SLAP Lesions: An Update on Recognition and Treatment

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Synopsis

Superior labral tears (SLAP lesions) can pose a significant challenge to orthopaedic surgeons and rehabilitation specialists alike. Although advancement in arthroscopic techniques has enhanced arthroscopic repair of SLAP lesions, the clinical diagnosis of SLAP lesions can still be difficult. There is a variety of etiologic factors associated with SLAP lesions and a thorough clinical evaluation is crucial to make the diagnosis. Concomitant injury to the capsular-labral complex or rotator cuff is not uncommon and can further confuse the clinical presentation. The purpose of this paper is to review the pathomechanics, diagnosis, and treatment of SLAP lesions. We will specifically review some of the physical examination tests that are used to diagnose SLAP lesions and report on our technique of arthroscopic repair. Additionally, we will discuss the operative management of associated intraarticular pathology and finally, we will briefly discuss our postoperative rehabilitation guidelines.

Introduction

Superior labral lesions are a common occurrence among 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.[1] 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.[28] Although both authors reported on similar lesions, the etiology remained unclear. Andrews proposed that the biceps tendon acted to "pull off" the labrum whereas the most common mechanism of injury in the Synder 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 the senior author's (DWA) 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.[1] Mileski and Snyder reported that 29% of their patients with SLAP lesions exhibited partial thickness 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.[18]

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 tendon during the arm deceleration and follow-through phases of throwing. The authors subsequently applied electrical stimulation to the bicep during arthroscopy and noted that contraction of the biceps caused the biceps anchor to separate from the glenoid.[2]

Burkhart and Morgan have hypothesized a "peel-back" mechanism that can 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.[4] 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.[24] Additional authors have also demonstrated 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.[11,30]

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).[26] 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 seen during the peel-back mechanism. These results support the theory of "peel back" as the predominant mechanism, but in reality it is most likely a combination of mechanisms that 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.[23] They also found that the presence of a simulated SLAP lesion resulted in a 6mm 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.[13] 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 with leads to instability.

Classification

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).[28] 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 consists 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 which are anteroinferior Bankart-

type labral lesions in continuity with SLAP lesions, type VI lesions which consist of biceps

tendon separation with an unstable flap tear of the labrum, and type VII lesions which consist of

an extension of the superior-labrum biceps tendon separation to beneath the middle

glenohumeral ligament.[16] 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 that can ultimately lead to pseudolaxity and partial

thickness articular sided rotator cuff tears that are lesion specific.[19] (ie posterior SLAP lesions

cause posterior cuff tears, anterior SLAP lesions casue 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 when present. 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 in 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 90º 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 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.[22] The shoulder is positioned in 20º of adduction and 90º of forward

elevation. 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 joint (AC). 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.[29] We typically perform this maneuver when assessing for labral pathology and find pain inside the shoulder when testing with the forearm pronated and 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 position.[28] The glenohumeral joint is then compressed and the humerus is rotated in an attempt to trap the labrum within the joint (Figure 3). The presence of an uncomfortable clunk may indicate a labral tear. The arm can be abducted with an anterior directed force or adducted with a posterior directed force to assess for anterior and posterior labral lesions respectively.

Myers et al recently described a test termed the resisted supination external rotation test.[20] The test is performed in the supine position with the shoulder in 90º of abduction, the elbow in 70º of flexion, and neutral forearm rotation. The shoulder is then passively externally rotated while the examiner resists against supination (Figure 4). Myers noted that this test simulates the peel-back mechanism of SLAP lesions. A preliminary study of 40 patients demonstrated a superior 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 have described a similar test that they have termed the "pronated load test".[31] The patient is placed supine and the shoulder is abducted to 90º and externally rotated. The

forearm is kept in a fully pronated position to increase tension on the biceps tendon and the patient is instructed to perform an isometric biceps contraction to simulate the peel-back mechanism (Figure 5). The authors have found this maneuver to cause pain and discomfort in patients with SLAP lesions.

Several authors have evaluated the ability of multiple provocative tests to predict the presence of labral pathology.[17,29] 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). 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.[9,15] Therefore, they recommend magnetic resonance arthrogram with an intraarticular 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.[3]

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 (Figure 6). 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.[8] 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.

Non Operative 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, however, be 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 which is focused on restoring normal shoulder motion. Strengthening of the shoulder girdle musculature is also crucial in order 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 whether or not GIRD is a risk factor for SLAP lesions, restoring shoulder internal rotation may improve pathomechanics which could possibly reduce some of the patients' symptoms. The patient is eventually advanced to a strengthening phase of the physical therapy program which includes trunk and core strengthening as well as rotator cuff and scapular muscles strengthening. 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 lesion 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 30º 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 boney bed at the superior rim of the glenoid via the anterosuperior lateral portal (Figure 10). 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 11). A No. 1 polydioxanone (PDS) suture (Ethicon, Somerville, NJ) is manually advanced through the spinal needle into arthroscopic view (Figure 12). An arthroscopic retriever is then used to retrieve the free end of the suture and bring it out via the standard anterior portal (Figure 13).

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 (Figures 14 and 15). The more medial limb of 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 16).

The 2 sutures are tied outside the shoulder and the opposite limb of the PDS suture is pulled. The medial limb of the suture from the suture anchor is then shuttled through the superior labral tissue and then retrieved into the anterosuperior lateral portal. A standard arthroscopic knot is then tied under direct arthroscopic visualization (Figures 17 and 18). The labrum is probed for stability and security of fixation (Figure 19); a second suture anchor, if needed, can be employed using the same technique (Figure 20). 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 21 and 22). 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 23-25).

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 speaking, we immobilize our patients for a short period followed by emphasis on restoring motion and, lastly, initiate strengthening exercises. The following paragraph describes our postoperative guidelines for patients who underwent SLAP repair with no associated pathology.

For the first 2-3 weeks postoperatively, patients are immobilized in internal rotation in a sling. During that time period, we prohibit any external rotation and limit abduction to 60º. Pendulum and elbow range of motion exercise are encouraged. At 4 weeks, shoulder motion is increased using active-assisted and passive techniques. At this point, the sling is discontinued but we still limit external rotation to 30º to minimize strain on the labrum through the peel-back mechanism. Over the ensuing 4 weeks, performance of internal rotation and external rotation range of motion activities is progressed to 90º of shoulder abduction. At approximately 8 weeks, we initiate resistance exercises with a focus on scapular strengthening provided adequate motion has been achieved (approximately 115º to 120º of shoulder external rotation). No resisted biceps activity (elbow flexion and forearm supination) is allowed for the first 2 months to protect the

healing of the biceps anchor. After 4 months, a sport directed throwing program is initiated in overhead athletes and contact sports are generally allowed after 6 months.

The postoperative rehabilitation of surgically repair 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 activites 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 Table.

Outcomes

Several studies have documented successful surgical treatment of SLAP lesions.[6,7,21,25,] The majority of publications address results following surgical repair of type II SLAP lesions because it is 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[14] 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[10]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 excellened 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 preinjury 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 to be a challenge for clinicians who treat 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 post-operative rehabilitation

program is mandatory for optimal results, and the patient undergoing such surgery should be

well aware of its importance.

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Figures

SLAP lesions (Type I-IV)
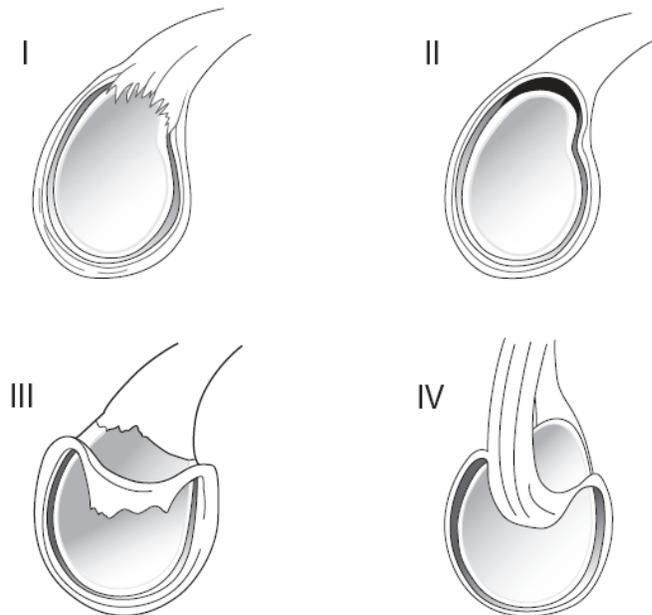
Figure 1

Figure 2

Figure 3

Figure 4

Figure 5
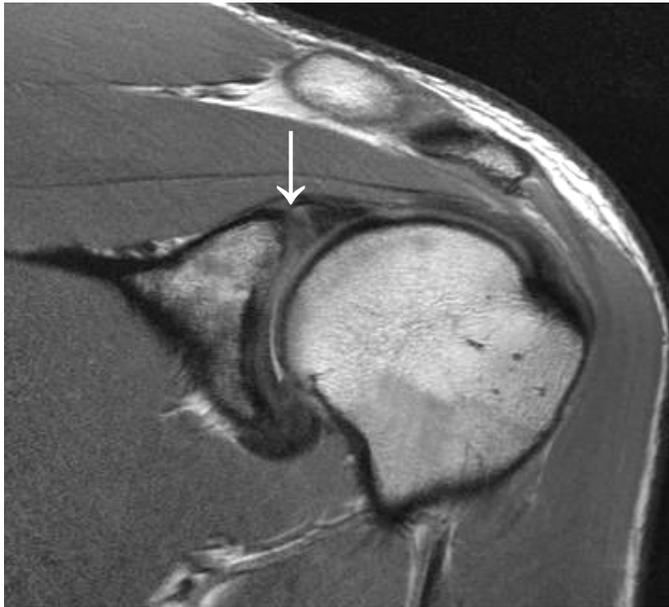
Figure 6

Figure 7

Figure 8

Figure 9
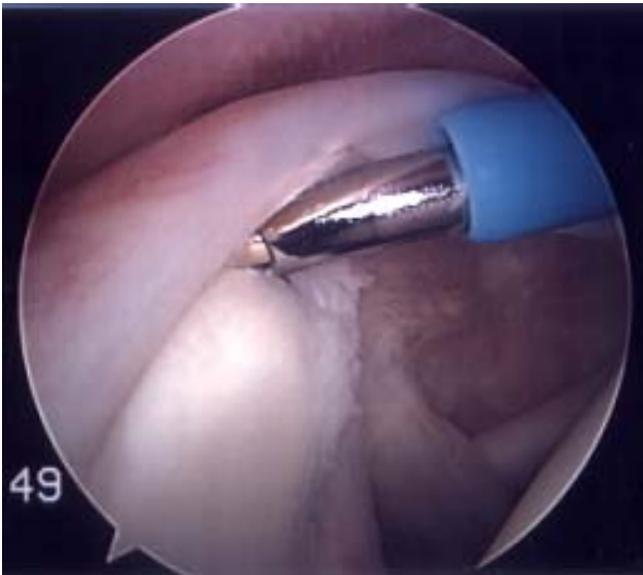
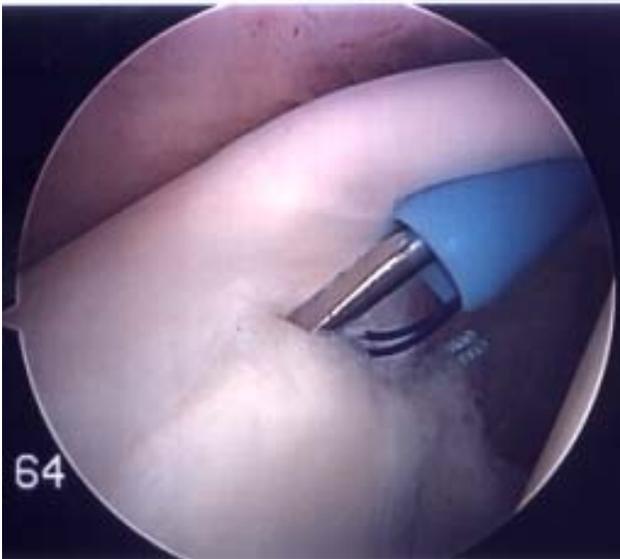

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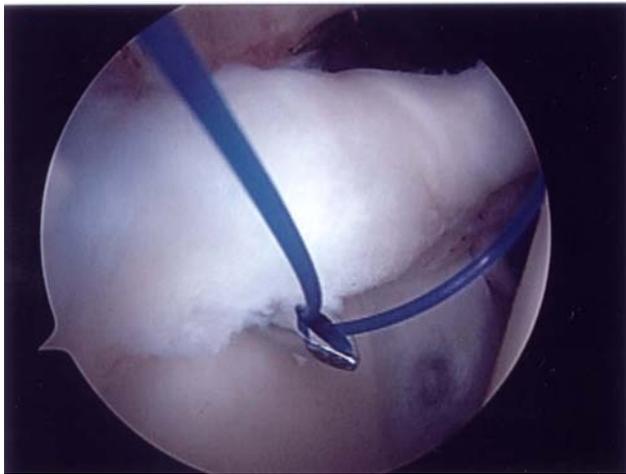
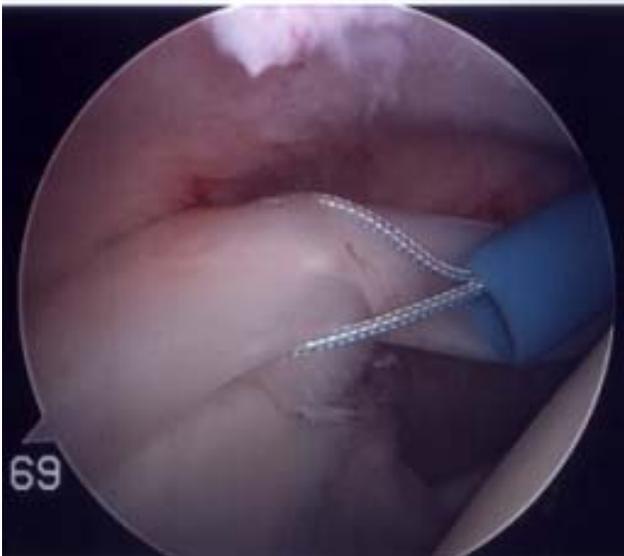Figure 12
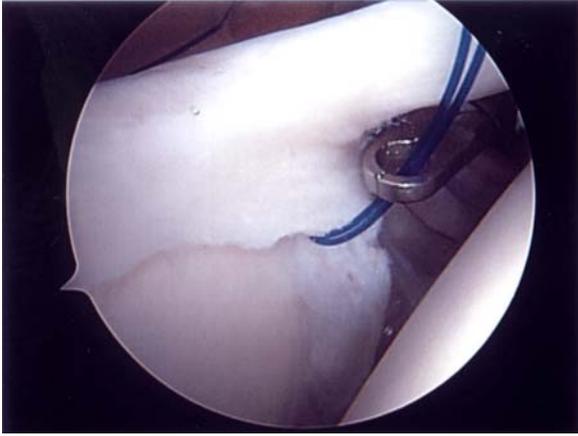
Figure 13

Figure 14

Figure 15

Figure 16

Figure 17

Figure 18

Figure 19

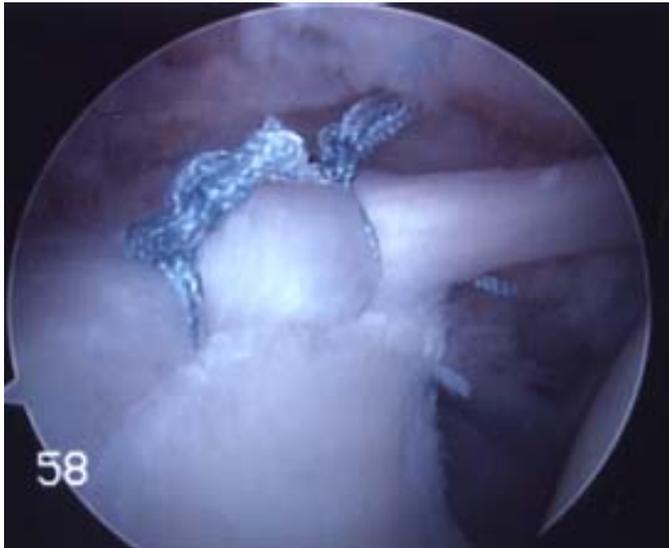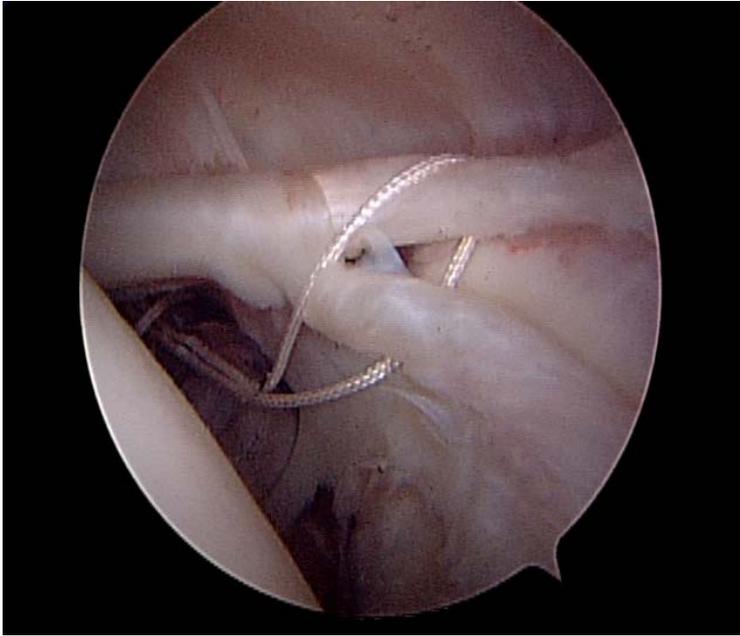

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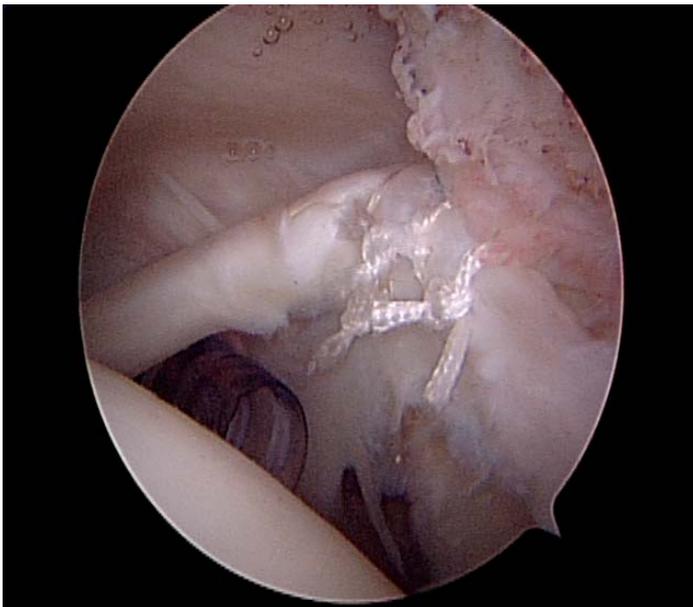
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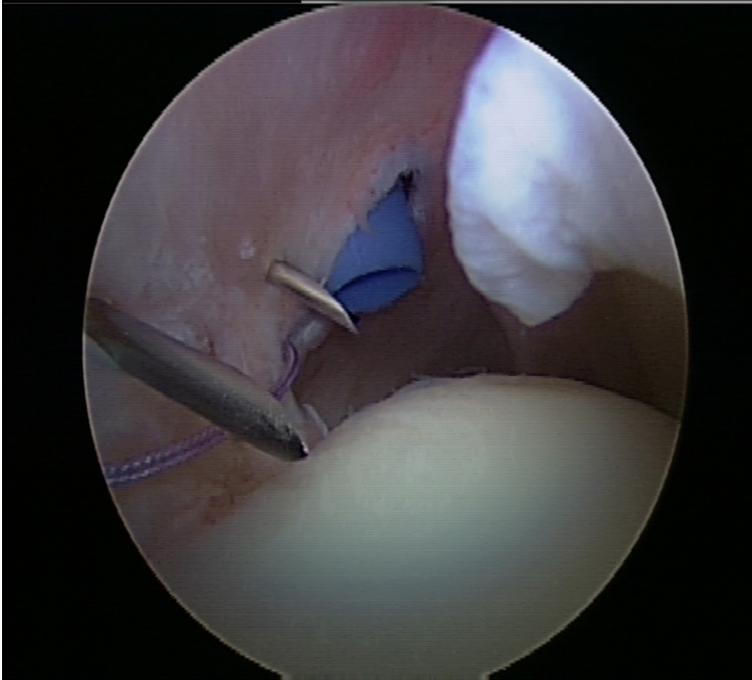
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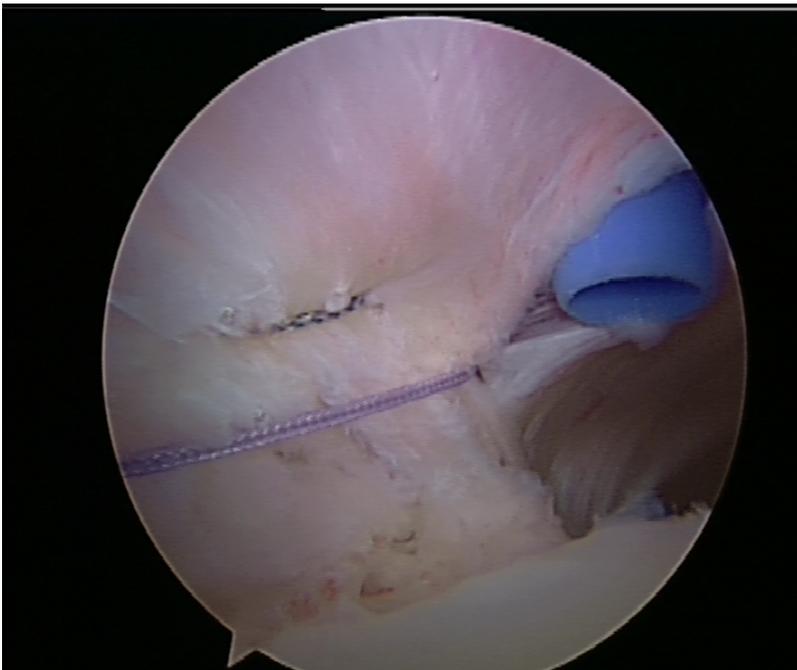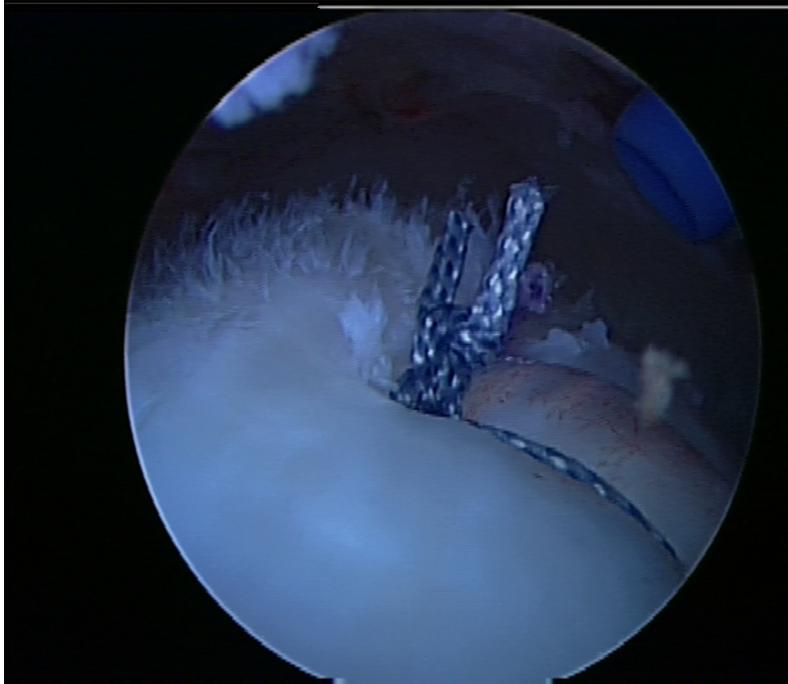

Figure 24

Figure 25

Table

Phase I (0-3weeks)**

Sling immobilization-MD directed

Codmans/Pendulum exercises

Hand/Wrist/Elbow ROM exercises

Gripping exercises

FF plane of scapula-PROM/AAROM (supine)-limit to 90 degrees

Passive ER to neutral

Passive elbow abduction to 30 degrees

Cryotherapy/Modalities PRN

** Patient begins above program as directed by MD starting on first post-op day

Phase II (3-6 weeks)

Discontinue sling-MD directed

Continue FF plane of scapula-PROM/AAROM (wand/pulleys)-rate of progression based on patient's tolerance

ER-PROM/AAROM to 30 degrees

Manual scapular stabilization exercise-sidelying

Begin pain-free IR/ER isometrics in modified neutral

No biceps strengthening

Cryotherapy/Modalities PRN

Phase III (6-8 weeks)

Progress PROM/AAROM

    -FF plane of scapula and abduction to 180º

    -ER to 90º

 Begin isotonic IR/ER strengthening in modified neutral

Begin latissimus strengthening-below 90 degree elevation

Begin upper body ergometer below 90 degrees elevation

Begin humeral head stabilization exercises (if adequate strength and ROM exists)

Phase IV (8-10 weeks)

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 V (10-14 weeks)

Continue full upper extremity strengthening

Restore normal shoulder flexibility

Begin activity specific polymeric program

Continue endurance training

Type II repairs-begin gentle resisted biceps isotonic strengthening

Phase VI (14-24 weeks)

Continue flexibility exercises

Continue full strengthening program

Begin return to interval throwing-MD directed

Type IV repairs-progress to isotonic biceps strengthening

Continue endurance training

Figure Legend

Figure 1: Illustration of Type I-IV SLAP lesions as described by Snyder.[21] Type I represents a

frayed or degenerative labrum with attachment of the labrum to the glenoid. Type II represents

detachment of the superior labrum and biceps from the glenoid rim. Type III represents a

bucket-handle tear of the labrum with an intact biceps anchor. Finally, type IV represents a

bucket handle tear of the labrum that extends into the biceps tendon.

Figure 2: O'Briens test (Active-compression test). Patients shoulder is positioned at 90° of

elevation, approximately 30° of adduction and full internal rotation. Resistance against elevation

is applied by examiner. A test is positive for labral involvement when pain is elicited with the forearm in pronation and relieved when the forearm is supinated.

Figure 3: Compression-rotation test. The examiner imparts a compressive force from the elbow through the long axis of the humerus as the shoulder is rotated in an attempt to grind the labrum and elicit pain.

Figure 4: The resisted supination external-rotation test. The patient's shoulder is abducted to approximately 90°, elbow flexed, and forearm in neutral. The examiner then simultaneously resists forearm supination while externally rotating the shoulder in an attempt to peel back the labrum.

Figure 5: A relatively new test for SLAP lesions: pronated load test. The patient's 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 peform an isometric biceps contraction in an attempt to peel back the labrum.

Figure 6: Coronal view of a non contrast MRI demonstrating a type II SLAP lesion. (red arrow)

Figure 7: MR arthrography in abduction and external roation (ABER) demonstrating an articular partial thickness rotator cuff tear.(red arrow) We prefer this view in suspected cases of associated cuff pathology because it allows for better visualization of the articular surface, labrum, and is helpful in detecting intratendinous lesions.

Figure 8: Arthrocopic view of a right shoulder demonstrating a type II SLAP lesion. A probe has entered the joint via the standard anterior interval portal and is used to confirm the diagnosis.

Figure 9: Once the SLAP lesion has been confirmed, a second anterior lateral portal(blue cannula) is created which will be the working portal for repairing the SLAP lesion. Establishing the proper position of this portal is crucial because the angle at which the suture anchor is inserted is completely dependent upon the location of the portal. We avoid penetrating the rotator cuff in creating this portal especially in throwing athletes.

Figures 10 and 11: An arthroscopic shaver is used to prepare the bed of the SLAP lesion and then an 18-gauge spinal needle is passed percutaneously through the superior labrum via the region of the portal of Neviaser.

Figures 12 and 13: A PDS suture is then manually advanced through the spinal needle and retrieved via the standard anterior interval portal.

Figures 14 and 15: A hole is drilled into the bed of the SLAP lesion via the anterior lateral portal and a suture anchor is then inserted.

Figures 16, 17, and 18: The medial limb of the suture anchor is tied to the inferior limb of the PDS suture from the anterosuperior lateral portal. The 2 sutures are tied outside the shoulder and the opposite limb of the PDS is pulled. 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.

Figures 19 and 20: After a standard arthroscopic knot is tied the labrum is probed for stability and security of fixation. For larger labral lesions this process can be repeated using the same technique.

Figure 21 and 22: Type IV lesions with significant biceps involvement can be repaired. We repair the torn fragment to the major portion of the biceps tendon first, followed by repair of the superior labrum at the biceps anchor.

Figure 23: With the tear reduced, 2 spinal needles are passed in successive fashion through the full thickness of the rotator cuff. A PDS suture is then advanced through each spinal needle. A grasper is used to shuttle these sutures out of the anterior cannula and the sutures are then tied to each other, pulled into the joint and the knot is then shuttled externally by pulling on one of the sutures.

Figure 24: For larger tears, we use multiple mattress sutures. A second set of spinal needles are passed superiorly and PDS sutures are advanced and shuttled in the same fashion.

Figure 25: The final step is to tie the sutures in the subacromial space.