Current Concepts Review

Shoulder Injuries in the Throwing Athlete

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- Pathologic conditions in the shoulder of a throwing athlete frequently represent a breakdown of multiple elements of the shoulder restraint system, both static and dynamic, and also a breakdown in the kinetic chain.
- Physical therapy and rehabilitation should be, with only a few exceptions, the primary treatment for throwing athletes before operative treatment is considered.
- Articular-sided partial rotator cuff tears and superior labral tears are common in throwing athletes. Operative treatment can be successful when nonoperative measures have failed.
- Throwing athletes who have a glenohumeral internal rotation deficit have a good response, in most cases, to stretching of the posteroinferior aspect of the capsule.

Throwing athletes are prone to shoulder injuries as a result of the high forces placed on the shoulder during the throwing motion. Dynamic stabilizers of the glenohumeral joint include the rotator cuff, the scapulothoracic muscles, and the long head of the biceps tendon. Static stabilizers include the osseous anatomy, the fibrocartilaginous labrum, and the glenohumeral joint capsule. While a single traumatic event may cause injury, more commonly it is repetitive overuse that leads to failure of one or more of these structures.

The act of throwing requires a coordinated motion that progresses from the toes to the fingertips. This sequence of events has been described conceptually as a kinetic chain[1]. For the kinetic chain to work effectively, sequential muscle activity is required so that the energy that is generated in the lower body can be transmitted to the upper body through the arm, hand, and fingers, and finally to the ball[2]. The ball velocity is determined by the efficiency of this process. Body rotation, timing, and positioning of the scapula are key elements in the kinetic chain. Any physical condition that alters the components of the kinetic chain, especially one that affects the so-called core (trunk, back, and proximal parts of the lower limbs), will alter more distal segments and may result in the development of a dysfunctional shoulder[1].

In elite-level throwers, there is a delicate balance between shoulder mobility and stability. The shoulder needs to be mobile enough to reach extreme positions of rotation so that velocity can be imparted to the ball, but at the same time the shoulder needs to remain stable so that the humeral head remains within the glenoid socket, creating a stable fulcrum for rotation; this is known as the "thrower's paradox."[3] With each pitch, the soft-tissue envelope that surrounds the shoulder is loaded at levels that approach the ultimate failure loads of the tissues, which are thus quite vulnerable to injury. The demands and repetition of high-velocity overhead throwing can alter this stability-mobility relationship and ultimately lead to injury.

While the injury patterns in the shoulders of high-level throwers are common and predictable, there is still some controversy about the exact mechanisms by which these injuries occur. Recent biomechanical studies have helped to enhance our understanding of the pathogenesis of injury in throwing athletes[4-8]. Furthermore, quantitative information about the biomechanics and kinematics, both normal and pathologic, has helped clinicians to develop effective prevention, treatment, and rehabilitation strategies for throwers[9-11].

The purpose of this article is to review the biomechanics of throwing and how it contributes to injury. A basic review

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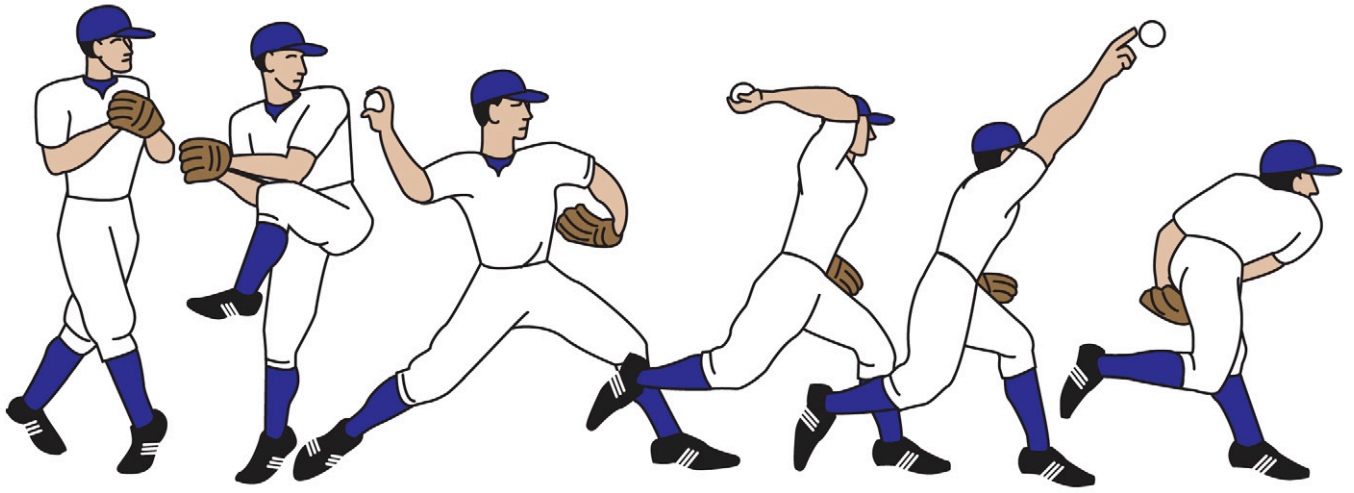

Fig. 1

The six phases of the throwing motion. Phase 1 is the wind-up phase. Phase 2 is the early cocking phase, ending with planting of the striding foot. Phase 3 is the late cocking phase, in which the arm reaches maximum external rotation. In Phase 4, the ball is accelerated until Phase 5 starts with release of the ball and deceleration of the arm. Phase 6, the follow-through, rebalances the body until the motion stops.

of the kinematics and skeletal adaptations that occur during throwing will serve as a foundation of information that is necessary for the evaluation and treatment of a throwing athlete's shoulder.

Kinematics of Throwing

The throwing motion has been divided into six phases, which usually take less than two seconds to occur (Fig. 1)[12-15]. The first three phases consist of wind-up, early cocking, and late cocking and take approximately 1.5 seconds in total. Although the duration of the fourth phase, acceleration, is only 0.05 second, the greatest angular velocities and the largest change in rotation occur during this phase. Consequently, most injuries manifest during this phase. The final two phases are deceleration and follow-through, and together they last approximately 0.35 second[16]. As certain injuries manifest in certain phases, it is important to determine when the pain or problem occurs.

To be successful, an overhead-throwing athlete needs to achieve both velocity and precision. Ball velocity depends on a variety of biomechanical factors but is most directly related to the amount of external rotation that the shoulder achieves[5,10]. Precision, which is the ability to throw the ball to a predetermined location, is related to the thrower's ability to create specific arm positions and exact timing of ball release in a reproducible way[17]. In order to generate maximum ball velocity in the most efficient manner, the lower and upper extremities must work in a synchronous and coordinated fashion. While high angular velocities can be achieved by elite throwers[10], there are limits, as the forces that are generated approach the fatigue strength of many of the soft-tissue structures that surround the shoulder[18,19]. Elite pitchers can generate ball velocities that exceed 90 mi (144.8 km)/hr; in order to create this velocity, the shoulder rotates at angular velocities of up to 7000°/sec[10]. At ball release, the shoulder of a professional pitcher can be exposed to distractive forces of up to 950 N[20]. In the deceleration phase, the compressive forces created by the rotator cuff and deltoid muscle are in the range of 1090 N with posterior shear forces of up to 400 N[20]. These forces approach the ultimate tensile strengths of the soft tissues that support the shoulder. For example, the anterior aspect of the capsule resists approximately 800 to 1200 N in twenty to thirty-year-old individuals[21]. Therefore, if compressive forces do not counteract the high distraction forces, injuries will occur.

The scapula functions to provide a stable platform for the humeral head during rotation and elevation, while transferring kinetic energy from the lower limbs and trunk to the upper extremity. The work of Kibler has added greatly to our understanding of scapular dynamics and injury prevention and treatment[22]. 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 lower-limb and trunk rotation and is transferred to the upper limb through the scapulothoracic joint, making that articulation an important, but frequently overlooked, part of the kinetic chain[23].

Because of the repetitive nature of throwing, the high velocities that occur, and the large forces that are generated, the shoulders of throwing athletes are at high risk for injury. Dynamic analysis of the shoulder during throwing has added to our current knowledge of normal and abnormal shoulder function and, by demonstrating which muscle groups are active during each phase of the throwing motion, has helped to guide the development of injury prevention and rehabilitation programs[24].

Anatomical Adaptations

The repetitive nature of pitching and the high forces that it causes result in adaptive changes of the dominant extremity[18,25].

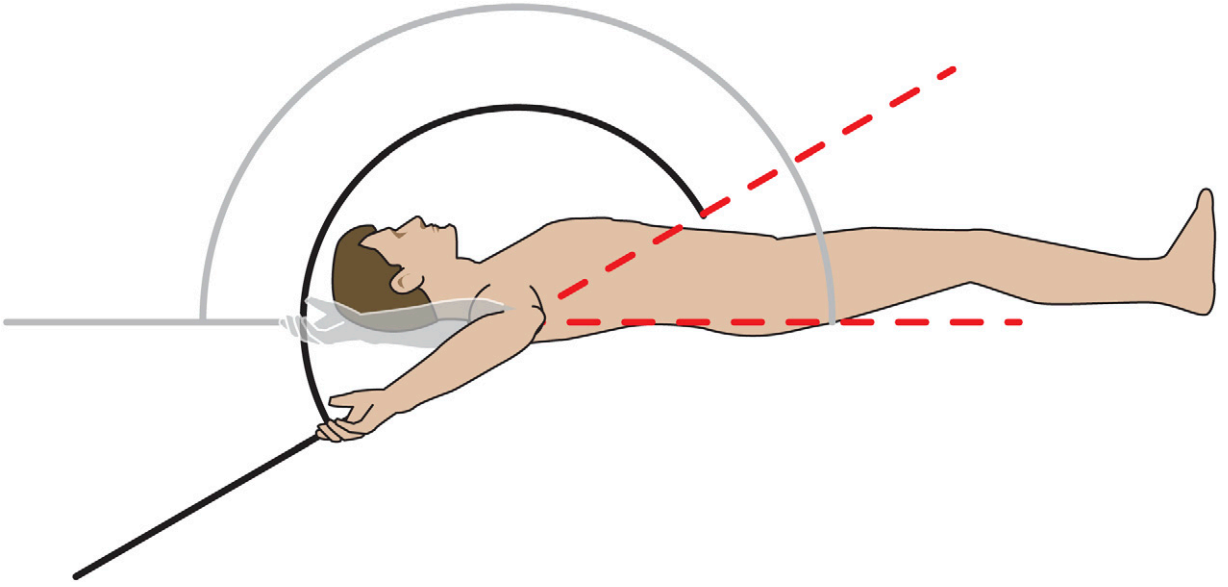

Fig. 2
The arc of motion of the throwing shoulder is shifted posteriorly, with increased external rotation and decreased internal rotation of the abducted shoulder.

These adaptations affect both the soft tissues and the bones. The laxity and range of motion (mobility) of the throwing arm change. The total arc of motion, including maximum internal and maximum external rotation of the abducted arm, is typically around 180° in healthy individuals[3]. The arc of motion of the dominant arm of an asymptomatic elite-level throwing athlete typically is shifted posteriorly, with increased external rotation and decreased internal rotation of the abducted shoulder (Fig. 2)[26-31]. One theory is that the increase in external rotation is caused by an adaptive increase in humeral retroversion and that any substantial internal rotation deficit (of >20°) is therefore related to soft-tissue adaptations. In addition to acquired retroversion of the humerus, there are increases in bone mineral density in the throwing arms of athletes[32,33].

In addition to osseous adaptations, there are soft-tissue adaptations that contribute to joint mobility. For example, baseball pitchers commonly demonstrate an increased sulcus sign on physical examination. The sulcus sign is manifested by dimpling of the skin underneath the acromion with inferior traction on the arm[34], and an excessive sulcus sign may be caused by laxity of the coracohumeral ligament and rotator interval structures that restrain external rotation of the abducted arm[26,35-37]. Another important restraint against maximum external rotation is the anteroinferior glenohumeral ligament. Repetitive ligament stresses may lead to microtears in the collagen fascicles and capsular laxity, which would also allow increased external rotation[36,38,39].

Adaptive changes of the muscles of the dominant shoulder and arm also occur in throwing athletes, particularly pitchers[29,40]. It is not unusual for throwing athletes to have hypertrophy of the shoulder girdle and arm muscles. However, there are also reports of loss of external rotation power of the dominant shoulders of pitchers, with simultaneous increases in the strength of the internal rotator muscles and adductor muscles[41-43].

Conditioning, Training, and Nonoperative Treatment

Because of the nature of shoulder injuries in throwers, the vast majority of them should be initially treated with nonoperative methods. Only certain diagnoses, such as traumatic injuries with an acute rotator cuff tear or a dislocation, may warrant earlier and more aggressive operative intervention.

As the kinetic chain is fundamental to the throwing motion, a training program that strengthens all elements of this chain and links them smoothly is very important. The athlete should be taught to work on a balanced distribution of training exercise for the agonist and antagonist muscles of the upper and lower extremities and the trunk[44,45]. A phased progression of rehabilitation has been suggested for the nonoperative management of overhead-throwing athletes[3,46].

Phases of Physical Therapy

In Phase 1, or the acute phase, the focus is on allowing the injured tissue to heal, activity modification, decreasing pain and inflammation, and normalizing range-of-motion deficits. Passive range-of-motion and active-assisted exercises, nonsteroidal anti-inflammatory drugs, massage therapy and manual lymphatic drainage, neuromuscular facilitation, and rhythmic stabilization exercises may be used, at the therapist's discretion, during this phase. Various levels of evidence support the use of these modalities[47]. Others, such as cryotherapy, are supported by Level-I evidence[48,49].

When pain and inflammation have decreased, the athlete may progress to Phase 2, in which strengthening and neuromuscular exercises are initiated and the range of motion is

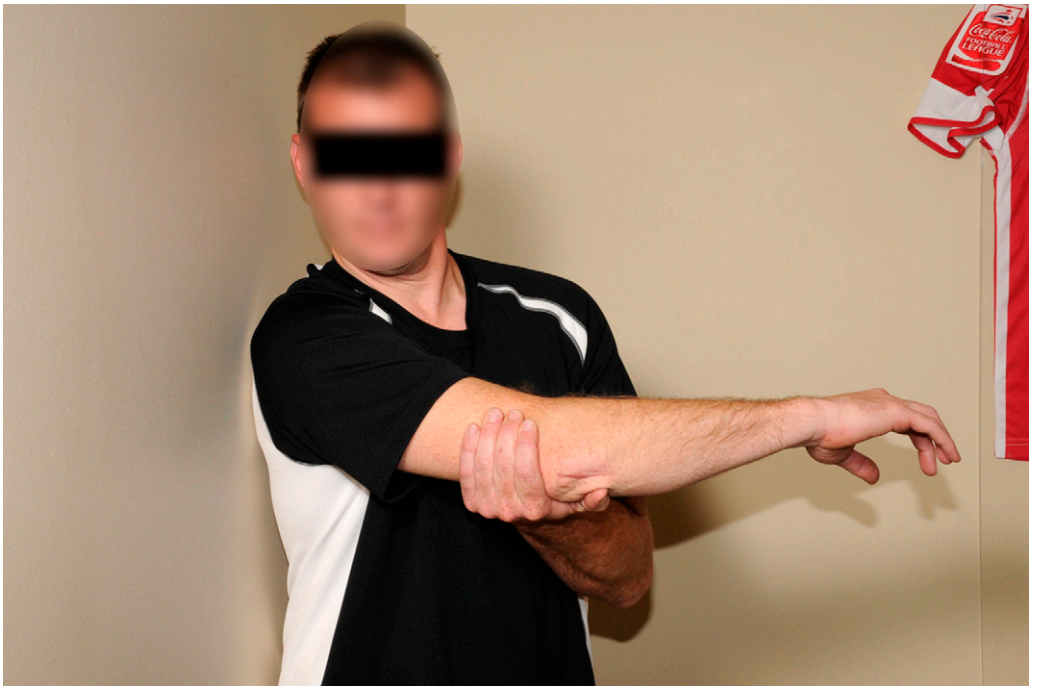

Fig. 3-A

For the cross-body stretch, the individual places the involved shoulder against a wall to prevent the scapula from rotating. The other arm is used to pull the involved arm across the body, placing a stretch on the posterior aspect of the shoulder.

Fig. 3-B

For the sleeper stretch, the individual lies on the involved side with the shoulder in approximately 90° of forward elevation. The other arm is used to internally rotate the involved shoulder until a stretch is achieved on the posterior aspect of the shoulder.

normalized to the preinjury level. Limitations of motion, particularly loss of internal rotation, should be addressed with a specific stretching regimen[3,50,51]. Contractures of the posterior structures, pectoralis minor, and short head of the biceps can contribute to glenohumeral internal rotation deficit and increased anterior tilting of the scapula[52]. McClure et al. showed that use of the cross-body stretch (Fig. 3-A) to treat patients with posterior shoulder tightness led to a significantly greater increase of internal rotation, in the position of 90° of shoulder abduction and 90° of elbow flexion (the 90/90 position), compared with that in a control group with normal shoulder motion who performed no exercises ($p = 0.009$); however, treatment with the sleeper stretch (Fig. 3-B) did not result in significant increases when compared with the findings in the control group[53]. Borstad and Ludewig found the unilateral corner stretch and the supine manual stretch to be effective for lengthening the pectoralis minor[54]. (The unilateral corner stretch is performed, in the 90/90 position with the forearm on a doorframe, by rotating the trunk away from the side that is being stretched. To perform the supine manual stretch, the patient lies supine on a treatment table with a towel roll along the upper thoracic spine, the arm is placed in a 90/90 position, and the therapist applies a posteriorly directed force on the coracoid process.) However, caution must be exercised to not overstretch the anterior aspect of the capsule in these positions.

A strengthening program should be developed on the basis of areas of weakness noted on physical examination. Isokinetic testing is recommended to assess normal strength ratios before the athlete is allowed to return to play[55]. Strength norms and ratios for overhead athletes have been established with isokinetic dynamometry[55,56], with external rotation strength found to be 65% of internal rotation strength in the 90/90 position[55].

The athlete may advance to Phase 3 when he or she has demonstrated minimum range-of-motion deficits, optimum rotator cuff and scapular strength and neuromuscular control, and neither pain nor apprehension on clinical tests meant to provoke those symptoms in the presence of injury. Phase 3 includes intensive strengthening and endurance drills, continued neuromuscular training, the introduction of plyometric training, and an initial interval throwing program (a progression of throwing that varies distances, rest periods, throwing intensities, and throwing on and off the baseball mound)[3]. Plyometric exercises entail a rapid transfer of eccentric to concentric contraction to allow stimulation of muscle spindles, which facilitates recruitment of muscle fibers[57]. Carter et al. compared an eight-week, high-load plyometric training program, referred to as the "Ballistic Six," with a general shoulder conditioning program for National Collegiate Athletic Association (NCAA) Division-I baseball players[58]. The throwing velocity in the group that received plyometric training was significantly increased compared with that in the control group ($p < 0.05$), whereas the isokinetic strength remained similar in the two groups. An interval throwing program may be initiated in this phase of rehabilitation. This program begins with short, flat-ground throwing at variable distances. When a throwing program is initiated, intensive strengthening should be replaced with a less-intensive, high-repetition, low-weight program to avoid overtraining[46].

Phase 4 continues with a strength and neuromuscular maintenance program and an advanced interval throwing program[3,46,56]. During this phase, the athlete is advanced to position-specific throwing provided that he or she remains asymptomatic. The goal is to return to full throwing velocity over the course of three months. Lack of improvement after three months, or an inability to return to competitive play within six months, constitutes failure of nonoperative management and should prompt additional diagnostic testing and, if necessary, consideration of operative intervention.

Common Pathologic Conditions and Options for Their Treatment

Laxity and Instability

In general, laxity is defined as the passive motion of a joint in a particular direction or rotation[55,59]. It may be a normal fundamental property of shoulder soft tissue or an adaptation resulting from the stresses and strains of the throwing motion. Excessive laxity may be physiological or pathologic, and it may predispose a joint to injury. The term *shoulder instability* on the other hand is generally reserved for the sensation of excessive humeral head movement in relation to the glenoid rim, which is usually associated with pain and discomfort. Very few throwing athletes have overt symptoms of instability, as defined above, although the term *instability* has been used in many studies to describe the syndrome that occurs in throwers[26,60,61]. Jobe et al. coined the term *subtle instability* to describe the so-called acquired laxity that occurred in many of the throwing athletes they were treating[62-64]. Neer described the development of laxity in the shoulder of an overhead-throwing athlete as an acquired type of laxity, a pathologic condition distinct from either traumatic or nontraumatic instability[65]. He theorized that this acquired laxity resulted from repetitive injury and microtrauma. While some degree of laxity is essential to compete at a high level in sports involving throwing, experts have speculated that excessive laxity may be responsible for the development of certain pathologic conditions of the shoulder. This has also been called *microinstability,* which is believed to be the result of repetitive shear stresses during the cocking and acceleration phases[66,67]. Kuhn offered the term *pathologic laxity,* which is a more precise description of what is actually happening[59]. Throwers do not typically complain of subluxation or symptoms of instability, but excessive joint translation and rotation are the clear causes of the injuries and associated pain. This pathologic laxity causes pain with the throwing motion, but there is typically no apprehension or feeling of dislocation.

The shear stresses that occur with throwing contribute to the development of pathologic conditions in the posterior-superior region of the glenoid and can result in lesions such as labral fraying and articular-sided rotator cuff tears involving the junction of the supraspinatus and infraspinatus tendons. Recent studies have shown that pathologic conditions of the

shoulder that were once attributed to microinstability are caused by a shift in the center of glenohumeral rotation with concomitant injuries to many of the surrounding structures[38,67]. The prevailing theory is that abnormal shoulder function in a thrower is multifactorial as opposed to being due to simple laxity in the anterior structures of the shoulder.

Noffal proposed that one possible mechanism that could lead to shoulder injury was an imbalance between eccentric and concentric strength of the rotator cuff muscles[41]. The internal rotator cuff muscles act concentrically during the acceleration phase of the throw, and the external rotator cuff muscles act eccentrically during the deceleration phase. Since the external rotator cuff muscles have the dual task of decelerating the arm and, at the same time, maintaining dynamic stabilization of the glenohumeral joint, muscular dysfunction from pain or delayed activation patterns could lead to shoulder injuries.

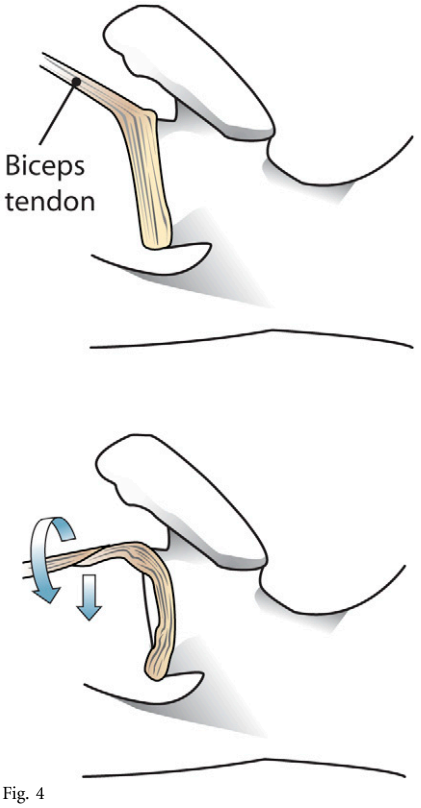

Fig. 4

SLAP lesions caused by a ''peel-back'' mechanism. The drawing on the top shows the biceps tendon and the biceps anchor at the superior aspect of the labrum in a resting position. The drawing on the bottom shows a view from superior with the biceps-labrum complex in an abducted-externally rotated arm position. The posterior rotation of the biceps tendon peels the biceps anchor and the superior aspect of the labrum from the superior part of the glenoid rim.

Superior Labrum Anterior-Posterior (SLAP) Lesions

Labral tears are common in throwing athletes, and these tears, especially those of the superior aspect of the labrum that involve the biceps anchor, can be quite debilitating. Snyder et al. described four types of SLAP lesions[68]. Type I appears as fraying of the superior aspect of the labrum with a stable attachment of the proximal part of the long head of the biceps tendon, the so-called biceps anchor. Type-II lesions show tearing of the biceps anchor in addition to the fraying and are frequently observed in throwers[26]. Type-III lesions appear as a bucket-handle tear, but the remainder of the labrum and the biceps anchor stay attached to the glenoid. Type-IV lesions show a bucket-handle tear of the superior part of the labrum that extends into the biceps tendon. Recent biomechanical studies and arthroscopic observations have suggested that extreme external rotation of the thrower's shoulder may be a cause[67,69,70]. Burkhart and Morgan proposed that SLAP lesions in throwers occur by a "peel-back" mechanism, which is defined as an increased strain at the biceps anchor during the late cocking phase at maximum external rotation (Fig. 4)[70]. Laboratory studies have shown that the long head of the biceps is an important dynamic restraint against external rotation when the arm is abducted[71]. Kuhn et al. showed an increased incidence of SLAP lesions in baseball pitchers, which supports the peel-back theory[71].

SLAP lesions cause vague pain that can be localized to the posterosuperior joint line. The symptoms are exacerbated by throwing and can manifest as locking, snapping, or instability, depending on the extent of the tear. Throwers with a superior labral injury frequently report pain in the late cocking phase and loss of velocity of the pitch. Posterior tightness and a positive active compression test (the O'Brien test) or Speed test are common physical findings with SLAP lesions[72-74]. Radiographic evaluation should include conventional radiographs and magnetic resonance imaging with or without contrast medium to confirm the lesion. Physical therapy may be considered for the initial management of SLAP lesions. When rehabilitation is unsuccessful, operative treatment is indicated.

While there is some difficulty in diagnosing and classifying SLAP lesions[75], the operative treatments are fairly consistent. Type-I SLAP lesions warrant simple débridement. Type-II SLAP lesions should be partially débrided, and the biceps-labral anchor should be secured back to the glenoid[76]. Type-III lesions should be débrided, and acute Type-IV lesions with a tear of the superior aspect of the labrum extending into the biceps tendon should be repaired with suture anchors[77]. Type-IV SLAP lesions with substantial degenerative changes of the biceps tendon and the superior aspect of the labrum may require extensive débridement or biceps tenodesis. Although thermal capsulorrhaphy alone has proven inadequate for the treatment of instability, thermal capsulorrhaphy combined with labral repair has yielded better results than has repair of the labrum alone[78]. This suggests that acquired laxity of the

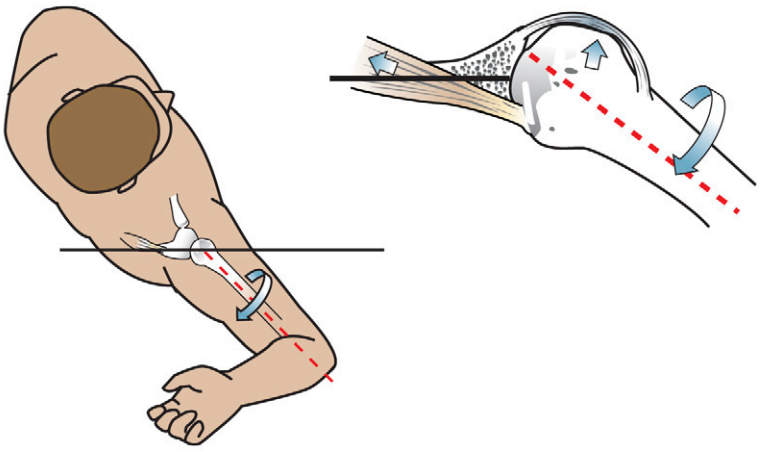

Fig. 5

Internal impingement of the undersurface of the rotator cuff against the posterior aspect of the labrum in maximum external rotation and abduction.

anterior aspect of the capsule may play a pathologic role in this condition.

After operative repair of the biceps-labral complex, the throwing shoulder should be protected and Phase 1 of the rehabilitation program should be initiated. Patients with a stable biceps anchor who were managed with limited débridement should not be treated with immobilization, and rehabilitation may progress more rapidly, although return to elite throwing may take close to one year[79]. It was reported that 87% of forty-four pitchers returned to their preinjury performance levels after operative treatment of a Type-II SLAP lesion[80].

Rotator Cuff Tears

The majority of throwers have articular-sided partial-thickness rotator cuff tears, which can result from acute tensile overload and/or repetitive microtrauma from eccentric failure[8]. Partial-thickness, articular-sided rotator cuff tears in throwers are commonly found posterosuperiorly, at the junction of the infraspinatus and supraspinatus tendon insertions[66,81,82]. Tearing of the superior fibers of the subscapularis tendon may result in subtle destabilization of the biceps tendon in the proximal groove, which may lead to anterior pain and mechanical symptoms such as snapping or locking of the joint.

Physical therapy should be considered for the initial management of throwing athletes who have a rotator cuff tear, and it should focus on tissue-specific stretching and strengthening of functioning rotator cuff muscles. Simple débridement of partial tears is effective in nonathletic patients, but it has shown less consistent results in throwing athletes, with less than half of forty-three athletes returning to their preinjury level of sports activity in one series[83]. Full-thickness rotator cuff tears are rarely seen in throwers, or even in pitchers. Another report, on forty-five athletes, provided evidence that, even when these complete tears were repaired and the procedure relieved pain, only half of all players were able to return to playing at their preinjury level[84].

Impingement

Several types of impingement, including "classic" subacromial impingement, "secondary" impingement, and internal impingement, have been described in the literature[62,63,85-88].

The so-called classic form of impingement, also known as *outlet or external impingement,* results from compression of the rotator cuff between the coracoacromial arch and the humeral head[89,90]. Anatomical variants such as a hooked acromion, arthritis of the acromioclavicular joint with osteophyte formation, and a laterally sloping acromion have been proposed as predisposing factors. Superior migration of the humeral head due to fatigue of the rotator cuff and an improper throwing technique can exacerbate symptoms. Subacromial impingement is typically diagnosed in older throwing athletes with a stable shoulder. These athletes often have loss of internal rotation without the increase in external rotation that is seen in younger throwers. Adaptive osseous changes may also play a role in this loss of internal rotation[87,91]. Patients present with a painful arc of shoulder motion and positive impingement signs and typically respond well to injection tests with analgesic solutions blocking the suspected source of pain.

Rotator cuff strengthening, stretching, and scapular kinesis should be emphasized, whereas arthroscopic subacromial decompression is reserved for patients for whom conservative management has failed. An older thrower will show an irritated and thickened bursa with fraying, matched excoriation, and hypertrophy of the coracoacromial ligament. If a bursal-sided partial or full-thickness rotator cuff tear is present, débridement or repair is recommended. Subacromial decompression alone has not been shown to be effective in enabling throwing athletes to return to their prior levels of activity[92]. It is relatively uncommon for outlet impingement to be the sole source of pain in throwing athletes[93]. This may help to explain why, in early studies, throwing athletes treated with subacromial decompression for apparent impingement rarely returned to their preinjury level of activity[93].

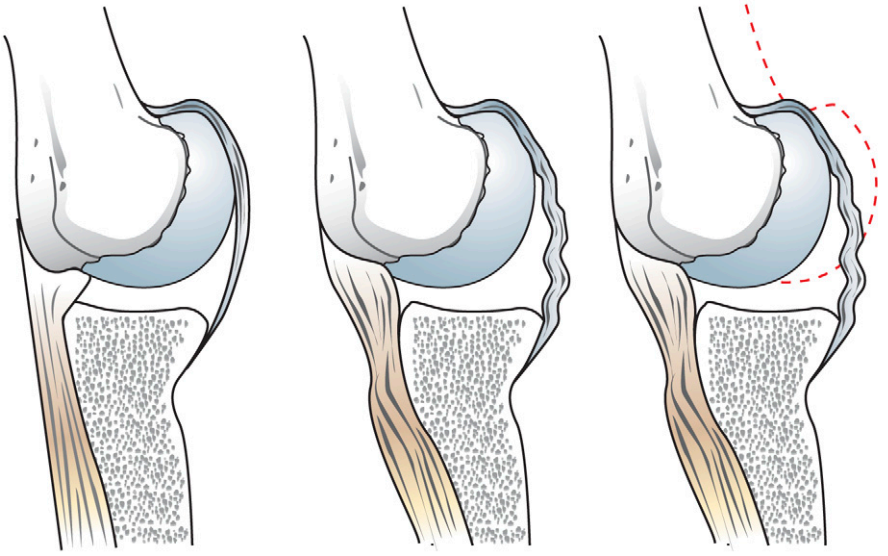

Fig. 6

Left: With the arm in a position of abduction and external rotation, the humeral head and the proximal humeral calcar produce a substantial cam effect of the anteroinferior aspect of the capsule, tensioning the capsule by virtue of the space-occupying effect. *Middle:* With a posterosuperior shift of the glenohumeral contact point, the space-occupying effect of the proximal part of the humerus on the anteroinferior aspect of the capsule is reduced (a reduction of the cam effect). This creates a relative redundancy in the anteroinferior aspect of the capsule that has probably been misinterpreted in the past as microinstability. *Right:* The 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.

Secondary impingement is a dynamic process in which the subacromial arch is normal but the rotator cuff is compressed against the acromion secondary to excessive translation of the humeral head relative to the glenoid socket. This is most commonly observed in athletes with a capsular contracture in whom forward elevation of the arm causes an obligatory anterosuperior translation of the humeral head relative to the glenoid socket[94]. Posterior capsular tightness can create a vector imbalance resulting in anterosuperior migration of the humeral head with secondary rotator cuff symptoms[95]. There is also a strong association between scapulothoracic dyskinesia and symptoms of impingement[85,86]. Weakness of the scapular stabilizers leads to improper scapular rotation during humeral elevation. As a result, the space available for the rotator cuff acutely narrows, causing the symptoms of impingement.

Recommendations for treatment of secondary impingement are based on the underlying pathologic condition. When scapular dyskinesia is the cause of secondary impingement, rehabilitation of the periscapular muscles is usually successful. When rehabilitation is unsuccessful, operative treatment with capsulotomy and débridement is recommended[96]. A strict postoperative rehabilitation program is mandatory to maintain the range of motion that was achieved intraoperatively. Open or arthroscopic repair can be considered for treatment of secondary impingement associated with partial-thickness rotator cuff tears that affect more than one-half of the rotator cuff thickness, but it is rarely indicated. Successful operative treatment reduces pain, but patients are rarely able to return to their preinjury level of athletic performance[84].

Internal impingement is a physiological phenomenon in which the undersurface of the rotator cuff contacts the posterosuperior aspect of the labrum with the arm in maximum external rotation and abduction (Fig. 5)[96,97]. Halbrecht et al. demonstrated this phenomenon in college baseball players and showed that internal impingement can occur even in the absence of symptoms[98]. Symptoms may result from the recurrent microtrauma, which leads to failure of the rotator cuff fibers and destabilization of the biceps-labral complex. Internal impingement presents as a variety of pathologic conditions that may include SLAP lesions, partial-thickness rotator cuff tears, hyperlaxity of the anterior glenohumeral ligaments, and posterior capsular contractures. Myers et al. showed that eleven throwing athletes with internal impingement had a significantly greater glenohumeral internal rotation deficit than a control group without symptoms of impingement ($p = 0.03$)[99].

Several reports have suggested that internal impingement is most likely caused by fatigue of the muscles of the shoulder girdle resulting from a lack of conditioning or from over-throwing[62,66,100,101]. These reports indicate that, during the acceleration phase of throwing, the humerus should be

aligned in the plane of the scapula. As the shoulder girdle muscles become fatigued, the humerus drifts out of the scapular plane. This has been termed *hyperangulation* or *opening up,* which can lead to tensile stressing of the anterior aspect of the capsule[102]. Loss of anterior capsular integrity compromises the obligatory posterior roll-back of the humeral head, leading to anterior translation and therefore causing the undersurface of the rotator cuff to abut the margin of the glenoid and labrum.

Conservative management of internal impingement may begin with Phase 1 of the rehabilitation program. Emphasis should be placed on improving the mechanics of throwing, a core strengthening program, scapular kinesis, and strengthening and stretching of muscles and tendons that appear to be weak or shortened on physical examination. Posterior glenohumeral tightness should be assessed and then addressed with a specific stretching regimen. The goals of operative treatment of internal impingement are to repair the superior aspect of the labrum if it is detached, débride partial-thickness tears of the rotator cuff, and reduce the laxity in the anteroinferior glenohumeral ligament. This approach has led to improved outcomes in several studies[64,103,104].

Glenohumeral Internal Rotation Deficit

The posterior shift in the total arc of motion is considered to be a physiological adaptation of the shoulder joint to throwing. In many cases, a glenohumeral internal rotation deficit is simply a muscular tightness that responds quickly to stretching[8,105]. Burkhart et al. described glenohumeral internal rotation deficit as an alternative mechanism for primary progression of "internal impingement-like" changes in the shoulder[80]. The glenohumeral internal rotation deficit model is based on the high prevalence of posterior capsular contractures and contractures of the posterior band of the inferior glenohumeral ligament in throwers' shoulders[8,50,106]. When a posterior capsular contracture develops, the center of rotation of the humerus, or the contact point of the humerus on the glenoid, is shifted posterosuperiorly[94]. This shift functionally increases the length of the anterior aspect of the capsule, which provides more clearance for the greater tuberosity, diminishing the glenohumeral contact point of the anteroinferior aspect of the capsule with the proximal part of the humerus. This results in excessive external rotation (Fig. 6)[107]. As a result, the biceps anchor is peeled back under tension, causing injury to the posterosuperior structures, especially the posterosuperior aspect of the labrum. The so-called peel-back progression mechanism permits further laxity of the anterior aspect of the capsule. The pathologic cycle culminates in torsional failure of the rotator cuff—not compressive failure, as occurs in the internal impingement model. The end results of this cascade of events are the articular-sided partial-thickness rotator cuff tears and SLAP lesions that are typically seen in the throwing shoulder.

With the glenohumeral internal rotation deficit model, one attempts to identify throwers at risk for shoulder injury by quantifying the internal rotation deficit. Individuals are considered to have a clinically relevant glenohumeral internal rotation deficit when there is a loss of ≥25° of internal rotation of the throwing shoulder as compared with the non-throwing side. Such deficits are commonly found in overhead throwers, with some studies having demonstrated average deficits of up to 50°, when compared with measurements on the contralateral side, as well as concomitant increases in external rotation on the order of 30°[80]. Shoulders with a total arc of motion of <180° and an internal rotation deficit of >25° seem to be at risk for the development of SLAP lesions as a result of the increased posterosuperior peel-back forces[80].

Verna demonstrated the association of glenohumeral internal rotation deficits with the development of shoulder dysfunction[108]. He followed thirty-nine professional pitchers over a single season and found that shoulder problems developed in more than half of the players with a glenohumeral internal rotation deficit of >35°. In a similar study, Kibler prospectively followed two groups of high-level tennis players for two years[109]. One group performed daily posteroinferior capsular stretching to minimize the glenohumeral internal rotation deficit, while the control group continued their routine exercise program. The study demonstrated a 38% decrease in the occurrence of shoulder problems in the group that performed the stretching as compared with the controls.

The majority of athletes respond to physical therapy programs that focus on stretching of the tight posterior aspect of the capsule. Burkhart et al. reported that the majority of throwers respond to these programs, with concomitant decreases in shoulder-related problems[80]. The few who do not respond are typically older elite players who are unresponsive to conservative treatment. These older players can be treated with selective arthroscopic posteroinferior capsulotomy in the zone of the posterior band of the inferior glenohumeral ligament[10].

Dyskinesia

Adaptive and pathologic changes have been noted in the scapulae of throwers. Clinical studies have documented alterations in dynamic scapular positioning and asynchrony of the scapula in patients with impingement or anterior instability[85]. Drooping of the shoulder, so-called ptosis, and scapular dyskinesia are becoming more commonly recognized, but their biomechanical causes and consequences are still largely unknown. The scapula can also impinge on the thorax during late cocking, creating a form of scapulothoracic impingement that is unique to throwing athletes[94,110,111]. Changes in scapular position, both dynamic and static, play critical roles in pathologic processes in the throwing shoulder, and yet the contribution of scapulothoracic motion to throwing is currently one of the least studied and understood entities in the throwing athlete. Scapular dyskinesia results from imbalances of the periscapular muscles secondary to fatigue, direct trauma, or nerve injury. It can negatively impact shoulder function in several ways. For example, in order for throwing athletes to reach the extremes of motion, the scapula must rotate counterclockwise (in the sagittal plane of the right arm)

so that the acromion elevates to prevent impingement. The scapula must also retract appropriately to keep the glenoid vault centered under the humerus, maintaining stability. If the scapula fails to retract appropriately, there is hyperangulation of the humerus relative to the glenoid and excessive stress is placed on the anterior aspect of the capsule. Normal function of the serratus anterior, trapezius, and rhomboid muscles is required to achieve the necessary scapular positioning. Loss of function due to nerve injury, weakness, and/or fatigue leads to glenohumeral 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[85,112].

Because the scapula is part of the kinetic chain, transferring energy derived from trunk rotation to the pitching arm, destabilization of the scapula results in inefficient throwing mechanics that lead to decreased ball velocity. Frequently, in an attempt to compensate for this loss of ball velocity, the pitcher subconsciously increases the effort of the shoulder muscles, which increases strain on the shoulder[22].

For these reasons, rehabilitation programs for throwing athletes must have a strong emphasis on strengthening and conditioning of the scapular stabilizers. The vast majority of scapula-related issues can be resolved by a physical therapy program. Sometimes, however, surgical intervention is required for entities such as scapular bursitis or a snapping scapula, which can be treated by excision of the offending tissues at the inferior margin of the scapula[110,111,113]. These procedures are consistently reported to have excellent results, with the athletes returning to sports at their previous level.

Overview

The performance of throwing athletes is frequently limited by shoulder injuries. These problems are very complex and therefore difficult to address. While overlapping of symptoms is common, there are typical patterns of injuries observed in throwers' shoulders. The problems occur as a result of a combination of muscle fatigue imbalances, anterior capsular laxity, posterior capsular contractures, abnormal mechanics, scapular dyskinesia, increased humeral retroversion, and repetitive microtrauma from compressive, tensile, and torsional forces. As a result, lesions that involve the posterosuperior aspect of the labrum, the articular side of the posterior part of the rotator cuff and the superior part of the subscapularis tendon, the biceps-labral complex, and the chondral surfaces commonly develop in throwers.

The mechanisms and etiologies of throwing injuries are becoming better defined. While there is some controversy about the initiating event—i.e., whether it is anterior capsular laxity or posterior capsular tightness—the typical injury patterns remain the same. Fortunately for the practitioner, the evaluation and treatment algorithms are also very similar. Before one begins thinking about treatment options, it is necessary to obtain a detailed history, a physical examination, and additional studies to arrive at a precise diagnosis. The treatment of shoulder injuries should start with a conservative protocol that initially focuses on restoring a full range of motion as a foundation for a smooth kinetic chain. Strengthening and specific stretching to address imbalances, proprioceptive and neuromuscular conditioning to provide optimum scapular and glenohumeral stability to enable the shoulder to endure the demands of throwing, and a program of core strengthening to allow optimum transfer of forces to the shoulder follow. Finally, shoulder conditioning and respect for the recovery period required between games are imperative for throwing athletes. It is the responsibility of coaches, trainers, and physicians to educate and provide guidance to prevent or minimize the potential for shoulder injuries. ■

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