SLAP Lesions: An Update on Recognition and Treatment

Superior labral lesions are a common occurrence in the athletic population, especially overhead athletes. The first description of labral tears involving the superior aspect of the glenoid was described by Andrews et al., who reported on 73 overhead throwing athletes who had labral tears specifically located in the anterosuperior quadrant of the glenoid, near the origin of the long head of the biceps tendon. Snyder would later coin the term “SLAP lesion” (superior labrum, anterior and posterior) to describe a similar injury pattern, located at the biceps anchor and extending anterior to posterior. Although both authors reported on similar lesions, the etiology remained unclear. Andrews et al. proposed that the biceps tendon acted to “pull off” the labrum, whereas the most common mechanism of injury in the Snyder report was compression loading, with the shoulder in a flexed and abducted position.

The advancement of arthroscopic techniques has led to a tremendous increase in our understanding of SLAP lesions. However, making the diagnosis clinically can still be a challenge. A comprehensive approach involving a thorough history and physical examination, adequate imaging, and ultimately diagnostic arthroscopy is often necessary to recognize and appropriately treat SLAP lesions. Untreated, SLAP lesions can be a potentially devastating injury that can lead to chronic pain, as well as a significant loss of function and performance. The purpose of this article is to review the classification, pathomechanics, clinical evaluation, and treatment of SLAP lesions. The recommendations regarding diagnosis and treatment presented in this article are based on clinical experience.

PATHOMECHANICS

A comprehensive discussion of the pathomechanics of SLAP lesions is beyond the scope of this article. However, we do feel that a basic review of some of the proposed mechanisms of SLAP lesions can be helpful in understanding their surgical treatment and rehabilitation.

It is not uncommon to encounter associated pathology when treating a SLAP lesion. Most notably, patients who have SLAP tears can also have concomitant rotator cuff tears and other labral pathology. Andrews et al. reported that 45% of patients (and 73% of baseball pitchers) with SLAP lesions also had partial-thickness tears of the supraspinatus portion of the rotator cuff. Mileski and Snyder reported that 29% of their patients with SLAP lesions exhibited partial-thickness tears of the supraspinatus and biceps anchor.
rotator cuff tears, 11% complete rotator cuff tears, and 22% Bankart lesions of the anterior glenoid. They also demonstrated that type I SLAP lesions are typically associated with rotator cuff pathology, while types III and IV are associated with traumatic instability. Finally, they noticed that in patients with type II lesions, older patients tended to have associated rotator cuff pathology, while younger patients had associated anterior instability.

Our recognition of these associated lesions may allow for insight with regard to the biomechanical etiology of SLAP lesions. There are several proposed mechanisms, although it is still unclear what exactly causes SLAP lesions. These mechanisms can be divided into acute traumatic events or chronic repetitive injuries that lead to failure. An example of an acute traumatic event would be falling onto an outstretched arm, which may result in a SLAP lesion, secondary to impaction of the humeral head against the superior labrum and the biceps anchor. Repetitive overhead activity has been hypothesized as a common mechanism for producing SLAP lesions. Andrews et al first theorized that SLAP lesions in overhead throwing athletes was the result of the high eccentric activity of the biceps muscle, creating tension on the long head of the biceps tendon, during the arm deceleration and follow-through phases of throwing. The authors subsequently applied electrical stimulation to the biceps during arthroscopy and noted that contraction of the biceps caused the biceps anchor to effectively separate from the glenoid.

Burkhart and Morgan have hypothesized that a “peel-back” mechanism may produce a SLAP lesion in the overhead athlete. They believe that when the shoulder is placed in a position of abduction and maximal external rotation, the rotation causes a torsional force at the base of the biceps. In a cadaveric study, Pradham et al measured superior labral strain during each phase of the throwing motion. They reported increased superior labral strain during the late cocking phase of throwing, which supports the concept of the “peel-back” mechanism. Additional authors have also demonstrated that there is contact between the posterior-superior labrum and the rotator cuff when the arm is in an abducted and externally rotated position, which simulates the late cocking phase of throwing.

Shepard et al simulated each of the aforementioned mechanisms in 9 pairs of cadaveric shoulders that were loaded to biceps anchor failure in either a position of in-line loading (similar to the deceleration phase of throwing) or in a simulated peel-back mechanism (similar to the late cocking phase of throwing). Their results showed that all of the simulated peel-back group failures resulted in a type II SLAP lesion, whereas the majority of the simulated in-line-loading group failures occurred in the midsubstance of the biceps tendon. Additionally, the biceps anchor demonstrated a significantly higher strength with in-line loading as opposed to the ultimate strength during the “peel-back” mechanism. These results support the theory of peel-back as the predominant mechanism; but, most likely, a combination of mechanisms result in SLAP lesions.

Finally, several authors have reported an association between SLAP lesions and glenohumeral instability. Pagnani et al found that a complete lesion of the superior portion of the labrum was associated with significant increases in glenohumeral translation. They also found that the presence of a simulated SLAP lesion resulted in a 6-mm increase in anterior glenohumeral translation. Kim et al reported that maximal biceps activity occurred when the shoulder was in the abducted and externally rotated position in patients with anterior instability. The exact cause-and-effect relationship of instability and SLAP lesions is still unclear. It may be that instability allows for a pathologic range of motion that facilitates the peel-back mechanism or, possibly, that SLAP lesions allow for excessive glenohumeral translation, which leads to instability.

The most common classification system for SLAP lesions was described by Snyder et al following a retrospective review of 700 shoulder arthroscopies. They identified 4 types of superior labral lesions involving the biceps anchor (FIGURE 1). Type I SLAP lesions have fraying of the edge of the superior labrum. Type II SLAP lesions, the most common, consist of frank detachment of the biceps anchor from the glenoid. Type III lesions consist of a bucket-handle tear of the superior labrum with, an otherwise normal biceps anchor. Finally, type IV lesions, the least common, consist of a bucket-handle tear of the superior labrum that extends into the biceps tendon, causing detachment of the biceps anchor.

Over time, many surgeons have encountered numerous combined or complex SLAP lesions that cannot be classified by Snyder et al’s original classification system. Maffet et al expanded the original classification to include type V lesions, anteroinferior Bankart-type labral lesions in continuity with SLAP lesions, type VI lesions, consisting of biceps tendon separation with an unstable
flap tear of the labrum, and type VII lesions, consisting of an extension of the superior-labrum biceps tendon separation to beneath the middle glenohumeral ligament. Morgan et al has subclassified type II slap lesions into (1) anterior, (2) posterior, and (3) combined anterior and posterior lesions. They hypothesize that SLAP lesions cause anterior or posterior microinstability, which can ultimately lead to pseudolaxity and partial-thickness, articular-sided rotator cuff tears that are lesion specific. In other words, posterior SLAP lesions cause posterior cuff tears, and anterior SLAP lesions cause anterior cuff tears.

CLINICAL EVALUATION

The clinical evaluation of any patient with shoulder pain should include a subjective history, a thorough physical examination, plain radiographs, and, when indicated, advanced imaging, which is usually magnetic resonance imaging (MRI). The goal is to make an accurate diagnosis, so that an appropriate treatment plan can be initiated.

History

A comprehensive history is essential and should try to precisely define the mechanism of injury. Patients usually complain of vague shoulder pain, often with clicking, popping, or snapping that is usually exacerbated with overhead activity. Patients may also complain of instability if the lesion extends to the anterior labrum/capsule. When a concomitant rotator cuff tear is present, patients may complain of shoulder weakness. It has been our experience that patients typically complain of pain and dysfunction, or limited function when the labrum is involved, and do not experience pain at rest or at night, which is more common with rotator cuff injuries.

The most commonly cited mechanisms of injury include traction and compression of the shoulder; although, in many instances, no antecedent trauma is remembered. Overhead athletes may complain of pain during a specific phase of throwing, most notably the late cocking phase. We have found that the most likely complaint in the thrower is the inability to perform at their optimal level, which should raise suspicion about an internal derangement in the shoulder.

Physical Examination

The physical exam begins with gross inspection of the involved extremity. Atrophy of the rotator cuff can be attributed to possible compression of the suprascapular nerve by a ganglion cyst and warrants evaluation with an MRI and possible electrodiagnostic testing (EMG). The cervical spine is then examined for range of motion and any evidence of nerve root compression.

The affected shoulder is then assessed and compared to the unaffected side. Bilateral passive and active range of motion is noted, with attention paid to any motion that elicits pain. Many patients with SLAP lesions will note pain with passive external rotation at 90° of shoulder abduction. Overhead athletes may exhibit excessive external rotation with posterior capsule tightness and resulting internal rotation deficits. Motor strength is then tested and the extremity is examined for gross neurovascular deficits. The presence of rotator cuff pathology or instability is then evaluated before proceeding towards specific diagnostic maneuvers for SLAP lesions.

Numerous tests have been described to be specifically designed to determine the presence of labral pathology, including the active-compression test, the compression-rotation or grind test, Speed’s test, the clunk test, the crank test, the anterior-slide test, the biceps load test, the biceps load test II, and the pain provocation test. Although many of these tests have been shown to accurately diagnose SLAP lesions, their reproducibility among multiple examiners is uncertain. Therefore, it is important to correlate the patient’s symptoms with the physical examination findings to make the diagnosis. It is not necessary to perform all these maneuvers on every patient; therefore, we will describe some of the more common tests that we typically use, as well as some of the more recently reported ones.

The active-compression test, as described by O’Brien, has been shown to be helpful in diagnosing SLAP lesions. The shoulder is positioned in 90° of forward elevation and 20° of horizontal adduction. The examiner then places a downward force on the forearm, while the forearm is pronated and then supinated (Figure 2). A test is positive for a labral involvement when pain is elicited with the forearm in the pronated position and relieved when the forearm is supinated. The authors also point out that a test is considered negative when the pain is localized to the acromioclavicular (AC) joint. O’Brien et al reported this maneuver to be 100% sensitive and 95% specific in diagnosing labral pathology. However, other authors have reported less success using this test.

We typically perform this maneuver when assessing for labral pathology and find pain inside the shoulder when testing with the forearm pronated and pain relieved with supination to be most indicative of a SLAP lesion. Pain in the posterior aspect of the shoulder or located to the AC joint is not specific for the presence of a SLAP lesion.

The compression rotation test is performed with the patient in the supine po-
CLINICAL COMMENTARY

The sensitivity (82.8%), specificity (81.8%), positive predictive value (92.3%), negative predictive value (64.3%), and diagnostic accuracy (82.5%), when compared to other provocative maneuvers.

Wilk et al. described a similar test that they have termed the “pronated load test.” With the patient in supine, the shoulder is abducted to approximately 90°, and the examiner passively externally rotates the shoulder with the forearm in pronation. At the point of maximal external rotation, the examiner instructs the patient to perform an isometric biceps contraction in an attempt to peel back the labrum.

Several authors have evaluated the ability of multiple provocative tests to predict the presence of labral pathology. Although many of the tests did provide some clinical accuracy, none was perfect. It is possible that each test may have varying specificity and sensitivity, depending on the type of slap lesion present. Clearly, further investigation regarding the accuracy of clinical tests is warranted.

Imaging Studies

Imaging evaluation begins with standard radiographs of the shoulder (AP, axillary, scapular-Y, and Stryker notch views). Radiographs are typically normal in cases of isolated SLAP lesions but may reveal bony abnormalities in cases of associated pathology (e.g., Hill-Sachs lesion).

MRI is the gold standard imaging modality for diagnosing SLAP lesions. However, the reliability of MRI to diagnose SLAP lesions has been disputed. Several authors have found difficulty diagnosing labral lesions with standard MR techniques. Therefore, they recommend magnetic resonance arthrogram with an intra-articular injection of gadolinium. Bencardino et al. demonstrated a sensitivity of 89%, a specificity of 91%, and an accuracy of 90% in detecting labral lesions using this technique.

At our institution, noncontrast MRI has become the standard advanced-imaging modality for diagnosing labral injuries to the shoulder. The SLAP lesions can typically be appreciated on a coronal sequence as a cleft between the superior labrum and the glenoid. Although previous studies have reported that noncontrast MRI is limited in the evaluation of the superior glenoid labrum, our experience has been that high-resolution noncontrast MRI can accurately diagnose superior labral lesions and aid in surgical management. In cases of suspected concomitant rotator cuff involvement, we do use MRI arthrography, with the arm in abduction and external rotation (ABER), to enhance the visualization of the articular surface of the rotator cuff, the superior glenoid labrum,
and the anterior inferior capsule-labrum complex. We have found this technique to be particularly helpful in detecting articular-sided partial thickness tears with intratendinous extension (FIGURE 7). We believe this is important because without preoperative identification of the extent of the delamination, the surgeon might have a difficult time making that determination in the operating room.

NONOPERATIVE MANAGEMENT

Conservative management of SLAP lesions is often unsuccessful, particularly when there is a component of glenohumeral joint instability or when a concomitant rotator cuff tear is present. There may be, however, a small subset of patients, particularly those with type I SLAP lesions, who are amenable to conservative treatment. The initial phase of conservative management consists of cessation of throwing activities, followed by a short course of anti-inflammatory medication to reduce pain and inflammation. Once the pain has subsided, we initiate physical therapy focused on restoring normal shoulder motion. Strengthening of the shoulder girdle musculature is also crucial to restore normal scapulothoracic motion. An excessive loss of glenohumeral internal rotation (GIRD) is common in overhead athletes and a particular emphasis should be placed on stretching the posterior capsule and restoring internal rotation, which may prevent pathologic contact between the supraspinatus tendon and the posterosuperior labrum. Although it is unclear if GIRD is a risk factor for SLAP lesions, restoring shoulder internal rotation may improve pathomechanics that could possibly reduce some of the patient’s symptoms. The patient is eventually advanced to a strengthening phase, which includes trunk, core, rotator cuff, and scapular musculature. In throwing athletes, we initiate a progressive throwing program after 3 months that is directed toward the patients’ specific sport and position. We have no published data regarding the success rate of nonoperative management; but as previously stated, it has been our experience that the majority of patients with symptomatic SLAP lesions will fail conservative management, particularly throwers. The goal of a nonoperative program is to reduce pain, improve motion, and restore strength in patients who do not wish to proceed to operative management.

SURGICAL MANAGEMENT

Our indications for surgery are patients who fail conservative management, patients who have a SLAP lesion with significant rotator cuff tears (>50%), and patients with large associated labral tears who exhibit severe mechanical symptoms. Generally speaking, we debride types I and III SLAP lesions and repair types II and IV. The following is a description of our SLAP repair technique.

We perform shoulder arthroscopy with the patient in the beach chair position. The patient is anesthetized under intrascalene block with sedation. After a complete clinical examination under anesthesia is performed, a 30° arthroscope (Smith & Nephew Dyonics, Andover, MA) is introduced into the glenohumeral joint via the posterior soft-spot portal. Then, a standard anterior portal is established in the rotator cuff interval and a complete diagnostic arthroscopy is performed. A probe is used to confirm a SLAP lesion and the extent of any other concomitant pathology is assessed (FIGURE 8). In cases of an isolated repairable SLAP lesion, a second anterosuperior lateral portal is established in the rotator cuff interval (FIGURE 9). We caution against the use of a transrotator cuff approach, especially in throwing athletes. Any unstable flaps of labrum are debrided to a stable rim, and an arthroscopic shaver or burr is used to create a bony bed at the superior rim of the glenoid via the anterosuperior lateral
portal (FIGURE 10A). After the bed of the SLAP lesion has been satisfactorily prepared, an 18-gauge spinal needle is passed via a transdermal puncture in the region of the portal of Neviaser into arthroscopic view above the superior labrum. Accurate suture placement is ensured by direct arthroscopic visualization of the spinal needle as it is passed through the superior labrum from superior to inferior (FIGURE 10B). A number 1 polydioxanone (PDS) suture (Ethicon, Somerville, NJ) is manually advanced through the spinal needle into arthroscopic view (FIGURE 11A). An arthroscopic retriever is then used to retrieve the free end of the suture and bring it out via the standard anterior portal (FIGURE 11B).

A suture anchor (Bio-Suture Tak, Arthrex, Naples, FL) is then inserted via the anterosuperior portal into the superior neck of the glenoid off the face (FIGURE 12). The more-medial limb of the suture from the suture anchor is then retrieved with the inferior limb of the PDS suture, and both sutures are brought out through the anterosuperior lateral portal (FIGURE 13A). The 2 sutures are tied outside the shoulder and the opposite limb of the PDS is pulled (C). This allows the medial limb of the suture anchor to be shuttled through the labral tissue. This limb can now be retrieved above the labral lesion and retrieved via anterosuperior lateral portal.
throscopic knot is then tied under direct arthroscopic visualization (FIGURES 13B AND 13C). The labrum is probed for stability and security of fixation (FIGURE 14A); a second suture anchor, if needed, can be employed using the same technique (FIGURE 14B). In cases of associated instability, where the SLAP lesion extends into the anterior labrum or a separate Bankart lesion is present, we use the exact same technique as described above at the location of the additional lesion.

Type IV lesions are complex and can pose a challenge when encountered intraoperatively. Our approach is that if the longitudinal tear in the biceps tendon is less than one third of the tendon diameter, then the fragment is excised. If the fragment is approximately one third of the diameter or greater, we repair the torn fragment to the major portion of the biceps tendon, followed by repair of the superior labrum at the biceps anchor, as described previously (FIGURES 15A AND 15B). A spinal needle or a suture-passing instrument with a sharp tip can be used to pierce the torn fragment, and the major portion of the biceps tendon, followed by suture passing and arthroscopic knot-tying.

**SLAP Lesions Associated With Rotator Cuff Tears**

In throwing athletes, it is not uncommon to encounter delaminated (split into layers), intratendinous, partial-thickness rotator cuff tears in conjunction with SLAP lesions. We generally debride tears less than 50% and repair those that are greater than 50%. We have adopted a technique of arthroscopic intratendinous repair for delaminated, articular-side, partial-thickness rotator cuff tears in overhead athletes using percutaneously placed mattress sutures. This technique accomplishes 3 essential goals: (1) restoration of the anatomy of the articular side of the rotator cuff, (2) repair of the delamination component of the tear, and (3) prevention of overconstraining the shoulder. The final effect is the placement of a suture that fixes the torn articular-side flap to the intact peripheral rotator cuff, reducing the articular-sided partial defect, and closing down the intrasubstance delamination. There is no bony fixation (FIGURES 16-18).

**POSTOPERATIVE REHABILITATION**

Postoperative rehabilitation following SLAP repair is determined by the type of SLAP lesion, the exact surgical procedure performed (debridement versus repair), and other concomitant pathology. Generally
The postoperative rehabilitation of a surgically repaired type IV SLAP lesion is similar to that of a type II repair, in that range-of-motion and exercise activities are progressed similarly. However, there are substantial differences regarding the progression of biceps activity based on the extent of bicipital involvement. In cases where the biceps is resected, biceps muscular contractions typically begin between 6 and 8 weeks post surgery. In cases where the biceps is repaired, no resisted biceps activities are allowed for at least 3 months following surgery. Light isotonic strengthening for elbow flexion is initiated between 12 and 16 weeks postoperatively and full resisted biceps activity is not initiated until between 17 and 20 weeks postoperatively. The progression to sports and related activities follows similar guidelines to those outlined for type II repair. A more detailed and comprehensive description of our postoperative guidelines is demonstrated schematically in the Appendix.

**OUTCOMES**

Several studies have documented successful surgical treatment of SLAP lesions. The majority of publications address results following surgical repair of type II SLAP lesions, because these are the most common type. In general, approximately 90% of patients demonstrate good or excellent results at the short to intermediate follow-up, but there is a paucity of long-term follow-up studies. Several recent publications have provided additional insight into our understanding of surgical results after SLAP repair. Kim et al. evaluated 34 patients at a mean of 33 months after surgical repair of type II SLAP lesions. While the overall results were good (94% satisfactory UCLA shoulder score, 91% return to preinjury shoulder function), significant differences were seen between patients who participated in different types of athletics. Specifically, throwing athletes had lower shoulder scores and a lower percentage of return to their preinjury level of shoulder function than patients who were not involved in overhead sports. Ide et al. evaluated 40 patients at a mean of 41 months after surgical repair of type II SLAP lesions. All subjects in this study were overhead athletes and, overall, results were favorable, with 90% good or excellent modified Rowe scores and 75% return to preinjury shoulder function. However, throwers without a specific traumatic injury had lower scores and a lower return to pre-injury function rate than throwers with a history of a specific traumatic event. These publications suggest that surgical repair of type II SLAP tears in overhead athletes with an overuse-related cause may be less successful than in other patients.

**SUMMARY**

SLAP lesions can be a source of pain and disability in patients, particularly during overhead activity. Diagnosing a SLAP lesion remains a challenge for clinicians treating shoulder disorders. Clinical examination to detect SLAP lesions can often be difficult because of the presence of concomitant pathology. A wide variety of physical examination “maneuvers” have been described to help diagnose the presence of SLAP lesions. A comprehensive approach, including a thorough history and physical examination, standard shoulder radiographs, and, often, MRI, will most likely allow the clinician to successfully diagnose this condition. Surgical intervention is often a successful option for the patient with a SLAP lesion who wishes to return to optimal function. Addressing concomitant shoulder disorders at the time of surgery is also crucial to ensure a successful outcome. Finally, a well-organized postoperative rehabilitation program is mandatory for optimal results, and the patient undergoing such surgery should be well aware of its importance.
REFERENCES


Patient begins program, as directed by physician, on first postoperative day

**Phase 1 (0-3 wk)**
- Sling immobilization, as directed by physician
- Codman’s/pendulum exercises
- Hand/wrist/forearm ROM exercises
- Gapping exercises
- FF plane of scapula PROM/AAROM (supine), limit to 90°
- Passive ER to neutral
- Passive elbow abduction to 30°
- Cryotherapy/modalities PRN

**Phase 2 (3-6 wk)**
- Discontinue sling, physician directed
- Continue FF plane of scapula PROM/AAROM (wand/pulleys), rate of progression based on patient’s tolerance
- ER PROM/AAROM to 30°
- Manual scapular stabilization exercise, side lying

Begin pain-free IR/ER isometrics in modified neutral
- No biceps strengthening
- Cryotherapy/modalities PRN

**Phase 3 (6-8 wk)**
- Progress PROM/AAROM
  - FF plane of scapula and abduction to 180°
  - ER to 90°
- Begin isotonic IR/ER strengthening in modified neutral
- Begin latissimus strengthening, below 90° elevation
- Begin upper body ergometer, below 90° elevation
- Begin humeral head stabilization exercises, if adequate strength and ROM exists

**Phase 4 (8-10 wk)**
- Continue aggressive scapula strengthening
- Advance strengthening for deltoid, biceps, triceps, and latissimus, as tolerated
- Begin PNF patterns
- Continue humeral head stabilization exercises

Advance IR/ER to elevated position in overhead athletes (must be pain free and have good proximal strength)
- Continue UBE for endurance training
- Begin general flexibility exercises

**Phase 5 (10-14 wk)**
- Continue full upper extremity strengthening
- Restore normal shoulder flexibility
- Begin activity-specific plyometric program
- Continue endurance training
- Type II repairs, begin gentle resisted biceps isotonic strengthening

**Phase 6 (14-24 wk)**
- Continue flexibility exercises
- Continue full strengthening program
- Begin return to interval throwing, physician-directed
- Type IV repairs, progress to isotonic biceps strengthening
- Continue endurance training

Abbreviations: AROM, active range of motion; ER, external rotation; FF, forward flexion; IR, internal rotation; PROM, passive range of motion; ROM, range of motion; UBE, upper body ergometer.