a patulous recess posteriorly. It is similar to a posterior Perthes or ALPSA lesion, depending upon whether it is medially displaced, which if present, would be the ALPSA lesion. POLPSA lesions are seen in association with posterior dislocation or subluxation of the glenohumeral joint and may predispose to recurrent instability. The surgical technique for repairing the POLPSA lesion differs from the reverse Bankart, since the periosteal sleeve must be reduced in order to reattach the labrum.

The *reverse Bankart* lesion and variants are similar to the anterior inferior labral lesions described above. These posterior inferior labral tears are detached from the scapular periosteum without adjacent cartilage damage ^{45 46,47}. In these cases, fluid or contrast may extend behind the glenoid posterior to the scapular neck.

Another lesion of the posterior inferior labrum is the deep/intrasubstance incomplete detachment that is associated with a defect at the chondrolabral junction. This is termed the *Kim's lesion*⁴⁸. These lesions are produced by a submaximal posterior force of the labrum at the attachment of the posterior band of the IGHL. This tears at the posterior inferior portion of the labrum, causing detachment of the inner portion without involving the chondrolabral junction. It is also common to see extensive posterior labral tears associated with tears of other portions of the labrum. This includes the types VIII and IX SLAP tears that involve the superior labrum and almost the entire labrum, respectively.

Glenohumeral ligament and capsular lesions associated with posterior instability

Posterior capsular laxity is the most common abnormality seen with posterior glenohumeral instability ⁴⁹. In addition one may see a *reverse glenoid avulsion of the glenohumeral ligament (GAGL)* lesion, which is a stripping of the capsule and synovium at the posterior inferior glenoid rim ⁴⁷.

The *Bennett lesion* is an extracapsular avulsive injury commonly seen along the posterior glenoid rim in 25% of throwing athletes such as baseball pitchers ⁵⁰. This lesion is characterized by heterotopic ossification near the insertion of the posterior band of the IGHL on the glenoid produced by traction of the inferior glenohumeral ligament during the cocking or follow-through phases of pitching ^{51 52}. The mineralization may occasionally be identified on MRI but is better seen on axillary radiographs and CT. At the other end of the posterior capsule, where it attaches to the humeral neck, the *reverse HAGL (RHAGL) lesion*, also called the posterior band inferior glenohumeral ligament avulsion from the humerus (*PHAGL*) lesion can be seen ^{53,54}. This injury has multiple etiologies. One mechanism of injury is a posteriorly directed force on an abducted shoulder and is often associated with posterior labral tears and cystic changes in the greater tuberosity. The RHAGL may also be related to multidirectional and microinstability and is characterized by a lack of attachment of the posterior soft tissues. The RHAGL lesion is important to identify on MRI pre-operatively since it can be missed at arthroscopy if no anterior portals are used to look posteriorly ⁵⁵⁻⁵⁷.

Osseous lesions associated with posterior instability

Some of the osseous lesions associated with posterior instability carry the familiar eponyms associated with anterior instability except that the word "reverse" is added to them. A *reverse osseous Bankart* lesion refers to an impaction fracture of the posterior glenoid resulting from a posterior dislocation of the humerus. A *reverse Hill Sachs* lesion results from an impaction fracture of the anterior medial humeral head which involves 10-30% of the articular surface of the anterior humeral head.. Also referred to as a trough or McLaughlin's fracture, it is seen in up to 86% of cases of acute traumatic posterior dislocation of the shoulder ⁵⁸.

The posterior glenoid usually has a pointed posterior margin. Osseous anomalies that affect the posterior glenoid can produce multidirectional and posterior instability. These range from the milder posterior glenoid rim deficiency to the denticulate glenoid. Posterior labral tears are common with these alterations in the normal glenoid configuration ^{59 60}.

SUPERIOR LABRUM

The long head of the biceps tendon attaches to the superior labrum forming the biceps labral complex. Three types of complexes have been described. Type I shows the labrum firmly attached to the glenoid rim with no cartilage interface or central free edge. Type II shows a more medial attachment of the glenoid labrum and biceps tendon to the glenoid with hyaline cartilage under the labrum and a sublabral recess. A Type II biceps labral complex demonstrates a triangular meniscoid labrum that extends into the joint space and results in a deep cleft (sublabral recess) that may be continuous with a sublabral foramen in the anterior superior segment. A normal variant, the sublabral recess may be difficult to distinguish from a labral tear. The

sublabral recess has been seen in 19 out or 26 (73%) cadaver shoulders in one study 61 . One can see accumulation of contrast material or fluid in this area.

Several features may aid in the distinction. These include location, contour and orientation. The sulcus typically extends only to the posterior attachment of the biceps tendon on the labrum and glenoid. The recess is usually less that 2.5 mm on MR arthrography and has smooth margins. The direction of the fluid parallels the glenoid margin in a direction towards the head and does not extend into the substance of the labrum¹⁹.

A superior labral tear with anterior and posterior components of the tear is called a SLAP lesion (Superior Labrum from Anterior to Posterior relative to the biceps tendon insertion on the supraglenoid tubercle)^{62,63}. This usually results when the bicipitolabral complex is overloaded from forceful traction or repetitive overhead motion or subject to direct compression, resulting in a tear or avulsion of the superior labrum from the glenoid. There may be involvement of the biceps tendon and associated rotator cuff tears. SLAP lesions are seen in the following situations: 1) during the follow-through phase of pitching and throwing when the biceps muscle contracts with deceleration; 2) following a fall on an outstretched abducted arm with associated superior glenohumeral compression and a proximal subluxation force; and 3) upon catching a heavy falling object . Repetitive stress on the biceps tendon and glenohumeral instability can also produce a SLAP lesion.

A lesion of the superior labrum can contribute to anterior shoulder instability since the long head of the biceps tendon and superior glenoid labrum play a role in glenohumeral joint stability⁶⁴. A retained biceps tendon stump at the superior glenoid from a tear can cause glenoid and humeral head chondromalacia via a "windshield wiper" mechanism and it is important to remove it before this occurs⁶⁵.

Clinical diagnosis of SLAP lesions can be a challenge. Patients with this disorder frequently present with a painful locking, pseudosubluxation, or snapping sensation. This can be difficult to distinguish from instability, biceps tendinosis and impingement.

Identification and classification of SLAP lesions by CT and MRI can be challenging. SLAP lesions can be seen by CT arthrography ⁶⁶ but in our opinion, they are best seen on MRI where there is routine multiplanar imaging and improved soft tissue contrast.

Four types of SLAP lesions were originally described by Snyder ⁶². Type I lesions consist of degenerative fraying that primarily involves the superior glenoid labrum. The biceps tendon is intact. These lesions present with labral irregularity and slight increase in signal intensity on MRI, and are the most difficult to identify with MRI. Type I SLAP lesions were seen in 11% in Cartland's retrospective series⁶⁷. These lesions are treated with arthroscopic debridement of the degenerative labrum. Type II lesions are the most common (seen in 41% of Cartland's series) and have similar superior labral fraying with detachment of the superior labrum and long head of the biceps tendon from the osseous glenoid, resulting in instability. The labral appearance is similar to Type I lesions on MRI with an additional globular region of high signal intensity between the superior glenoid and the labrum, representing the biceps anchor tear. Treatment of the Type II SLAP lesion involves debridement of the labrum and reattachment of the detached biceps anchor to the superior glenoid. A Type III SLAP lesion is seen in about a third of SLAP lesions and presents as a crescentic vertical tear that involves the central portion of the superior labrum with displacement or buckling of the labrum into the joint (similar to a bucket handle tear). The biceps tendon is normal. The treatment of this type of tear is variable. The labrum may be debrided, reattached, or resected. A Type IV SLAP lesion is a Type III tear of the superior labrum with extension of the tear into the proximal long head of the biceps tendon. It presents on MRI as a

diffuse high signal intensity lesion in the superior portion of the labrum and proximal portion of the biceps tendon. These patients have functional instability. Fifteen percent of SLAP lesions in Cartland's series were Type IV. Treatment of the Type IV SLAP lesion may involve resection of the torn labrum or suture repair of bucket handle tears. If the biceps tear involves more than half of the tendon, and the patient has symptoms referable to the tendon, biceps tenodesis may also be performed. A complex SLAP lesion may consist of a combination of two or more types, usually types II and IV.

Since the original description of the four SLAP lesions in the superior segment of the glenoid, Morgan subdivided the type II SLAP lesions into three different subcategories ⁶⁸. Type IIA is an anterior tear, IIB is a posterior tear, and IIC is a combined anterior and posterior tear of the superior labrum.

Three more types of SLAP lesions have been described by Maffet et al ²⁶. Type V lesions represent an anteroinferior Bankart lesion that extends superiorly to a torn biceps tendon. Type VI lesions are unstable radial or flap tears associated with separation of the biceps anchor. Type VII lesions involve anterior extension of the superior labral tear beneath the middle glenohumeral ligament. Type VIII (extending posteriorly), IX (diffuse involvement of the labrum) and X (SLAP lesion associated with a reverse labral Bankart lesion) have also been described. Another type of SLAP lesion that extends into the rotator interval involving the SGHL, coracohumeral ligament, capsule or synovium has also been separately described as a type X SLAP lesion.

Various mechanisms of injury lead to certain types of SLAP lesions ⁶⁹. Traction injuries often produce a type II SLAP lesion. Those patients who fall on an outstretched hand more commonly have a type III, IV, or V lesion. Athletes who use overhead shoulder motion and patients with atraumatic instability tend to get type I or II SLAP lesions whereas, patients who have traumatic instability are prone to type V or VII lesions.

One can evaluate the superior labrum and biceps tendon in all three planes, with the tear most easily characterized in the oblique-coronal and axial planes. SLAP lesions are best demonstrated on MRI with the shoulder in external rotation which provides traction on the long head of the biceps, enhancing the detection of imbibition of contrast, posterior and superior extent of the tear and separation at the site of the tear. Traction can also be placed on the biceps tendon in the form of a weight or placement of the shoulder in the ABER position.

It is difficult sometimes to completely characterize the lesions according to the various subtypes using MRI. The main function of the MRI is to identify the lesions of the superior labrum and biceps tendon. SLAP lesions are best evaluated when there is fluid or contrast in the joint. One should also look for SLAP lesions when imaging in the abduction external rotation (ABER) position. This provocative position may enhance evaluation of the SLAP lesion by causing fluid to be further pushed into the superior labrum.

Fifteen to twenty five percent of SLAP lesions are associated with a full-or partial thickness tear of the rotator cuff, anterior instability, humeral head fracture, chondromalacia of the humeral head, and AC joint arthritis⁷⁰. It is especially important to remember to carefully scrutinize the superior labrum and biceps tendon in these situations.

Labral fraying and irregularity can be seen in asymptomatic shoulders, often in patients of older age. This appearance can simulate a Type I or II SLAP lesion. It is crucial to keep this in mind and to correlate symptoms with the appearance on MRI.

Sensitivity, specificity and accuracy of routine noncontrast MRI and direct MR arthrography for identifying tears of the superior labrum have been reported at between 66-

98%,71-90%, and 96% respectively for routine MRI ^{71,72} and at 82%-100%, 69-98% and 74-94%, respectively for MR arthrography^{22,73-75}. Indirect MR arthrography with intravenous contrast has also been advocated, but is rarely done in the shoulder.

PARALABRAL CYSTS

A unique subset of shoulder ganglion cysts may arise from glenoid labral tear ⁷⁶. The mechanism for formation of these paralabral cysts is similar to a meniscal cyst with extrusion of joint fluid through labrocapsular tears into adjacent tissue planes. The most common locations for the labral cyst/tear complex are posterior (associated with a posterosuperior labral tear) and superior (associated with a SLAP (superior labrum anterior to posterior) lesion. Extra-articular extension of the labral cyst into the spinoglenoid notch and/or suprascapular notch is common. Intraosseous extension can also occur in the bony glenoid. When located adjacent to SLAP or posterior labral lesions, these cysts can produce suprascapular nerve entrapment, which is a cause of shoulder pain that can be evaluated with MRI⁷⁷. In addition, cysts associated with inferior labral tears can produce axillary nerve compression in the quadrilateral space ⁷⁸. Anterior dislocation of the glenohumeral joint may also damage the axillary nerve or its branches. In one series 43% of patients with anterior shoulder dislocation developed axillary nerve damage ⁷⁹.

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