SLAP LESIONS IN THE OVERHEAD ATHLETE

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The authors report an 87% rate of return to preinjury levels of throwing in 54 baseball players, and an 84% rate of return to preinjury performance levels in pitchers after repair of type II SLAP lesions. The etiology, biomechanics, surgical repair, and rehabilitation are discussed in detail.

KEY WORDS: shoulder, superior labral tears, dead-arm syndrome, peel-back mechanism

The "dead arm" has recently come alive as a topic of interest in sports medicine. Poorly understood for decades, it now appears that one potential cause of the "dead arm" is the type II superior labrum anterior and posterior (SLAP) lesion. This condition, which causes the thrower to be unable to throw with his preinjury velocity and control because of a combination of pain and subjective unease in the shoulder, is extremely disabling and potentially career-ending to the overhead athlete. The ability to successfully diagnose, surgically manage, and rehabilitate this condition is a recent development and is the focus of this article.

HISTORICAL PERSPECTIVE: PAST AND PRESENT TREATMENT AND RESULTS

Rowe and Zarins[1] called attention to the psychological aspects of shoulder dysfunction when they described the traumatic voluntary shoulder dislocator as a potential psychopathic individual who willfully dislocated his shoulder despite the surgeon's best efforts. They warned against surgery in these individuals. Subsequently, Gibbons et al,[2] in a long-term follow-up study of voluntary shoulder dislocators, found that none had true psychological abnormalities.

In 1972, Neer[3] described subacromial impingement as the most common cause of shoulder pain, so it was only natural that overhead throwing injuries would be viewed as potential impingement problems in the 1970s. However, Tibone et al[4] examined results of open acromioplasty as treatment for shoulder pain in athletes and found a success rate (good and excellent results) of only 43% overall, with only 22% of throwing athletes returned to their preinjury level of competition. They concluded that acromioplasty should be approached with caution in the overhead athlete. This reinforced the conclusions of Kennedy et al,[5] who did simple coracoacromialligament division in swimmers with impingement symptoms and found a very low long-term success rate.

Dr. Frank Jobe et al[6] have described impingement-instability overlap. They postulated that repetitive throwing gradually stretched out the anterior capsuloligamentous complex, allowing anterosuperior migration of the humeral head during throwing, thus causing subacromial impingement symptoms. They reported[7] some success with open anterior capsulolabral reconstruction (50% return to pitching in a report of 12 pitchers).

Andrews et al[8] arthroscopically observed anterosuperior glenoid labrum tears in throwers and debrided them initially. They postulated this labral injury as a deceleration injury that occurred in the follow-through phase of throwing, with the biceps decelerating the elbow as it went into extension, thereby causing a traction injury to the anterosuperior labrum by virtue of the biceps root attachment to the anterosuperior labrum and glenoid. Snyder et al[9] subsequently described SLAP lesions in the general population but did not specifically relate them to the overhead athlete.

The next step in the evolution of our understanding of the dead arm in the overhead thrower came from Dr Christopher Jobe.[10] He described posterosuperior glenohumeral impingement ("internal impingement") in throwers with the arm in abduction and external rotation (the cocked position). He credited Walch et al[11] as describing this "internal impingement." Internal impingement refers to a spectrum of injury to the rotator cuff, glenoid labrum, and even bone as a result of abnormal contact between the superior-posterior glenoid and the articular side of the rotator cuff. Jobe hypothesized that the "internal impingement" in throwers might progressively worsen by gradual repetitive stretching of the anterior capsuloligamentous structures. His theory of anterior microinstability aggravating internal impingement further validated the premise of the treatment of this problem by anterior capsulolabral reconstruction even though the results of treatment of throwers by this procedure were unpredictable (50% return to pitching[7]).

Morgan et al[12] reported on 102 type II SLAP lesions (53 in throwers) without associated anterior instability that underwent arthroscopic repair of the SLAP lesions alone. They described three subtypes of type II SLAP lesions: anterior, posterior, and combined anterior-posterior (Fig 1). Burkhart and Morgan[13] described the dynamic "peel-back sign" with the shoulder in abduction and external rotation as the

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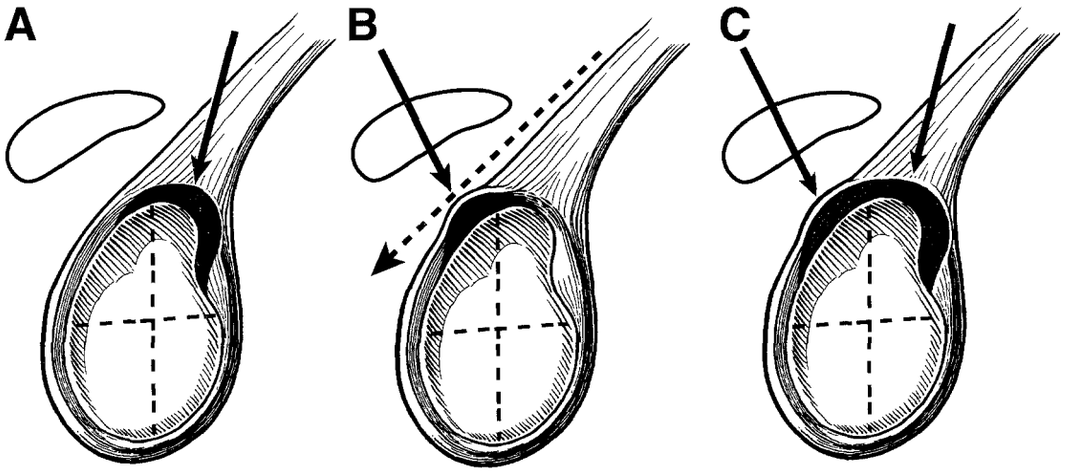

Fig 1. The subtypes of type II SLAP lesions by anatomic location: (A) anterior; (B) posterior; (C) combined anterior-posterior.

prime arthroscopic indicator of dysfunction of the biceps-labral complex, and advised SLAP lesion repair with suture anchors to eliminate the "peel-back sign." Interestingly, SLAP lesion repair not only eliminated the "peel-back" sign but also eliminated the "drive-through" sign that had been seen before repair in all patients. In the 53 throwers who had SLAP lesion repair only, all returned to throwing, and 87% returned to their preinjury level of throwing. Of the 53 throwers, 44 were baseball pitchers, of which 84% returned to their pre-injury level of activity or better. The remaining 9 pitchers who did not reach their preinjury level of success had associated undersurface rotator cuff tears. In this study, it was further noted that all throwers who presented with SLAP lesions had marked internal rotation deficits, as measured with the arm in the 90° abducted position, attributable to an acquired tight posterior-inferior capsule, which was implicated as a potential cause of the SLAP lesion in this population.

Nonsurgical prevention of SLAP lesions in throwers has focused on stretching of the tight posterior-inferior capsule that exists even in asymptomatic throwers. A preseason stretching program among professional baseball pitchers is the subject of an ongoing study by one of us (C.D.M.).

THE PEEL-BACK SIGN

We have observed a dynamic peel-back phenomenon[13] arthroscopically in throwers with posterior and combined anterior and posterior SLAP lesions. When the arm is removed from traction and brought into abduction and external rotation, the biceps tendon assumes a more vertical and posterior angle. This dynamic angle change produces a twist at the base of the biceps, which then transmits a torsional force to the posterior superior labrum, causing it to rotate medially over the corner of the glenoid onto the posterior superior scapular neck. **In** addition, the biceps root shifts medially to the supraglenoid tubercle (Fig 2). This is a consistent finding in patients with posterior SLAP lesions or combined anterior/posterior SLAP lesions, and it is absent in normal shoulders. Furthermore, the dynamic peel-back sign is eliminated by repair of the SLAP lesion. **In** anterior SLAP lesions, the dynamic peel-back sign may

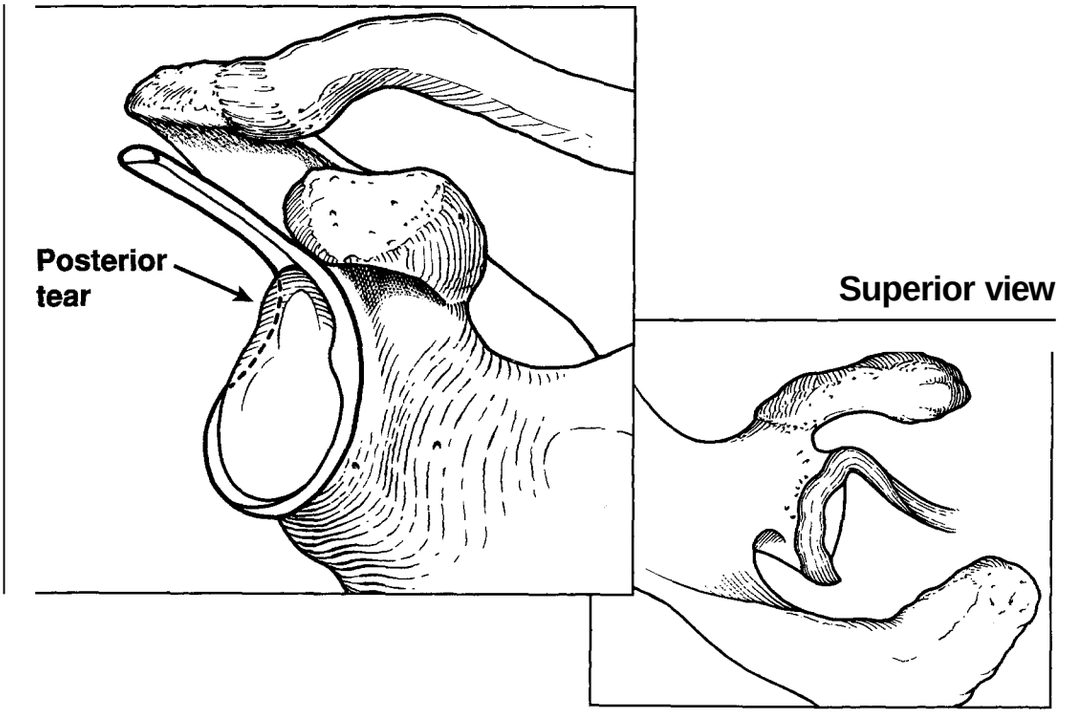

Fig 2. Peel-back mechanism. The biceps vector shifts posteriorly as the tendon twists at its base, causing the unstable posterior-superior and labrum to shift medially on the glenoid neck. In combined anterior-posterior type II SLAP lesions, the biceps root also shifts medially during the peel-back.

be diminished or even absent, because the posterior-superior labral attachment may be secure enough to prevent the labrum from rotating medially over the edge of the glenoid.

There are 2 clinical implications of this peel-back mechanism, the first regarding surgical repair and the second regarding post-SLAP repair rehabilitation. To surgically repair the posterior SLAP lesion, this torsional peel-back must be neutralized. This requires at least 1 suture anchor stabilizing the labrum posterior to the biceps to effectively cmillter the torsion. Suture anchors should be placed at the corner of the glenoid at a 45° angle of insertion to most effectively restore the anatomy in a mechanically effective way. Usually, a separate posterolateral portal should be used for the posterior anchor, and an anterolateral portal can be used for an anterior anchor.

The second clinical implication applies to postoperative rehabilitation. An important part of the postoperative program in many of our shoulder arthroscopy patients, particularly those with rotator cuff repairs, is to emphasize early passive external rotation. However, in posterior SLAP lesion repair patients, we now avoid early (first 3 weeks postoperatively) external rotation past 0° because that is where we have observed the peel-back phenomenon, even with no abduction (that is, peel-back occurs with external rotation only). Because external rotation stresses the repaired posterosuperior labrum, we now wait 3 weeks before allowing external rotation beyond 0°.

BIOMECHANICS: POSTEROSUPERIOR INSTABILITY, THE "CIRCLE CONCEPT," AND THE TIGHT POSTERIOR INFERIOR CAPSULE

Type II SLAP lesions, as originally described by Snyder et al,[9] were the most common type of labral injury and constituted approximately 50% of all SLAP lesions.[14] Based on our experience with overhead/throwing athletes, which constituted approximately 50% of our series, we have observed 2 subtypes of type II SLAP lesions (posterior and combined anterior-posterior) that have a posterior component. Type II SLAP lesions with a posterior component represented 62% of all SLAP lesions in our series and were 3 times more common in throwers than in the nonthrowing trauma group. These posterior type II SLAP lesions can cause extreme dysfunction in the throwing/overhead athlete as well as produce significant symptoms with the activities of daily living in the nonthrower.

In 1985, Andrews et al[8] reported on superior labral injuries in high-level baseball pitchers. They recommended debridement of these lesions rather than repair, but one must remember that suture anchor fixation was not available in 1985. Snyder et al[14] have reported good results from repair of type II SLAP lesions. However, they did not distinguish these lesions by anatomic location: anterior, posterior, or combined (anterior-posterior) type II SLAP lesions.

In their elegant macroscopic and microscopic study of the glenoid labrum, Huber and Putz[15] concluded that the tendon fibers of the long head of the biceps continued posteriorly as periarticular fiber bundles, and that the bulk of the "labrum" in the posterior superior quadrant of the glenoid actually consisted of these extended biceps periarticular fiber bundles. Vangsness et al[16] have reported similar anatomic findings and have additionally shown an anatomic variation in which the biceps fibers predominantly course posterior to the root of the biceps attachment (posterior-dominant biceps configuration). Huber and Putz,[15] based on their anatomic study, also postulated a "periarticular fiber system" consisting of the labrum, glenohumeral ligaments, and inserting tendons (biceps, triceps). They suggested that this system of parallel collagen fibers surrounding the circumference of the glenoid acted as a "basket" or "tension-brace" to provide hoop stresses at the periphery. In effect, the labrum would act as a bumper in all directions to resist dislocation or subluxation of the humeral head. The "bumper" function of the labrum and glenohumeral ligaments has long been recognized as an important restraint against anterior instability. In support of this concept, Lippitt and Matsen[17] showed that the glenoid labrum was important in resisting subluxation in all directions, including superior subluxation. In addition to the "bumper" effect, a circumferentially intact labrum creates a "suction-cup" effect to further enhance stability.

In view of these anatomic features of the biceps-Iabral complex, one can postulate the peel-back mechanism of the biceps (Fig 2) in a posterior-dominant labral configuration as the cause of disruption of the posterior superior labral attachment from the glenoid as well as the root of the biceps from the supraglenoid tubercle, leading to posterosuperior instability or combined anterior/posterior superior instability. During glenohumeral arthroscopy, we have observed a dynamic torsion of the root of the biceps in the abducted and externally rotated position (cocked throwing position) with peel-back of a pathologic posterior superior labrum off the glenoid (Fig 3).

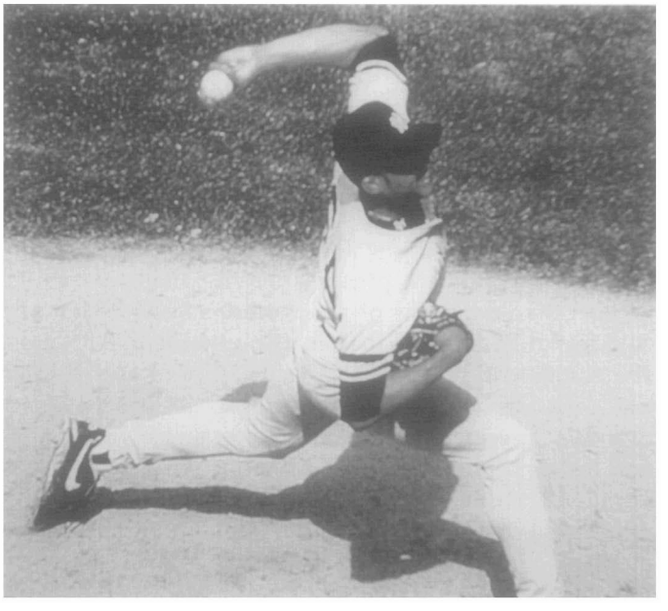

Fig 3. This baseball pitcher is in the late cocking phase of throwing, with the shoulder maximally externally rotated at 90° abduction. In this position, the peel-back forces are maximized.

It is our impression that detachment of the posterosuperior labrum causes a disabling secondary posterosuperior instability problem. The humeral head cannot dislocate superiorly because the acromion blocks proximal humeral translation before it can dislocate. However, repetitive superior translation or subluxation could damage the rotator cuff from inside the joint, and we have noted a high incidence (31%) of injury to the rotator cuff in patients with chronic SLAP lesions. Conversely, no associated rotator cuff pathology was seen in patients treated for acute SLAP lesions.

In our series, partial or complete rotator cuff tears were found in 31% of shoulders with type II SLAP lesions. These tears were lesion-specific in location. The partial tears associated with anterior type II SLAP lesions were all undersurface and were located within the anterior to mid-portion of the rotator crescent. The partial tears associated with posterior SLAP lesions were all undersurface and were located in the posterior part of the rotator crescent. This lesion-specific location of the cuff tear is consistent with the concept of superior instability as the main factor causing the cuff to tear from inside-out, with the tear location corresponding to the location of increased tensile forces that are created in the muscle-tendon units that are maximally stressed in the zone of superior instability.

In our series, the patients with posterior superior type II SLAP lesions all had some degree of positive drive-through sign at the time of arthroscopy but no Bankart lesion or anterior inferior labral pathology. Repair of the posterior SLAP lesion eliminated the drive-through sign in all of these patients. This observation is consistent with the "circle concept" of the periarticular labral fibers acting as a unit, so that the disruption of the fibers in one part of the labrum may manifest itself as instability on the opposite side of the glenoid. Pagnani et al[18] have recently reported in a cadaveric model "that a complete lesion of the superior portion of the labrum that destabilized the insertion of the biceps resulted in significant increases in anteroposterior and superior inferior glenohumeral translation." Repairing these posterosuperior labral fibers results in restoration of the circle and elimination of the anterior-inferior pseudolaxity. In all of our cases, repair of the posterior SLAP lesion eliminated the drive-through sign.

One of the authors (C.D.M.) has noted and recently reported a consistent physical finding that aids in the preoperative diagnosis of the posterior SLAP lesion and clinically differentiates it from the anterior SLAP lesion. The standard tests for SLAP lesions (Speed and O'Brien tests) are negative with posterior SLAP lesions and positive with anterior SLAP lesions. However, the Jobe relocation test is positive with the posterior SLAP lesions and negative with the anterior SLAP lesions. The Jobe relocation test is highly sensitive and highly specific for the posterior SLAP lesion. The combined SLAP lesions had features of both types of lesions on physical examination, with less specificity.

Posterosuperior glenoid impingement (internal impingement) has been postulated to result from anterior instability in throwers[6] who then developed posterior-superior rotator cuff pathology as a direct mechanical effect of contact between the cuff and the glenoid.[10] Furthermore, it has been postulated that entrapment of the cuff between the glenoid and greater tuberosity occurs in the abducted and externally rotated position, resulting in damage to the rotator cuff. If this is true, we ask, why do not all throwers develop this problem? We disagree with this concept. Instead, we suggest that this internal impingement lesion of the rotator cuff is secondary and part of the spectrum of pathology associated with the posterior SLAP lesion. Repetitive posterior superior subluxation combined with repetitive twisting of the cuff fibers during the cocking phase of the throwing motion probably results in fiber fatigue and failure of the cuff in the anatomic zone of instability.

To repair a posterior SLAP lesion, a unique posterosuperior lateral acromial portal (Port of Wilmington) (Fig 4) must be used to allow an adequate angle of approach for suture anchor placement into the posterosuperior glenoid. This area cannot be adequately approached through an anterosuperior portal because the angle of approach through that portal is almost parallel to the bone surface, precluding anchor placement. We recommend placing the anchors at the corner of the glenoid articular margin at a 45° angle (Fig 5). For anterior SLAP lesions, this necessitates a portal at the anterolateral tip of the acromion. For combined lesions, we suggest placement of anchors, both posterior and anterior to the biceps root, to stabilize the lesion to bone. In our experience, placement of anchors within the body of the biceps is unnecessary and may cause biceps fiber injury within the body of the biceps tendon substance.

The posterior SLAP lesion can prevent the athlete from effectively using the shoulder in skilled sports. Repair of the posterior SLAP lesion can return the overhead athlete to preinjury levels of activity with an 84% success rate.[12] We hypothesize that SLAP lesions with a posterior component develop posterosuperior instability that manifests itself by an anterior pseudolaxity (drive-through sign), and that chronic superior instability leads to lesion-specific rotator cuff tears that begin from inside the joint as partial-thickness tears, which then may enlarge and become full-thickness tears.

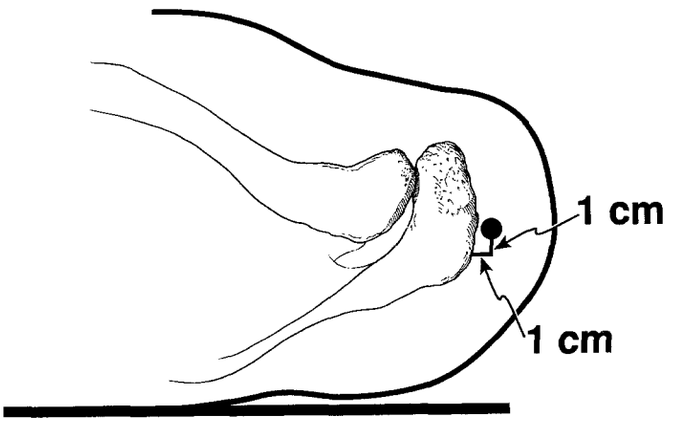

Fig 4. The posterolateral acromial portal used to approach posterior type II SLAP lesions is 1 cm lateral and 1 cm anterior to the posterior acromial angle.

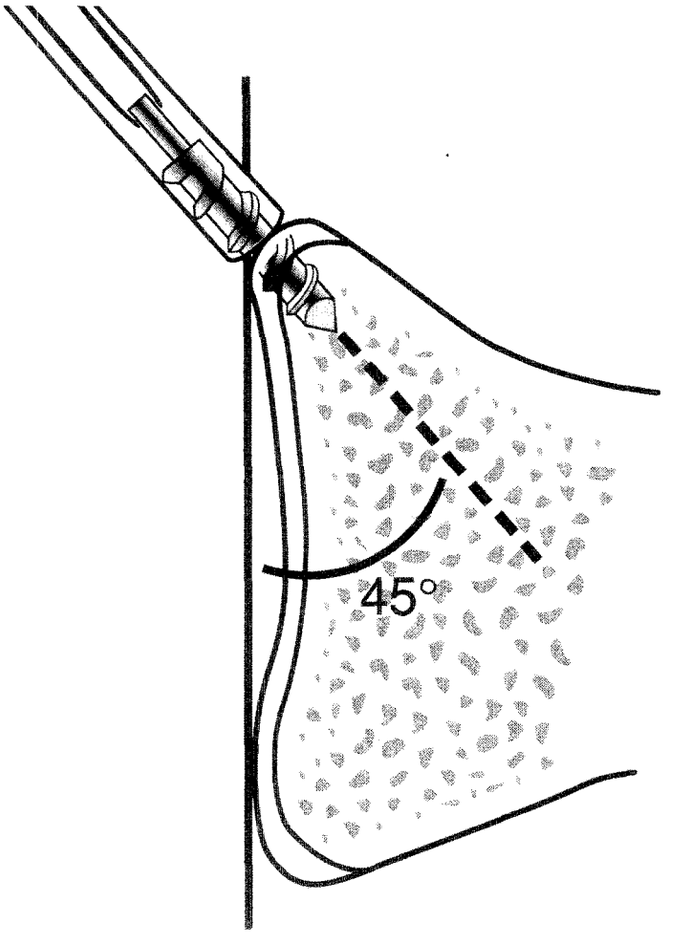

Fig 5. Posterior type II SLAP repair through the posterolateral acromial portal accesses the glenoid at a 45° angle to the glenoid articular surface.

Lastly, we believe, based on our preoperative range of motion data in throwing/overhead athletes, that a tight posterior capsule with marked lack of internal rotation greater than 40° compared with the nonthrowing shoulder predisposes this select group to develop type II SLAP lesions. This relationship is the subject of an ongoing prospective study. Supporting this concept, we have identified severe loss of internal rotation in the throwing/overhead athlete as a risk factor for the development of shoulder problems. Furthermore, maintenance of a flexibility program to minimize lack of internal rotation can decrease the incidence of shoulder problems in the throwing athlete.

THE SLAP LESION IN THROWERS: ACCELERATION VERSUS DECELERATION INJIURY AND PROPOSED MECHANISM

Andrews et al[18] postulated a deceleration mechanism for labral injuries in throwers as the biceps contracts to slow down the rapidly extending elbow in follow-through. Kuhn et al[19] have produced a SLAP lesion by applying a tensile force simulating the above scenario of a biceps force during deceleration. However, the labral avulsion was produced in only 20% of specimens, with a large tensile force (346 ± 40 N). They also loaded cadaver specimens in the abducted-externally rotated position of late cocking and consistently produced type II SLAP lesions at a force of 289 ± 39 N, 20% less than for the deceleration protocol. Importantly, they were able to produce type II SLAP lesions in 9 of 10 of the specimens in the abducted-externally rotated position, but in only 2 of 10 of those in the deceleration position ($P = .055$).

In the throwers who recall the pitch that caused their injury, we have found that they invariably relate the severe sudden onset of pain to the abducted-externally rotated position of late cocking, as the arm begins to accelerate forward. If one assumes that the thrower has a "shoulder at risk" with a tight posterior inferior capsule, then as the shoulder abducts and externally rotates into the cocked position, the tight posterior inferior capsule of a right shoulder would go through an arc of motion from 7 o'clock to 4 o'clock. As this tight band passes inferior to the humeral head, it pushes superiorly against the humeral head and causes a posterosuperior shift of the glenohumeral fulcrum, or contact point between the glenoid and humerus. Once this shift occurs, the shoulder will then externally rotate fully around this new rotation point, which causes an increased contact at the internal impingement zone of cuff-labrum contact and causes increased forces at the posterior superior biceps/labralattachment with the peel-back mechanism. This force could produce the SLAP lesion, which in turn magnifies the posterior superior shift/instability problem. It is at this point in the late cocking phase that the peel-back forces are maximized. Therefore, we believe that these lesions are not deceleration injuries, but more likely acceleration injuries with the shoulder in abduction and external rotation. It seems likely that the biceps-superior labrum complex is not pulled from bone, but rather is peeled from bone.

EVALUATION

The history in throwers is typical: the athlete has an acute searing pain in the late cocking phase as the arm begins to accelerate from its position of maximal external rotation. A significant number of athletes will have prodromal symptoms of posterior superior shoulder pain, often accompanied by medial elbow pain, which is a secondary phenomenon in the thrower with a tight posterior-inferior capsule.

On physical examination, there is often a dynamic winging of the scapula caused by a tight posterior-inferior capsule and periscapular muscle weakness leading to scapular muscle dyskinesis. The winging of the scapUla is most noticeable if the examiner has the patient lie face-down on the examining table and place his hands on his hips.

Three tests have been shown to have predictive value for distinguishing anterior type II SLAP lesions from posterior type II SLAP lesions. These tests and their significance are:

1. Positive Speed test producing anterior shoulder pain in the bicipital groove region with resistance to forward flexion with the shoulder flexed 90° and the forearm supinated. A positive Speed test suggests an anterior type II SLAP lesion.
2. Positive O'Brien cross-arm test producing anterior shoulder pain against resistance with the shoulder forward flexed 90°, adducted to the midline, and the forearm

supinated. A positive O'Brien test is common with anterior type II SLAP lesions.

3. Positive Jobe relocation test in which the posterosuperior shoulder pain produced by abduction and external rotation is relieved by a posteriorly directed force applied to the proximal humerus. This is the most reliable test on physical examination for posterior type II SLAP lesions.

Radiographic evaluation is usually normal. Posterior calcification has been previously noted in throwers, but is not commonly seen with type II SLAP lesions. Magnetic resonance imaging (MRI) scans, including gadolinium-enhanced MRI, usually do not show these lesions. Definitive diagnosis of type II SLAP lesions is generally possible only by direct arthroscopic evaluation.

ARTHROSCOPIC EVALUATION AND TREATMENT

Arthroscopic findings vary according to the location of the type II SLAP lesion (anterior, posterior, or combined anterior-posterior). Anterior type II SLAP lesions are characterized by 2 arthroscopic findings:

a) "Uncovered" glenoid for 5 mm or more medial to the corner of the glenoid under the biceps root
b) "Displaceable vertex" of the biceps root, indicating an unstable biceps anchor

Posterior Type II Slap lesions display the following arthroscopic features:

a) "Uncovered" glenoid for 5 mm or more medial to the corner of the glenoid
b) Positive "peel-back" sign with the shoulder in 60° abduction and full external rotation
c) Positive "drive-through sign" in which the scope sheath can be passed superior to inferior

Combined anteroposterior type II SLAP lesions have findings of both anterior and posterior lesions. It is important to recognize that anterior lesions typically do not display a positive peel-back sign because the posterosuperior labral attachment is intact and prevents peel-back of

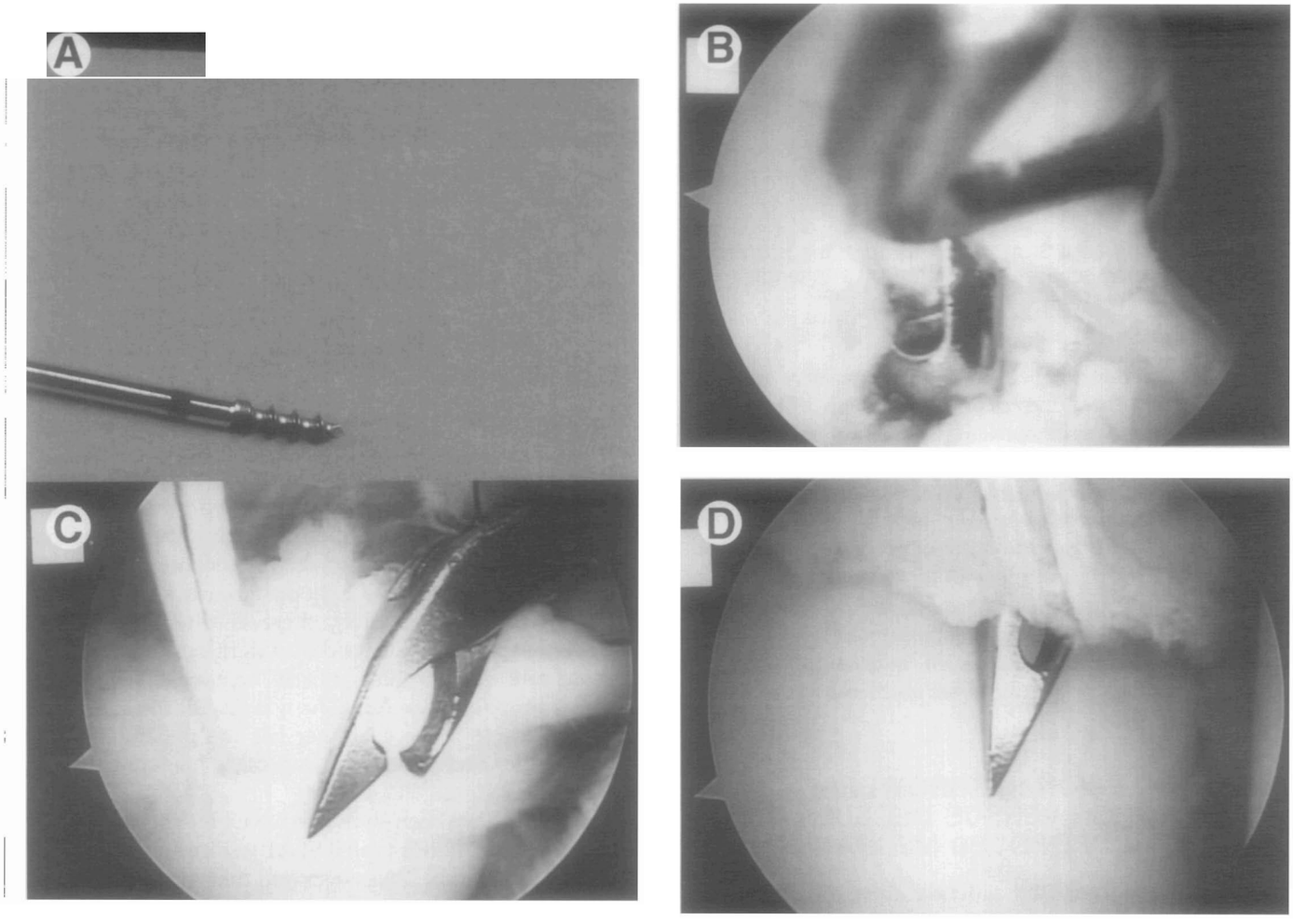

Fig 6. Arthroscopic repair by suture anchors: (A) Small 2-mm diameter suture anchor with inserter (FasTak; Arthrex, Inc., Naples, FL). (B) A 2.5-mm delivery device (Spear; Arthrex, Inc, Naples, FL) is used for translabral insertion of the suture anchor into the corner of the glenoid at the base of the biceps in a right shoulder with a combined anterior-posterior lesion. (C) A posterior anchor has been placed through a posterolateral acromial portal. The anchor was placed directly into bone, and now one suture limb must be placed translabrally with the 45° suture passer (Bird Beak; Arthrex, Inc, Naples, FL). (0) One limb of posterior suture is passed through labrum with 45° suture passer (Bird Beak; Arthrex, Inc, Naples, FL), which has penetrated the labrum to retrieve the suture limb position for the stretch, places the upper arm perpendicular to the trunk, with the elbow flexed 90° and the forearm vertical.

TABLE 1. Rehabilitation Protocol for Throwers

Week 1	Sling immobilization at all times
Weeks 2-3	Codman circumduction, PROM: 0°-90° abduction, and external rotation in adduction. No external rotation in abduction because of the peel-back mechanism. Sling immobilization when not doing PROM regimen.
Weeks 3-6	Discontinue sling. Progressive PROM to full as tolerated in all planes. Begin passive posterior capsular and internal rotation stretching. Begin passive and manual scapulothoracic mobility program. Begin external rotation in abduction. Allow use of the operative extremity for light activities of daily living.
Weeks 6-16	Continue all stretching and flexibility programs as above. Begin progressive strengthening of the rotator cuff, scapular stabilizers, and biceps.
4 Months	Begin interval throwing program, on level surface. Continue stretching and strengthening regimen, with particular emphasis on posterior capsular stretching.
6 Months	Begin throwing from the mound.
7 Months	Allow full-velocity throwing from the mound. Continue strengthening and posterior capsular stretching long-term (indefinitely). Remember an occult tight posterior capsule caused the SLAP lesion to begin with, and recurrence of the tightness can be expected to place the repair at risk in a throwing athlete.

the labrum with abduction and external rotation of the shoulder.

ARTHROSCOPIC REPAIR AND REHABILITATION

All 102 of our patients underwent arthroscopic repair of their type II SLAP lesions by use of suture anchors. Anterior lesions were repaired by placing the anchor from a standard anterosuperior approach, just off the anterior lateral tip of the acromion. However, this standard anterior portal does not provide a satisfactory angle of approach for placement of suture anchors to the posterosuperior glenoid. For labral repair in the posterosuperior quadrant of the glenoid, we used a posterosuperior lateral acromial portal (Port of Wilmington) for suture anchor placement. The skin incision for this portal was made 1 cm lateral and 1 cm anterior to the posterior acromial angle adjacent to the lateral acromial margin (Fig 4). The vector of entry into the shoulder began at this skin incision and angled toward the coracoid tip anteriorly. Suture anchors were placed at the articular margin and angled 45° medially to assure bony purchase (Fig 5). The anchors were placed through the repositioned soft tissue into bone in a single maneuver by using a small-diameter screw-in suture anchor deployed through a 2.5-mm delivery device (FasTak; Arthrex, Inc., Naples, FL) (Fig 6). Knot tying was accomplished in a vertical fashion by bringing one limb of the double-limb sutures through the SLAP defect with a crochet hook before knot tying. One of the authors (C.D.M.) used absorbable no. 1 polydiaxone (PDS) sutures in an attempt to avoid any permanent irritant in the shoulder, particularly in a throwing athlete. Another author (S.s.B.) used permanent no. 2 Ethibond sutures (Ethicon, Somerville, NJ) in 21 cases.

The postoperative rehabilitation protocol used for overhead/throwing athletes (group 1) after SLAP repair is summarized in Table 1. The rehabilitation program for nonthrowers (group II) was similar with regard to the range of motion and stretching program but obviously was devoid of the interval throwing regimen. The nonthrowers were allowed to return to stressful biceps activity, heavy lifting, and manual labor at 3.5 to 4 months.

THE SHOULDER AT RISK: THE 180° RULE

Finally, there is the matter of the "shoulder at risk." Pitchers with an acquired tight posterior inferior capsule and tight scapulothoracic articulation are the most likely to develop the "dead arm syndrome."[20] This posteroinferior capsular contracture is acquired in the throwing athlete and presents as internal rotation deficit with the arm in the 90° abducted position. The healthy throwing shoulder will present with increased external rotation in abduction at the expense of internal rotation. If the gain of external rotation equals the loss of internal rotation, allowing a 180° arc of motion, problems will be avoided. The shoulder with a posterior inferior capsular contracture that restricts the total arc to less than 180° is truly a "shoulder at risk." Most pitchers who develop an acute posterior superior SLAP

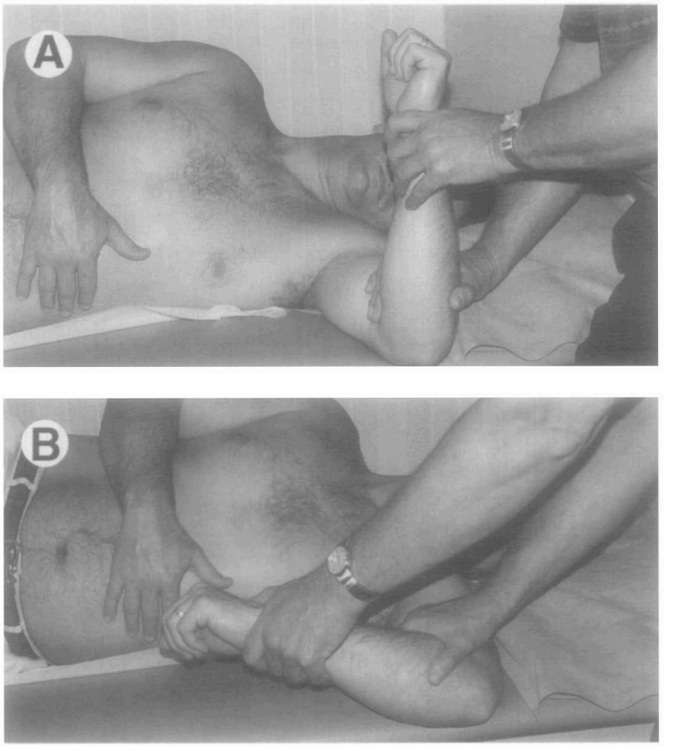

Fig 7. (A) To effectively isolate and stretch the posterior-inferior capsule, the athlete must be positioned in a 45° rotated position (midway between supine and lateral decubitus) so that his scapula is stabilized by direct pressure from the body onto the table. Note that the scapula is parallel to the table. The starting position for the stretch places the upper arm perpendicular to the trunk, with the elbow flexed 90° and the forearm vertical. (B) The trainer or therapist then stabilizes the elbow and upper arm as he internally rotates the shoulder. The goal is to obtain enough stretch so that the forearm will lie down flat on the table.

lesion go through a prodromal phase of mild posterior pain with a sense of posterior tightness before the event occurring. In addition, some may complain of associated activity-related bicipital groove pain. Pitchers in this prodromal phase can be successfully treated with a focused posterior capsular stretching program (Fig 7) and scapulothoracic stretching program to resolve the clinical condition. Trainers have directed a great deal of attention to this area and have developed stretching protocols for these throwers. A tight posteroinferior capsule will tend to push the humeral head toward the posterior superior quadrant of the glenoid as the shoulder goes into full external rotation in the late cocking phase of throwing, as already has been described. This humeral head force can potentially overload the posterior superior labrum and undersurface of the posterosuperior rotator cuff at the exact moment that it is most vulnerable from the peel-back mechanism that produces its maximum torsional effect in the late cocking phase of throwing (abduction plus extreme external rotation of the shoulder).

CONCLUSIONS

1. There are 3 distinct categories of type II SLAP lesions: anterior, posterior, and combined anteroposterior.
2. Posterior type II SLAP lesions have distinct clinical and anatomic features that distinguish them from anterior type II SLAP lesions.
3. Posterior and combined Type II SLAP lesions can be disabling to overhead athletes because of posterosuperior instability and anteroinferior pseudolaxity.
4. The Jobe relocation test is positive with posterosuperior pain in patients with posterior or combined anteroposterior type II SLAP lesions and is negative in patients with anterior type II SLAP lesions.
5. Rotator cuff tears are frequently associated with posterior or combined anteroposterior SLAP lesions, are lesion-location-specific, and typically begin from inside the joint as undersurface tears.
6. Repair of posterior SLAP lesions can return the overhead athlete to full overhead athletic function.
7. The peel-back mechanism is a likely cause of posterior type II SLAP lesions.
8. To securely repair the posterosuperior labrum to resist torsional peel-back, suture anchors must be placed posterior to the biceps at the corner of the glenoid.
9. The posterior SLAP lesion repair must be protected against external rotation greater than 0° for 3 weeks to avoid undue premature torsional stresses on the repair from the peel-back mechanism.
10. A tight posterior inferior capsule predisposes to a type II SLAP lesion in the overhead athlete.
11. The "shoulder-at-risk" for developing the dead-arm syndrome is one that has a marked loss of internal rotation caused by contracture of the posteroinferior capsule such that there is less than a 180° arc of rotation with the arm abducted 90° (The 180° Rule).
12. Type II SLAP lesions that cause the "dead arm" in overhead athletes are most likely acceleration injuries that occur in late cocking rather than deceleration injuries in follow-through.
13. Rehabilitation of athletes with the "dead arm" must include the entire kinetic chain.
14. The root cause of the "dead-arm syndrome" is the type II SLAP lesion.

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