

Current Controversies
Point Counter Point

Labrum/Biceps/Cuff Dysfunction in the Throwing Athlete

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What is the essential lesion of the dead arm syndrome in throwing athletes? Some high-performance throwers find it impossible to throw after an injury. Why is that? What are the findings at arthroscopy for this condition and why do they occur? Answers to these questions are essential to understand the most appropriate treatment for this condition. The following debate sets out two opposing positions about the etiology of this condition. These opposing views center on the issue of which side of the shoulder is the principal lesion and which needs the repair: the anterior inferior or the posterior superior. This debate has clinical significance because understanding the correct cause will necessarily point to which surgical solution holds the greater promise for success.

The classic position advanced by Dr. Frank Jobe¹ is that these athletes have anterior stretching of their capsules and that this leads to internal impingement. A capsulolabral repair will treat the laxity component found anteriorly, resolving the internal impingement. Dr. Christopher Jobe believes that there are several causes of glenoid impingement, not just anterior capsular stretching. Usually more than one of these is applicable to a specific clinical case. He feels it is this glenoid impingement that leads to the SLAP tear.

Drs. Morgan and Burkhart counter that the posterior superior SLAP is the essential lesion and that any impingement is secondary to that. A positive drive-

through sign will be eliminated and the thrower returned to throwing in a very high percentage of cases with the repair of the SLAP lesion. The laxity observed is a "pseudolaxity" due to the breaking of the other side of the ring.

It is only with the data provided by the arthroscope that we learn more about this condition. Developing a clear understanding of the etiology and resultant pathology allows us to develop surgical strategies to correct the condition. Debates such as this one help us to focus on these issues and achieve a greater understanding of the problem.

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In approaching the issue of superior labrum/biceps/rotator cuff dysfunction in the throwing athlete, one can either try to describe the pathology or explain the pathology. If all we do is describe the pathology, we are no better than the blind man describing the elephant. We must use dynamic observations to gain insight into the cause of the pathology. This insight can then be used to formulate logical treatments that prevent or specifically correct the pathology. We feel that our approach^{1,2} provides a plausible explanation for the mechanism of injury (acquired tight posterior inferior capsule and peel back mechanism) that explains the dysfunction (posterosuperior glenohumeral

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instability and anteroinferior pseudolaxity) and targets the specific pathology that needs to be corrected (biceps/labral stabilization and posteroinferior capsular flexibility). On the other hand, the Jobe model³ is primarily a descriptive model. It properly describes posterosuperior glenoid impingement and it properly describes the anterior instability in the dead arm syndrome. However, it improperly ascribes the instability to progressive stretching of the middle and inferior glenohumeral ligaments and, therefore, improperly advises treatment by anterior capsulolabral plication.

POSTEROSUPERIOR GLENOID IMPINGEMENT MODEL—WALCH, JOBE, SIDLES

Walch⁴ observed contact between the posterosuperior glenoid and the intra-articular humeral neck junction with the arm at 90° of abduction and 90° or more of external rotation. He observed that this occurred in the normal shoulder without instability. Jobe³ expanded on this concept and applied it to the throwing athlete. He correctly noted injury to the under surface of the rotator cuff, superior labrum, greater tuberosity, and the biceps root. He also correctly observed anterior laxity in many of these patients. So far, so good. The descriptions are accurate. What is missing is failure to recognize the importance and biomechanical sequelae of an acquired tight posterior inferior capsule with marked lack of internal rotation in these throwing athletes. The Jobe model breaks down in its leap from a description of the pathology to treatment of the pathology. The critical flaw in this model is that it does not direct treatment toward repair of the abnormal structures (the damaged superior labrum and biceps root anchor and the excessively tight posterior inferior capsule), and it does not recognize that disruption of these structures alone (without stretching of the glenohumeral ligaments) can cause anterior pseudolaxity. Therefore, this model espouses an indirect approach to the problem of instability. Even though the instability is caused by a posterosuperior labral detachment, Jobe approaches it on the opposite (wrong) side of the shoulder by anterior-inferior capsulolabral plication. A seamstress would never try to repair a ripped seam in a shirtsleeve by reinforcing the opposite side of the sleeve; she would repair the ripped seam. We would do well to follow her example.

MORGAN-BURKHART MODEL

We have based our model on 3 critical observations:

1. A type II posterosuperior glenoid labrum tear causes anterior pseudolaxity with a positive arthroscopic drive-through sign.
2. Combined abduction and external rotation in a shoulder with a type II posterosuperior glenoid labrum tear and unstable biceps anchor will cause the biceps-superior labral complex to “peel back” over the posterior superior corner of the glenoid. We believe that the anterior instability that we have observed with this lesion is a result of a “channeling” phenomenon of anterior-inferior pseudolaxity due to disruption of the labral “circle” posterosuperiorly (i.e., there is a channel of laxity from the area of disruption of the labral ring to the opposite side of the glenoid).⁵
3. A contracted posterior inferior capsule that presents clinically with marked lack of internal rotation in abduction is seen in all throwers who develop posterosuperior SLAP lesions. In our series of 53 overhead/throwing athletes (44 of whom were baseball pitchers) all presented with 25° or greater lack of internal rotation compared to their nonthrowing shoulder. We believe that this acquired, tight posterior capsule predisposes the shoulder to develop a posterior SLAP lesion by the following mechanism. In the presence of a tight posterior inferior capsule, as the shoulder abducts and externally rotates into the cocked position of throwing, the tight, posterior capsule will not allow full normal external rotation required for throwing. It acts as a check rein, which causes a posterosuperior shift of the glenohumeral rotation point. Once this shift occurs, the shoulder will then externally rotate fully around this new rotation point, which causes increased contact at the internal impingement zone and causes increased forces at the posterior superior biceps/labral attachment via the peel-back mechanism and produces the SLAP lesion that in turn magnifies the posterior superior shift/instability problem. Lastly, as this pathological sequence continues, the cuff begins to fail in tension from its undersurface in the zone of instability (posteroinferior).

The most critical point to understand about our model is that we have shown that suture anchor repair of the biceps-superior labral complex will immediately result in (1) elimination of the anterior pseudo-instability as

demonstrated by elimination of the drive-through sign, and (2) stabilization of the biceps/superior labrum complex demonstrated by elimination of the peel-back sign. In short, our model has pinpointed the pathology, directly treated the pathology, and shown that our treatment has eliminated the causes of dysfunction (instability and peel-back). In addition, it is critical in the rehabilitation of the SLAP repairs that a focused posterior capsular stretching program be continued indefinitely to avoid the development of an acquired tight posterior capsule, which initiates the pathological sequence.

The mechanism of injury is complex. We do not necessarily believe that a tensile force from the biceps during follow-through causes this lesion as postulated by Andrews et al.⁶ In fact, most of the athletes who recall the acute event that caused the injury will describe a sudden severe pain in late cocking, just as the arm begins to accelerate forcibly forward. It is at this point that the peel-back forces, which are magnified by a posterosuperior glenohumeral shift caused by a tight posterior inferior capsule, are maximized. Therefore, we believe that this is not a deceleration injury, but more likely an acceleration injury 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.

Finally, there is the matter of the “shoulder at risk.” It has been amply shown that pitchers with an acquired tight posterior inferior capsule and tight scapulothoracic articulation are the most likely to develop the dead arm syndrome.⁷ Most pitchers who develop an acute posterior superior SLAP lesion go through a prodromal phase of mild posterior pain with a sense of posterior tightness prior to 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 and scapulothoracic stretching program to resolve the clinical condition. Trainers have directed a great deal attention to this area and have developed stretching protocols for these throwers. A tight posterior inferior 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 has already been described. This humeral head force can potentially overload the posterior superior labrum and undersurface of the posterior-superior rotator cuff at the exact moment that it is most vulnerable from the peel-back mechanism that pro-

duces its maximum torsional effect in the late cocking phase of throwing (abduction plus extreme external rotation of the shoulder).

PARTING SHOTS

We agree with Walch⁴ that physiological posterior-superior glenoid contact occurs in normal individuals when the arm is in abduction and full external rotation. Therefore, this internal impingement, which is normal and physiological, cannot be the cause of the dysfunction in the throwing athlete or else all throwing athletes would develop this clinically pathological problem. Although there is a component of laxity in many of these throwers, we believe that this is a “pseudolaxity” from disruption of the labral ring around the glenoid, as well as loss of a stable biceps root anchor. Therefore, the correction of that laxity by anterior shoulder reconstruction is not logical, particularly in view of the fact that we have been able to eliminate this anterior “pseudolaxity” by simply repairing the biceps/labral detachment. Clinical results of SLAP repair alone in conjunction with a focused postoperative posterior capsular stretching program in our series of 44 pitchers showed a 100% return to sports with 84% at or better than their preinjury level of performance long term. These results are far superior to those reported by Jobe et al.⁸ with anterior capsulolabral repair in their series of baseball pitchers (50% return to their preinjury level of pitching). Athletic performance is the benchmark for success in the overhead athlete. These athletes will not accept less than a full return to their preinjury performance level. Neither should we.

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My understanding of the model that Morgan and Burkhart propose is as follows: The throwing athlete begins with normal anatomy and tension overload on the biceps in either the deceleration or follow-through phase of throwing produces, a type II SLAP lesion. By detaching the bony anchor of this portion of the labrum, instability is produced in the shoulder. This instability then allows glenoid impingement, which produces the incomplete rotator cuff tear. Normal shoulder (biceps tension overload) leads to a type II SLAP. The SLAP allows for shoulder instability, which leads to glenoid impingement and rotator cuff damage.

GLENOID IMPINGEMENT MODEL—WALCH-JOBE-SIDLES

According to this model, the throwing athlete begins with normal anatomy, which is capable in its normal condition of making glenohumeral contact. Overapplication of this glenohumeral contact either on an acute or a chronic basis, through increased frequency or increased load, leads to damage to 1 or more of 5 tissues: (1) superior labrum, (2) biceps origin—internal fibers of rotator cuff, (3) bony glenoid, (4) greater tuberosity, and (5) inferior glenohumeral ligament. As a rule, more than 1 of these tissues is damaged producing a variable pattern of injury depending on which of these structures is predisposed to damage. In throwers, it is our current belief that it is the internal fibers of the rotator cuff and the superior labrum that tend to be injured earlier in the course of the disease in comparison with the inferior glenohumeral ligament. Initially, this injury tends to produce stiffness but, if continued loading is allowed, a posteriorly located pain will be produced (an anterior pain on the reduction test may point to a SLAP lesion). Continued loading after the development of pain will

lead to stretching of the inferior glenohumeral ligament and subsequent instability and the need for surgical reconstruction rather than correction through physical rehabilitation.

DISCUSSION

In the Morgan-Burkhart model, the first step is tension overload on the biceps versus posterior glenoid impingement in our model. There are 2 research projects that support this mechanism of injury. The first of these was done by Dr. Andrews,¹ who found that stimulating the biceps tendon during the course of surgery would pull open a type II SLAP lesion (this was before the type II SLAP lesion became known as the type II SLAP lesion). The second study was recently reported by Soslow'sky's group at the 1998 American Shoulder and Elbow Meeting (unpublished data).

Soslow'sky was able to produce a SLAP lesion by overloading the biceps tendon in a shoulder that was in neutral position but inferiorly subluxated. Note that in this position they were unable to produce a SLAP lesion if the shoulder was not subluxated. These 2 studies hint that it is possible to produce a SLAP lesion by this mechanism and that this SLAP lesion can be pulled open through tension in the biceps tendon.

I would cite 2 arguments against this mechanism of injury. The first is that there is no electromyographic evidence that there is a great deal of tension within the biceps tendon during the throwing motion. I believe that electromyographic studies show that, during the deceleration phase, the biceps exhibits 44% of the maximum muscle test in terms of its activity and, in the deceleration phase, the activity is 10% of a maximum muscle test. This is actual research data. Second, we understand biologic tissues are anisotropic. Therefore, mechanically, collagen is much more likely to fail when the line of force is not parallel to the fiber. Damage to a tissue is less likely to occur in the course of that tissue performing a function for which it is designed. The orientation of the collagen within the structures would make it much more likely that they would be damaged at a lower rate of loading by being loaded in an unnatural fashion. For example, Soslow'sky had to place the shoulder in an inferior subluxated position in order to pull on this origin in an abnormal fashion and thereby produce a SLAP lesion. Granted that there are instances in orthopaedics where tendons are actually avulsed off of the bone by tension overload. The patellar tendon and the Achilles tendon

come to mind. These fail under a great deal of force performing some type of eccentric contraction that generates greater than a maximal muscle test activity within the muscle. I think it is more likely that our SLAP lesions are produced by the mechanism I am suggesting or by the mechanism that Steve Snyder² suggested in which the humeral head is shoved superiorly. In these types of loading, the humerus no longer conforms to the confines of the glenoid and may compress the superior labrum, which is really designed to bear tensile stresses from the biceps tendon.

That instability arises from a type II SLAP lesion cannot be denied. This is based on reliable laboratory work and we have all seen this at arthroscopy. However, I am uncomfortable with the idea that a disruption of a particular region of capsule, such as the posteriorly directed SLAP lesion, would thereby increase the likelihood of an angulation of the joint in that direction and lead to a more posteriorly located internal impingement. This direction of angulation and secondary glenoid impingement would be the least affected by a disruption of the labrum in the posterior superior corner because the reduction-external rotation would tend to stretch all other parts of the capsule and would put decreased tension on the portion of the capsule to which the SLAP lesion is attached. Conversely, the tear in the anterior superior corner would seem to have little effect on anterior-superior contact in abduction-external rotation. This is another way of saying that Morgan and Burkhart's explanation is too pretty: that the anterior-superior SLAP creates an anterior tear of the rotator cuff, and the posterior-superior SLAP creates a posterior-superior cuff injury. I think a simpler explanation would be that this is produced by direct contact with these structures and that the association of the 2 lesions is based on anatomic proximity.

In their rebuttal to my argument, Morgan and Burkhart suggest that if glenoid impingement were the primary cause of shoulder problems then all of the throwers would have ended their careers. But it is not necessary to be unstable to produce posterior-superior glenohumeral contact; none of the 10 cadaver shoulders that I dissected had instability; none of the 8 pin brothers studied by Dr. Sidles³ had instability; and none of Walch's⁴ original 16 patients were unstable. This is contact of which normal shoulders are capable. Ed McFarland⁵ found that 85% of patients at arthroscopy can make contact.

The difference between the normal and the pathological is the degree, either in total force or in the

frequency, with which it is applied that leads to pathology. Another argument in favor of the glenoid impingement model is the spectrum of diseases that we see with other mechanisms of injury. We rarely get a tight grouping of injuries occurring together. There is a spectrum of disease here much as we see of the valgus external rotation injury at the knee that occurs with clipping. Some players will develop a medial collateral ligament injury, others will develop an anterior cruciate ligament tear, some will tear both, some will tear the medial meniscus, others will not. The published data on glenoid impingement point to a spectrum of injuries. If the SLAP II lesion is the primary lesion then we would see it universally in those patients who have glenoid impingement. Its frequency would obviously be greater than the number of patients who have the partial rotator cuff tear. Arguing counter to this are the published statistics on partial rotator cuff tear. Dick Caspari published 1 of the earlier series in which he only debrided a partial rotator cuff tear and did nothing to the labrum and obtained good results in his patients. Also pointing to compression as the primary cause are the radiographic findings in the glenoid discussed by Walch⁴ in his report. I think these are obviously more from compression than from tension. There are also the 2 throwing patients reported by Iannotti and Wang⁶ who had a bony SLAP lesion in which they had a fatigue fracture of the superior labrum. Iannotti and Wang treated these patients by performing a biceps tenodesis and did nothing about instability.

Morgan and Burkhart suggest that patients who got well with exercise alone had some other problem and that we did not document this by arthroscoping them. Actually, we arthroscoped several patients who developed some erythema of the superior labrum and a partial rotator cuff tear. A debridement of the rotator cuff was performed. There was nothing done about any instability, and these patients subsequently had a good response to rehabilitation. These are anecdotal reports. Nobody has assembled a large series of them because the patient who is going to respond to physical therapy will not reach the operating room.

In short, all of the evidence cited by Morgan and Burkhart in support of his model can also be used to support the glenoid impingement model. The fact that the glenoid impingement disappeared after repair of the SLAP lesion just means that there has been some capsular contracture because the ability to make glenohumeral contact in the posterior superior corner is seen

in the majority of shoulders without instability or SLAP lesions.

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