The baseball pitch places tremendous stress upon the shoulder. These stresses lead to microtrauma in the soft tissues surrounding the shoulder and, with repetitive throwing, can result in overuse injuries. A delicate balance exists between mobility and stability at the shoulder, and a fine line separates optimal athletic performance and unwanted injury.

A variety of different shoulder injuries can occur from the chronic stresses of pitching. The first part of this presentation described the shoulder biomechanics and injuries that occur during the late cocking and acceleration phases of the baseball pitch, including anterior instability, bicipital tendinitis, and subacromial impingement, common causes of shoulder pain in the baseball pitcher. This presentation describes the shoulder biomechanics and injuries that occur during the deceleration phase of the pitch. Deceleration involves tremendous stresses on the posterior structures of the shoulder, contributing to rotator cuff tears and posterior instability. Considerable traction forces on the glenoid labrum and shoulder joint capsule can lead to superior labral lesions, including injury to the superior labrum anterior to posterior (SLAP lesion), and the Bennett lesion, a lesion peculiar to baseball players. Finally, we return to the topic of internal impingement and review Burkhart’s theory that it is part of the spectrum of disorders associated with and secondary to posterior Type II SLAP lesions. Despite varying etiologies, shoulder injuries in baseball pitchers usually present with pain. A thorough understanding of the biomechanical sources for injury has enabled the development of effective treatment and rehabilitation regimens, often allowing baseball pitchers to return to their sport at their previous levels of competition.

**Deceleration**

The primary purpose of the deceleration and the subsequent follow-through phases is to decelerate the throwing arm comfortably, safely dissipating the excess kinetic energy not transferred to the ball, minimizing the risk of injury.\(^1,2\) The deceleration phase occurs during the first 50 ms following ball release and is characterized by a powerful deceleration force generated by the posterior shoulder girdle musculature, with deceleration values approaching -500,000 deg/sec\(^2\) (Fig. 1F).\(^1,2\) The shoulder continues its internal rotation while the elbow continues its extension. From its maximal value, occurring 5 ms before ball release, the shoulder internal rotation angular velocity decreases to zero. From a position of neutral horizontal abduction at the time of ball release, the shoulder horizontally adducts across the front of the body. After ball release, the arm rapidly abducts about the shoulder to a position of about 110°. The relative constancy of the 90° to 110° abduction position of the shoulder throughout the pitching motion suggests that this is a strong, dynamic position for the arm and shoulder. Deviations greater than 10° outside this range (less than 80° or greater than 120°) from foot contact to ball release may indicate abnormal positioning of the arm.\(^2\) The deceleration phase is complete when the arm reaches a position of 0° internal rotation.

**EMG Analysis**

In general, opposing muscles around the shoulder fire simultaneously to control deceleration. Reaching peak activity, the teres minor demonstrates the highest level of ac-
tivity (84% of MVC) of all the poster rotator cuff muscles, which act in concert to decelerate the arm by eccentric contraction.\(^3\) After ball release, a posterior force of about 400 N, a compressive force of about 1090 N, and a horizontal abduction torque of about 97 N-m are applied to the humerus at the shoulder (Fig. 1).\(^4\) These forces are generated primarily by the posterior rotator cuff muscles as they try to resist glenohumeral distraction, internal rotation, and horizontal adduction at the shoulder. The teres minor also serves to stabilize the posterior shoulder joint to limit humeral head translation. As with the acceleration phase, the posterior cuff pain that occurs during the deceleration phase can often be isolated to the high activity of the teres minor.\(^3\)

The middle and posterior heads are the most active of the three heads of the deltoid because of their position antagonistic to the motion of the decelerating upper extremity. The pectoralis major loses its mechanical advantage once the humerus drops below 90° elevation; thus, of the two main anterior shoulder muscles, the latissimus dorsi is more active than is the pectoralis major during deceleration. Continuing its role in positioning the humeral head in the glenoid during contractions by the latissimus dorsi and pectoralis major, the subscapularis demonstrates high activity safeguarding against subluxation of the humeral head.

**Shoulder Injuries**

**Posterior Instability**

Analogous to the stretching of anterior structures that occurs during the late cocking phase, posterior structures may be stretched and damaged during the deceleration phase of the pitching motion as they resist glenohumeral distraction and horizontal adduction. Again, the stresses due to stretching and eccentric contraction of the posterior cuff muscles can lead to micro-tears that accumulate with repetitive throwing.\(^5\) This, in combination with stretching of and injury to the posterior capsule and labrum from repetitive throwing, can lead to posterior shoulder instability and subluxation.\(^5\) Alternatively, a single traumatic event involving a forceful posterior displacement of the upper extremity while the arm is flexed, adducted, and internally rotated, could lead to stretching of the posterior capsule, posterior labral and glenoid injury, and subsequent posterior instability and subluxation.\(^8\) Although the mechanisms of injury are similar, posterior instability is far less common than is its anterior counterpart.

Patients with posterior subluxation typically will present with posterior shoulder pain during the deceleration phase of the pitching motion when the humerus in a position of 90° forward flexion, adduction, and internal rotation.\(^8\) In this position, patients may also complain of a sensation of looseness or instability of the shoulder joint. On physical examination, it may be possible to sublux the shoulder by placing the arm in forward elevation and applying a posterior load on the proximal humerus. In contrast to patients with anterior instability, apprehension as a complaint or during physical examination is uncommon in patients with posterior instability.\(^8\) Some athletes are able voluntarily to sublux the shoulder posteriorly, either by horizontally adducting the arm or by selective activation of shoulder muscles. When the subluxed shoulder is subsequently reduced, an audible click or palpable crepitus in the shoulder may be noted. In addition, many athletes with posterior instability have concomitant inferior instability (a condi-
tion termed multidirectional instability), which may be demonstrable on physical examination by eliciting the sulcus sign, a noticeable dimple inferior to acromion upon inferior traction on the shoulder.7,8,10 Motion about the shoulders is usually symmetric and range of motion is full.11

Standard anteroposterior radiographs are usually normal in patients with posterior instability. Stress radiographs, however, may demonstrate the posterior subluxation.11 Transaxillary and Stryker notch radiographs may reveal calcification along the posterior capsule or erosion of bone from the posterior glenoid rim.8 Support for the routine use of computerized tomography (CT) or magnetic resonance (MR) imaging in the evaluation of a patient with posterior instability has not been documented. Bigliani and colleagues12 reported that findings on CT scans taken after injection of arthrographic contrast medium correlated with operative findings in less than two-thirds of 20 shoulders studied. They suggested that CT may be limited in its ability to delineate a posteroinferior labral lesion precisely and consistently because of the capsular redundancy in this region. Capsular redundancy may limit the ability of MR imaging to identify posteroinferior labral lesions as well.13 These studies may be reserved for patients whose arthrotomographs suggest severe glenoid abnormalities and those in which a repair has failed.12

As with anterior instability, the initial treatment of posterior instability is nonoperative.5,8,11 Because such tremendous stresses are placed on the shoulder during the deceleration phase, for any injury sustained during this phase, each muscle playing a prominent role in this phase of the throw should be strengthened.14 More specific to posterior instability, physical therapy is directed at strengthening the infraspinatus and teres minor with external rotation exercises performed at the side, as well as the posterior deltoid to add stability to the posterior aspect of the shoulder joint. Follow-through technique may be adjusted so that the leg muscles can accept a portion of the stress being placed on the posterior shoulder. With a non-operative approach, approximately two-thirds of athletes note subjective improvement. Although the correction of instability is frequently incomplete, the athlete’s functional performance often improves.6 Physical therapy generally lasts for at least six months.

For patients with recurrent posterior subluxation who do not respond to non-operative treatment after this period of time, surgical treatment may be indicated. Examination under anesthesia and arthroscopy will usually be necessary preoperatively to confirm the diagnosis.8 Examination under anesthesia usually reveals the increased posterior translation compared with the normal shoulder. Arthroscopic findings demonstrate an abnormally redundant posterior capsule, excessive volume of the glenohumeral joint, and minor posterior labral damage. Frank tears or detachments of the labrum may be seen, but the true reverse Bankart lesion is seen in less than 5% of cases, usually in athletes involved in contact sports with traumatic injuries.8,15 The major goal of arthroscopy is to assess the anterior glenohumeral ligaments and labrum to determine the major direction of instability.6

The selection of the best operative procedure remains controversial. A number of operative repairs have been suggested, including a reverse Bankart repair, capsulorrhaphy with biceps tendon transfer, reverse Putti-Platt repair, and opening-wedge glenoid osteotomy. Many investigators recommend posterior capsularrhaphy, in which a T-shaped incision is made in the posterior aspect of the capsule, with the inferior flap advanced superiorly and the superior flap, inferiorly, to adjust the laxity of the posterior capsule.6,8,12,16 During exposure of the shoulder joint, the status of the posterior labrum is assessed. If it is detached, the posterior labrum is reattached to the osseous glenoid rim with non-absorbable nylon sutures. Success rates of 96% have been reported in patients who had not had previous surgical attempts at stabilization, as judged by shoulder stability and pain relief.12 Arthroscopic capsulolabral augmentation to reduce posterior capsular laxity and restore the depth of the glenolabral concavity has also been found to be an effective means of surgical stabilization of posterior instability.17

Postoperatively, the patient’s arm and shoulder are immobilized for three weeks in a position of 30° abduction, slight extension, and neutral rotation. Isometric exercises for the deltoid and rotator cuff muscles are begun in the immediate postoperative period. After three weeks, active and active-assisted range of motion exercises are begun. Forward elevation is allowed after six weeks, and at 12 weeks resistance beyond 90° elevation can be started to increase strength and endurance. The ultimate goal of rehabilitation is to regain synchrony of the scapular rotators, rotator cuff, and deltoids.6 When this is re-established, the athlete may return to pitching. To develop the endurance necessary for competitive throwing, the athlete will usually require an additional six months of training. With adequate rehabilitation following surgery, an athlete can often return to his former throwing ability.

**Rotator Cuff Tears**

Rotator cuff injury can occur due to anterior instability and impingement during late cocking and subacromial impingement during acceleration. Injury to the rotator cuff can also occur during deceleration as it resists horizontal adduction, internal rotation, anterior translation, and distraction forces.7 If eccentric forces and stretch lead to overloading of the rotator cuff tendons, microtraumatic injuries with repetitive throwing accumulate. Degeneration over several years of repeated stress eventually leads to rotator cuff tears.18 This can occur even without impingement.

Patients will typically present with a complaint of pain during pitching that is either poorly localized or localized superiorly near the acromion. On physical examination,
active abduction and external rotation usually reproduces the pain. Pain may also be elicited by having the patient accelerate the arm from an abducted and externally rotated position into internal rotation. Atrophy, particularly of the infraspinatus, may be present, representing chronic rotator cuff dysfunction. On palpation of the supraspinatus and/or infraspinatus, tenderness may be elicited. Gross weakness of the rotator cuff, however, is usually not present. The drop-arm test, performed by placing the arm in maximum forward elevation and instructing the patient to lower the arm, is positive when the patient cannot lower the arm slowly but instead drops it to the side. While a positive drop-arm test usually indicates a rotator cuff tear, a negative test does not exclude the diagnosis.

Plain radiographs add little to the diagnostic evaluation of rotator cuff tears. As mentioned in the first part of this review, MR imaging allows the diagnosis of partial-thickness and full-thickness tears.

Initial treatment consists of rest, anti-inflammatory medications with possible steroid injections for refractory pain and inflammation, and rehabilitation. Physical therapy should emphasize strengthening the rotator cuff muscles with internal and external rotation exercises at the side. If pain persists after two to three months of nonoperative treatment, arthroscopy may be performed. Upon arthroscopic evaluation, partial-thickness tears are often found on the undersurface of the supraspinatus tendon, sometimes extending posteriorly to the infraspinatus tendon (Fig. 2). Associated partial tears of the glenoid labrum and biceps tendon are often found as well. Partial-thickness tears should be treated with debridement to healthy bleeding tissue of the torn tendon edge with a motorized shaver. Chronic situations may lead to full-thickness rotator cuff tears, which, if demonstrated arthroscopically, are an indication for repair of the rotator cuff tendon back to bone. After surgery, the shoulder is kept in a sling for one to two weeks. On the first postoperative day, rehabilitation is started, consisting of passive abduction, and internal and external rotation. Active abduction, forward elevation, and internal and external rotation exercises can begin one month after surgery. At two months, muscle strengthening can begin, which should progress slowly. Throwing can begin at about six months postoperatively, although a full year is usually required before the patient can return fully to competitive throwing.

**SLAP Lesion**

Snyder and colleagues described a lesion of the superior glenoid labrum beginning posteriorly and extending anteriorly (SLAP lesion). These investigators described four types of SLAP lesions based on arthroscopic findings (Fig. 3). Type I lesions involve fraying and degeneration of the superior labrum; the labrum and biceps anchor, however, remain firmly attached to the glenoid. In addition to the fraying and degeneration that occurs in Type I lesions, Type II lesions include detachment of the superior labrum and
biceps anchor from their insertion on the superior glenoid, resulting in arching of the labral-biceps complex away from the glenoid neck. In Type III lesions, a bucket-handle tear occurs in the superior labrum, while the peripheral portion of the labrum and biceps tendon remain firmly attached to the underlying glenoid. The bucket-handle tear of the superior labrum extends into the biceps tendon in Type IV lesions, leaving portions of the labral flap and biceps tendon displaceable into the glenohumeral joint. In a study of 140 patients with injuries to the superior labrum, Snyder and associates\textsuperscript{22} reported that 21% were type I SLAP lesions; 55%, type II; 9%, type III; 10%, type IV; and 5%, complex injuries involving multiple lesions. Maffet and coworkers\textsuperscript{23} added three additional types to Snyder’s classification: Type V lesions involve an anteroinferior Bankart lesion extending upward to include separation of the biceps tendon; Type VI lesions consist of an unstable radial or flap tear associated with separation of the biceps anchor; and Type VII lesions involve extension of the SLAP lesion beneath the middle glenohumeral ligament.

As mentioned, SLAP lesions are often associated with other lesions. Benincardo and colleagues\textsuperscript{24} found that SLAP lesions were associated with partial rotator cuff tears in 42% of patients, frayed or lax inferior glenohumeral ligaments in 26%, Bankart lesions in 16%, Hill-Sachs lesions in 16%, chondral lesions in 16%, loose bodies in 10%, complete rotator cuff tears in 5%, and posterior labral tears in 5%. Because their presence affects the choice of treatment, associated lesions should be identified at arthroscopic evaluation and appropriately treated.

Snyder and colleagues\textsuperscript{21} stated that SLAP lesions usually result from a compression force to the shoulder, such as a fall on an abducted and flexed arm. However, in throwing athletes without evidence of trauma, SLAP lesions may be attributed to repetitive traction forces from overuse.\textsuperscript{25,26} Serving to decelerate the rapidly extending elbow, the force supplied by eccentric contraction of the long head of the biceps during the deceleration phase places tremendous stress on the biceps-labral complex. This force may be transmitted proximally to the origin of the biceps long head on the superior glenoid tubercle and labrum, exerting considerable traction on the anterosuperior labrum. With repetitive throwing, labral tears and biceps-labral complex avulsions can eventually ensue.

Burkhart and associates\textsuperscript{27} subclassified type II SLAP lesions into three types: anterior, posterior, and combined anterior and posterior. In a study of 102 Type II SLAP lesions, 37% were located anteriorly; 31%, posteriorly; and 31%, combined anterior and posterior.\textsuperscript{27} As an alternative to the avulsion mechanism, the peel-back mechanism was proposed as a cause of posterior and combined anterior-posterior Type II SLAP lesions in pitchers.\textsuperscript{28} In this mechanism, as the arm is brought into abduction and external rotation during late cocking, the long biceps tendon assumes a more vertical and posterior angle. This angle change produces a twist at the base of the long biceps tendon, in turn producing a torsional force at the base of the biceps that is transmitted to the posterior labrum. This torsional force “peels back” the biceps and posterior labrum off of the glenoid rim, demonstrable arthroscopically with the arm abducted and externally rotated.\textsuperscript{29} The torsional peel-back is then repeated with each pitch, causing progressive enlargement of the lesion.

Interestingly, in a study by Burkhart and coworkers\textsuperscript{29} that included 44 pitchers with Type II SLAP lesions, all of the subjects were found to have tight posterosuperior capsules, evident on physical exam as marked loss of internal rotation with the arm abducted to 90°. The authors concluded that a tight posterosuperior capsule would contribute to the development of, and therefore predispose to, Type II SLAP lesions. As the shoulder abducts and externally rotates during late cocking, a tight posterosuperior capsule shifts anteriorly, inferior to the humeral head. In this position, the tightened posterosuperior capsule pushes superiorly against the humeral head and causes a posterosuperior shift of the glenohumeral fulcrum. When the shoulder then externally rotates fully around the new center of rotation into the cocked position, the force at the posterosuperior biceps-labral attachment is increased. This increased force contributes to the effect of the torsional “peel-back” force and to the development of a posterior or combined anterior-posterior Type II SLAP lesion.

As discussed in Part 1 of this review, internal impingement is thought by many to be a result of anterior instability caused by repetitive microtrauma to anterior shoulder structures and secondary loss of effective dynamic stabilizing function. Suggesting that internal impingement instead is part of the spectrum of disorders associated with and secondary to posterior Type II SLAP lesions, Burhart and colleagues\textsuperscript{29} proposed an alternative mechanism for internal impingement. In the posterior Type II SLAP lesion, detachment of the posterosuperior labrum and biceps origin could lead to posterosuperior instability that is clinically similar to anterior instability.

Repeated posterosuperior translation or subluxation during late cocking would stress the undersurface of the posterior rotator cuff tendons, eventually leading to rotator cuff damage. Indeed, 31% of shoulders examined by Burkhart and colleagues\textsuperscript{29} had associated partial or complete undersurface tears of the posterior rotator cuff. Furthermore, according to the “circle concept,” disruption of one part of the labrum may manifest itself as instability on the opposite side of the glenoid, creating a type of pseudolaxity.\textsuperscript{29} Thus, detachment of the posterosuperior labrum could create a corresponding anteroinferior pseudolaxity. Evidence for the existence of anteroinferior pseudolaxity is provided by the finding on arthroscopic exam of the drive-through sign in all patients with posterior Type II SLAP lesions in Burkhart and colleagues’ study.\textsuperscript{29} Moreover, repair of the posterior...
Type II SLAP lesion eliminated the drive-through sign and anterior instability in all of these patients. Through this mechanism, posterosuperior labral damage could lead to a clinical picture similar to anterior instability, with subsequent internal impingement. While initially attractive, based on Paley and coworker’s finding that only 10% of patients with symptomatic internal impingement had associated SLAP lesions, however, Burkhart’s explanation for internal impingement may not be complete.

Athletes with SLAP lesions will usually present with shoulder pain during the pitching motion. Pain may also be present while lying on the affected shoulder or during activities of daily living. The pain from a SLAP lesion is often indistinguishable from the pain associated with impingement. The mechanical symptoms of catching, locking, popping, or grinding are also commonly experienced by athletes with SLAP lesions. In addition, athletes may complain of decreased strength or range of motion at the shoulder. Burkhart and colleagues attribute the classic “dead arm” syndrome to the Type II SLAP lesion. In this syndrome, the pitchers feel a sudden sharp anterior or posterior shoulder pain and subjective uneasiness upon abduction and maximal external rotation during late cocking, which prevents them from throwing with their pre-injury velocity and control.

On physical examination, audible popping or snapping is frequently heard on shoulder motion. Crepitation is commonly palpable in patients with isolated SLAP lesions. During the compression-rotation test, while the patient is lying laterally with the arm in 90° abduction, pain may be experienced with internal or external rotation of the humerus. During the biceps tension test, with the elbow extended and forearm supinated, pain may be elicited with resisted shoulder flexion. Pain may also be elicited with resisted supraspinatus strength testing.

Although pain is frequently present during the apprehension test, it may arise from traction on the torn labrum, rather than being due to pure instability. Indeed, the relocation test is usually negative in patients with SLAP lesions, allowing some differentiation between pure SLAP lesions and pure anterior instability. Because partial and complete rotator cuff tears are often associated with SLAP lesions, Neer and Hawkins signs are often positive in these patients.

Some distinctions can be made between the different sub-classifications of Type II SLAP lesions based on physical exam findings. As discussed, patients with Type II SLAP lesions often have loss of internal rotation in 90° of abduction due to underlying contracture of the posteroinferior capsule. In addition, patients with posterior and combined anterior-posterior Type II SLAP lesions will often have positive relocation tests, while those with anterior Type II SLAP lesions will not. The anterior lesion may be predicted by eliciting bicipital groove tenderness and positive Speed and O’Brien’s cross-arm tests, which are usually negative in the posterior lesion. In O’Brien’s test, resisted shoulder adduction and forward-flexion to 90° across the chest produces anterior shoulder pain. None of these tests, however, are entirely sensitive or specific for any particular Type II SLAP lesion.

Since they do not detect intra-articular labral disorders, standard radiographs add little in the evaluation of these patients. Offering high resolution of soft tissues, MR imaging has proven useful in detecting labral tears and detachments. Findings on MR imaging that may indicate a labral or superior labral lesion include high signal intensity either in the labrum-biceps anchor or between the superior glenoid labrum and the superior glenoid fossa, inferior or anterior and medial labral displacement, deformity, or the presence of a superior glenoid labral cyst. A glenoid labral cyst should also be considered a possible indicator of associated shoulder instability. When the results of standard MR imaging are unclear in a patient with a suspected labral lesion, MR arthrography may be indicated. With reported sensitivities of 96% for detecting both labral tears and detachments, MR arthrography is gaining more widespread use.

False positive diagnoses of SLAP lesions still occur with MR imaging and MR arthrography, diminishing the reliability of these tests in the diagnosis of labral pathology. The definitive diagnosis of SLAP lesions can therefore be made only with diagnostic arthroscopy (Fig. 4). In addition, after definitive diagnosis, the SLAP lesion can then be treated arthroscopically. Arthroscopic evaluation may reveal hemorrhage or granulation tissue
beneath the biceps tendon, the presence of a space between the articular cartilage margin of the glenoid and the attachment of the labrum and biceps tendon, and arching of the superior labrum more than three to four millimeters away from the glenoid upon traction on the biceps tendon. Variations in normal glenohumeral anatomy, however, necessitate caution when diagnosing SLAP lesions. A sublabral hole at the two o’clock position or meniscoid-appearing labrum are commonly seen in normal shoulders. Present in about 1.5% of shoulders, the “Buford complex” is another normal variant. This complex consists of a cordlike middle glenohumeral ligament that attaches to the base of the biceps anchor, along with the absence of labral tissue on the anterior superior glenoid. Mistakenly reattaching this middle gleno-humeral ligament as if it were a SLAP lesion could result in considerable limitations in shoulder rotation.

Treatment is based on appropriate classification of the SLAP lesion. Types I and III SLAP lesions are treated with arthroscopic debridement. In Type I lesions, the frayed and degenerated labral tissue should be debrided back to intact labrum, preserving the attachment of the labrum and biceps tendon to the glenoid. The bucket-handle portion of the labral tear in Type III lesions should be excised. After debridement of frayed labral tissue, Type II lesions are treated with arthroscopic fixation of the biceps anchor and superior labrum to the glenoid, usually with a screw-in suture anchor. Treatment of Type IV lesions depends on the extent of tearing of the biceps tendon. If the biceps tear involves less than 30% of the biceps tendon, the detached portions of the labrum and biceps tendon should be resected. In a young athlete, if the biceps tear involves 30% or more of the tendon, then arthroscopic suture repair of the biceps tendon and torn labrum and fixation should be performed. For each type of lesion, glenohumeral instability should be assessed and any associated lesions should be appropriately treated. Postoperatively the patient is placed in a sling for three weeks and immobilized for one week. Rehabilitation initially involves elbow, wrist, hand, and pendulum exercises. Exercises to maintain the range of motion and strengthen the rotator cuff, deltoid, and scapular muscles are also emphasized. Active biceps strengthening is begun slowly at five to six weeks after surgery. Stressful biceps activity is allowed after three months. Throwing is begun at four months, with full velocity throwing initiated at seven months. Most patients report good to excellent symptomatic relief after surgery, and many are able to return to pitching at their previous levels of performance.

Specifically for posterior and combined anterior-posterior Type II SLAP lesions, the peel-back mechanism of injury has direct implications on both treatment and postoperative rehabilitation. In order to repair securely the posterosuperior labrum, suture anchors must be placed posterior to the origin of the long head of the biceps at the corner of the glenoid. Since the peel-back phenomenon occurs when external rotation exceeds 0°, the repair must be protected against external rotation beyond 0° for 3 weeks to avoid premature torsional stresses. Patients with Type II SLAP lesions demonstrate tightened posteroinferior capsules, and postoperative rehabilitation should therefore emphasize stretching of this structure. In their study that included 44 pitchers with Type II SLAP lesions, Burkhart and colleagues reported that after arthroscopic repair and rehabilitation all pitchers returned to competition and 84% returned to their preinjury levels of activity or better. The remaining seven pitchers who failed to reach pre-injury levels after SLAP repair alone had associated undersurface rotator cuff tears.

Most pitchers who develop acute posterior Type II SLAP lesions experience a prodromal phase of mild posterior pain with a sense of posterior tightness before the acute injury occurs. If caught during this prodromal phase, the condition may be treated with posterior capsular and scapulothoracic stretching. Moreover, it has been suggested that preseason stretching of the tight posteroinferior capsule may prevent posterior and combined anterior-posterior Type II SLAP lesions, an idea which is currently under study. The elucidation of more non-avulsion mechanisms should allow for specific treatments, rehabilitation regimens, and preventive measures for other types of SLAP lesions in the future.

**Bennett Lesion**

Bennett described a posterior inferior glenoid calcific lesion that he thought was caused by traction stresses at the insertion of the long head of the triceps in the posterior inferior glenoid. He suggested that the proximity of the calcific deposits to the axillary nerve results in nerve irritation; this, in combination with synovial membrane and capsular irritation, was thought to cause subsequent shoulder pain. However, it is now known that the Bennett lesion is an extraarticular ossification of the posterior inferior capsule arising at the insertion of the capsule in the posterior inferior glenoid, unrelated to the triceps insertion. This lesion seems to be peculiar to the baseball pitcher.

The etiology of the Bennett lesion remains incompletely understood. The lesion may result from a traction injury to the posterior inferior capsule that causes capsular micro-tearing, bleeding, and subsequent new bone formation at the insertion of the joint capsule in the posterior inferior glenoid. Traction injury to the posterior inferior capsule could occur during the deceleration phase of the pitching motion as posterior structures strain to decelerate the throwing arm. Alternatively, subluxation and impingement of the humeral head on the posterior capsule may lead to posterior capsular calcification. As mentioned, patients with posterior
subluxation occasionally have associated posterior capsular calcification.\textsuperscript{3,39,40}  

Athletes typically present with posterior shoulder pain during the late cocking or deceleration phases, or both. In some patients, the shoulder pain may be generalized and occur throughout the pitching motion. Shoulder pain may develop gradually with increasing severity or may be sudden in onset. Jobe and colleagues\textsuperscript{4} have suggested that the ossified lesion is often asymptomatic, becoming associated with pain when there is a fracture of the ossified lesion or when a fibrous union forms at its base. Motion imparted to the lesion during the pitching motion causes irritation and subsequent pain. Furthermore, Bennett lesions are frequently associated with posterior labral injuries and posterior rotator cuff tears, which may contribute to the patient’s symptoms.\textsuperscript{39}  

On physical examination, the most consistent finding is tenderness on palpation of the posterior inferior glenoid region. Posterior shoulder pain may be elicited with shoulder abduction and external rotation, as occurs during the late cocking phase, or with shoulder adduction and internal rotation of the arm, as occurs during the deceleration phase. Findings consistent with posterior subluxation may be demonstrable in those patients with this condition.  

Diagnosis of a Bennett lesion is usually made with plain radiographs or CT scans. A conventional axillary lateral view of the shoulder often demonstrates the lesion.\textsuperscript{39} The Bennett view, an anteroposterior view of the glenohumeral joint with the X-ray beam tilted five degrees cephalad and the arm positioned in abduction and external rotation, may improve radiographic detection.\textsuperscript{36-38} CT arthrography demonstrates extraarticular crescentic calcification originating from the posterior inferior glenoid and extending toward the humeral head.\textsuperscript{39} CT and MR imaging may allow earlier visualization of the lesion, as well as any associated posterior labral and rotator cuff lesions.\textsuperscript{39}  

Non-operative treatment consisting of rest and nonsteroidal antiinflammatory medications is usually effective for Bennett lesions.\textsuperscript{6} Exercises to strengthen the posterior cuff muscles can be initiated once the pain and tenderness resolve. For patients who fail non-operative treatment, arthroscopic resection of the spur may be indicated.\textsuperscript{6} After arthroscopic resection and rehabilitation, the athlete may return to competitive throwing within about six months. However, some investigators state that operative removal is not necessary, and that arthroscopic treatment should be aimed only at repairing the associated intraarticular injuries (posterior labral and rotator cuff tears, posterior instability) that may be present.\textsuperscript{39} After surgery, rehabilitation can begin once shoulder pain and tenderness cease.  

**Follow-Through**  
The follow-through has been described as a “passive phase, with the body merely catching up with the [throwing] arm.”\textsuperscript{1} This phase involves extension of the stride leg, shoulder adduction, horizontal adduction, and elbow flexion (Fig. 1F). The pivot leg moves forward to a contact point, allowing the pitcher to assume a balanced fielding position, and thus completing the pitching motion. With adduction, the anterior and superior portions of the capsule limit inferior and posterior motion of the humeral head.\textsuperscript{39} As determined by EMG analysis, all shoulder girdle and upper extremity muscles exhibit low to moderate activity during this phase.\textsuperscript{5}  

**Summary**  
The extreme range of motion at the shoulder, the high angular velocities and torques, and the repetitious nature of the pitching motion combine to make the shoulder vulnerable to injury during the baseball pitch. An understanding of the biomechanics that contribute to shoulder injuries during each phase of the pitching motion can facilitate the athlete’s diagnosis, treatment, and rehabilitation. The athlete’s symptoms and signs, as well as radiographic imaging, are key elements in arriving at a diagnosis of shoulder injuries. Nonoperative treatment consisting of an initial period of rest and NSAIDs, followed by physical therapy and a gradual return to activity, is usually successful. When this approach fails, surgical intervention, either arthroscopic or open, may be necessary. Physical therapy and rehabilitation are directed toward restoring the integrity and strength of the dynamic and static stabilizers of the shoulder joint, yet preserving the range of motion necessary for performance. Through rehabilitation, the dedicated athlete can often return to the pitching mound at his previous level of performance.  

**References**  


