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Current Concepts in the Recognition and Treatment of Superior Labral (SLAP) Lesions

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Pathology of the superior aspect of the glenoid labrum (SLAP lesion) poses a significant challenge to the rehabilitation specialist due to the complex nature and wide variety of etiological factors associated with these lesions. A thorough clinical evaluation and proper identification of the extent of labral injury is important to determine the most appropriate nonoperative and/or surgical management. Postoperative rehabilitation is based on the specific surgical procedure as well as the extent, location, and mechanism of labral pathology and associated lesions. Emphasis is placed on protecting the healing labrum, while gradually restoring range of motion, strength, and dynamic stability of the glenohumeral joint. The purpose of this paper is to provide an overview of the anatomy and pathomechanics of SLAP lesions and review specific clinical examination techniques used to identify these lesions, including 3 newly described tests. Furthermore, a review of the current surgical management and postoperative rehabilitation guidelines is provided. *J Orthop Sports Phys Ther 2005;35:273-291.*

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The inherently complicated nature of injuries involving the superior aspect of the glenoid labrum can present a substantial clinical challenge. Successful return to unrestricted function requires integrating the appropriate diagnosis, surgical management, and rehabilitation in a coordinated effort. The advent of new arthroscopic techniques has helped to provide a better understanding of normal labral anatomy, capsulolabral anomalies, and the pathomechanics of conditions involving this structure. Likewise these techniques have also drastically improved the surgical treatment options available to successfully address these pathologies. Andrews et al[3] originally described the detachment of

the superior labrum in a subset of throwing athletes in 1985. Later Snyder et al[49] introduced the term SLAP lesion, indicating an injury located within the superior labrum extending anterior to posterior. They originally classified these lesions into 4 distinct categories based on the type of lesion present, emphasizing that this lesion may disrupt the origin of the long head of the biceps brachii[49] (Figure 1). Subsequent authors have added additional classification categories and specific subtypes, further expanding on the 4 originally described categories.[15,29,33] Based on these subtle differences in labral pathology an appropriate treatment plan may be developed to adequately address the specific pathology present.

In recent years it has become clear that symptomatic superior labral lesions and detachments can be treated effectively with either arthroscopic debridement or repair, depending on the specific type of pathology present.[14,39,45,51,65] We believe that it is critical to carefully follow a postoperative rehabilitation program that has been based on an accurate diagnosis that specifies extent of superior labral pathology to ensure a successful outcome. The purpose of this paper is to

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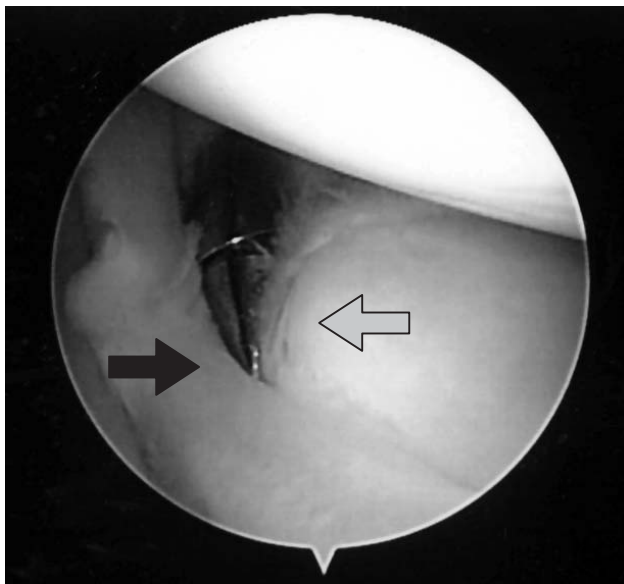
FIGURE 1. Arthroscopic view of a superior labral (SLAP) lesion. Note the detachment of the biceps tendon anchor (dark arrow) from the glenoid labrum (light arrow).

describe the normal anatomy, biomechanics, pathomechanics, physical exam, surgical management, and rehabilitation of lesions involving the superior labrum, which will assist rehabilitation professionals in effectively managing patients presenting with these complex lesions. The recognition and specific treatment of these lesions presented in this paper is based on our collective clinical experience and numerous published materials.

Normal Anatomy and Biomechanics of the Glenoid Labrum

The total surface of the humeral head is approximately 4 times larger than that of the glenoid, contributing to the tremendous joint mobility available at the glenohumeral joint. Glenohumeral stability is the result of the interplay between multiple anatomical structures that include the capsule, ligaments, muscles, tendons, osseous configuration, and glenoid labrum. Each of these elements contributes in controlling glenohumeral joint translation via a sophisticated biomechanical system that allows the shoulder to function successfully as the most mobile joint of the human body. The glenoid labrum plays an important role in this process.[38,46,59,60] Perry[40] demonstrated that the depth of the glenoid fossa across its equatorial line is doubled (from 2.5 to 5 mm) by the presence of the labrum.

The labrum is a fibrous structure strongly attached around the edge of the glenoid that serves to increase the contact surface area between the glenoid and the humeral head.[10] Although it is commonly stated that the glenoid labrum consists mainly of fibrous cartilage,[6,9,13] some studies have shown that it is composed of dense fibrous collagen tissue.[10,34]

Moseley and Overgaard[34] also noted that the superior and inferior labrum exhibit significantly different anatomy and that the labrum changes appearance in varying degrees of humeral rotation. The superior labrum is rather loose, mobile, and has a "meniscal" aspect, while the inferior labrum appears rounded and more tightly attached to the glenoid rim. Histologically, the attachment of the labrum to the glenoid rim consists of loose connective fibers above the equator of the glenoid while the inferior portion of the labral attachment is fixed by inelastic fibrous tissue.[10] The labrum is attached to the lateral portion of the biceps anchor superiorly. Additionally, approximately 50% of the fibers of the long head of the biceps brachii originate from the superior labrum and the remaining fibers originate from the supraglenoid tubercle of the glenoid.[10] The fibers of the biceps tendon blend with the superior labrum and continue posteriorly to become a periarticular fiber bundle, making up the bulk of the labrum.[22] The anterosuperior labral fibers appear to be attached more to the middle and inferior glenohumeral ligaments than directly to the glenoid rim itself.

Vascular supply to the labrum arises mostly from its peripheral attachment to the capsule and is from a combination of the suprascapular circumflex scapular branch of the subscapular and the posterior circumflex humeral arteries.[10] The anterosuperior labrum appears to generally have poor blood supply, whereas the inferior labrum exhibits significant blood flow.[10] Vascularity of the labrum decreases with increasing age.[10] No mechanoreceptors have been identified within the glenoid labrum.[53] However, free nerve endings have been isolated in the fibrocartilagenous tissue of the labrum, the biceps-labrum complex, and the connective tissue surrounding the labrum.[19,53]

The glenoid labrum enhances shoulder stability in 4 distinct ways: (1) it produces a "chock-block" effect between the glenoid and the humeral head that serves to limit humeral head translation[10,38,57,60]; (2) it increases the "concavity-compression" effect between the humeral head and the glenoid[10,31,38,57,60]; (3) it contributes to the stabilizing effect of the long head of the biceps anchor[46,57,60]; and (4) it increases the overall depth of the glenoid fossa.[10,57,60]

Normal Anatomic Variations

The cross-sectional shape of the superior labrum is similar in appearance to a knee meniscus. It is normally triangular, with the sharp free edge pointing to the center of the joint.[10] Sometimes the free edge of the labrum is more prominent and may extend into the center of the joint without any pathological significance. The presence of this finding is termed a "meniscoid-type" superior labrum and must not be considered pathological unless frayed or torn.[10,15] The presence of a meniscoid superior labrum may

lead to an incorrect diagnosis of a SLAP lesion during magnetic resonance imaging (MRI) interpretation. A meniscoid superior labrum may also tear in some athletes performing overhead sports or following a trauma and evolve into a type III SLAP lesion.

Another common normal finding is a minimal recess or anterior sublabral hole that must not be confused with a SLAP lesion.[34] This anterior sublabral hole can exist below the biceps attachment at or just above the 3-o'clock position (in a right shoulder) on the glenoid rim where a small notch is typically found.[13,34] Occasionally the labrum in front of this opening looks detached from the bone without any signs of a lesion. This is a normal anatomical variation and does not appear to contribute to glenohumeral joint instability.

In addition to the meniscoid superior labrum and sublabral hole variations, a third normal anatomical variation of the glenoid labrum, the Buford complex, also exists.[64] Williams et al[64] noted this variation in 1.5% of shoulders evaluated and described it as a cord-like middle glenohumeral ligament that blended with the anterior superior labrum, with the absence of any anterior superior labrum from the 12- to the 3-o'clock position (in a right shoulder) on the glenoid. The authors recommended not treating this variation surgically as it does not appear to lead to instability and/or pain when present in isolation.

Pathomechanics of SLAP Lesions

There are several injury mechanisms that are speculated to be responsible for creating SLAP lesions. These mechanisms range from single traumatic events to repetitive microtraumatic injuries. Traumatic events, such as falling on an outstretched arm or bracing oneself during a motor vehicle accident, may result in SLAP lesions due to compression of the superior joint surfaces superimposed with subluxation of the humeral head. Snyder et al[49] referred to this as a pinching mechanism of injury. Other traumatic injury mechanisms include direct blows, falling onto the point of the shoulder, and forceful traction injuries of the upper extremity.

Repetitive overhead activity, such as throwing a baseball, is another common mechanism of injury frequently responsible for producing SLAP injuries.[3,7,33] Andrews et al[3] first hypothesized that SLAP pathology in overhead throwing athletes was the result of the high eccentric activity of the biceps brachii during the arm deceleration and follow-through phases of the overhead throw. The authors applied electrical stimulation to the biceps during arthroscopic evaluation and noted that the biceps contraction raised the labrum off of the glenoid rim, simulating the hypothesized mechanism.[3]

Burkhart and Morgan[7] and Morgan et al[33] have hypothesized a "peel-back" mechanism that produces SLAP lesion in the overhead athlete. They suggest that when the shoulder is placed in a position of abduction and maximal external rotation (ER), the rotation produces a twist at the base of the biceps, transmitting torsional force to the anchor (Figure 2). Pradham et al[41] recently measured superior labral strain in a cadaveric model during each phase of the throwing motion. They noted that increased superior labral strain occurred during the late cocking phase of throwing. Furthermore, Jobe[23] and Walch et al[54] have also demonstrated that when the arm is in a maximally externally rotated position there is contact between the posterior-superior labral lesions and the rotator cuff.

A recent study at our center[47] simulated each of these mechanisms using cadaveric models. Nine pairs of cadaveric shoulders were loaded to biceps anchor complex failure in either a position of simulated in-line loading (similar to the deceleration phase of throwing) or simulated peel-back mechanism (similar to the cocking phase of overhead throwing). Results showed that 7 of 8 of the in-line loading group failed in the midsubstance of the biceps tendon, with 1 of 8 fracturing at the supraglenoid tubercle. However, all 8 of the simulated peel-back group failures resulted in a type II SLAP lesion. The ultimate strength of the biceps anchor was significantly different when the 2 loading techniques were compared. The biceps anchor demonstrated significantly higher ultimate strength with the in-line loading (508 N) as opposed to the ultimate strength seen during the peel-back loading mechanism (202 N).

A subsequent follow-up study at our center evaluated the same mechanisms of injury pattern (peel-back versus in-line loading) in 7 paired cadaveric models following repair of a SLAP II lesion.[12] The results were similar to those published by Sheppard et al on intact structures,[47] with a 51% lower load to failure in the peel-back group compared to the
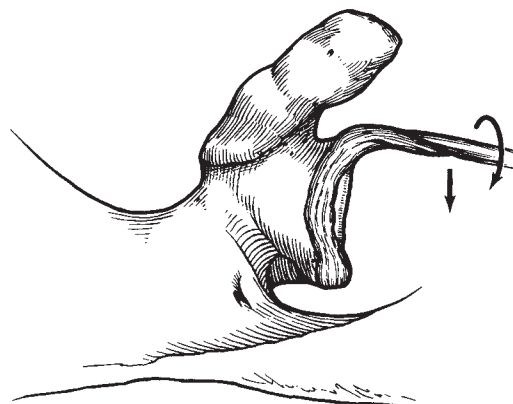
FIGURE 2. Peel-back mechanism of SLAP injury. When the shoulder is placed in a position of maximal external rotation, the rotation produces a torsional force to the base of the biceps anchor (reproduced with permission from Burkhart and Morgan[7]).

in-line loading group. However, the mean load to failure was 77% of the load to failure of the intact biceps labral complexes, as determined by Shepard et al.[47] Interestingly, the location of failure was found to occur at the biceps attachment to the glenoid tubercle, rather than at the posterosuperior labrum, in 5 of 7 specimens, suggesting that the strength of the SLAP repair was stronger than the biceps insertion on the glenoid tubercle.

In theory, SLAP lesions most likely occur in overhead athletes from a combination of these 2 previously described forces. The eccentric biceps activity during deceleration may serve to weaken the biceps-labrum complex, while the torsional peel-back force may result in the posterosuperior detachment of the labral anchor.

Several authors have also reported a strong correlation between SLAP lesions and glenohumeral instability.[7,25,38,43,46,63] Normal biceps function and glenohumeral stability is dependent on a stable superior labrum and biceps anchor. Pagnani et al[38] found that a complete lesion of the superior portion of the labrum large enough to destabilize the insertion of the biceps was associated with significant increases in anterior-posterior and superior-inferior glenohumeral translation. Reinold et al[43] reported that in a series of 130 overhead athletes with symptomatic hyper laxity undergoing thermal-assisted capsular shrinkage of the glenohumeral joint (TACS), 69% exhibited superior labral degeneration, while 35% had type II SLAP lesions. Furthermore, Pagnani et al[38] reported that the presence of a simulated SLAP lesion in 7 cadaveric shoulders resulted in a 6-mm increase in anterior glenohumeral translation. These studies are in agreement with the results of Glousman,[16] who showed increased EMG activity of the biceps brachii in baseball pitchers with anterior instability. Furthermore, Kim et al[25] reported that maximal biceps activity occurred when the shoulder was abducted to 90° and externally rotated to 120° in patients with anterior instability. Because this position is remarkably similar to the cocking position of the overhand throwing motion, the finding of instability may cause or facilitate the progression of internal impingement (impingement of the infraspinatus on the posterosuperior glenoid rim) in the overhead athlete.

CLASSIFICATION OF SLAP LESIONS

Because the glenoid labrum is involved in the stability of the glenohumeral joint, pathological conditions of the labrum appear in cases of instability, whether due to repetitive loads or frank traumatic injury.[6,29,31,34] Clinically these instabilities may be either gross or subtle. Although instability may occur, SLAP lesions most often result in symptoms of mechanical pain and dysfunction, rather than instability.

The prevalence of SLAP lesions is disputed in the published literature. Some authors have reported encountering SLAP lesions in as many as 26% of shoulders undergoing arthroscopy.[20,27,29,48,49,52,55] These percentages rise dramatically in reports specific to overhead throwing athletes. Andrews et al[3] noted that 83% of 73 throwers exhibited labral lesions when evaluated arthroscopically. Reinold et al[43] noted 91% of overhead athletes undergoing TACS for glenohumeral instability had superior labral pathology of some type.

Following a retrospective review of 700 shoulder arthroscopies, Snyder et al[49] identified 4 types of superior labrum lesions involving the biceps anchor (Figure 3). Collectively they termed these SLAP lesions, in reference to their anatomic location (superior labrum extending from anterior to posterior). Type I SLAP lesions were described as being indicative of isolated fraying of the superior labrum, with a firm attachment of the labrum to the glenoid. These lesions are typically degenerative in nature. Type II SLAP lesions are characterized by a detachment of the superior labrum and the origin of the tendon of the long head of the biceps brachii from the glenoid resulting in instability of the biceps-labral anchor. A bucket-handle tear of the labrum with an intact biceps insertion is the characteristic presentation of a type III SLAP lesion. Type IV SLAP lesions have a bucket-handle tear of the labrum that extends into the biceps tendon. In this lesion, instability of the biceps-labrum anchor is also present, similar to that seen in the type II SLAP lesion.

Maffet et al[29] noted that 38% of the SLAP lesions identified in their retrospective review of 712 arthroscopies were not classifiable using the I-IV terminology previously defined by Snyder et al.[49] They suggested expanding the classification scale for SLAP lesions to a total of 7 categories, adding descriptions for types V through VII.[29] Type V SLAP lesions are characterized by the presence of a Bankart lesion of the anterior capsule that extends into the anterior superior labrum. Disruption of the biceps tendon anchor with an anterior or posterior superior labral flap tear is indicative of a type VI SLAP lesion. Type VII SLAP lesions are described as the extension of a SLAP lesion anteriorly to involve the area inferior to the middle glenohumeral ligament. These 3 types typically involve a concomitant pathology in conjunction with a SLAP lesion. Thus, the surgical treatment and rehabilitation will vary based on these concomitant pathologies. A detailed description of these variations is beyond the scope of the current paper.

Three distinct subcategories of type II SLAP lesions have been further identified by Morgan et al.[33] They reported that in a series of 102 patients undergoing
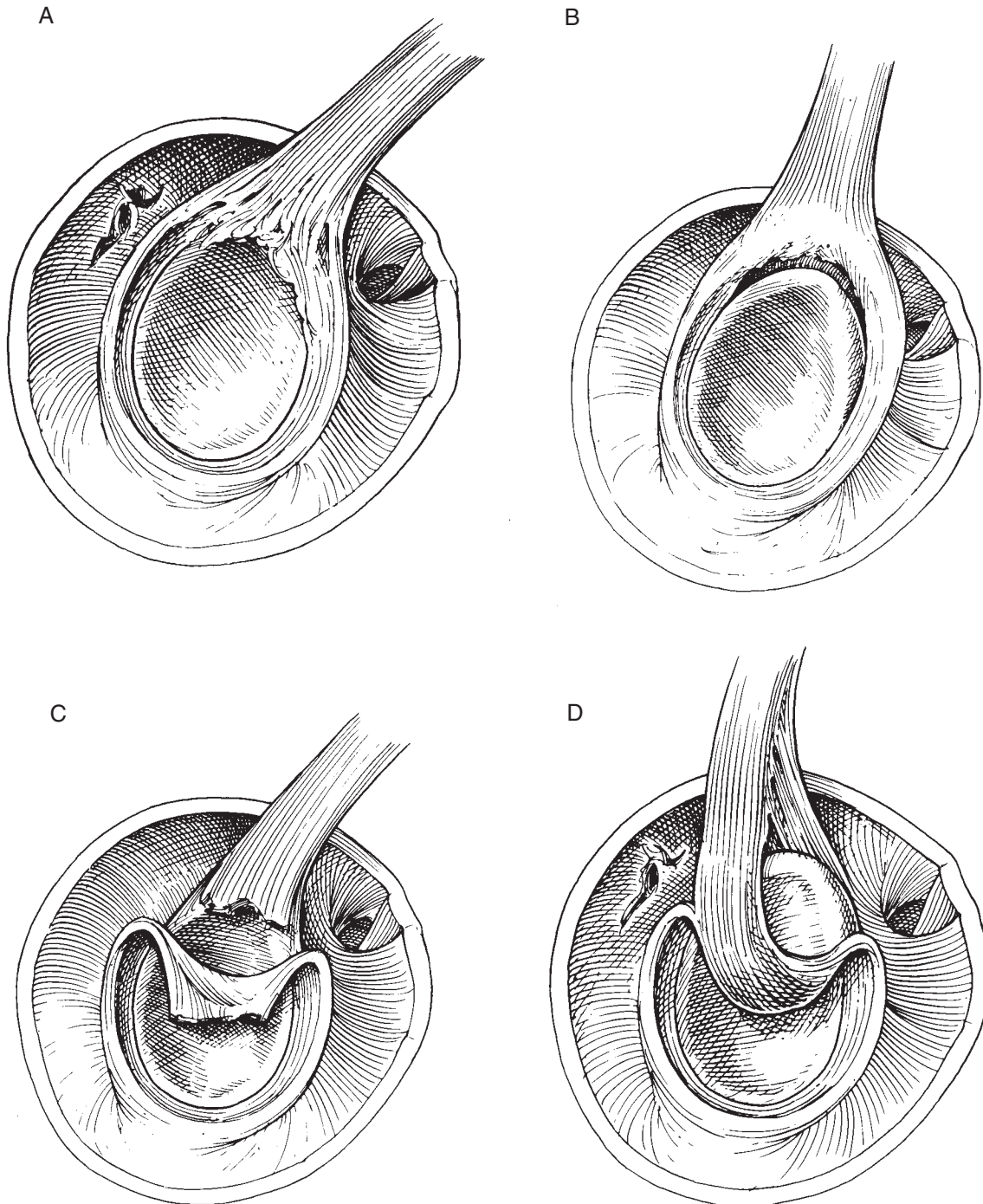
A

B

C

D

FIGURE 3. Illustration of type I-IV SLAP lesions. Type I represents a frayed and/or degenerative labrum with a firm attachment of the labrum to the glenoid (Figure 3A). Type II represents a detachment of the superior labrum and biceps attachment from the glenoid rim (Figure 3B). Type III represents a bucket-handle tear of the labrum with an intact biceps anchor (Figure 3C). Type IV represents a bucket-handle tear of the labrum that extends into the biceps tendon (Figure 3D). Reproduced with permission from Snyder et al.[49]

arthroscopic evaluation 37% presented with an anterosuperior lesion, 31% presented with a posterosuperior lesion, and 31% exhibited a combined anterior and superior lesion.[33] These findings are consistent with our clinical observations. In the authors' experience, the majority of overhead athletes present with posterosuperior lesions, while individuals who have traumatic SLAP lesions typically present with anterosuperior lesions. These variations may become important when selecting which special tests to perform based on the patient's history and mechanism of injury.

CLINICAL COMMENTARY

CLINICAL EVALUATION

As with appropriately assessing any pathology, a thorough clinical examination is essential to establishing the potential presence of glenoid labral pathology. Clinical examination to detect SLAP lesions is often difficult because of the common presence of concomitant pathology in patients presenting with this type of condition. Andrews et al[3] reported that 45% of patients (and 73% of baseball pitchers) with superior labral lesions had concomitant partial thickness tears of the supraspinatus portion of the rotator cuff. Mileski and Snyder[31] 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. Kim et al[27] prospectively analyzed the clinical features of different types of SLAP lesions as they varied with patient population in 139 cases. They demonstrated that type I SLAP lesions are typically associated with rotator cuff pathology, while types III and IV are associated with traumatic instability. They also note that injuries presenting concomitant with type II SLAP lesions vary by patient age, with older patients presenting more often with rotator cuff pathology and younger patients with instability.

The clinical examination should include subjective history, physical examinations, specific special tests, and an enhanced MRI. In combination, the goal of these measures is to make an accurate clinical diagnosis. A comprehensive history, including the exact mechanism of injury, must be obtained and should clearly define all overhead activities and sports participation. The clinician should keep in mind that while labral pathologies frequently present as repetitive overuse conditions, such as those commonly seen in overhead athletics, the patient may also describe a single traumatic event such as a fall onto the outstretched arm or an episode of sudden traction, or a blow to the shoulder. A patient with a superior labral injury may have nonspecific complaints. Pain complaints are typically intermittent and are most frequently associated with overhead activity. Often patients exhibit mechanical symptoms of painful clicking or catching of the shoulder.[2] Pain is typically elicited with specific movements and the condition is not painful at rest. We refer to this as ''mechanical pain'' as opposed to pain at rest, which is often present when rotator cuff pathology is present. Overhead athletes typically report a loss of velocity and accuracy along with general uneasiness of the shoulder. Snyder et al[48] have reported that this type of subjective complaint is present in 50% of patients. Probably the most predictive subjective complaint in the athlete is the inability to perform sporting activities at a high level.

The physical examination should include a complete evaluation of bilateral passive and active range

FIGURE 4. Active-compression test. The patient's shoulder is positioned at 90° of elevation, 30° of horizontal adduction, and full internal rotation. Resistance against elevation is applied by the examiner in an attempt to compress the labrum.

FIGURE 5. Compression-rotation test. The examiner imparts a compressive force through the long axis of the humerus as the shoulder is rotated in an attempt to grind or trap the labrum within the joint.

of glenohumeral motion with particular emphasis on determining the presence, persistence, and behavior of any painful arc of motion. Our experience suggests that patients with a SLAP lesion will often exhibit pain with passive ER at 90° of shoulder abduction, especially with overpressure. Furthermore, pain may also be present with active arm elevation. A wide variety of potentially useful special-test maneuvers have been described to help determine the presence of labral pathology, including the active-compression test,[37] compression-rotation or grind test,[49] Speed's

test,[49] the clunk test,[3] the crank test,[28] the anterior-slide test,[24] the biceps-load test,[26] the biceps-load test II,[25] and the pain provocation test.[32]

The active-compression test, as described by O'Brien et al,[37] is used to evaluate labral lesions and acromioclavicular joint injuries. The shoulder is placed into approximately 90° of elevation and 30° of horizontal adduction across the midline of the body. Resistance is applied, using an isometric hold in this position, with both full shoulder internal rotation (IR) and ER (altering humeral rotation against the glenoid in the process) (Figure 4). A positive test for labral involvement is when pain is elicited during testing, with the shoulder in IR and forearm in pronation (thumb pointing toward the floor). Symptoms are typically decreased when tested in the externally rotated position or the pain is localized at the acromioclavicular (AC) joint. O'Brien et al[37] found this maneuver to be 100% sensitive and 95% specific as it relates to assessing the presence of labral pathology. Pain provocation using this test is common, which challenges the validity of the results. In the authors' experience, the presence of deep and diffuse glenohumeral joint pain is most indicative of the presence of a SLAP lesion. Pain localized in the AC joint or in the posterior rotator cuff is not specific for the presence of a SLAP lesion. The posterior shoulder symptoms are indicative of provocative strain on the rotator cuff musculature when the shoulder is placed in this position.

The compression-rotation test[49] is performed with the patient in the supine position. The glenohumeral joint is manually compressed through the long axis of the humerus while the humerus is passively rotated back and forth in an attempt to trap the labrum within the joint (Figure 5). When performing this maneuver, the authors typically perform a variety of small and large circles, while providing joint compression, in an attempt to grind the labrum between the glenoid and the humeral head. Furthermore, the examiner may attempt to detect anterosuperior labral lesions by placing the arm in a horizontally abducted position while providing an anterosuperior-directed force. In contrast, the examiner may also horizontally adduct the humerus and provide a posterosuperior-directed force when performing this test.

The Speed's biceps tension test has been found to accurately reproduce pain in instances of SLAP lesions.[14,45,49,51] It is performed by resisting downwardly applied pressure to the arm when the shoulder is positioned in 90° of forward elevation with the elbow extended and forearm supinated. Clinically, we also perform a new test for SLAP lesions. This is a variation of the original Speed's test, which we refer to as the "dynamic Speed's test" (Figure 6). During this maneuver, the examiner provides resistance against both shoulder elevation and elbow flexion

FIGURE 6. A new test for detecting SLAP lesions, the dynamic Speed's test. The examiner resists forward shoulder elevation and elbow flexion simultaneously as the arm is elevated overhead in an attempt to provide tension on the superior labrum. (Video available at www.jospt.org.)

FIGURE 7. Clunk test. The examiner applies a compressive or anterior force proximally while rotating the shoulder overhead in an attempt to trap the labrum.

simultaneously as the patient elevates the arm overhead. Deep pain within the shoulder is typically produced with shoulder elevation above 90° if this test is positive for labral pathology. Anecdotally, we have found this maneuver to be more sensitive than the originally described static Speed's test in detecting SLAP lesions, particularly in the overhead athlete.
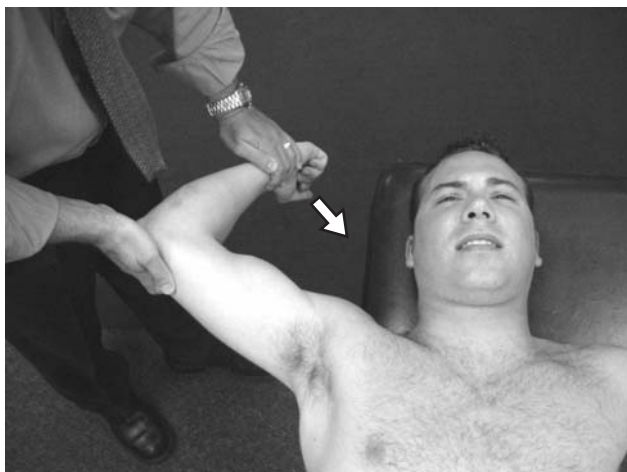
FIGURE 8. Biceps load II test. The patient is passively positioned in maximal external rotation at 120° of abduction, with the forearm in a supinated position. In this position, an isometric biceps contraction is performed in an attempt to peel back the labrum.

The clunk test[3] is performed with the patient supine. The examiner places one hand on the posterior aspect of the glenohumeral joint while the other grasps the bicondylar aspect of the humerus at the elbow. The examiner's proximal hand provides an anterior translation of the humeral head while simultaneously rotating the humerus externally with the hand holding the elbow (Figure 7). The mechanism of this test is similar to that of a McMurray's test of the knee menisci, where the examiner attempts to trap the torn labrum between the glenoid and the humeral head. A positive test is produced by the presence of a clunk or grinding sound and is indicative of a labral tear.[4]

The crank test[28] can be performed with the patient either sitting or supine. The shoulder is elevated to 160° in the plane of the scapula. An axial load is then applied by the examiner while the humerus is internally and externally rotated in this position. A positive test typically elicits pain with ER. Symptomatic clicking or grinding may also be present during this maneuver.

The anterior-slide test was originally described by Kibler.[24] To perform this test the arm to be examined is positioned with the hand on the ipsilateral hip with the thumb forward. The examiner then stabilizes the scapula with one hand and provides an anterosuperiorly directed axial load to the humerus with the other hand.[24] The test is considered positive if there is a click or deep pain in the shoulder during this maneuver.

The biceps load test was originally described by Kim et al.[26] During this test, the shoulder is placed in 90° of abduction and maximally externally rotated. At maximal ER and with the forearm in a supinated position, the patient is instructed to perform a biceps contraction against resistance. Deep pain within the shoulder during this contraction is indicative of a

SLAP lesion. The original authors further refined this test with the description of the biceps load II maneuver.[25] The examination technique is similar, although the shoulder is placed into a position of 120° of abduction rather than the originally described 90° (Figure 8). The biceps load II test was noted to have greater sensitivity than the original test.[25]

Mimori et al[32] described the pain provocation test. During this maneuver, the shoulder is passively abducted 90° to 100° and passively externally rotated with the forearm in full pronation and then full supination. The authors determined that a SLAP lesion was present if pain was produced with shoulder ER with the forearm in the pronated position or if the severity of the symptoms was greater in the pronated position. The authors note that positive symptoms with this test are due to the additional stretch placed on the biceps tendon when the shoulder is externally rotated with the forearm pronated.

We have recently begun utilizing 2 new tests to detect SLAP lesions during clinical examination. The first test, which we refer to as the "pronated load test," is performed in the seated position with the shoulder abducted to 90° and externally rotated. However, the forearm is in a fully pronated position to increase tension on the biceps and subsequently the labral attachment. When maximal ER is achieved, the patient is instructed to perform a resisted isometric contraction of the biceps to simulate the peel-back mechanism (Figure 9). This test combines the active

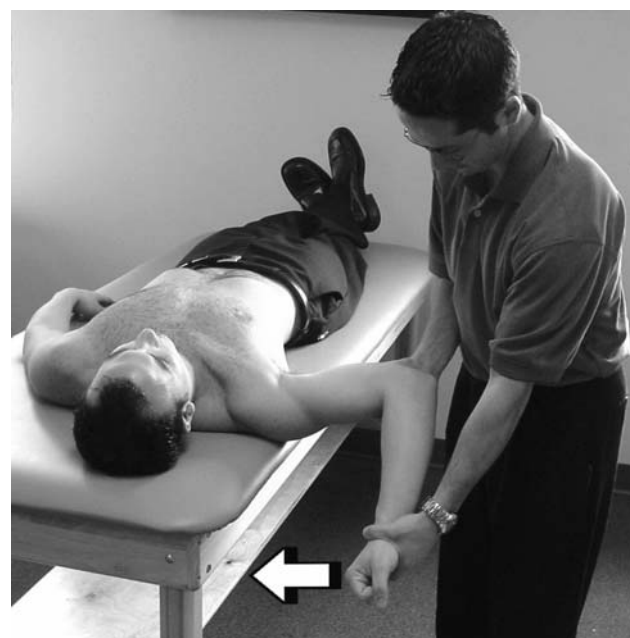**FIGURE 9.** A new test for detecting SLAP lesions, the pronated load test. The patient's shoulder is abducted to 90° to 110°. The examiner passively externally rotates the shoulder with the forearm in pronation. When maximal external rotation is achieved, the patient is instructed to perform an isometric biceps contraction in an attempt to peel back the labrum. (Video available at www.jospt.org.)

FIGURE 10. A new test for detecting SLAP lesions, the resisted supination external-rotation test. The patient is passively positioned at 90° of abduction, 65° to 70° of elbow flexion, and neutral rotation. The examiner simultaneously resists forearm supination during passive shoulder external rotation in an attempt to peel back the labrum. (Video available at www.jospt.org.)

bicipital contraction of the biceps load test with the passive ER in the pronated position similar to the pain provocation test.

The second new test, recently described by Myers et al,[35] is called the resisted supination external rotation test (Figure 10). During this test, the patient is positioned in 90° of shoulder abduction, and 65° to 70° of elbow flexion, and the forearm in neutral position. The examiner resists against a maximal supination effort while passively externally rotating the shoulder. Myers et al[35] note that this test simulates the peel-back mechanism of SLAP injuries by placing maximal tension on the long head of the biceps. A preliminary study of 40 patients revealed that this test had better sensitivity (82.8%), specificity (81.8%), positive predictive value (92.3%), negative predictive value (64.3%), and diagnostic accuracy (82.5%), compared to the crank test and active-compression test (Table 1).[35]

Anecdotally, we have found these tests (the pronated load and the resisted supination external rotation tests) to be 2 of the most sensitive tests in detecting SLAP lesions, particularly in the overhead athlete.

McFarland et al[30] evaluated the ability of 3 clinical tests to predict the presence of labral pathology. In this investigation 3 tests (active-compression test, anterior-slide test, and compression-rotation test) were performed on 426 patients who subsequently underwent arthroscopic examination. Of these patients, 39 had type II through IV SLAP lesions, while 387 had type I lesions. The active-compression test was found to be the most sensitive and have the highest predictive value, although both values were low (47% sensitivity, 10% positive predictive value). The anterior-slide test was the most specific maneuver, with an 84% specificity. All 3 tests were found to

TABLE 1. Diagnostic accuracy of special tests associated with SLAP lesions.

Test	N	Sensitivity	Specificity	PPV*	NPV†
Active compression[18]	33	54	47.0	55.0	45
Active compression[30]	426	47	55.0	10.0	91
Active compression[37]	318	100	99.5	94.6	100
Active compression[35]	37	78	11.0	70.0	14
Active compression[51]	65	54	31.0	34.0	50
Anterior slide[24]	226	78	92.0		
Anterior slide[30]	426	8	84.0	5.0	90
Biceps load II[25]	127	90	97.0	92.0	96
Compression rotation[30]	426	24	76.0	9.0	90
Crank[18]	33	39	67.0	59.0	47
Crank[28]	62	91	93.0	94.0	90
Crank[35]	36	35	70.0	75.0	29
Crank[51]	65	46	56.0	41.0	61
MRI[5]	52	89	91.0	90.0	
MRI[8]	46	89	88.0	89.0	
MR[51]	65	42	92.0	63.0	83
Pain provocation[32]	32	100	90.0	97.0	
Resisted supination ER[35]	40	83	82.0	92.0	64
Speed's[18]	33	9	74.0	30.0	40
Speed's[21]	50	32	75.0	50.0	58

* Positive predictive value.
† Negative predictive value.

TABLE 2. Selection of SLAP tests based on mechanism of injury.

Mechanism	Test
Compressive injury	Active compression
	Compression-rotation
	Clunk
	Anterior slide
Traction injury	Speed's
	Dynamic Speed's
	Active compression
Peel-back injury (overhead athlete)	Pronated load
	Resisted supination external rotation
	Biceps load I and II
	Pain provocation
	Crank

have high associated accuracy, although the majority of patients presented with only type I lesions. It is also interesting to note that the presence of clicking and the location of pain was not a reliable predictor of the presence or severity of labral involvement.

Stetson et al[51] similarly examined the reliability and validity of the crank test, active-compression test, and enhanced MRI. MRI was determined to have the greatest specificity (92%), positive predictive value (63%), and negative predictive value (83%). Of the 2 clinical tests, the crank test was found to have better specificity, positive predictive value, and negative predictive value. Sensitivity was low (42%-54%) for all 3 tests.

The reliability of MRI for the diagnosis of SLAP lesions is disputed[17,28] and definitive diagnosis requires arthroscopy. Several authors recommend MR-enhanced arthrography to detect SLAP lesions.[5,36] Bencardino et al[5] retrospectively reviewed preoperative MR arthrography following shoulder arthroscopy. The authors report MR arthrography has a sensitivity of 89%, a specificity of 91%, and an accuracy of 90% (47 of 52 patients) in detecting SLAP lesions. Thus, enhanced MR arthrography is routinely utilized at our center to assess the glenoid labrum.

Thus, it appears that each of the current SLAP tests have limited diagnostic accuracy (Table 1). A limitation of previous studies is the lack of differentiation among the different types of SLAP lesions. In most studies, several variations of SLAP lesions are grouped together to obtain enough statistical power to analyze the data. It is the authors' opinion that different tests will result in different specificity and sensitivity results, based on the variation of SLAP lesion present. For example, overhead athletes with a type II or IV posterosuperior peel-back SLAP lesion may be more symptomatic during tests that simulate the aggravating position and mechanism of injury, such as the biceps load II, clunk, crank, and pain provocation tests; whereas patients with type I or III SLAP lesions due to a traumatic type of injury may be more symptomatic during tests that provide compres-

sion to the labral complex, such as the active-compression, compression-rotation, and anterior-slide tests. Further investigation on the diagnostic characteristics of these tests based on the type of SLAP lesion is warranted.

Furthermore, the authors feel it is imperative to correlate the clinical examination findings to the patient's complaints, symptoms, and injury mechanism. The selection of specific SLAP tests to perform may be based on the symptomatic complaints as well as the mechanism of injury described by the patient (Table 2). It is our opinion that the clinician should correlate the clinical examination findings to the chief complaint.

SURGICAL MANAGEMENT

Conservative management of SLAP lesions is often unsuccessful, particularly of type II and IV lesions with labral instability and underlying shoulder instability. Therefore, surgical intervention is most often warranted to repair the labral lesion while addressing any concomitant pathology. In the event that an athlete does undergo conservative rehabilitation, many of the same principles discussed in the upcoming sections may be applied.

Our experience suggests that a type I SLAP lesion may represent age-related fraying of the superior labrum and does not necessarily require specific treatment. Often the overhead athlete may exhibit fraying of the superior and posterior labrum due to internal impingement.[54] Isolated debridement of labral fraying has not been shown to reliably relieve symptoms over the long term.[1,12] However, if symptoms are progressive in nature or warrant surgical intervention, type I SLAP lesions are generally debrided back to a stable labral rim.

FIGURE 11. SLAP II repair using suture anchors.

Type III SLAP lesions should also be excised and debrided back to a stable rim, much like some bucket-handle meniscus tears in the knee. The exception to this is a type III lesion involving a Buford complex, which should be treated as a type II SLAP lesion.[50]

The outcomes following debridement (without repair) of unstable type II and IV SLAP lesions have been poor and thus should be repaired to restore the normal anatomy.[1,12] In the presence of a type II SLAP lesion, the superior labrum should be reattached to the glenoid and the biceps anchor stabilized (Figure 11). The type II lesion is often stabilized utilizing suture anchors or a bioabsorbable tack. Treatment of type IV SLAP lesions is generally based on the extent to which the biceps anchor is involved. When biceps involvement is less than approximately 30% of the entire anchor, the torn tissue is typically resected and the superior labrum reattached. If the biceps tear is more substantial, a side-to-side repair of the biceps tendon, in addition to reattachment of the superior labrum, is generally performed. However, if the biceps tear is extensive enough to substantially alter the biceps origin, a biceps tenodesis is more practical than a direct repair. In addition to the treatment of the SLAP lesion, associated rotator cuff pathology or glenohumeral joint instability should be independently evaluated and treated at the time of surgery.

Operative SLAP Repair Surgical Technique

The goal of surgical repair of a SLAP lesion is to obtain a strong repair that allows the patient to aggressively rehabilitate the shoulder and return to full activities or sports competition.

Using arthroscopic surgical techniques, the superior labrum is mobilized along the entire area of detachment using a 4.5-mm motorized shaver to take down any fibrous adhesions. This area usually extends from approximately the 11- to the 1-o'clock positions of the glenoid (in a right shoulder). The bony area of attachment is abraded to create a bleeding bed to facilitate healing. The repair surface of the labrum is also gently debrided to stimulate a healing response. Two suture anchors are usually adequate to secure the biceps anchor and superior labrum. Our center prefers to use bioabsorbable suture anchors with number 2 braided nonabsorbable sutures loaded on the eyelet. The number of anchors utilized is based on the size of the SLAP lesion present. The suture anchors are positioned so that each one splits the difference between the biceps and the normal area of labral insertion, usually at the 11:30 and 12:30 positions on a clock face. The suture anchors are placed at the junction of the articular cartilage and cortical bone. The security of anchor fixation is tested with a firm pull on the sutures. Once the suture anchors are in place, one end of each suture is passed through the labrum. The surgeon may choose to incorporate some of the biceps tendon near the junction of the biceps and labrum if necessary to secure the biceps anchor. Arthroscopic knot-tying techniques are utilized. In general, the placement of anchors and tying knots progresses from posterior to anterior.

The outcomes following repair of unstable SLAP II and IV lesions have been good with satisfactory results in over 80% of patients in the majority of published articles.[2,3,14,39,45,51,53,65] At our center, Reinold et al[43] reported that 87% of athletes undergoing TACS with concomitant debridement of a SLAP lesion and 84% of athletes with a concomitant SLAP repair returned to competition with good to excellent outcomes using the Modified Athletic Shoulder Outcome Scale.

SLAP LESION REHABILITATION GUIDELINES

The specific rehabilitation program following surgical intervention involving the superior glenoid labrum is dependent on the severity of the pathology and should specifically match the type of SLAP lesion, the exact surgical procedure performed (debridement versus repair), and other possible concomitant procedures performed because of the underlying glenohumeral joint instability that is often present. Overall, emphasis should be placed on restoring and enhancing dynamic stability of the glenohumeral joint, while at the same time ensuring that adverse stresses are not applied to healing tissue.

Prior to rehabilitation, we believe that it is imperative that a thorough subjective and clinical exam be performed to determine the exact mechanism and nature of labral pathology. For patients who sustained a SLAP lesion via a compressive injury, such as a fall on an outstretched hand, weight-bearing exercises should be avoided to minimize compression and sheer on the superior labrum. Patients with traction injuries should avoid heavy resisted or excessive eccentric biceps contractions. Furthermore, patients with peel-back lesions, such as overhead athletes, should avoid excessive amounts of shoulder ER while the SLAP lesion is healing. Thus the mechanism of injury is an important factor to individually assess when determining appropriate rehabilitation guidelines for each patient.

Although the efficacy of rehabilitation following SLAP repairs has not been documented, the following sections will overview guidelines based on our clinical experience and basic science studies on the mechanics of the glenoid labrum and pathomechanics of SLAP lesions.[2,7,10,11,35,36,38,43,44,46,47,53,60,62]

Debridement of Type I and III SLAP Lesions

Type I and type III SLAP lesions normally undergo a simple arthroscopic debridement of the frayed

TABLE 3. Rehabilitation protocol following arthroscopic debridement of type I and III SLAP lesions.

I. Phase 1: motion phase (days 1-10)

Goals
- Re-establish nonpainful range of motion
- Retard muscular atrophy
- Decrease pain/inflammation

Range of motion (PROM/AAROM)
- Pendulums exercise
- Rope and pulley
- L-bar exercises
  - Flexion/extension
  - Abduction/adduction
  - ER/IR (begin at 0° AB, progress to 45° AB, then 90° AB)
- Self-stretches (capsular stretches)

Exercises
- Isometrics
- No BICEPS isometrics for 5 to 7 days postoperative
- May initiate tubing for ER/IR at 0° AB late phase (usually 7 to 10 days postoperative)

Decrease pain/inflammation
- Ice, NSAIDs, modalities

II. Phase 2: intermediate phase (weeks 2-4)

Goals
- Regain and improve muscular strength
- Normalize arthrokinematics
- Improve neuromuscular control of shoulder complex

Criteria to progress to phase 2
- Full PROM
- Minimal pain and tenderness
- Good MMT of IR, ER, flex

Week 2
Exercises
- Initiate isotonic program with dumbbells
  - Shoulder musculature
  - Scapulothoracic
  - Tubing ER/IR at 0° abduction
  - Sidelying ER
  - Prone rowing ER
  - PNF manual resistance with dynamic stabilization
- Normalize arthrokinematics of shoulder complex
  - Joint mobilization
  - Continue stretching of shoulder (ER/IR at 90° of abduction)
- Initiate neuromuscular control exercises
- Initiate proprioception training
- Initiate trunk exercises
- Initiate UE endurance exercises

Decrease pain/inflammation
- Continue use of modalities, ice, as needed

Week 3
Exercises
- Thrower's ten program
- Emphasis rotator cuff and scapular strengthening
- Dynamic stabilization drills

III. Phase 3: dynamic-strengthening phase, advanced-strengthening phase (weeks 4-6)

Goals
- Improve strength, power, and endurance
- Improve neuromuscular control
- Prepare athlete to begin to throw, etc

Criteria to enter phase 3
- Full nonpainful AROM and PROM
- No pain or tenderness
- Strength 70% compared to contralateral side

Exercises
- Continue thrower's ten program
- Continue dumbbell strengthening (supraspinatus, deltoid)
- Initiate tubing exercises in the 90°/90° position for ER/IR (slow/fast sets)

TABLE 3. (continued)

- Exercises for scapulothoracic musculature
- Tubing exercises for biceps
- Initiate plyometrics (2-hand drills progress to 1-hand drills)
- Diagonal patterns (PNF)
- Initiate isokinetic strengthening
- Continue endurance exercises: neuromuscular control exercises
- Continue proprioception exercises

IV. Phase 4: return-to-activity phase (week 7 and beyond)

Goals
- Progressively increase activities to prepare patient for full functional return

Criteria to progress to phase 4
- Full PROM
- No pain or tenderness
- Isokinetic test that fulfills criteria to throw[63]
- Satisfactory clinical exam

Exercises
- Initiate interval sport program (ie, throwing, tennis, etc)
- Continue all exercises as in phase 3 (throw and train on same day, LE and ROM on opposite days)
- Progress interval program

Follow-up visits
- Isokinetic tests
- Clinical exam

Abbreviations: AB, abduction; AAROM, active assisted range of motion; ER, external rotation; IR, internal rotation; LE, lower extremity; MMT, manual muscle test; NSAIDS, nonsteroidal anti-inflammatory drugs; PNF, proprioceptive neuromuscular facilitation; PROM, passive range of motion; ROM, range of motion; UE, upper extremity.

labrum without an anatomic repair. Table 3 outlines the rehabilitation program following this type of procedure. This program can be somewhat aggressive in restoring motion and function because the biceps-labral anchor is stable and intact. The rate of progression during the course of postoperative rehabilitation is based on the presence and extent of concomitant lesions. If, for example, significant rotator cuff fraying (partial thickness tear) is present and treated with arthroscopic debridement, the rehabilitative program must be appropriately adapted. Generally, a sling is worn for comfort during the first 3 to 4 days following surgery. Active-assistive range of motion (AAROM) and passive range of motion (PROM) exercises are initiated immediately following surgery, with full PROM expected within 10 to 14 days postoperatively. Flexion ROM is performed to tolerance. ER and IR in the scapular plane are initiated at 45° of glenohumeral abduction and advanced to 90° of abduction usually by postoperative day 4 or 5. ROM exercises may be performed early because an anatomical repair has not been performed.

Isometric strengthening in all planes of shoulder motion is performed submaximally and pain free during the first 7 days after surgery to minimize muscular atrophy. Light isotonic strengthening for the shoulder and scapular musculature (with the exception of the biceps) are initiated approximately 8 days following surgery. This includes ER/IR exercise tubing, sidelying ER, prone rowing, prone horizontal abduction, and prone ER. Active elevation exercises, such as scapular plane elevation (full can) and lateral raises, are also included. Weighted resistance begins at 0.45 kg (1 lb) and advances by 0.45 kg per week in a gradual, controlled, progressive-resistance fashion. This progression is used to gradually challenge the musculature. Light biceps resistance is usually not initiated until 2 weeks following surgery in an attempt to prevent debridement site irritation. Furthermore, caution should be placed on early over-aggressive elbow flexion and forearm supination exercises, particularly eccentric exercises.

As the strengthening program progresses after this type of surgical procedure, the emphasis of rehabilitative interventions should be on obtaining muscular balance and promoting dynamic shoulder stability. This is accomplished through a variety of manual resistance and end range rhythmic stabilization drills performed in conjunction with isotonic strengthening and core stabilization exercises. The primary goal of these drills is to re-establish dynamic humeral head control, especially if the pathomechanics of the labral lesion was due to excessive glenohumeral laxity.

The individual is advanced to controlled weight-training activities between postoperative weeks 4 and 6. The athlete is instructed on proper technique, such as avoiding excessive shoulder extension during bench press and seated rows to minimize strain on the shoulder. Plyometric exercises are initiated between weeks 4 and 5 to train the upper extremity to absorb and develop forces. Two-hand plyometrics, such as chest passes, side throws, and overhead throws are performed initially, progressing to include 1-hand drills, such as baseball throws, in 7 to 10 days. The athlete is allowed to begin a gradual return to

sport-specific activities between the seventh and 10th postoperative week, typically using an interval sport program. The rate of return to overhead sports is often dependent on the extent of concomitant injuries. For example, an athlete with rotator cuff debridement involving 20% to 30% penetration of the rotator cuff will usually begin an interval sport program following these guidelines, while an athlete with more extensive pathology may need to delay initiation of the interval sport program for up to 4 months. An interval sport program is used to ensure that the athlete allows a gradual application of applied loads to the healing tissues.[44] The start date for initiating any interval sport program is often varied based on the time of year, the goals of the patient, and the competitive athletic season. The ultimate success of return to high-level activity following this procedure is dependent on the individual's ability to dynamically stabilize the glenohumeral joint during the performance of high-demand activities, thus appropriate and adequate rehabilitation is paramount.

Criteria to begin an interval return to sports activity includes minimal pain, full ROM, adequate strength and dynamic stability, and an appropriate rehabilitation progression as previously described.[39] To determine if the athlete has adequate strength, we perform isokinetic testing with the goals of achieving an ER peak torque/body weight ratio of 18% to 23%, an ER/IR ratio of 66% to 76%, and an ER/abduction ratio of 67% to 75% at 180°/s.[43,61-63]

Repair of Type II SLAP Lesions

Overhead-throwing athletes commonly present with a type II SLAP lesion, with the biceps tendon detached from the glenoid rim. Frequently a peel-back lesion is also present. The initial rehabilitative concern is to ensure that forces and loads on the repaired labrum are appropriately controlled. We believe that it is important to determine the extent of the lesion and understand its exact location and number of suture anchors in constructing an appropriate rehabilitation program. For instance, the rate of rehabilitation progression would be slower for a SLAP repair completed with 3 anchors than for a 1-anchor repair, based on the extent of the pathology and tissue involvement. Postoperative rehabilitation is delayed to allow healing of the more extensive anatomical repair required to reattach the biceps tendon anchor in a type II lesion, in comparison to type I and III lesions (Table 4).

The patient is instructed to sleep in a shoulder immobilizer and wear a sling during the daytime for the first 4 weeks following surgery to protect the healing structures from excessive motion. Gradual ROM in a protective range is performed for the first 4 weeks below 90° of elevation to avoid strain on the labral repair.[60] During the first 2 weeks, internal and ER ROM exercises are performed passively in the scapular plane to approximately 10° to 15° of ER and 45° of IR. Initial ER ROM is performed cautiously to minimize strain on the labrum through the peel-back mechanism. Performance of IR and ER ROM activities is progressed to 90° of shoulder abduction at week 4. Motion is gradually increased to restore full range of motion (90° to 100° of ER at 90° of abduction) by 8 weeks and progressed to thrower's motion (approximately 115° to 120° of ER) through week 12. Restoration of motion is usually accomplished with minimal difficulty.

Isometric exercises are performed immediately postoperatively. Exercises are initially performed with rhythmic stabilization drills for ER/IR and flexion/extension. These rhythmic stabilizations theoretically promote dynamic stabilization and cocontraction of the shoulder and rotator cuff musculature.[59,61-63] This concept is important when considering the underlying glenohumeral joint instability often observed with SLAP lesions. Rhythmic stabilizations may also be performed with manual resistance ER exercises by incorporating the alternating isometric contractions within sets of ER (Figure 12). Other exercises designed to promote proprioception, dynamic stability, and neuromuscular control include joint repositioning exercises and proprioceptive neuromuscular facilitation (PNF) drills.

ER/IR exercise tubing is initiated during weeks 3 through 4 and progressed to include lateral raises, full can, prone rowing, and prone horizontal abduction by week 6. As the patient progresses, a full isotonic exercise program, such as the thrower's ten program,[58,59,62,63] is initiated by weeks 7 through 8. Emphasis is placed on strengthening exercises for the external rotators and scapular stabilizations, such as sidelying ER, prone rowing, and prone horizontal abduction.[42] No resisted biceps activity (both elbow flexion and forearm supination) is allowed for the first 8 weeks to protect healing of the biceps anchor. Neuromuscular control drills are integrated, as tolerated, to enhance dynamic stability of the shoulder. These include rhythmic stabilization and perturbation drills incorporated into manual-resistance and exercise tubing exercises (Figures 13 and 14).

Aggressive strengthening of the biceps is avoided for 12 weeks following surgery. Furthermore, weight-bearing exercises are typically not performed for at least 8 weeks to avoid compression and shearing forces on the healing labrum. Two-hand plyometrics, as well as more advanced strengthening activities, are allowed between 10 and 12 weeks, progressing to the initiation of an interval sport program at postoperative week 16. The same criteria described previously are utilized to determine if an interval sport program is initiated. Return to play following the surgical repair of a type II SLAP lesion typically occurs at approximately 9 to 12 months following surgery.

TABLE 4. Rehabilitation protocol following arthroscopic type II SLAP repair.

I. Phase 1: immediate postoperative phase "protected motion" (day 1-week 6)

Goals
- Protect the anatomic repair
- Prevent negative effects of immobilization
- Promote dynamic stability
- Diminish pain and inflammation

Week 0-2
- Sling for 4 weeks
- Sleep in immobilizer for 4 weeks
- Elbow/hand PROM
- Hand-gripping exercises
- Passive and gentle shoulder active assistive ROM exercise
    - Flexion to 60° (week 2, flexion to 75°)
    - Elevation in scapular plane to 60°
    - ER/IR with arm in scapular plane
    - ER to 10°-15°
    - IR to 45°
    - No active ER or extension or abduction
- Submaximal isometrics for shoulder musculature
- No isolated biceps contractions
- Cryotherapy, modalities as indicated

Week 3-4
- Discontinue use of sling at 4 weeks
- Sleep in immobilizer until week 4
- Continue gentle ROM exercises (PROM and AAROM)
    - Flexion to 90°
    - Abduction to 75°-85°
    - ER in scapular plane to 25°-30°
    - IR in scapular plane to 55°-60°
    (Note: rate of progression based on evaluation of the patient.)
- No active ER, extension, or elevation
- Initiate rhythmic stabilization drills
- Initiate proprioception training
- Tubing ER/IR at 0° abduction
- Continue isometrics
- Continue use of cryotherapy

Week 5-6
- Gradually improve ROM
    - Flexion to 145°
    - ER at 45° abduction: 45°-50°
    - IR at 45° abduction: 55°-60°
- May initiate stretching exercises
- May initiate light (easy) ROM at 90° abduction
- Continue tubing ER/IR (arm at side)
- PNF manual resistance
- Initiate active shoulder abduction (without resistance)
- Initiate "full can" exercise (weight of arm)
- Initiate prone rowing, prone horizontal abduction
- No biceps strengthening

II. Phase 2: intermediate phase: moderate-protection phase (weeks 7-12)

Goals
- Gradually restore full ROM (week 10)
- Preserve the integrity of the surgical repair
- Restore muscular strength and balance

Week 7-9
- Gradually progress ROM
    - Flexion to 180°
    - ER at 90° abduction: 90°-95°
    - IR at 90° abduction: 70°-75°
- Continue to progress isotonic strengthening program
- Continue PNF strengthening
- Initiate thrower's ten program
- May begin AROM biceps

TABLE 4. (continued)

Week 10-12
- May initiate slightly more aggressive strengthening
- Progress ER to throwers motion
  - ER at 90° abduction: 110°-115° in throwers (weeks 10-12)
- Progress isotonic strengthening exercises
- Continue all stretching exercises
  - Progress ROM to functional demands (ie, overhead athlete)
- Continue all strengthening exercises

III. Phase 3: minimal protection phase (weeks 12-20)

Goals
- Establish and maintain full PROM and AROM
- Improve muscular strength, power and endurance
- Gradually initiate functional activities

Criteria to enter phase III
- Full nonpainful AROM
- Satisfactory stability
- Muscular strength (good grade or better)
- No pain or tenderness

Weeks 12-16
- Continue all stretching exercises (capsular stretches)
- Maintain throwers motion (especially ER)
- May begin resisted biceps and forearm supination exercises
- Continue strengthening exercises
  - Throwers ten program or fundamental exercises
  - PNF manual resistance
  - Endurance training
  - Initiate light plyometric program
  - Restricted sport activities (light swimming, half golf swings)

Weeks 16-20
- Continue all exercise listed above
- Continue all stretching
- Continue throwers ten program
- Continue plyometric program
- Initiate interval sport program (throwing, etc)
  - See interval throwing program

IV. Phase 4: advanced strengthening phase (weeks 20-26)

Goals
- Enhance muscular strength, power, and endurance
- Progress functional activities
- Maintain shoulder mobility

Criteria to enter phase IV
- Full nonpainful AROM
- Satisfactory static stability
- Muscular strength 75%-80% of contralateral side
- No pain or tenderness

Weeks 20-26
- Continue flexibility exercises
- Continue isotonic strengthening program
- PNF manual-resistance patterns
- Plyometric strengthening
- Progress interval sport programs

V. Phase 5: return-to-activity phase (months 6 to 9)

Goals
- Gradual return to sport activities
- Maintain strength, mobility and stability

Criteria to enter phase V
- Full functional ROM
- Muscular performance isokinetic (fulfills criteria)
- Satisfactory shoulder stability
- No pain or tenderness

Exercises
- Gradually progress sport activities to unrestrictive participation
- Continue stretching and strengthening program

Abbreviations: ROM, range of motion; ER, external rotation; IR, internal rotation; PROM, passive range of motion; AAROM, active assisted range of motion; PNF, proprioceptive neuromuscular facilitation.

FIGURE 12: Sidelying manual resistance external rotation. The clinician may resist external rotation as well as scapular retraction with the proximal hand (A). End range rhythmic stabilizations and perturbations may be incorporated to enhance neuromusclar control (B).

FIGURE 13. Rhythmic stabilizations performed on a wall in the scapular plane with the hand placed on a unstable surface.

FIGURE 14. Perturbation and rhythmic stabilization drills incorporated into external rotation at 90° abduction with exercise tubing.

Often a type II SLAP repair may be performed with a concomitant glenohumeral stabilization procedure, such as a thermal capsular shrinkage, arthroscopic plication, or Bankart repair. In these instances the rehabilitation program must be a combined approach that considers the healing constraints inherent to both procedures. The reader is encouraged to review several published articles by the authors to learn more about these approaches.[56,61,62,63]

Repair of Type IV SLAP Lesion

The surgical repair of a type IV SLAP lesion with either a biceps repair, biceps resection of frayed area, or tenodesis follows much the same postoperative rehabilitation course as that outlined for a type II lesion, in that the ROM and exercise activities are progressed similarly. However, there are substantial differences related to controlling both active and resistive biceps activity, based on the extent of bicipital involvement. In cases where the biceps is resected, biceps muscular contractions may begin between 6 and 8 weeks postsurgery. Conversely, in the cases of repaired biceps tears or biceps tenodesis, we recommend no resisted or active biceps for 3 months following surgery, when the soft tissue is most likely healed. Light isotonic strengthening for elbow flexion is initiated between weeks 12 and 16 postoperatively and progresses gradually, as tolerated from that point. Full resisted biceps activity is not incorporated until weeks 16 to 20. Progression to sport-specific activities, such as plyometrics and interval sport programs, follows similar guidelines to those outlined for type II SLAP repairs.

SUMMARY

A wide variety of pathology may affect the superior aspect of the labrum. Clinical examination is often difficult due to the numerous injury mechanisms and various extent of labral pathology. Proper identification of the exact mechanism and specific severity of pathology is vital to accurately diagnose and manage these injuries. Surgical procedures to address SLAP

CLINICAL COMMENTARY

lesions vary from minimal debridement to extensive labral repair. We suggest postoperative rehabilitation based on the specific injury and surgical procedure performed, as well as an understanding of basic science related to injury and tissue healing. Rehabilitation places emphasis on gradually restoring ROM, strength, and dynamic stability of the glenohumeral joint while controlling forces on the healing labrum. The aim is for the patient to return to full functional activities as quickly and safely as possible. Because no outcome data exist, research regarding the efficacy of the rehabilitation guidelines that are provided in this article is warranted.

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