ANTEROINFERIOR INSTABILITY

The overhead athlete typically places demands on his or her shoulder that far exceed activities for the normal population. Intra-articular pathology in the overhead athlete includes microinstability, SLAP tears, internal impingement, biceps tendinopathy, and partial articular surface rotator cuff tears. The fact that these pathologies are interrelated and often coexist creates a challenge in identifying specific etiologies. The pathomechanics involved in the overhand throwing motion and the internal impingement phenomenon in particular are complex. Treatment of these entities in the past has produced mixed results. As the results of valuable research accumulate, more unified models are evolving that begin to better explain the breadth of clinical findings. With a more complete understanding comes the hope of more effective treatment strategies.

MICROINSTABILITY

Progressive acquired capsular and ligamentous attenuation unrelated to specific traumatic events may create progressive dysfunction in the shoulder. Symptomatic microinstability can be anteroinferior, straight anterior, or anterosuperior. Although posterosuperior instability has been described, the pathology is not truly one of instability. Rather than being caused by capsular and ligamentous laxity, it is due to capsular tightness and aberrant posterosuperior glenohumeral translation. This is addressed later with internal impingement.

Repetitive overhead sports activities including throwing, volleyball, tennis, and gymnastics may create anteroinferior glenohumeral instability, which is the most common acquired symptomatic laxity. On examination, the “load and shift” test identifies excessive anteroinferior laxity but is difficult to quantitate. The anterior Jobe relocation test is a helpful sign. It is considered to be positive when the patient’s apprehension created by the examiner placing the shoulder in abduction and external rotation is eliminated by a posteriorly directed force on the upper arm. Arthroscopic findings may include a positive “drive-through” sign in which the arthroscope can be passed through a generous glenohumeral interval from posterior to anterior across the shoulder joint. Depression or concavity of the labrum and glenoid chondromalacia are footprints of excessive translation of the humeral head on the glenoid. Occasionally, an incomplete Bankart lesion is present and may also create subtle anteroinferior instability.

Treatment options include an arthroscopic capsular plication that allows the surgeon, with some “guesstimation,” to roughly quantitate the magnitude of capsuloligamentous shortening that is produced. A rasp or whisker shaver blade is used to lightly excoriate the capsule along a 1.5-cm band adjacent to the labrum. A tuck or fold of capsule is then initiated by inserting a curved, cannulated suture hook into the capsule 1.0 to 1.2 cm lateral to the glenoid rim, passing it immediately deep to the capsule, and exiting approximately 5 mm lateral to the glenoid rim. The fold of capsule is then created by delivering the suture hook beneath the intact labrum to exit centrally. Long-term follow-up is not available for this technique, but early results are encouraging and the risk is relatively low because minimal tissue destruction is created.
A second option to stabilize the shoulder is to perform a thermal capsulorrhaphy. A great deal of debate has surrounded this technique in the past several years regarding its safety and efficacy. The limited clinical studies that are available show widely varying success rates. Levitz et al.\(^1\) reported 85% success rate in 122 throwing athletes when thermal capsulorrhaphy was used as an adjunctive tool. Others have reported failure rates from 15% to 60% depending on the primary clinical pattern of instability.\(^2\) The visible tissue response to the heat probe is quite variable and is an unreliable guide to the magnitude of the thermal effect on the tissues. Reports of permanent capsular damage have led to recommendations for “striping” or creating a grid pattern rather than painting the tissues. It is advisable to leave as much healthy untreated tissue as that which is thermally altered. Capsular necrosis, stiffness, and axillary nerve injury are concerns and this modality must be used with caution.\(^3\)

The open capsulolabral reconstruction has been reported to permit return of up to 75% of professional baseball players for at least one full season subsequent to shoulder repair. A transverse incision rather than a vertical detachment of the subscapularis avoids much of the morbidity associated with an open procedure and permits earlier and more aggressive rehabilitation. A “pants over vest” imbrication of the capsule along the glenoid rim is created to reduce the capsular volume and restore stability to the shoulder.

**STRAIGHT ANTERIOR INSTABILITY**

Straight anterior glenohumeral instability is relatively uncommon and may result from tearing of the midlbralum and detachment of the middle glenohumeral ligament origin. Associated partial articular surface rotator cuff tears are identified in approximately 2 thirds of patients. In addition to repetitive overhead activities, glenohumeral hyperextension at neutral rotation and 45° abduction may also produce this pathology. Examination findings include a positive load and shift test and a positive anterior Jobe relocation test. A positive Whipple test denotes pain on resisted elevation of the arm in the scapular plane if associated supraspinatus tearing is present. Treatment includes a suture anchor repair of the anterior labrum and associated middle glenohumeral ligament complex along with arthroscopic debridement of the articular surface rotator cuff tear.

**ANTEROSUPERIOR INSTABILITY**

Anterosuperior instability is also relatively rare. The eponym, SLAC (superior labrum anterior cuff) has been used to describe this lesion.\(^4\) An anterosuperior labral lesion, a superior glenohumeral ligament tear, and a partial articular surface supraspinatus tear have been noted. Occasionally, chondromalacia in the superior glenoid quadrant is detected. Approximately 50% of the patients who have been recognized with this entity have been overhead athletes, and 50% have sustained significant shoulder trauma. The superior labral and glenohumeral ligament damage is either repaired or debrided along with the rotator cuff.

**SLAP-BICEPS LESIONS**

The superior labrum is typically more meniscoid in appearance than the inferior region. The biceps anchor has a variable attachment to the supraglenoid tubercle with approximately 25% to 50% attaching to the bony tubercle and 50% to 75% attaching predominantly to the posterosuperior labrum. Normal variants include an anterosuperior sublabral foramen and the “Buford complex” or cord-like middle glenohumeral ligament. Snyder\(^5\) was the first to classify superior labral tears. Type I consists of superior labral fraying (20%); type II, biceps-labral detachment (55%); type III, a superior bucket-handle tear (9%); type IV, a bucket handle tear with extension into the biceps tendon (10%) and complex (5%). Microinstability, internal impingement (discussed later), marked external rotation of the abducted arm,\(^6\) and traction on the long head tendon of the biceps during deceleration of the throwing arm are possible mechanisms of injury creating SLAP lesions in the overhead athlete.\(^7,8\)

Many tests have been described to diagnose superior labral tears but lack specificity and sensitivity. The following are more reliable. The posterior Jobe relocation test (for posterior or superior SLAP lesions) is begun by placing the patient’s arm in 90° abduction and full external rotation. If posterosuperior pain is relieved by the examiner creating a posteriorly directed force on the upper arm, the test is considered positive. The O’Brien test is performed by placing the patient’s arm in 90° flexion, 25° adduction, and full internal rotation. Downward pressure on the arm by the examiner may create anterosuperior pain. The test is considered positive for an anterosuperior labral tear if the pain on resisted flexion is eliminated with the arm in a similar position but with the forearm supinated. Kibler’s anterior slide test is begun by asking...
the patient to their place hands on their hips with the elbows directed posteriorly. With one hand, the examiner supports the scapula. The other hand creates an anterosuperiorly directed force on the patient’s elbow. If anterosuperior pain is generated, an anterior or superior labral tear is suspected.

A challenge is often presented to the arthroscopist in deciding which superior labral tears are significantly pathologic and require treatment. A large recess between the superior labrum and glenoid may be a normal occurrence. The findings that suggest significant pathology include hemorrhage and irregularity at the biceps anchor, superior labral arching with biceps traction, biceps “peelback” with abduction and external rotation of the shoulder, and a positive “drive-through” sign seen arthroscopically. Treatment includes debridement for type I tears, suture anchor repair for type II tears, resection versus repair for type III lesions, and repair, debridement, or occasionally tenodesis for type IV tears.9

INTERNAL IMPINGEMENT

The constellation of pathology found with internal impingement includes posterosuperior SLAP tears, a partial articular surface tear of the posterior supraspinatus, and posterosuperior glenoid chondromalacia. Walch et al.10 were among the first to describe this entity. Though contact between the greater tuberosity and posterosuperior glenoid was thought to occur normally in full abduction and external rotation, it was believed that the repetitive frequency and intensity with which it occurred, especially during throwing, led to labral and rotator cuff damage. It was also believed that decreased humeral retroversion may exacerbate the problem. Jobe11 and Davidson et al.12 attributed the pathologic findings of internal impingement to acquired anteroinferior microinstability that compromises the obligate posterior rollback of the humeral head during abduction and external rotation.13 The anterior translation and lack of roll-back of the humeral head were believed to permit increased impact of the greater tuberosity on the posterosuperior glenoid. In addition, hyperangulation of the glenohumeral joint in the transverse plane was thought to increase the frequency and magnitude of the greater tuberosity-rotator cuff contact on the posterosuperior glenoid. Kibler14 suggested that a loss of scapular synchrony with inefficient scapular elevation and retraction also created hyperangulation of the glenohumeral articulation. Eventually, the increased stress on the anterior capsuloligamentous structures was believed to create an acquired anteroinferior microinstability. Components of several of these models likely coexist in any one particular shoulder patient suffering from internal impingement.

Recently, Burkhart et al.15 offered a model that unifies a number of these concepts used to explain internal glenohumeral impingement. A key finding thought to initiate the pathologic cascade is a glenohumeral internal rotation deficit (GIRD) due to a contracted posterior capsule. As the arm comes into abduction and external rotation during the throwing motion, the contracted posterior capsule “slings” beneath the humeral head. After the excursion in the posterior capsule reaches its limit, the humeral head then begins to “roll up” the capsule much like a tire on a rope and results in an aberrant posterosuperior shift of the humeral head. This shift creates shearing forces that produce posterosuperior labral tearing and glenoid chondromalacia. Accompanying the posterosuperior shift in the axis of humeral head rotation is a pseudolaxity of the anterior capsule. As the humeral head translates posterosuperiorly, it no longer “fills” the anterior capsule and permits a capsular redundancy. Both this anterior pseudolaxity and the increased clearance between the greater tuberosity and the posterosuperior glenoid, caused by the posterior humeral head shift, permit hyperexertial internal rotation of the shoulder. As the humeral head externally rotates excessively, there are 2 consequences. First, along with excessive torsion of the biceps tendon, the vector of the tendon becomes more posteriorly directed than normal and leads to a “peelback” of the posterior and superior labrum.16,17 Second, excessive torsion of the rotator cuff may contribute to tearing of the articular surface fibers. Finally, a “break in the ring” of the posterosuperior labrum (circle concept) is thought to add to the anterior capsular pseudolaxity that may be manifested as a positive “drive-through” sign during arthroscopy.

Examination findings of significance include a loss of internal rotation greater than 25° and a positive posterior Jobe relocation test. Excessive anteroinferior glenohumeral translation may be detected but is often difficult to quantitate.

The initial treatment is directed toward activity modification, nonsteroidal anti-inflammatory medication (NSAIDs), focused posterior capsule stretching, and rotator cuff and periscapular strengthening. If posterior capsular stretching is unsuccessful, a limited arthroscopic posterior capsular release may be indicated in a small number of patients.18 If there appears to be significant anterior inferior microinstability, con-
sideration may need to be given for an arthroscopic capsular plication, thermal capsulorrhaphy, or open anterior capsule labral reconstruction. When a posterior or superior SLAP II lesion is present, it should be repaired and will usually eliminate the drivethrough sign seen at diagnostic arthroscopy. Partial articular surface cuff tears are debrided to stable healthy tissue.

PARTIAL ARTICULAR SURFACE ROTATOR CUFF TEARS

The etiology for partial articular surface rotator cuff tears is likely multifactorial but may include repetitive traction on articular surface fibers such as during deceleration of the throwing arm and internal impingement as described above. Biomechanically, the articular surface of the cuff may be more likely to fail under tensile rather than compressive forces. A grade 1 tear describes a defect less than 3 mm (< 25%); grade 2, 3 to 6 mm defect thickness (< 50%); and grade 3, greater than 6 mm defect thickness (> 50%). Treatment includes an arthroscopic debridement to stable healthy rotator cuff tissue. For the few that are grade 3 tears, consideration may need to be given for an arthroscopic or mini-open rotator cuff repair. An arthroscopic subacromial decompression may be considered part of the management for grade 1 and 2 articular surface tears as suggested by Payne et al. Great caution should be exercised if instability is a component of the pathology because a subacromial decompression may worsen symptoms.

SUMMARY

The pathology in the overhead athlete’s shoulder is often complex, with substantial overlap between microinstability, labral pathology, internal impingement, and partial articular surface rotator cuff tears. An accurate diagnosis demands the careful integration of the history, physical examination findings, imaging studies, examination under anesthesia, and findings at diagnostic arthroscopy. The treatment options described have relatively little intermediate or long-term follow-up and remain controversial.

REFERENCES