Advancements in shoulder arthroscopy have led to a better understanding of the anatomy and disorders of the superior labrum biceps tendon anchor complex and the role that lesions of the superior labrum anterior and posterior lesions play in pain and instability of the shoulder. Various injury mechanisms have been suggested and studied and it is likely that different mechanisms produce different types and areas of damage to the superior labrum. Classification systems have been proposed to describe the specific pathoanatomy of lesions of the superior labrum anterior and posterior lesions and to guide treatment. Presenting symptoms often are nonspecific and physical examination maneuvers have varying degrees of sensitivity and specificity making diagnosis challenging. Diagnostic ability is enhanced by the ultimate diagnostic test, arthroscopy. A clear appreciation for the various lesions and the potential resulting joint dysfunction is necessary to determine the appropriate treatment of this complex region of the shoulder. The current authors review the anatomy, classification, presentation, evaluation and treatment results of superior labrum anterior and posterior lesions, and includes novel evaluation methods and treatment guidelines useful in treating these lesions.

The use of shoulder arthroscopy in the diagnosis and treatment of shoulder disorders has lead to increased understanding of superior labrum anterior and posterior (SLAP) lesions. The current authors reviewed current knowledge particularly in the areas of classification, presentation, evaluation, treatment, and results. Also included are novel evaluation and treatment guidelines.

Andrews and coworkers described a superior labral lesion in a group of 73 throwing athletes, in which the anterosuperior labrum was pulled off the glenoid by traction from the biceps tendon. They thought that the traction occurred as the elbow was decelerated during the follow-through phase of throwing. In 1990, Snyder et al coined the term SLAP lesion in a retrospective review of 700 shoulder arthroscopies in which 27 SLAP lesions were identified and classified into a four-part scheme.

The awareness of SLAP lesions and their potential role in shoulder pain and disorders has increased rapidly during the past 10 years. Concomitant advancements in shoulder arthroscopy also have resulted in significant improvement in diagnosis and treatment of SLAP lesions. Yet, the pathoanatomy sometimes is more complex than previously described and treatment remains uncertain. The current authors will review classification, presentation, evaluation, treatment, and results, and will include novel
evaluation methods for SLAP lesions and guidelines that have aided in treatment.

Classification

Many authors use the four-part classification of Snyder and coworkers to describe SLAP lesions. Type I lesions have degeneration or fraying of the labrum without instability. Type II lesions are most common, accounting for more than 50% of SLAP lesions, and involve detachment of the superior labrum from the glenoid. A Type III lesion is characterized by a bucket-handle tear of the superior labrum with firm attachment of the remainder of the labrum. Type IV lesions also remain attached to the labrum but have an associated bucket-handle tear of the labrum that extends into the biceps tendon. Snyder et al developed this scheme from a retrospective evaluation of 700 shoulder arthroscopies in which only 27 patients had SLAP lesions. Therefore, it is not surprising that all SLAP lesions do not fall into the initial classification of Snyder et al.

Morgan and coworkers developed a secondary classification of Type II lesions from a retrospective review of 102 SLAP lesions. Three Type II lesions were described based on the anatomic location (anterior, posterior, or combined anterior and posterior.) Posterior and combined lesions occurred three times more frequently in throwing athletes and anterior lesions usually were seen in patients with trauma. Morgan et al think that patients with SLAP lesions with posterior components develop posterosuperior instability that leads to chronic superior instability and rotator cuff tears.

Maffet and coworkers retrospectively reviewed 84 superior labral lesions and found that 38% could not be classified according to a four-part scheme. Types V to VII were added to the four-part classification. A Type V lesion is an anteroinferior Bankart lesion that continues superiorly to include separation of the biceps tendon. A Type VI lesion included a biceps separation with an unstable flap tear of the labrum. Finally, a Type VII lesion is a superior labrum-biceps tendon separation that extended anteriorly beneath the middle glenohumeral ligament.

The current authors think that the classification of SLAP lesions can be simplified by noting whether the lesion would contribute to instability of either the biceps tendon anchor or the glenohumeral ligaments. Lesions producing significant labral defects at the site of attachment of critical capsuloligamentous structures generally require repair of these structures back to the bony glenoid rim. Lesions producing significant defects extending into the biceps tendon may require biceps tenotomy with or without tenodesis.

Presentation

Mechanism of Injury

Most patients with SLAP lesions present with nonspecific shoulder pain associated with activity. The complicating factor in the presentation is that the majority of SLAP lesions reported in the literature are associated with other shoulder disorders such as rotator cuff tears, acromioclavicular joint disorders, and instability. In a review of 140 lesions, which is the largest review in the literature, only 28% of the SLAP lesions were isolated.

Traction and compression injuries are thought to be the source of the majority of SLAP lesions. Most compression injuries are traumatic in nature, whereas traction injuries occur most often in overhead athletes. Bey and coworkers showed, in a biomechanical model, that traction on the biceps tendon can create Type II SLAP lesions and that inferior subluxation of the shoulder may lead to the creation of more unstable lesions. Seven of the eight shoulders with inferior subluxation led to lesions but only two of the shoulders that were reduced had unstable lesions develop. Several authors described the peel-back mechanism for the creation and extension of Type II lesions. They stated that when the shoulder is in the abducted and external rotated position, the biceps assumes a more vertical and posterior angle, which produces a twist at the base of the
biceps. This transmits a force to the posteroinferior labrum, which causes it to rotate medially and peel off the glenoid.

Pathoanatomy
The superior labrum-biceps-glenohumeral ligament complex is attached to the superior and peripheral portions of the glenoid rim. The labrum is a fibrocartilaginous tissue that is anatomically and histologically distinct from the shoulder capsule. The superior and anterosuperior portions of the labrum are attached more loosely to the glenoid than the inferior portion of the labrum. Although the inferior labrum is a rounded, elevated structure closely adherent to the glenoid, the superior labrum is attached less firmly, more mobile, and meniscoid in appearance. The labrum adds to the depth of the glenoid and increases the diameters of the glenoid surface to 75% of the humeral head vertically and 57% in the transverse direction.

The overall anatomy of the shoulder has been described by Cooper and coworkers in an exhaustive anatomic study of 17 cadavers. Steinbeck and coworkers additionally delineated the intraarticular anatomy of the shoulder. The current authors think that there is a strong relationship and interplay between the superior labrum, biceps tendon, and superior and middle glenohumeral ligaments. In a study of 24 shoulders from cadavers, the biceps tendon was found to blend with the superior labrum 75% of the time, with only a small portion inserting on the supraglenoid tubercle. In a study of 100 shoulders from cadavers, Vangsness et al found that approximately 50% of the biceps arose from the supraglenoid tubercle and the remainder arose directly from the superior labrum. In more than ½ of the specimens, the main labral origin was posterior but a small percentage (8%) had attachment mainly anterior to the biceps tendon.

The anatomic association between the superior labrum and glenohumeral ligaments has been well documented. In an anatomic study of 104 cadavers by Steinbeck and coworkers, all of the superior glenohumeral ligaments originated from the superior labrum, whereas approximately 30% of the middle glenohumeral ligaments also arose from the anterosuperior labrum. In 17% of the cases, the superior and middle glenohumeral ligaments originated together from the 1 o’clock position of the labrum. Some authors have observed that the anteroinferior glenohumeral ligaments can insert directly onto the superior labrum.

Prior studies indicated there was a relationship between the superior labrum-biceps-glenohumeral ligament complex and shoulder instability. Rodosky and coworkers determined that the superior labrum and biceps tendon contributed to anterior shoulder stability by increasing resistance to torsional forces in the abducted and externally rotated position. They also found that SLAP lesions created a significant increase in the strain in the inferior glenohumeral ligament. Kumar and coworkers showed that the long head of the biceps acted to prevent humeral head migration when the arm was at the side. Itoi and coworkers concluded that the long head and the short head of the biceps contribute to the anterior stability of the shoulder in the abducted and externally rotated positions and showed that their role as anterior stabilizers only increased as shoulder instability worsened. In a biomechanical model creating tension through the biceps tendon, Bey and coworkers showed that the incidence of SLAP lesions was increased if the shoulder was unstable. Finally, Pagnani and coworkers found that complete lesions of the superior labrum resulted in significant increases in anteroposterior and superoinferior glenohumeral translation.

The current authors think and the literature supports the concept that the superior labrum, long head of the biceps, and the superior and middle glenohumeral ligaments work together to provide shoulder stability. Currently, the exact contribution of each structure is not completely clear but the relationship between superior labral lesions and instability has been well documented.
a132 reported that patients with SLAP lesions with a posterior component have posterosuperior instability develop, which results in undersurface rotator cuff tears. The incidence of rotator cuff disorders, which includes incomplete undersurface tears and complete tears, has been reported between 10% and 48% of the time to occur with a SLAP lesion.1,10,18,28,32,39,44,45 The question remains, however, whether the SLAP lesion leads to shoulder instability or whether subtle shoulder instability leads to the SLAP lesion and then increased shoulder instability.

Evaluation

Physical Examination

The studies regarding SLAP lesions generally indicate that the diagnosis of SLAP lesions is difficult.29 The current authors have found that the diagnosis of SLAP lesions in isolated cases is more straightforward but, as the current authors have described, it is much more common to have associated disorders that complicate and cloud clinical findings. Numerous authors describe the use of Hawkins’ test, Neer’s test, Speed’s test, Jobe’s relocation test, and Yergason’s tests and generally report that they are nonspecific.10,15,18,28,29,32,44,45 As a result, numerous authors have developed specific tests that they reported to be sensitive and specific in the diagnosis of SLAP lesions.5,22,23,26,31,33

Mimori and coworkers30 developed the pain provocation test and reported a 100% sensitivity and 90% specificity regarding Type II SLAP lesions. However, ½ of the 22 patients in the study who were thought to have a Type II SLAP lesion had their definitive diagnosis made by magnetic resonance imaging (MRI) only. Liu and coworkers27 described the crank test in a group of 62 patients in which 30 had SLAP lesions; however, the type of tear was unclear. The crank test was reported to have a sensitivity of 91%, a specificity of 93%, a positive predictive value of 94%, and a negative predictive value of 90%. Kibler22 developed the anterior slide test and described its use in a study with 88 arthroscopically-confirmed SLAP lesions, although the lesions were not classified. He found that the anterior slide test had a sensitivity of 78.4% and a specificity of 91.5%.

Kim and coworkers23 described the biceps load test in patients with instability and SLAP lesions. Although only 10 patients had Type II lesions they determined that the test had 90.9% sensitivity and a specificity of 96.9% with a positive predictive value of 83% and a negative predictive value of 98%. Finally, O’Brien and coworkers33 described the active compression test that they thought could be used to diagnose SLAP lesions and acromioclavicular joint disorders. In their study, which included 53 SLAP lesions that were repaired but not classified, the active compression test had a sensitivity of 100%, a specificity of 98.5%, a positive predictive value of 94.6%, and a negative predictive value of 100%.

Morgan and coworkers32 thought that the Speed and O’Brien tests were helpful in the diagnosis of anterior lesions with sensitivities of 100% and 88% and specificities of 70% and 42%, respectively. The Jobe relocation test was particularly useful for posterior lesions with 85% sensitivity and 68% specificity.

The current authors recently completed a prospective clinical study to determine the most effective provocative maneuver with which to diagnose SLAP lesions. The following tests were included: active compression, anterior slide, pain provocation, crank, relocation, Hawkins’, Neer, Speed’s, and Yergason’s tests. A series of 132 consecutive patients scheduled to have diagnostic arthroscopy were examined preoperatively with each of the aforementioned tests. The final diagnosis in each case was made arthroscopically. Forty patients had either a Type I (17 patients) or a Type II (23 patients) SLAP lesion. The remainder of the diagnoses included complete rotator cuff tears, partial rotator cuff tears, impingement, and instability. The two most sensitive tests for Type II SLAP lesions were the active compression (65.2%) and Hawkins’ tests (65.2%) followed by Speed’s (47.8%), Neer (47.8%), and the relocation test (43.5%) (Table 1). None of the remaining tests had a sensitivity greater than 17.4% for Type II lesions. When Type I and Type II lesions were
combined the sensitivities were similar for all of the provocative tests (Table 2). However, none of the sensitive tests were specific for either Type II lesions alone or when Type I and II lesions were combined. There were no significant differences between the tests when positive and negative predictive values were assessed.

The current authors’ results may have differed from those previously reported because unbiased investigators evaluated numerous tests in a random population and arthroscopy was used as the final diagnostic tool in all patients. These results indicated that no test is sensitive and specific for diagnosis of SLAP lesions and diagnostic arthroscopy remains the best means to definitively diagnose SLAP lesions. Yet the active compression test although not as sensitive or specific as described by O’Brien and coworkers,33 may be the most useful test. The mechanism of this test was reported to be that when the shoulder was in the internally rotated position, the superior labrum was tensioned from medial and inferior displacement of the biceps tendon. Tension then could lead to mechanical symptoms by displacing an unstable superior labrum into the joint. O’Brien and coworkers33 also reported that the active compression test was an excellent test for evaluation of the incongruous or degenerative acromioclavicular joint. The active compression test was 100% sensitive and 96.6% specific for detecting acromioclavicular joint disorders. They thought the greater tuberosity “locked and loaded”33 an unstable or degenerative acromioclavicular joint by elevating the lateral aspect of the acromion in the internally rotated position.

To better understand the mechanism, the current authors did a study to describe the intraarticular and extraarticular anatomic relationships during the active compression test. Four matched pairs of fresh-frozen shoulders from cadavers were positioned statically in the internally or externally rotated positions of the active compression test. The shoulders then were embedded in polyurethane and sequentially sectioned at 3-mm intervals in the axial and coronal planes using an industrial planer. Each section was reviewed independently to determine anatomic contact areas.

In the axial plane, shoulders in the internally rotated position had consistent contact between the lesser tuberosity and subscapularis tendon and the superior aspect of the glenoid and the labrum (Fig 1A–B). In the externally rotated position, there was no contact between the superior structures of the shoulder (Fig 1C–D). The lesser tuberosity and subscapularis tendon likely displace an unstable or torn superior labrum, leading to a positive clinical result in the internally rotated posi-

<table>
<thead>
<tr>
<th>Test</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
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<tbody>
<tr>
<td>Yergason’s test</td>
<td>92.7*</td>
<td>13.0</td>
</tr>
<tr>
<td>Pain provocation test</td>
<td>89.9*</td>
<td>17.4</td>
</tr>
<tr>
<td>Anterior slide test</td>
<td>83.5</td>
<td>13.0</td>
</tr>
<tr>
<td>Crank test</td>
<td>82.6</td>
<td>8.7</td>
</tr>
<tr>
<td>Speed’s test</td>
<td>67.9</td>
<td>47.8*</td>
</tr>
<tr>
<td>Relocation test</td>
<td>51.4</td>
<td>43.5*</td>
</tr>
<tr>
<td>Neer test</td>
<td>51.4</td>
<td>47.8*</td>
</tr>
<tr>
<td>Active compression test</td>
<td>48.6</td>
<td>65.2*</td>
</tr>
<tr>
<td>Hawkins’s test</td>
<td>30.3</td>
<td>65.2*</td>
</tr>
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* = p < .05

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**TABLE 1. The Sensitivity and Specificity of Nine Provocative Tests for Type II SLAP Lesions**

<table>
<thead>
<tr>
<th>Test</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
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<tr>
<td>Yergason’s test</td>
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</tr>
<tr>
<td>Pain provocation test</td>
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<td>Relocation test</td>
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<tr>
<td>Active compression test</td>
<td>50</td>
<td>62.5*</td>
</tr>
<tr>
<td>Hawkins’s test</td>
<td>30.4</td>
<td>67.5*</td>
</tr>
</tbody>
</table>

* = p < .05
Fig 1 A–F. (A) A photograph of a 3-mm section and (B) a schematic drawing show the active compression test in the axial plane with the shoulder in internal rotation. (C) A photograph of a 3-mm section and (D) a schematic drawing show the active compression test in the axial plane with the shoulder in external rotation. (E) A photograph of a 3-mm section and (F) a schematic drawing show the active compression test in the coronal plane with the shoulder in internal rotation. AC = acromioclavicular (continues)
tion. However, when the shoulder is rotated externally, contact from the lesser tuberosity and subscapularis tendon is relieved and the clinical result is a negative test.

In the coronal plane, the internally rotated specimens revealed contact between the supraspinatus tendon and the lateral aspect of the acromion (Fig 1E–F). There was no contact between the supraspinatus tendon and the acromion when the shoulders were rotated externally in the coronal plane (Fig 1G–H). In the case of a Type II or Type III acromion, one would expect periacromial pain in the internally rotated position. The pain should improve in the externally rotated position because no contact was seen between the tendon and the acromion in the externally rotated position. This provides an anatomic explanation for the clinical results of the active compression test. It can be used to assist in diagnosis of SLAP lesions and impingement including rotator cuff disorders.

**Radiographic Analysis**

Short of arthroscopic evaluation, the clinical history and physical examination are the best indicators of the presence of a SLAP lesion but contrast magnetic resonance imaging (MRI) can improve the diagnostic acumen of the clinician. Numerous studies have been done in which the use of noncontrast and contrast MRI was used to diagnose superior labral lesions of the shoulder. Retrospective study was used to ascertain the effectiveness of noncontrast MRI in the diagnosis of SLAP lesions. Smith and coworkers reviewed six MRI scans from shoulders with SLAP lesions confirmed at arthroscopy and found only one to have been read positive. Even with retrospective review, only four of six could be labeled positively torn. All six had increased signal intensity in the superior labrum. Monu and coworkers similarly reviewed eight patients with proven SLAP lesions and found that noncontrast MRI of the superior labrum was abnormal on all coronal images, 88% of axial images, and 50% of sagittal images. These studies are problematic in that they are purely retrospective and therefore give little information to the true accuracy of noncontrast MRI in the diagnosis of SLAP lesions.

Karzel and Snyder compared noncontrast MRI with contrast MRI in seven lesions and found that four were diagnosed correctly with contrast MRI whereas all seven were missed with the noncontrast MRI. They also reported false positive results in three patients who had contrast MRI scans. They thought that the ad-
dition of contrast material to standard MRI could aid in diagnosis of SLAP lesions. Recently, Bencardino and coworkers found MRI arthrography to have a sensitivity of 89%, specificity of 91%, and accuracy of 90% in the diagnosis of 19 SLAP lesions in 52 patients. Furthermore, the MRI classification was correct in 13 of 17 cases. Chan and coworkers added arm traction in a cadaver model and found that this improved the diagnostic accuracy of contrast MRI, most likely by displacing the unstable superior labrum.

The current authors use MRI arthrography as an adjunctive tool to clinical history and physical examination. Some studies support that the addition of contrast improves the accuracy of MRI in the diagnosis of unstable superior labral lesions. One must be aware, however, that false positive results occur. In a study on cadavers by Smith and coworkers, 73% of the 26 shoulders had a sublabral recess that can be the source of false positive MRI arthrography results. Furthermore, anatomic variants, such as the cordlike middle glenohumeral ligament, or Buford complex, can complicate results.

**Treatment**

The treatment of SLAP lesions has evolved significantly since first described. At the time SLAP lesions first became recognized, most were treated by either debridement alone or removal if the SLAP lesion was unstable. Cordasco and coworkers reviewed 52 cases of labral debridement in which there were 27 SLAP lesions. They found that with debridement alone, 78% of patients had good results at 1 year but this decreased to 63% at the second year. Seventy percent of the patients with SLAP lesions had shoulder instability on physical examination and only 44% returned to prior athletic levels. Similarly, Altchek and coworkers debrided 40 anterosuperior labral lesions and found that only 7% of patients had symptomatic relief at 2 years. Segmuller and coworkers repaired 17 unstable SLAP lesions with the Suretac device and 83.3% of patients had good results at 17 months but only 53% returned to preinjury levels of activity. Field and Savoie prospectively reviewed 20 unstable SLAP lesions that were repaired and found good results in all patients at 21 months. All of the patients in that study returned to their preinjury activity level but only six were athletes. Yoneda and coworkers arthroscopically stapled 10 unstable SLAP lesions and had 80% good results at 2 years. Second look arthroscopy at an average of 4 months revealed that all of the lesions had healed solidly to bone. Finally, Resch and coworkers reviewed 18 cases of unstable SLAP lesions in which 14 were repaired and four were debrided only. Five lesions were repaired with an arthroscopic screw and nine were repaired with a Suretac. The mean follow-up was only 18 months and revealed that all of the patients who had lesions repaired, eight patients had excellent results, four had improved results, and two patients had failed results. Only one patient with debrided lesions had improvement in symptoms. Morgan and coworkers described preliminary 1-year results on 102 repaired Type II lesions in pitchers with 83% excellent, 14% good, and 3% fair results. All of the 37 pitchers returned to pitching and 83% thought they returned to the same level.

Although the current authors do not think that Snyder’s four-part classification is completely inclusive, treatment recommendations are described according to this scheme because most orthopaedists are familiar with it. Type I lesions by definition are stable and only should require gentle debridement of frayed labral tissue. Type II lesions, or any lesion determined to be unstable, should be repaired using either arthroscopic sutures or tacks (Fig 2). The suction test can greatly assist with the determination of whether the superior labrum is unstable. In this test, the distended joint is suctioned and an unstable superior labrum is pulled away from the superior glenoid rim. Various methods to repair the superior labrum have been described in the literature.

Type III lesions only may require debridement of the damaged portion of the labrum involved in the bucket handle tear. Type IV le-
sions are characterized by a bucket-handle tear of the superior labrum that extends into the biceps tendon. The treatment algorithm for these lesions is determined by the extent of injury to the biceps tendon because the remainder of the superior labrum is attached by definition, to the glenoid. The biceps can be debridged if less than 40% is involved and either should be repaired or tenodesed if greater than 40% to 50% is involved. In the current authors’ experience, however, bucket-handle tears of the superior labrum (Type III and Type IV) often are superimposed on a Type II avulsion of the remaining labral tissue. If this is observed, and the labral remnant is adequate, repair as described for a Type II lesion should be done.

Gartsman and Hammerman stated that one must be aware that a normal superior labrum is not always well attached to the superior glenoid. They reported that if the glenoid underlying the superior labrum is covered with smooth cartilage and the labrum and glenoid show no outward signs of trauma, the superior labrum should be considered a normal variant.

**Fig 2 A–D.** Sequential repair of the superior labrum with suture anchor is shown. (A) Posterior to anterior arthroscopic view shows superior labral avulsion from the glenoid rim. (B) The suture anchor is shown in place in the anterosuperior quadrant. The anterior limb of the suture is shown through rotator cuff interval cannula and the posterior limb of the suture is shown through superolateral periacromial portal. (C) The suture passer with prolene loop penetrating the anterosuperior labrum is shown. The prolene loop is used to pull the posterior suture limb through the labrum. (D) The completed repair with simple arthroscopic knots is shown.
The current authors have observed a strong association between the presence of paralabral or spinoglenoid notch cysts and tears of the superior labrum. Therefore, when such cysts are found on diagnostic imaging studies, the superior labrum should be evaluated carefully for the presence of a tear. Successful arthroscopic cyst decompression has been reported and in the current authors’ experience, repair of the torn labrum results in resolution of the cyst.

Shoulder surgeons need to be aware of the existence of superior labral lesions and the normal anatomy of the superior labrum-biceps-glenohumeral complex when treating patients with shoulder pain. Hopefully, the next decade will provide as much growth in the understanding of these lesions as has the past. The authors think that the superior labrum works as a complex with the long head of the biceps and the glenohumeral ligaments to provide stability to the anterosuperior shoulder. The continuum of superior labral disorders and instability is a result of the interplay between the superior labrum, biceps tendon, and superior and middle glenohumeral ligaments.

References


