The first report on superior labral lesions associated with the long head of the biceps origin, in a population of throwing athletes, was done by Andrews and colleagues, in 1985. Several years later, Snyder and coworkers described a superior glenoid labral lesion that “begins posteriorly and ends anteriorly,” and coined the term SLAP lesion. Since then, clinical and basic science research into the significance, etiology, diagnosis, and management of these injuries has proliferated.

**Anatomy**

The labrum is a triangular fibrocartilaginous structure that occupies the transition zone between the underlying articular cartilage of the glenoid and the fibrous tissue of the joint capsule. The morphology of the superior labrum is different than the inferior labrum. The superior labrum is more meniscal in nature and is loosely attached via a thin elastic connective tissue to the underlying glenoid.

At the twelve o’clock position, the superior labrum inserts directly into the long head of the biceps tendon, distal to the insertion of the biceps onto the supraglenoid tubercle. In addition, the articular cartilage of the glenoid extends five millimeters over the edge of the rim, with a synovial reflection, which creates a normal recess beneath the long head of the biceps and the superior labrum.

Two normal anatomic variants can be arthroscopically and radiographically confused with SLAP lesions. A sublabral recess or sublabral foramen, defined as a physiologic detachment of the superior labrum, has been observed in up to 73% of normal shoulders. Secondly, the appearance of a cord-like middle glenohumeral ligament, with an absent anterosuperior labrum, coined the Buford complex, has also been described. This variant is less common, with reported incidence varying from 1.5% to 5%. Although these anatomic variants exist, the extent to which they predispose or protect the superior labrum from injury is not well understood.

The blood supply of the glenoid labrum has been well described by Cooper and colleagues. It consists of branches from the suprascapular artery, the circumflex scapular artery, and posterior humeral circumflex artery. These branches also supply the capsule, synovium, and periosteum of the scapula neck and glenoid. Similar to the menisci of the knee, the vascular penetration of the labrum is limited to the peripheral attachment, which has implications for healing of tears based on location. In general, the superior and anterosuperior labrum is less vascular than the posterior and inferior labrum.

The long head of the biceps attaches to both the superior glenoid labrum and supraglenoid tubercle (located approximately five millimeters medial to the superior rim of the glenoid at the twelve o’clock position). The attachment is predominately to the posterosuperior labrum with extension to the anterosuperior labrum. Vangsness and associates studied the insertion of the long head of the biceps tendon in 100 cadavers. They found that 50% of the tendon arises from the supraglenoid tubercle, and 50% arises from the labrum. In addition, they classified the attachment of the long head
of the biceps onto the glenoid/labrum into four types: Type I (22%), entirely posterior; Type II (33%), mostly posterior; Type III (37%), equal; and Type IV (8%), mostly anterior.

Biomechanics

The long head of the biceps tendon is generally regarded as a humeral head depressor. Other studies also suggest that it plays a role in providing anterior stability to the glenohumeral joint. Kumar and coworkers, in a cadaveric study, stimulated the short head of the biceps alone and found that the humeral head migrated 15.5 mm superiorly. However, when both the long head and short head were simultaneously stimulated, there was less migration, and the investigators concluded that the long head on the biceps functions as a humeral head depressor. Itoi and colleagues created cadaveric SLAP lesions and found an increase in anterior-posterior and superior-inferior translation with applied loads. They then stimulated the long head of the biceps tendon and found a significantly decreased translation with an anteriorly applied force. Rodosky and coworkers looked at the contribution of the long head of the biceps and superior labrum to anterior stability by testing cadaveric shoulders before and after the creation of SLAP lesions. They found that stimulation of the long head of the biceps stabilizes the humeral head to an anterior force and increases resistance to torsional force (resists excessive external rotation). They also found that a SLAP lesion places increased strain on the inferior glenohumeral ligament, which could theoretically lead to damage of the ligament and subsequent anterior instability. A recent study by Healey and colleagues evaluated the relative contribution of stiffness and displacement under load of the two origins of the long head of the biceps tendon. They concluded that the biceps anchor is the primary restraint and the superior labrum is a secondary restraint, and that disruption of both is required to produce significant laxity.

Classification

Snyder and associates classified SLAP lesions into four types (Fig. 1). The type I lesion (21%) is characterized by fraying and degeneration of the edge of the superior labrum, with maintenance of a firm attachment of the labrum and biceps anchor to the glenoid. The type II lesion (55%) consists of fraying and degenerative changes similar to those seen in a type I lesion, but the superior labrum and attached biceps tendon are detached from the underlying glenoid, resulting in an unstable labral-biceps anchor. The type III lesion (9%) is a bucket handle tear of the superior labrum, with the remaining portions of the labrum and biceps anchor remaining firmly attached. In a type IV lesion (10%), there is a bucket handle tear of the superior labrum that extends into the biceps tendon, and the partial biceps tendon tear can displace with the labral tear into the joint.

A study by Maffet and coworkers found that 32 of 84 patients with SLAP lesions did not have lesions that fit Snyder’s classification scheme, and they described three additional types. The type V lesion consists of a Bankart lesion that continues superiorly to include separation of the biceps tendon. The type VI lesion involves a type II lesion with an unstable flap tear of the superior labrum. In a type VII lesion, the biceps tendon-superior labrum separation extends anteriorly to involve the middle glenohumeral ligament.

Pathology

The exact etiology of superior labral lesions remains uncertain. Several causative mechanisms have been proposed as being responsible for creation of these injuries. The initial description of these lesions by Andrews and colleagues postulated that tensile overload of the biceps tendon occurred during the follow-through phase of throwing. As the elbow rapidly extends during this phase, the biceps actively
fires to decelerate the elbow, causing a sudden tensile load with the potential to avulse the biceps or labral complex. The internal impingement theory,\textsuperscript{17,18} may also contribute to superior labral pathology. With the shoulder abducted to 90° and maximally externally rotated (late cocking or acceleration phase), the greater tuberosity can cause fraying of the posterosuperior labrum as well as impingement of the rotator cuff against the glenoid. These events can occur with a perfectly reduced head, but are more likely to occur in patients with anterior instability. Lastly, Burkhart and Morgan\textsuperscript{19} proposed a particular mechanism for the creation of type II SLAP lesions. These investigators believe that tensile overload of the posterosuperior labrum is not the mechanism for biceps anchor failure. They state that type II labral tears occur when an acquired posterior and inferior capsular contracture develops. When this acquired contracture occurs, the humeral head undergoes a posterosuperior shift as the shoulder abducts and externally rotates in the wind-up phase of throwing. With this new point of rotation, when the shoulder goes into extreme abduction and external rotation (cocking phase of throwing), the biceps assumes a more vertical and posterior angle that produces a twist, which causes the labrum to rotate medially and “peel back” from the superior glenoid.

**Diagnosis**

The diagnosis of SLAP lesions is difficult and challenging for several reasons. First, the incidence is relatively low. Snyder, in a retrospective review of over 2,300 shoulder arthroscopies, found only 140 patients with superior labral pathology (6.1%).\textsuperscript{20} Second, the symptoms are similar to other shoulder disorders, and thus may mimic other disorders, such as rotator cuff disorders and glenohumeral instability. Lastly, there is a high incidence of associated pathology and injuries with SLAP lesions, including rotator cuff disorders (partial-thickness rotator cuff tears, 40% to 29%, and full-thickness rotator cuff tears, 11%), Bankart lesions (22%), acromioclavicular joint arthrosis (16%), and glenohumeral chondromalacia (10%).\textsuperscript{21} Only 28% of SLAP lesions are isolated without other associated injuries.

The symptoms caused by SLAP lesions are vague and inconsistent, so the clinician must have a high index of suspicion. Pain is the most common complaint, especially with overhead activities, similar to impingement-type pain. Patients may also describe mechanical symptoms such as catching, popping, locking, or grinding. Other common complaints include pain when lying on the affected shoulder, pain with activities of daily living, loss of strength, loss of motion, and a sense of the arm “going out.” Throwing athletes may complain of a “dead arm.”

The mechanism of injury can be divided into compression and traction. The most common compression injury is incurred through falling onto the outstretched hand with the arm positioned in slight flexion and abduction, forcing the humeral head superiorly; this accounts for 23% to 31% of compression injuries.\textsuperscript{20} Another compressive mechanism is a direct blow to the shoulder, which is the cause of injury in 17% of patients with an isolated SLAP lesion. Traction injuries occur when there is a sudden pull on the arm or in conjunction with a dislocation. A traction injury can occur in an inferior direction when one loses hold of a heavy object, or in an anterior direction (water skiing), or in an upward direction, such as when grabbing overhead while falling (gymnastics). Another common mechanism of injury, particularly in athletes, is repetitive overhead activity, such as pitching. Up to 33% of patients, who ultimately prove to have a SLAP lesion of the shoulder, will simply describe an insidious onset of shoulder pain without any specific history of trauma or overuse.\textsuperscript{21}

The physical exam findings in patients are subtle, as there is no physical finding specific for SLAP lesions of the shoulder. The exam should always begin with inspection, palpation, evaluation of the range of motion of the shoulder, a detailed neurovascular exam, and evaluation of the neck. The physician must be aware of concomitant associated shoulder pathology. Another important component of the exam is evaluation of the scapula. Kibler and colleagues\textsuperscript{22} found that 60 out of 64 patients with type II SLAP lesions demonstrated scapular dyskinesis.

Numerous provocative tests have been described to aid in the diagnosis of SLAP lesions (Table 1). The compression-rotation test, also known as the “grind test” is performed by having the patient lay in either the supine or lateral position, with the shoulder abducted 90°, the elbow flexed 90° with a compressive force applied to the humerus with internal and external rotation of the shoulder (similar to the McMurray’s test for meniscal tears in the knee). Pain on testing is considered a positive test result. This test is relatively sensitive for labral tears, but is not specific for SLAP lesions. The biceps tension test, also known as “Speed’s test,” is performed by having the patient resist forward flexion with the shoulder flexed 90° with the elbow extended and the forearm supinated. The test is positive if this maneuver elicits anterior shoulder pain. This test is more useful for anterior type II SLAP lesions than posterior type II SLAP lesions.\textsuperscript{19} The biceps tension test is also not very specific for SLAP lesions; false positive tests are found in patients with biceps tendinitis.

The anterior slide test (Kibler’s test) is performed with the patient either sitting or standing, with the hand on the ipsilateral hip with the thumb posterior and fingers anterior. The examiner pushes upward and forward on the elbow against resistance. A positive test produces anterior shoulder pain and/or a pop or click, as well as a subjective reproduction of the symptoms felt during overhead activity. This test is reported to have a 78.4% sensitivity and a 91.5% specificity for superior glenoid labral tears.\textsuperscript{23}

The crank test, described by Liu and associates,\textsuperscript{24} is performed with the patient either upright or supine. The arm is elevated 160° in the scapular plane, and the humerus is then
loaded axially with maximal internal and external rotation. A positive test produces pain (usually in the externally rotated position) with or without a click, and also reproduces the patient’s subjective symptoms that occur during offending activities. This test has a reported sensitivity of 93% and specificity of 94%, with a 94% positive predictive value and 90% negative predictive value for glenoid labral tears, but is not specific for SLAP lesions.

The active compression test (O’Brien test) is performed with the patient standing, the arm forward flexed 90° with the elbow extended and the arm adducted 10° to 15°, and maximal internal rotation. The examiner then applies a resisted downward force to the arm. The patient then maximally supinates the arm and the maneuver is repeated. A positive test elicits pain during the first maneuver, which is reduced or eliminated with the second maneuver. Pain localized to the AC joint must be differentiated from pain or painful clicking inside the shoulder. This test has a reported sensitivity of 100% and specificity of 98.5%, with a 94.6% positive predictive value, and a 100% negative predictive value for labral abnormalities.  

The pain provocation test (Mimori’s test) is performed with the patient sitting and the arm abducted 90° to 100°. The examiner externally rotates the shoulder and places the arm in maximal pronation and then maximal supination. A positive test provokes pain only when the forearm is in the pronated position or when pain in the pronated position is greater than pain in the supinated position. This test is reported to have a sensitivity of 100%, a specificity of 90%, and 97% accuracy for diagnosing superior labral tears.

Kim and coworkers developed the Biceps load test to evaluate the integrity of the superior glenoid labrum in shoulders with recurrent anterior dislocations, in order to differentiate a type V SLAP lesion from anterior instability alone. The test was evaluated in 75 patients with anterior dislocations by externally rotating the shoulder to the apprehension position, then having the patient perform resisted elbow flexion and assessing for a change in apprehension. If the apprehension was unchanged or the shoulder was more painful, this was considered positive for a SLAP lesion, and if the apprehension was less or the patient felt more comfortable, this was a negative test. Using this test, the investigators reported a 100% sensitivity, a 90% specificity, and a 97% accuracy for diagnosing a superior labral tear. Kim and coworkers then developed the biceps load test II to specifically evaluate patients for the presence of

<table>
<thead>
<tr>
<th>Test</th>
<th>General Position</th>
<th>Positive Result</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression-rotation test (Grind test)</td>
<td>Supine</td>
<td>Pain with internal and external rotation of the shoulder*</td>
<td>Not specific for SLAP lesion</td>
</tr>
<tr>
<td>Biceps tension test (Speed’s test)</td>
<td>Standing</td>
<td>Anterior shoulder pain on flexing the shoulder against resistance*</td>
<td>More useful for anterior type II SLAP lesions; not specific for SLAP lesions</td>
</tr>
<tr>
<td>Anterior slide test (Kibler’s test)</td>
<td>Sitting</td>
<td>Pain and/or pop/click and possibly subjective symptoms on resistance to upward/forward push to elbow*</td>
<td>Patient hands should be placed on their hips</td>
</tr>
<tr>
<td>Crank test</td>
<td>Supine or upright</td>
<td>Pain and possibly subjective symptoms, usually on external rotation of humerus, sometimes internal*</td>
<td>Not specific for SLAP lesion</td>
</tr>
<tr>
<td>Active compression test (O’Brien test)</td>
<td>Standing</td>
<td>Pain on 1st maneuver (resisted downward force on arm), pain reduced or eliminated on 2nd maneuver*</td>
<td>Must differentiate pain in AC joint from shoulder pain</td>
</tr>
<tr>
<td>Pain provocation test (Mimori’s test)</td>
<td>Sitting</td>
<td>Pain when forearm is placed in pronated position with externally rotated shoulder, or pronated position is more painful than supinated*</td>
<td>Arm must be placed in “maximal” for both positions</td>
</tr>
<tr>
<td>Biceps load test</td>
<td>Standing</td>
<td>Painful shoulder or apprehension unchanged during resisted elbow flexion and external shoulder rotation*</td>
<td>Differentiates type V SLAP from anterior instability alone</td>
</tr>
<tr>
<td>Biceps load test II</td>
<td>Supine</td>
<td>Pain on resisted elbow flexion with forearm supinated</td>
<td>Developed to test presence of type II SLAP lesion</td>
</tr>
</tbody>
</table>
a type II SLAP lesion. The exam is performed with the patient supine, with the arm elevated 120° and maximally, externally rotated. The elbow is then flexed 90°, the forearm is supinated, and the patient is then asked to flex his elbow against resistance. A positive test elicits pain during resisted elbow flexion. This test has a reported sensitivity of 90%, a specificity of 97%, with a 92% positive predictive value and a 95% negative predictive value for diagnosing type II SLAP lesions.

Imaging Studies

Radiologic investigation of the shoulder should begin with conventional radiographs, including an AP, supraspinatus outlet, and axillary lateral to identify associated pathology such as glenohumeral dislocation/subluxation, acromioclavicular disease, or subacromial spurs. However, radiographs will not help disclose any possible intra-articular labral disorders. Additional studies to consider include CT arthrography, magnetic resonance imaging (MRI), and MR arthrography.

CT arthrography is better for detecting bone abnormalities, and MRI is superior for evaluating labral lesions. MRI has a reported sensitivity of 95% in detecting labral lesions, with decreased sensitivity in identifying superior lesions compared to middle and inferior labral tears.29 Another report shows MRI to have 86% sensitivity and 100% specificity in detecting superior labral lesions.30 MR arthrography was developed to improve the ability of MRI to detect superior labral lesions. The introduction of fluid into the joint increases contrast differences between soft tissue structures and the glenohumeral joint. MR arthrography was initially performed with saline, but is now performed with gadopentetate dimeglumine (gadolinium), a highly magnetic contrast agent, which has been shown to be nontoxic to the synovial lining. Chandani and colleagues31 reported 89% sensitivity, 88% specificity, and 89% accuracy in detecting superior labral lesions, while Lee and colleagues,32 in a recent study, reported a 92% sensitivity, and 83% specificity in identifying these lesions. MR arthrography findings suggestive of a SLAP lesion include: contrast extending into the long head of the biceps insertion on the oblique sagittal and oblique coronal images; irregularity of the long head of the biceps insertion on the oblique sagittal and oblique coronal images; contrast extending between the labrum and the glenoid on the axial images; displacement of the superior labrum on oblique sagittal and oblique coronal images; displacement of a fragment of the labrum between the humeral head and glenoid fossa; and presence of a glenoid labral cyst in the suprascapular or spinoglenoid notch.29-32

Treatment

Conservative treatment of SLAP lesions has been found to be generally unsuccessful; however, these lesions are amenable to arthroscopic treatment. Appropriate treatment of SLAP lesions depends on accurate arthroscopic classification of the lesion. The original article by Snyder and colleagues2 proposed a treatment algorithm based on the classification system that, for the most part, remains true today. They recommended debridement of type I lesions, repair of type II lesions (not feasible at the time so they recommended abrasion to bleeding bone), and excision of the bucket handle tear in type III lesions. For type IV lesions, they recommended excision of the bucket handle tear and the involved portion of the biceps tendon, if less than 50% of the tendon. If the involved portion was greater than 50% of the tendon, biceps tenodesis and excision of the tear were recommended. The indication for tenodesis was later changed to involvement of greater than 30% of the biceps tendon.20

Altchek and associates33 reported only 7% good results for debridement alone for SLAP lesions in ten patients in two years. Cordasco and coworkers34 looked at 27 patients with SLAP lesions treated with debridement alone. At one year, 78% of patients had good results, which declined to 63% at two years, with only 44% of patients returning to prior athletic levels. Other authors also reported relatively poor results with debridement alone, with 100% poor results noted when labral resection without glenohumeral stabilization was performed for labral lesions with concomitant shoulder instability.35,36 In summary, debridement should be performed with caution, as excessive debridement may be associated with the development of instability of the biceps-labral complex. Debridement of types I and III is appropriate, but the surgeon must also look for concomitant shoulder pathology and address it in the same surgical setting.

Yoneda and colleagues37 first reported on repair of type II SLAP lesions in 10 patients using an arthroscopic staple, which was removed 3 to 6 months postoperatively, with 80% good to excellent results. Field and Savoie38 described labral debridement and repair using a transglenoid suture technique in 20 patients; they reported 100% good results at 21 months. Their technique is technically difficult with significant neurovascular risk.

Resch and associates39 reported on 18 patients with unstable SLAP lesions, 14 of which were repaired using either titanium screws or Suretac® anchors, and four of which were treated with debridement alone. In the follow-up period of 18 months, there were 12 good to excellent results in the repair group, while in the debridement group only one patient improved.

Several other investigators have also reported the results of treatment of unstable SLAP lesions using a biodegradable tack. Pagnani and coworkers40 reported on 22 patients with type II SLAP lesions repaired with Suretac® anchors. Twelve out of 13 overhead athletes (6 out of 6 professional athletes) were able to return to sport activities, and 86% overall were satisfied. A study by Segmüller and colleagues40 looked at 70 patients with labral lesions, 17 of which were stabilized using Suretac® anchors. At 17-month follow-up, results were good to excellent in 82% of cases, with 53% of these athletes returning to their pre-injury level of performance. The most
favorable report of Suretac® anchors was published recently by Samani and associates. They looked at 25 repairs of type II SLAP lesions with 22 having good to excellent results at a minimum of 24 months and 23 of 25 returning to sport activities.

There have been problems associated with the use of Suretac® anchors. Burkhardt and coworkers3 performed a biomechanical-SM 11/15 study comparing the Suretac® to a suture anchor (Ultimate SuperAnchor, Mitek®). The anchors were tested to determine single load to failure (i.e., pullout strength). The Suretac® anchors failed at a mean of 122 N, and the suture anchors failed at a mean of 217 N.

Only one published study reports the results of repair of unstable SLAP lesions using suture anchors. Burkhart and Morgan19 repaired 102 type II SLAP lesions with arthroscopic suture anchors. Fifty-three of the patients were overhead athletes and 49 of the patients were non-throwers who had experienced a single-event traumatic injury. At one-year follow-up, they had 97% good to excellent results, with 84% of pitchers returning to their pre-injury level of performance.

In summary, SLAP lesions are treated based on the type of lesion. Type I lesions are debrided, with preservation of the biceps anchor attachment to the superior glenoid. Type II lesions are repaired using one of several arthroscopic repair techniques. Type II lesions are treated with excision of the bucket handle tear of the labrum. Treatment of type IV lesions depends on the patient’s age and degree of involvement of the biceps tendon. Excision of the bucket handle tear and involved portion of the biceps is performed for older patients with less than 30% biceps tendon involvement. For young patients with a small but significant biceps tendon tear and for older patients with more than 30% biceps tendon involvement, biceps tenodesis is recommended. Arthroscopic labral repair may be considered for young patients with extensive type IV lesions. Lesions types V to VII are treated similarly to type I to IV, with additional treatment required for the additional pathology.

Summary

SLAP lesions are becoming a more recognized cause of shoulder pain and disability. The diagnosis of these lesions is difficult due to vague symptoms and high degree of overlap with other shoulder disorders, and this requires a high index of suspicion. Advances in MR arthrography may lead to advances in preoperative diagnosis of labral tears, but definitive diagnosis, classification, and management is greatly facilitated with the use of the shoulder arthroscopy. Further basic science and clinical research should enhance our ability to manage patients with these lesions effectively.

References