

SHOULDER INJURIES IN THROWING ATHLETES

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Shoulder injuries, both traumatic and chronic, are a relatively common occurrence in overhead athletes. Shoulder injuries can be career-changing, or even career-ending events, especially for overhead athletes such as baseball pitchers, football quarterbacks, tennis players, and swimmers. Efficient throwing requires a coordinated effort that progresses from the toes to the fingertips, and this has been described by Kibler as the kinetic chain concept. The sequence of body segment motions begins with the lower body and moves to the upper body and arm. Energy is generated in the legs and trunk and is then transferred through the shoulder to the arm, which delivers the force to the ball. Any condition that affects a component of the chain, especially those located more proximally in the kinetic chain, may produce changes in later segments, possibly resulting in the development of pathology.

Due to ongoing controversy regarding the exact causes of injury in the thrower's shoulder, the authors will not attempt to provide a single unifying theory. Instead, we will provide an overview clarifying the terminology and describing common pathologic findings, and presenting the various theories on injury in the throwing shoulder. The purpose of this chapter

is to discuss the biomechanics, presentation, diagnosis, and treatment of common shoulder injuries in overhead athletes.

SHOULDER BIOMECHANICS AND KINEMATICS

Overhead athletes perform a majority of their upper extremity activity in the inherently unstable position of maximal abduction and external rotation. A thorough understanding of the biomechanics and joint kinematics of the shoulder is a necessary prerequisite to diagnose and treat shoulder injuries successfully in these athletes. Due to the frequency of shoulder injuries in baseball pitchers, we will review the mechanics of throwing.

During a baseball pitch, ball velocities frequently exceed 90 miles per hour, and the shoulder of a professional player will rotate at speeds of up to 7,000 degrees per second, with distractive forces equal to body weight. These are among the fastest angular velocities created in all of sport. Throwing has been divided into six phases (Table 16-1 and Fig. 15-18 in Chapter 15), and the entire throw usually takes

TABLE 16-1 THE SIX PHASES OF THE BASEBALL PITCH

PHASE	ACTION	DESCRIPTION
1	Wind-up	Readying phase
2	Early cocking	Concluding with the shoulder positioned in 90 degrees of abduction, with the elbow positioned slightly behind the plane of the body
3	Late cocking	Arm reaches maximum external rotation
4	Acceleration	Internal rotation of the arm with highest angular velocity
5	Deceleration	Eccentric contraction of all muscle groups to slow the arm results in greatest loads across the joint
6.	Follow-through	Rebalancing phase

less than 2 seconds. The first three phases occupy 1.5 seconds, acceleration occupies only 0.05 seconds, and the last two occupy approximately 0.35 seconds. Also see Chapter 15 for further details on the kinematics of throwing.

A model of the throwing shoulder during the baseball pitch has been created through a combination of in vitro biomechanical studies, electromyographic analysis, and clinical observation. Although football throwing follows the same basic phases, there are slight differences, imparted by the greater weight of the football, and mainly resulting in lower angular velocities of approximately 5,000 degrees per second.

ADAPTATION

Generally, most upper extremity throwing sports require repetitive motion of the shoulder under high loads at the extremes of motion. Because of these loads, adaptive changes occur in the dominant extremity of overhead athletes. These changes affect passive stabilizing structures such as the capsule, ligaments, and bone, as well as dynamic stabilizers such as the rotator cuff, shoulder girdle, and chest wall musculature. It is widely believed that repetitive subfailure loads can lead to acquired laxity of the shoulder, and this has been demonstrated in cadaveric models.

In throwing athletes, the ability to rotate the humerus externally to generate high ball velocities is paramount. Studies have shown a direct correlation between the amount of external rotation of the abducted arm and the subsequent speed of the pitched ball. With repetitive throwing in a developing skeleton, adaptation of the osseous and ligamentous anatomy occurs, which results in increased humeral retroversion and acquired ligamentous laxity, allowing increased external rotation in the throwing arm.

Examination of the dominant arm of asymptomatic highlevel overhead athletes (baseball pitchers and tennis players) has shown increased external rotation and decreased internal rotation in the abducted shoulder. It is commonly accepted that the majority of these changes result from laxity in the anterior inferior glenohumeral ligament and contracture of the posterior capsule. In the throwing position, the anterior inferior glenohumeral ligament is the primary restraint to external rotation. Therefore, it appears likely that this ligament would be repetitively stressed and could develop laxity, allowing for increased external rotation. Interestingly, baseball pitchers commonly have an increased sulcus sign on physical exam, which may be related to laxity of the coracohumeral ligament, another restraint to external rotation in both the abducted and adducted arm.

Another factor contributing to increased external rotation in the throwing arm is acquired retroversion of the humeral head. Multiple studies have associated this with throwing. This osseous adaptation has been described in professional handball and baseball players, especially when intense training was started before skeletal maturity. An average increase in humeral retroversion of 10 to 20 degrees was observed compared with the nondominant arm.

As a result of the adaptive changes—both ligamentous and osseous—range of motion (ROM) is altered. Some authors suggest that increased humeral retroversion is the predominate cause of this altered ROM. Others believe that laxity of the anterior inferior glenohumeral ligament is the main factor and recommend capsular plication as part of the surgical treatment. Still, other studies suggest that the posterior capsular contracture is the initiating and primary cause of pathology and recommend release of the posterior capsule as part of surgical treatment. Clearly, this area remains in need of additional study.

In addition to bony and ligamentous adaptation, throwing athletes typically demonstrate muscular asymmetry between the dominant and nondominant arm as a result of muscle adaptation. It is not uncommon for athletes to develop hypertrophy of the shoulder girdle musculature, humeral head, cortex, and arm musculature of the throwing arm. In chronic shoulder conditions such as suprascapular nerve dysfunction or rotator cuff pathology, however, subtle atrophy can sometimes be found, especially in the infraspinatus and supraspinatus fossa. Overhead athletes, particularly volleyball players, can demonstrate significant atrophy of the infraspinatus with weakness in external rotation as a result of suprascapular neuropathy. This neuropathy is thought to represent a repetitive traction injury, with constriction occurring at the spinoglenoid notch (often associated with labral cysts) or more proximally at the scapular notch.

Several investigators have examined muscle strength in the overhead throwing athlete with varying results and conclusions. External rotation strength as a function of the infraspinatus and teres minor muscles in the dominant shoulder of professional baseball pitchers has been found to be significantly weaker than the nonthrowing shoulder. The shoulder abductors, the deltoid, and supraspinatus muscles usually do not demonstrate marked hypertrophy in throwers, and some studies have even demonstrated significantly weaker supraspinatus strength in the throwing arm of pitchers compared with the nondominant arm.

Conversely, testing of internal rotation in the dominant shoulders of pitchers has demonstrated significantly increased strength of the internal rotators and adductor muscles. The subscapularis, latissimus dorsi, pectoralis major, teres major, coracobrachialis, and the long head of the triceps act in concert to internally rotate and adduct the arm during the acceleration phase of throwing.

EVALUATION

History

- A detailed history is the basis for a successful diagnosis and treatment.
- Duration, location, and timing of symptoms, as well as associated symptoms, provide essential clues to the diagnosis.
- Patient age and history of other injuries are also important in creating a differential diagnosis.
 - Patient age is relevant in that certain diagnoses are more common in particular age groups.
 - For example, shoulder pain in young athletes should raise concerns for physeal injury.
 - Younger athletes are also more likely to have problems with laxity.
 - Older players, especially pitchers, are more likely to suffer from rotator cuff pathology.
 - Pitchers in the middle of their careers may experience both laxity and rotator cuff pathology.
- Timing of symptoms during the throwing cycle is important in formulating a differential diagnosis (Table 16-2).
 - Pain during cocking can suggest labral pathology, internal impingement, laxity, and/or instability.
 - Pain during late cocking or the early acceleration phase is seen with anterior instability.
 - Pain after ball release or during deceleration is frequently associated with rotator cuff pathology.
 - Posterior instability typically presents with pain during follow-through.
- Timing of symptoms during a game is also important.
 - Symptoms occurring late in the game or after repeated pitching starts suggest fatigue, typically of the rotator cuff. These symptoms may respond well to rest and rehabilitation.
- History of associated symptoms and/or other nonshoulder injuries should also be obtained.

TABLE 16-2 RELATIONSHIP OF PHASE OF THROW WITH DIFFERENTIAL DIAGNOSIS

Phase in Throwing Cycle	Possible Differential Diagnosis	
Wind-up	,	
Early cocking	•	
Late cocking	Labral pathology	
Acceleration	Internal impingement	
•	Anterior instability	
Deceleration	Rotator cuff pathology	
Follow-through	Posterior instability	

- It is important to consider the kinetic chain concept, as injuries to the lower extremities, spine, and other areas may alter throwing mechanics and in turn cause shoulder pain.
- A history of numbness, tingling, or discoloration in the fingers should raise concern for a neurologic or vascular problem.
- Distal paresthesias or "dead arm" may also be associated with shoulder instability.

Physical Examination

Observation

- The majority of injuries seen in the throwing athlete will present with an insidious onset; therefore, the examiner must be attuned to the presence of vague complaints and subtle findings on physical exam, as opposed to gross deformity and overt distress.
- Inspection of both symptomatic and asymptomatic throwing athletes at rest will typically reveal some asymmetry—frequently, hypertrophy of the dominant shoulder and arm.
- Chronic shoulder conditions can present with very subtle atrophy that can be detected with careful inspection of the supraspinatus and infraspinatus fossa, in addition to the scapular stabilizers bilaterally.
- Atrophy within the infraspinatus fossa can signal the presence of suprascapular neuropathy, which occurs in overhead and throwing athletes presumably from traction
- General posture and alignment of the shoulder girdle should also be noted.

Palpation

- Many throwing athletes with shoulder pathology will hold the scapula in a depressed and protracted position.
- Palpation can be helpful in distinguishing between disorders of the subacromial space or supraspinatus, the long head of the biceps, and the teres major tendons.
- All bony prominences around the shoulder should be palpated, especially the acromioclavicular (AC) joint, where tenderness and swelling can indicate degeneration.
- Acute AC joint disruptions are uncommon unless there has been a history of trauma.
- Attention should also be directed to the bicipital groove and coracoid process.
- Tenderness of the bicipital groove is typical for biceps tendonitis, whereas pain with deep palpation of the coracoid can indicate an impingement process.
- The exam should always include palpation of the posterior joint line, where pain from both rotator cuff and labral pathology can sometimes be elicited.
- Additionally, pain from the presence of posterior glenoid osteophytes (e.g., Bennett's lesions) can be appreciated with deep palpation of the posteroinferior glenohumeral joint.

Range of Motion

- ROM, both glenohumeral and scapulothoracic, must be evaluated.
- Scapulothoracic motion should be smooth and symmetrical.

- Asymmetry or winging of the scapula should alert the examiner to the presence of periscapular muscle weakness and overuse or, less commonly, nerve injury or tightness of the pectoralis minor muscle.
- Painful crepitus with scapulothoracic motion may suggest inflammation of the scapulothoracic bursa.
- Rotation of the abducted arm in overhead athletes typically shows loss of internal rotation and increased external rotation due to posterior capsular tightness and stretching of the anterior structures.
- Posterior capsular tightness is best assessed in the prone position, where maximum internal rotation of the shoulder can result in inferior scapular winging.
- Frequently, there is a net loss in ROM due to a comparatively larger loss of internal rotation than gain of external rotation.
- Limitations in internal rotation beyond the normal-butshifted range may place the athlete at risk for the development of shoulder problems, which will be discussed in more detail later in the chapter.

Any discrepancy between active and passive ROM may be a sign of muscle dysfunction or inhibition by pain.

Strength Testing

- Strength testing of the rotator cuff, deltoid, and periscapular muscles should always be performed.
- Internal (subscapularis and pectoralis major muscles) and external rotation (infraspinatus and teres minor muscles) should be evaluated with the arm at the side and in 90 degrees of abduction.
- The supraspinatus may be evaluated with resisted abduction with the 90 degrees in the plane of the scapula and the thumbs pointing to the ground.
- The subscapularis is evaluated with the lift-off test and the belly press test.
- Any pain elicited during testing will help identify the source of the patient's symptoms.
- More subtle muscular dysfunction can frequently be detected by using specific tests (Table 16-3).

TABLE 16-3 SUMMARY OF FUNCTIONAL TESTS

Muscle and Test	Description
Supraspinatus	
Jobe	Patient asked to bring arm to 90 degrees of flexion and full pronation with thumbs pointing to floor, and examiner compares resistive strength to downward directed force; pain and/or weakness indicates supraspinatus dysfunction (see Fig. 14-3 in Chapter 14). Sensitivities range from 0.8 to 0.9; specificities from 0.5 to 0.6°.
Drop arm sign	Patient asked to bring arm to 90 degrees of flexion and full pronation with thumbs pointing to floor; examiner lifts arm and lets drop; unable to maintain position in supraspinatus dysfunction. Sensitivity of 0.2; specificity of 1.0 ^a .
Infraspinatus and teres	
minor Gross strength	Resisted external rotation with arm by the side and at 90 degrees of abduction.
Hornblower's sign	Patient asked to externally rotate arm from a position of 90 degrees of abduction; unable to maintain position in infraspinatus dysfunction.
External rotation lag sign	Patient asked to maintain position of maximum passive external rotation; unable to maintain position in infraspinatus dysfunction. Sensitivity quoted as 0.7; specificity as 1.0 ^a .
Biceps	D. H. A. J. H. Connected at the Control of C
Speed's test	Patient asked to forward elevation from 90 degrees of forward flexion; pain indicates bicep tendonitis (see Fig. 14-10 in Chapter 14).
Subscapularis	Resisted internal rotation with arm by the side.
Gross strength	nesisted internal rotation with aim by the side.
External rotation	Increased passive external rotation, compared with contralateral side—suspicious for subscapularis rupture.
Lift-off test	Patient places back of hand on buttock; unable to lift hand off in subscapularis dysfunction (see Fig. 14-8 in Chapter 14).
Lift-off lag test	Patient places back of hand on buttock. Examiner lifts hand maximally away from buttock; unable to maintain position in subscapularis dysfunction.
Belly press	Useful in patients with limited internal rotation. Patient firmly pushes hand into lower abdomen with elbow held forward of body; while maintaining forward position of elbow in subscapularis dysfunction (see Fig. 14-9 in Chapter 14).

^a Sensitivity and specificity provided were available from Dinnes (2003); these figures are sometimes based on a single study and may not be completely accurate.

Stability

- Glenohumeral joint translation should be evaluated in all directions (anterior, posterior, and inferior).
- This should be done in multiple positions with the athlete standing, sitting, and lying supine.
- Although increased laxity in the dominant arm may not necessarily be the source of pathology, reproduction of pain with any of these maneuvers is helpful in identifying the presence and direction of glenohumeral instability.

Provocative Tests

- Provocative tests are a very important tool when trying to determine the source of a patient's shoulder pain.
- The Neer and Hawkins' impingement tests are routinely used to evaluate the subacromial space and supraspinatus muscle (Table 16-4).
- The apprehension and relocation tests are sensitive tools in diagnosing classic anterior instability if true apprehension is elicited.
 - They are less specific when only pain is produced.
- Placing the arm in abduction and external rotation reproduces the symptoms of pain in many throwing athletes.
- A positive relocation test—in which posterior shoulder pain is diminished when a posteriorly directed force is applied to the maximally abducted and externally rotated arm—may be a sensitive means of diagnosing occult anterior instability and internal impingement, which can contribute to rotator cuff disease and posterior-superior labral pathology.
 - Some have speculated that, rather than testing true instability, the anterior-posterior force used in the

TABLE 16-4 COMMONLY USED TESTS FOR IMPINGEMENT

Test	Description
Neer's impingement sign	Pain with forced forward elevation (see Fig.14-4 in Chapter 14).
Hawkins impingement sign	Pain with internal rotation from a position of 90 degrees of forward elevation and 90 degrees of elbow flexion (see Fig. 14-5 in Chapter 14). Sensitivities range from 0.8 to 0.9; specificities from 0.2 to 0.6 ^a .
Neer's impingement test	Repeated Neer's sign after subacromial injection—increases specificity of impingement diagnosis. Sensitivities range from 0.7 to 0.9; specificities from 0.23 to 0.60 ^a .

^a Sensitivity and specificity provided were available from Dinnes (2003); these figures are sometimes based on a single study and may not be completely accurate.

TABLE 16-5 COMMONLY USED TESTS FOR SUPERIOR LABRAL LESIONS

Test	Description
Clunk test	With the shoulder in maximum elevation, circumduct the humeral head. A clunk that recreates the symptoms is a positive test.
Anterior slide test	The patient places hand on hip. The examiner places one hand on top of the acromion, the other behind the elbow to create a superior force, which the patient resists. Pain in the anterior shoulder constitutes a positive test.
O'Brien's test (active compression test)	Arm is forward flexed to 90 degrees and adducted 10 degrees (see Fig.14-10 in Chapter 14). A positive test demonstrates pain with resisted downward pressure on the internally rotated arm, whereas external rotation alleviates the pain.

relocation test may represent an "unlocking" of internally impinged tissues.

- A variety of provocative tests for the superior labral pathology have been described (Table 16-5).
 - Although these tests may be sensitive for detecting labral tears, none have shown great specificity, and therefore may also be positive in other pathology.
- We prefer the active compression test.

Other Tests

- Examination of the cervical spine is a necessary part of any shoulder exam due to the high frequency of referred pain from this location.
- Furthermore, the lower extremities and trunk should also be carefully examined.

Imaging Studies

Imaging should start with plain radiographs, adding cross-sectional studies such as computed tomography (CT) or magnetic resonance imaging (MRI) as needed to obtain additional information about the bony anatomy and condition of soft tissues.

Radiography

- Basic radiographs should include a true anteroposterior, axillary, and outlet views of the shoulder, with specialized radiographs for the detection of specific lesions added as needed.
- The Stryker notch view is useful in the evaluation of posterior humeral lesions and in the diagnosis of a Bennett's lesion (exostosis of the posterior glenoid).

A West Point view can be used to identify bony Bankart lesions, whereas specialized views are available to evaluate the AC joint for arthritic or traumatic changes.

Computed Tomography

CT scans have specific but limited applications in the evaluation of the thrower's shoulder.

It is the study of choice for the evaluation of glenoid abnormalities, such as bony Bankart lesions and, in conjunction with a contrast arthrogram, it allows for the evaluation of labral tears.

Magnetic Resonance Imaging

- In addition to plain radiographs, MRI is the imaging modality of choice for most conditions of the thrower's shoulder.
- Ideally suited for soft-tissue imaging, MRI is particularly useful in evaluating rotator cuff pathology and injury to the glenoid labrum.
- MRI, when used in conjunction with gadolinium arthrography, has reached a sensitivity of 90%, even for the evaluation of partial thickness rotator cuff tears.
- It also allows for the assessment of muscle degeneration, an important consideration before surgical treatment of chronically retracted rotator cuff tears, and the evaluation of labral cysts.
- To detect intra-articular pathology such as labral tears, the sensitivity of MRI can be augmented by the intra-articular injection of gadolinium. It is important to note that even MRI scans of asymptomatic throwing athletes commonly show pathologic changes; therefore, the MRI findings should be used primarily to support a diagnosis suggested by the history and physical exam findings, rather than as a screening tool.

Diagnostic Arthroscopy

- Diagnostic arthroscopy remains the gold standard for the diagnosis of pathology in the thrower's shoulder.
- Intra-articular pathology can be clearly defined, and the integrity of the rotator cuff and biceps—labral anchor complex can be directly tested.
- By using what some have termed "dynamic-assessment arthroscopy," the diagnosis of internal impingement can be made.
- Viewed from the posterior portal with the shoulder in the ABER (abduction-external rotation) position combined with extension, contact between the undersurface of the rotator cuff and the posterior-superior labrum is easily identified, along with any associated lesions of these and other surrounding structures.
- Diagnostic arthroscopy should be reserved, however, for the throwing athlete who has failed conservative management for 3 to 6 months and still continues to have an unclear diagnosis.

CONDITIONING, TRAINING, AND NONOPERATIVE TREATMENT

With very few exceptions, the treatment of shoulder injuries, especially in professional athletes, should start with a conservative program.

- Conservative management is divided into four phases: rest, stretching, strengthening, and a throwing program.
 - The first phase consists of activity restriction or modification, nonsteroidal anti-inflammatory drugs, ice, massage, and gentle passive ROM exercises.
 - Once the acute pain has diminished, the program should aim to increase motion with the goal of full motion before advancing to the next phase.
 - Focus is typically on contracted structures, such as the posterior capsule and pectoralis minor muscle in throwers.
 - Only after full motion has been restored, the athlete should begin strengthening, with an emphasis on dynamic stabilizers at first but also including trunk and lower extremity musculature in the program.
- The goal is to return to full throwing velocity over the course of 3 months.
- Lack of significant improvement after 3 months, or the inability to return to competitive play within 6 months, constitutes failure of conservative management, and should prompt additional diagnostic tests and consideration of surgical intervention.
- Certain diagnoses such as acute rotator cuff tears or dislocations may warrant earlier and more aggressive surgical intervention on a case by case basis.

SHOULDER CONDITIONS AND SURGICAL CONSIDERATIONS

Laxity and Instability

The development of laxity in the athlete was first described by Neer (1990), was thought to be "acquired," and as such, thought to be a distinct entity separate from traumatic and atraumatic instability. Neer theorized that this acquired laxity resulted from repetitive injury and microtrauma. This concept of acquired laxity gained widespread acceptance. However, there was no solid evidence to demonstrate whether laxity represented a failed repair mechanism or a remodeling response.

Glenohumeral instability and associated internal impingement are probably the most studied but least understood components of pathology in the thrower's shoulder. The definitions of laxity and instability are often blurred in the literature leading to much confusion. Although the terms are related, they are distinct entities. Laxity does not equal instability. Laxity is excessive motion for a particular direction or rotation for a particular joint. It may represent a normal inherent property of the soft tissues or it may be an adaptation for a given sport. For many authors, the term "instability" is generally reserved for the sensation of humeral head translation in the glenoid, associated with pain and discomfort. Taking this into account, the nomenclature of "subtle instability" may have led to some confusion. Others have called this microinstability. Kuhn (2002) recommended that a better description might have been "pathologic laxity.

Although it is obvious that some laxity is essential to compete in high-level overhead sports, excessive laxity may be responsible for the development of shoulder pathology. For example, excessive laxity of the glenohumeral ligaments could predispose the athlete to injury to the labrum and/or rotator cuff. However, this athlete may not have a sensation of instability. This pathologic laxity is the "subtle instability" described by Jobe et al. (1983). It presents as pain with certain motions, but does not result in true apprehension or a feeling of impending dislocation.

Instability presents either as primary, posttraumatic, or microinstability. Primary instability is the result of generalized ligamentous laxity, whereas posttraumatic instability is caused by a distinct traumatic event. Microinstability is the result of repetitive stresses, especially in shear, during the cocking and acceleration phases. Initially, the stretching of anterior structures permits athletes to attain higher degrees of external rotation, thus allowing them to perform at a higher level. Over time, increasing loads lead to further stretching and failure of the anterior capsule. Microinstability develops with increased anteroposterior translation of the humeral head that can lead to labral fraying, subacromial impingement, and rotator cuff tears.

Superior Labrum Anterior-posterior Lesions

The labrum is a fibrocartilaginous lip surrounding and deepening the glenoid. It also serves as the attachment site for the long head of the biceps and the superior and middle glenohumeral ligaments. Labral tears are common in athletes and can be quite debilitating, especially tears of the superior labrum affecting the biceps anchor. Superior labral tears have received increased attention and have been termed superior labrum, anterior—posterior, or SLAP lesions. The original reference describes four types of SLAP lesions (Figs. 16-1 through 16-3; Table 16-6).

Type I SLAP lesions of the superolabral complex are common in throwers, whereas true avulsions of the biceps anchor (type II SLAP lesions) are less frequent. Several theories exist regarding their etiology. Classically, SLAP lesions were thought to be the result of traction or compressive mechanisms, such as sudden pulling on the arm or falls on the outstretched arm. It was thought that traction on the biceps was likely responsible for the development of these lesions during the deceleration phase of throwing, but recent biomechanical studies and arthroscopic observations have suggested the extreme external rotation seen in the thrower's shoulder as the causative factor. Increased strain at the biceps anchor during the late cocking phase with the arm in maximum external rotation results in a "peel-back" effect, which has been suggested as the mechanism behind

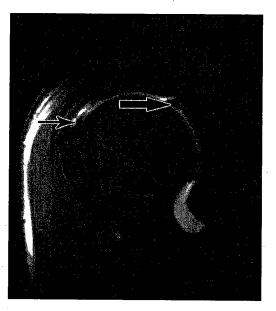
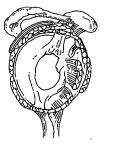


Figure 16-2 Shoulder MRI demonstrating partial thickness rotator cuff tear (small arrow) and SLAP tear (large arrow). (From Magee T, Williams D, Mani N. Shoulder MR arthrography: which patient group benefits most? Am J Roentgenol 2004;183:969-974.)

the development of SLAP lesions in throwers (see Fig. 16-5). This is supported by laboratory studies that have shown the long head of the biceps to be an important dynamic restraint to external rotation of the abducted arm. As part of the "peel-back" theory, the authors have noted an increased incidence of SLAP lesions in patients with decreased total arc of motion, such as seen in baseball pitchers who often have internal rotation deficits greater than the concomitant gain in external rotation (Fig. 16-4). Burkhart and Morgan (1998) have developed a theory regarding the association between decreased glenohumeral internal rotation and the development of pathology in the shoulder. This model is known as glenohumeral internal rotation deficit (GIRD) and will be discussed in more detail later in the chapter.

Diagnosis

- SLAP lesions present with vague pain, which sometimes localizes to the posterosuperior joint line and can be exacerbated with overhead activities.
- They can produce symptoms of locking or snapping and, depending on tear size, instability.







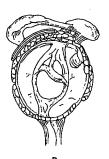


Figure 16-1 SLAP types. A: Type 1. B: Type 2. C: Type 3. D: Type 4.

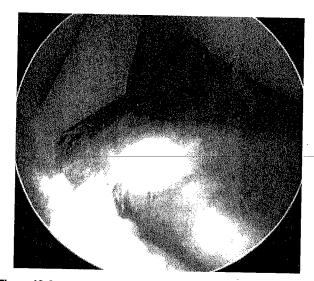


Figure 16-3 Shoulder arthroscopy, view from posterior portal, demonstrating the biceps anchor with a SLAP tear.

- Throwers frequently report pain in the late cocking phase and loss of velocity.
- Posterior tightness and positive provocative tests are common physical findings.
- Radiographic workup should include conventional radiographs and MRI arthrogram to delineate the lesion further.

Treatment

- Treatment of SLAP lesions is typically conservative at first, with many players responding to rest and rehabilitation in the acute period.
- If the acute inflammation in the shoulder has subsided, and the player has completed a course of rehabilitation

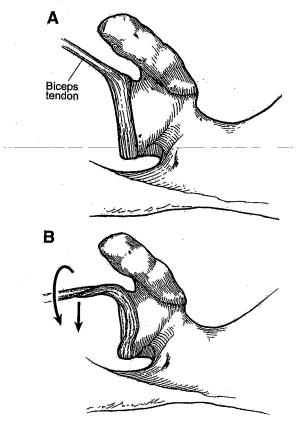


Figure 16-4 Peel-back effect. A: Superior view of the biceps and labral complex of a left shoulder in a resting position. B: Superior view of the biceps and labral complex of a left shoulder in the abducted, externally rotated position, showing peel-back mechanism as the biceps vector shifts posteriorly. (From Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part I: pathoanatomy and biomechanics. Arthroscopy 2003;19:404–420; with permission from the Arthroscopy Association of North America.)

TABLE 16-6 SLAP LESIONS

Туре	Description
1 .	Fraying of the labrum with a stable biceps anchor
11	Detachment of the biceps anchor from the superior glenoid in addition to the fraying observed in type I lesions
Α	Anterior
B _i	Posterior
С	Combined
Ш	Bucket-handle tear of the superior labrum with a preserved biceps anchor
IV	Extension of the bucket-handle tear into the biceps tendon but no instability of the anchor itself

but is still unable to resume throwing, serious consideration should be given to surgical intervention.

- Surgical treatment of symptomatic SLAP lesions consists of shoulder arthroscopy, which frequently demonstrates a positive "drive-through" sign, a displaceable biceps vertex and, in up to 60% of cases, associated rotator cuff pathology, mostly partial-thickness undersurface tears.
- If the biceps-labral anchor is avulsed, it is partially debrided and secured back to the glenoid with suture anchors, followed by a postoperative rehabilitation program for posterior capsular stretching.
- If minor tearing and fraying are present, but no true avulsion of the biceps anchor, a simple labral debridement can be performed.
- Although thermal capsulorrhaphy has fallen into disfavor in most cases, there have been some favorable results in the throwing athletes with superior labral tears.
 - When thermal capsulorrhaphy was combined with labral repair, better results were seen than with labral repair alone, and 87% of overhead athletes were able to return to play.

- After formal repair of the biceps—labral complex, throwers undergo a brief period of immobilization, followed by a rehabilitation program that focuses on throwing mechanics.
- Return to play is typically 4 to 6 months postoperatively, although return to elite throwing may take closer to 1 year.
- Patients with a stable biceps anchor at the time of surgery who have undergone only limited labral debridement are not immobilized after surgery and can typically resume play after 4 to 6 weeks of rehabilitation.
 - Authors have reported on the outcome of this type of program, with return to preinjury performance levels in more than 80% of pitchers.

Rotator Cuff Disorders and Impingement

- Most rotator cuff tears in this population are partialthickness, articular-sided tears.
- Some result from acute tensile overload but more commonly the cause is repetitive microtrauma and eccentric failure of the fibers.
- Whereas cuff tears may occur in the setting of impingement, in this population they are more commonly the result of subtle instability.
- Subacromial decompression alone has not been effective in the athletic population, with return to previous activity levels in only half the patients.
- Similarly, simple debridement of partial tears, a largely effective procedure in lower-demand patients, produces less consistent results especially in the overhead athlete.
- Full-thickness rotator cuff tears are a rare event in the overhead athlete but have a very poor prognosis even when repaired, with only half of all players able to return to play.
- Several types of impingement have been described in the literature:
 - "Classic" subacromial or outlet impingement
 - "Secondary" or nonoutlet impingement
 - Subcoracoid impingement
 - Internal impingement

Classic Subacromial Impingement or External Impingement

- The "classic" form of impingement as described by Neer (1983) is the result of compression of the rotator cuff between the coracoacromial arch and the humeral head.
- Anatomical variants such as a hooked acromion, acromioclavicular joint arthritis with osteophyte formation, and a laterally sloping acromion have been proposed as predisposing factors.
- Subacromial impingement is typically diagnosed in the older throwing athlete who has a stable shoulder.
- These overhand athletes will often have loss of internal rotation without concomitant increase in external rotation as seen in many younger throwers.
- Adaptive bony changes may also play a role in this loss of internal rotation.
- Subacromial impingement can be further exacerbated by weakness of the rotator cuff from fatigue or improper technique, leading to superior migration of the humeral head.

- These patients have a painful arc, positive impingement maneuvers, and will typically respond well to subacromial injections.
- Radiographs in older throwers usually show varying degrees of an acquired, or congenitally prominent anterior acromion that predisposes to outlet stenosis.
- Many patients improve with anti-inflammatory medications combined with a well-supervised physical therapy program focusing on rotator cuff rehabilitation and scapular dynamics.
- Arthroscopy with subacromial decompression is reserved for those who fail conservative management.
- Unlike the subacromial space of a younger thrower, which is typically smooth and white in appearance, the older thrower can demonstrate an irritated and thickened bursa with fraying, matched excoriation and hypertrophy of the coracoacromial ligament.
- If a significant bursal-sided partial or full-thickness rotator cuff tear is present, consideration for repair is recommended, either through a "mini-open" or arthroscopic approach.
- It is imperative to inform patients before surgery, however, that a return to the same premorbid level of competition is unlikely.
- External impingement as the sole source of pain appears to be relatively uncommon in throwing athletes, with the exception of the older thrower.
 - This may help explain why a high failure rate of almost 80% was seen in early reports for throwing athletes being treated with subacromial decompression for apparent impingement.

Secondary Impingement

- Secondary, or nonoutlet, impingement is a dynamic process in which a normal subacromial arch is present but there is abnormal proximity between the arch and the underlying rotator cuff.
- There is a strong association between scapulothoracic dyskinesia and impingement symptoms.
 - Weakness in the scapular stabilizers leads to lack of proper rotation of the scapula during humeral elevation.
 - As a result, the space available for the rotator cuff is acutely narrowed and thus causes impingement symptoms.
- Posterior capsular tightness can also create a vector imbalance resulting in posterior-superior migration of the humeral head with secondary rotator cuff symptoms.
- Malunion from displaced fractures of the greater humeral tuberosity, and massive rotator cuff tears with loss of the humeral head depressors, can also result in secondary impingement.
- Treatment recommendations are based on the primary pathology.
 - If the secondary impingement is associated with a partial-thickness rotator cuff tear affecting more than half the cuff thickness, the recommended treatment includes a formal open, mini-open, or arthroscopic cuff repair.
 - When scapular dyskinesia is the cause of secondary impingement, rehabilitation of the periscapular musculature is typically successful.

When impingement is caused by tightness involving the capsule, as in adhesive capsulitis, or by adhesions in the subacromial space, as seen in trauma or postsurgical cases, surgical correction with lysis of adhesions is recommended.

Coracoid Impingement

- Coracoid impingement occurs when the subscapularis tendon is compressed between the lesser tuberosity and the coracoid tip.
- Possible causes include postoperative changes (e.g., Bristow procedure), previous trauma, anterior instability, and idiopathic impingement.
- Coracoid impingement is typically a diagnosis of exclusion.
 - Patients present with localized anterior shoulder pain, which can mimic or occur in combination with subacromial impingement.
 - The test most often cited in the literature is pain localized to the coracoid when the shoulder is passively forward flexed, adducted, and internally rotated.
 - This test differs from O'Brien's test, because the latter requires active resistance in this position.
- Injections in the subcoracoid space have been recommended to aid in the diagnosis and treatment of the condition.
- A shortened coracohumeral distance, the distance between the coracoid and the lesser tuberosity with the arm in maximum internal rotation (average 11 mm in normal vs. 5.5 mm in symptomatic shoulders) has been described in association with subcoracoid impingement. This, however, is not specific to this problem.
- If conservative measures fail, a coracoidplasty is the next appropriate step in treatment.
 - This has been described both open and arthroscopically, with the goal being to debride the tip of the prominent coracoid to increase the space between the coracoid and the lesser tuberosity.

Internal Impingement

Walch et al. (1992) first described internal impingement as a physiologic phenomenon in which the undersurface of the rotator cuff contacts the posterior-superior labrum when the arm is placed in maximum external rotation and abduction (Fig. 16-5). Halbrecht et al. (1999) demonstrated this phenomenon in college baseball players and showed that internal impingement can occur even in the absence of symptoms. This is thought to result from recurrent microtrauma, which can ultimately lead to rotator cuff tearing and destabilization of the biceps—labral complex. Internal impingement presents as a spectrum of pathologies with significant overlap that typically involves SLAP lesions, partial thickness rotator cuff tears, hyperlaxity of the anterior glenohumeral ligaments, and posterior capsular contractures.

Several authors have postulated that internal impingement is most likely caused by shoulder girdle muscle fatigue resulting from a lack of conditioning or overthrowing and/ or anterior capsular stretch resulting in anterior capsular insufficiency. The authors believe that, during the acceleration phase of throwing, the humerus should be aligned in the plane of the scapula and that with fatigue of the shoulder girdle muscles the humerus drifts out of the scapular plane. This has been termed "hyperangulation" and is called opening up" by many pitching coaches. This hyperangulation of the humerus in turn stresses the anterior capsule (Fig. 16-6). Loss of anterior capsular integrity compromises the normal posterior rollback of the humeral head, leading to anterior translation, therefore causing the undersurface of the rotator cuff to abut against the margin of the glenoid and labrum. Reducing the laxity in the anterior inferior glenohumeral ligament seems to improve outcome significantly in the throwers with internal impingement.

Glenohumeral Internal Rotation Deficit

Burkhart et al. (2003) have recently questioned whether or not internal impingement actually occurs. They described their own model (GIRD) as the primary cause behind the pathologic changes seen in the "internal impingement" patient. The Morgan-Burkhart model is based on the frequency of posterior capsular contractures in throwers. Combined with the possibility of acquired humeral retroversion, the tight posterior capsule shifts the center of rotation of the humerus in the posterior-superior direction. This permits greater clearance of the greater tuberosity. Because of

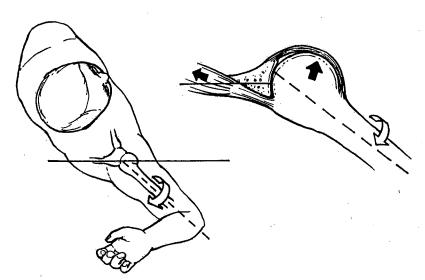


Figure 16-5 Internal impingement of the undersurface of the rotator cuff against the posterior labrum in maximum external rotation/abduction. (From Meister K. Injuries to the shoulder in the throwing athlete. Part I: biomechanics/pathophysiology/classification of injury. Am J Sports Med 2000;28:265–275.)

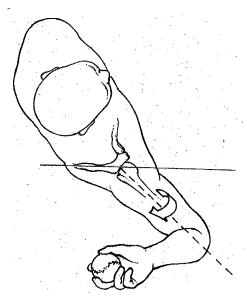


Figure 16-6 Hyperangulation of the humerus.

the diminished "cam" effect, the anterior capsule becomes functionally lengthened (Fig. 16-7). With a functionally lengthened anterior capsule allowing clearance of the greater tuberosity, excessive external rotation is achieved. As a result, the biceps anchor is "peeled back" under tension, causing injury to the posterior-superior structures, most notably to the posterosuperior labrum. The progres-

sion of the "peel-back" mechanism allows further "pseudo-laxity" of the anterior capsule to occur. The pathologic cycle culminates in torsional failure of the rotator cuff, not compressional failure as in the internal impingement model. The end results are articular-sided partial rotator cuff tears and SLAP lesions typically seen in the throwing shoulder.

The GIRD model attempts to quantify the internal rotation deficit to identify those players at risk for pathology. Defined as a greater than 25-degree loss of internal rotation of the dominant shoulder, compared with the contralateral side, GIRD is a common phenomenon in throwing athletes. Some studies have found average deficits of up to 50 degrees when compared with the contralateral side, with concomitant increases in external rotation on the order of 30 degrees. Shoulders with a total arc of motion less than 180 degrees and an internal rotation deficit of greater than 25 degrees seem to be at risk for developing SLAP lesions as a result of increased posterosuperior "peel-back" forces.

Verna (1991) is credited with first recognizing the relationship of GIRD with the development of shoulder dysfunction. By following 39 professional pitchers over a single season, he demonstrated that the development of shoulder problems occurred in more than half of the players with GIRD greater than 35 degrees.

In a similar study by Kibler (1998), high-level tennis players were divided in two groups and prospectively followed for 2 years. One group performed daily posteroinferior capsular stretching to minimize GIRD, whereas the control group continued their routine exercise program. Over the course of the study period, those in the stretching group

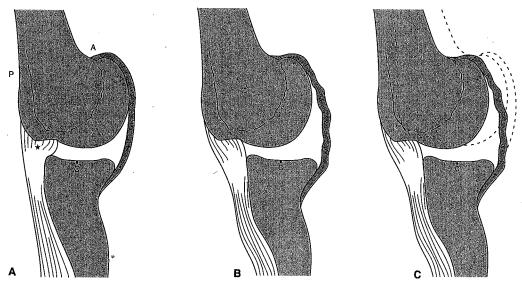


Figure 16-7 Cam effect. A: With the arm in a position of abduction and external rotation, the humeral head and the proximal humeral calcar produce a significant cam effect of the anteroinferior capsule, tensioning the capsule by virtue of the space-occupying effect. B: With a posterosuperior shift of the glenohumeral contact point, the space-occupying effect of the proximal humerus on the anteroinferior capsule is reduced (reduction of cam effect). This creates a relative redundancy in the anteroinferior capsule that has probably been misinterpreted in the past as microinstability. C: Superimposed neutral position (dotted line) shows the magnitude of the capsular redundancy that occurs as a result of the shift in the glenohumeral contact point. (After Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part I: pathoanatomy and biomechanics. Arthroscopy 2003;19:404–420.)

had a 38% decrease in the incidence of shoulder problems compared with controls.

Approximately 90% of throwers with GIRD respond to a physical therapy program focused on stretching of the tight posterior capsule, with a concomitant decrease in shoulder-related problems. The remaining 10%, frequently older elite players, who are unresponsive to conservative treatment can be treated by selective arthroscopic postero-inferior capsulotomy in the zone of the posterior band of the inferior glenohumeral ligament.

Increased Humeral Retroversion

Recent studies have investigated the issue of acquired humeral retroversion, its contribution to throwing, and its relevance to internal impingement. Increased humeral retroversion allows for increased external rotation with an obligate loss of internal rotation. Interestingly, Riand et al. (1998) reported that a loss of normal humeral retroversion (normally 25 to 35 degrees) to less than 10 degrees total humeral retroversion will increase the risk of contact between the greater tuberosity and the posterior-superior glenoid labrum (e.g., internal impingement). In patients with a loss of humeral retroversion (as opposed to throwing athletes who typically have increased retroversion), the subsequent internal impingement was corrected with humeral osteotomy.

Scapular Dyskinesia

The work of Kibler (1998) has added greatly to our understanding of scapular dynamics and its role in preventing injuries in the throwing athletes. The scapula functions to provide a stable platform for the humeral head during rotation and elevation, while transferring kinetic energy from the legs and trunk to the upper extremity. It has been estimated that only half of the kinetic energy imparted to the ball results from arm and shoulder action. The remaining half is generated by leg and trunk rotation, and is transferred to the upper limb through the scapulothoracic joint, making it an important, but frequently overlooked part of the kinetic chain.

Scapular dyskinesia results from imbalances of the periscapular musculature secondary to fatigue, direct trauma, or nerve injury (e.g., the long thoracic nerve). It can negatively impact shoulder function in several ways. To reach the extremes of motion needed in overhead athletics, elevation of the acromion is required or else impingement results. Normal function of the serratus anterior, trapezius, and rhomboid muscles is required to achieve the necessary scapular positioning. Loss of function from nerve injury, weakness, and/or fatigue leads to scapular hyperangulation and a relative increase in glenoid anteversion, placing the anterior capsular structures at risk. Associations between scapular dyskinesia and anterior instability and impingement have been documented by several authors.

Because the scapula transfers energy derived from trunk rotation to the pitching arm, destabilization of the scapula results in energy losses that decrease velocity. In an attempt to compensate for the loss of power, the pitcher tries to regain velocity by increasing the effort of the shoulder muscles, which results in increased strain on the shoulder. For these reasons, rehabilitation of the throwing athlete must

have a strong emphasis on strengthening and conditioning the scapular stabilizers.

- The vast majority of scapula-related issues can be resolved by a physical therapy program directed at the scapular stabilizers.
- Sometimes, however, surgical intervention can be required for entities such as scapular bursitis or a snapping scapula, which can be treated by excision of the offending tissues at the inferior and/or superior margin of the scapula.

Bennett Lesion

- The Bennett lesion is a mineralization at the posteroinferior glenoid present in approximately 20% of major league pitchers, best seen on the Stryker-Notch view.
- The lesion is thought to be the result of enthesopathic changes of the posterior capsule and inferior glenohumeral ligament.
- It is an infrequent cause of pain in the overhead athlete and can be associated with tears of the posterior labrum and rotator cuff.
- The diagnosis of a symptomatic Bennett lesion is difficult but frequently presents with posterior shoulder pain during throwing, especially in the follow-through phase.
- Tenderness to palpation of the posteroinferior glenohumeral joint is common, whereas resolution of pain with local injection can be both diagnostic and therapeutic.
- Symptomatic Bennett lesions can be treated by arthroscopic debridement.

Chondral Injuries

- True osteochondritis dissecans of the shoulder is a very rare occurrence, with less than 20 cases described in the literature.
- Traumatic osteochondral defects are seen more frequently as impression fractures of the humeral head (Hill-Sachs lesion), and fractures involving the glenoid rim (Bankart lesion) after anterior glenohumeral dislocation.
- Both can be the cause of recurrent dislocations, in which case they should be corrected by grafting of the Hill-Sachs lesion and fixation of the glenoid fracture.

Neurovascular Conditions

Vascular Injuries

- Vascular compromise after shoulder injury is rarely seen outside major trauma such as scapulothoracic dissociation injuries.
- Presenting predominantly as arterial thrombosis rather than transection, these injuries occur in less than 1% of shoulder dislocations and proximal humerus fractures.

Effort Thrombosis

- Effort thrombosis is a rare entity presenting with symptoms of tiredness, heaviness, and gradual development of swelling over the course of a few days.
- It has been described in a wide range of activities, including baseball, softball, hockey, swimming, wrestling, and backpacking.

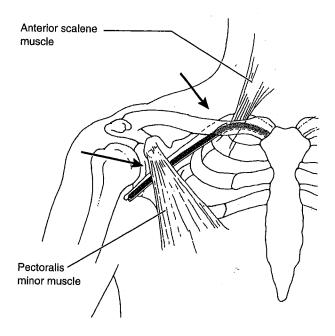


Figure 16-8 Common compression sites in thoracic outlet syndrome. (After Meister K. Injuries to the shoulder in the throwing athlete. Part 2: evaluation/treatment. Am J Sports Med 2000;28: 587-601.)

- Exam findings include slight discoloration, venous engorgement, and size difference, compared with the contralateral extremity.
- Venography or more modern CT or MRI-based imaging typically demonstrate thrombosis of the subclavian vein at the level of the first rib.
- The cause, although still not conclusively proven, is likely compression of the vascular structures between the first rib and the clavicle, especially with the arm in maximum abduction.
- Treatment options include catheter-directed thrombolysis, balloon venoplasty, and staged resection of the first rib with good results and return to preinjury level of play within 6 to 36 months.

Thoracic Outlet Syndrome (TOS)

- This term describes the compression of neurovascular structures that traverse the thoracic outlet, which is formed by the clavicle, first rib, and the anterior scalene muscle (Fig. 16-8).
- A subset of patients has an identifiable cause for the compression such as cervical ribs, exostosis of the first rib, or malunions of the first rib or clavicle.
 - In most cases, however, no such abnormality can be identified.
- Presenting complaints are neurological in greater than 90% of patients, and include pain, paresthesias, and weakness—especially in a lower plexus distribution.
- Vascular symptoms occur rarely and commonly present as activity-related claudication, pulse, or blood-pressure deficits.

- The workup is complicated by the lack of any specific diagnostic tests.
- Several provocative tests have been described, such as placing the affected extremity in maximum abduction and external rotation, which leads to recreation of symptoms in more than 80% of patients.
- Management should be conservative initially, with activity modification, nonsteroidal anti-inflammatory drugs, strengthening of the shoulder girdle, and scapular stabilizers. This is successful in more than 70% of patients.
- Surgical treatment is reserved for those severely affected or for those with refractory pain after conservative management.
- Studies have demonstrated a greater than 90% success rate with surgical decompression, frequently by resection of the first rib through a transaxillary approach.

Quadrilateral Space Syndrome

- Quadrilateral space syndrome is defined as compression of the axillary nerve and posterior humeral circumflex artery as they traverse the quadrilateral space.
 - This space is defined by the humerus laterally, the long head of the triceps medially, and the teres minor and major muscles superiorly and inferiorly, respectively.
- This rare condition presents in overhead athletes with nonspecific symptoms such as dull, aching, or burning pain in the posterolateral aspect of the shoulder, exacerbated by activity, especially with repetitive exercise with the arm abducted and externally rotated.
- Physical findings include deltoid weakness and wasting, pain to palpation over the quadrilateral space, and reproduction of symptoms with the arm in the flexionabduction-external rotation (FABER) position.
- Angiography frequently demonstrates occlusion of the posterior humeral circumflex artery when the arm is placed in the FABER position, whereas electromyographic studies can demonstrate denervation in the deltoid and teres minor muscles.
- Due to the rarity of the syndrome, no definite treatment guidelines have been established, but current recommendations include conservative treatment initially, with surgical exploration and release of the neurovascular structures reserved for refractory cases.

SUMMARY

The etiology of injuries seen in the thrower's shoulder is multifactorial. Overlapping signs and symptoms exist, as well as numerous causes of disability. The problems appear to be a combination of abnormal mechanics, muscle fatigue and imbalance, scapular dyskinesia, increased humeral retroversion, posterior capsular contractures, anterior capsular laxity, and repetitive microtrauma. As a result, throwers commonly develop multiple areas of pathology involving the posterior superior labrum, the articular surface of the rotator cuff, cartilage lesions and bony exostoses of the posterior glenoid, cystic changes at the insertion of the rotator cuff, thickening of the posterior capsule, and redundancy of the anterior capsule.

The etiology and exact pathomechanics of throwing remain controversial and are complicated by the difficulty of recreating an accurate in vitro model of the complex kinetic chain. Different schools of thought exist regarding the initiating event for many of the problems seen in the thrower's shoulder—whether it is anterior capsular laxity or posterior capsular tightness. Fortunately, for the practitioner, regardless of the conflicting theories regarding the pathomechanics at work in the throwing shoulder, the evaluation and treatment algorithms of the injured athlete are, with few exceptions, very similar.

Regardless of the specific cause, the repetitive stresses experienced typically during the late cocking and early acceleration phase result in damage to the posterior glenoid, the biceps-labral complex, and the articular surface of the rotator cuff. The forces acting on these posterior structures are a combination of compressive, tensile, and torsional forces, which culminate in actual fiber failure of both the biceps—labral complex and the rotator cuff. Conditioning of the entire kinetic chain, and respecting adequate recovery periods between games, is imperative, and it is the responsibility of the coaches, trainers, and physicians to educate the players.

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