Instructional Course 206

Comprehensive Evaluation and Treatment of the Shoulder in the Throwing Athlete

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ROTATOR CUFF INJURIES IN THE THROWING ATHLETE

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That rotator cuff injuries occur on a frequent basis in the throwing athlete is a well-known phenomenon. The challenge for the clinician is to make an accurate diagnosis and to be aware of the various treatment algorithms that govern expedited care. There are many ways to organize a discussion of rotator cuff injuries, but the most compelling one is to use causation as the primary determinant:

1. "Classic" subacromial impingement
2. Internal impingement
3. Coracoid impingement
4. "Secondary" impingement
5. Primary tensile failure

Each of these entities will be discussed in terms of causation followed by a brief treatment algorithm.

"Classic" Subacromial Impingement

Subacromial impingement is typically diagnosed in the older throwing athlete who has a stable shoulder. Often these overhand athletes will have a loss of internal rotation that may be refractory to stretching. Some have postulated bony adaptive changes in the thrower leading to internal rotation loss.[1,2] These patients have a painful arc, positive impingement maneuvers, and respond affirmatively to a subacromial injection. Radiographs usually show some form of an acquired or congenitally prominent anterior acromion that predisposes to outlet stenosis. Some may also exhibit lateral downsloping of the acromion. Many of these patients will improve with anti-inflammatory medication combined with a well-supervised physical therapy program focusing not only on cuff rehabilitation, but also on scapular dynamics. There is no conclusive data supporting acromioclavicular (AC) joint spurring as a cause of subacromial impingement.

Treatment

Treatment for this disorder begins with a conservative program as noted above, and for those who fail these measures, a subacromial decompression is the next appropriate intervention.[3-5] At the time of surgery, the findings usually consist of bursal fraying, matching excoriation of the coracoacromial (CA) ligament, a thickened bursa, and occasionally a hypertrophic CA ligament. Excision of the distal clavicle is a common associated procedure if preoperative symptoms implicate the AC joint. If a significant bursal-sided partial-thickness tear is present, consideration of a rotator cuff repair, either arthroscopically or through a mini-open approach is recommended. It is imperative that patients be forewarned that returning to the same premorbid level of competition is unlikely in most instances.[3]

Internal Impingement

Internal impingement can be a very confusing topic. By definition, internal impingement represents contact between the undersurface of the rotator cuff and the posterior-superior glenoid and labrum. It may be help-

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ful to consider the phenomenon of internal impingement as being analogous to a "rash," as espoused by Michael Gross (personal communication). Just as a rash can be the result of any number of causes such as a primary contact dermatitis, or an allergic reaction or perhaps as part of a systemic disease entity, several different conditions can lead to internal impingement which simply represents the final common denominator.

Walch et al.[6] have described internal impingement as a physiologic phenomenon in which contact between the posterior-superior glenoid and labrum with the undersurface of the rotator cuff is a normal finding. Halbrecht et al.[7] studied this contact in the asymptomatic throwing athlete using magnetic resonance imaging (MRI) and confirmed this common occurrence absent symptoms in a population of college baseball players. In the at-risk population, namely the throwing or overhand athlete, this physiologic phenomenon can, but not always, progress to pathology on the basis of recurrent microtrauma leading to cuff failure.

Anterior capsular insufficiency proposed by Jobe and by others[8-11] commonly results from attenuation of the anterior stabilizers due to relative hyperangulation when pitching. Poor throwing mechanics allow the arm to become co-linear with the body axis instead of remaining co-linear with the scapula. Loss of capsular integrity compromises the obligate posterior rollback leading to anterior translation and internal impingement forces.

In the Morgan-Burkhart model,[12-14] ubiquitous posterior capsular contractures combined with the possibility of acquired humeral retroversion cause humeral head rotation to shift in a posterior-superior position. This in turn leads to a "peel-back" of the posterior-superior structures, most notably the posterior-superior labrum. Although direct impact of the humeral head on the labrum can occur, the degree of angulation at wind-up and acceleration causes the type II SLAP to occur in a "peel-back" mechanism as opposed to the previously theorized deceleration-avulsion type mechanism. A "pseudolaxity" ensues due to obligate anterior-inferior instability (labral "ring" is compromised), and the internal impingement forces are magnified.

While recent studies[1,2] have clarified the contribution of acquired humeral retroversion to gains in external rotation with obligate losses in internal rotation, Riand et al.[15] noted that loss of humeral retroversion of up to 20° or 30° increased the risk of contact between the greater tuberosity and the posterior-superior glenoid and labrum. Restoration by means of a humeral osteotomy was the only successful intervention.

Kibler[16] has helped us appreciate the role of scapular dynamics in preventing shoulder injuries in the throwing athlete. Scapular dyskinesia can create a "relative" anteverted glenoid during the throwing motion, eventually leading to anterior capsular compromise. A careful and thoughtful assessment of the scapula is critical in the evaluation of the throwing athlete as either weakness or asynchronous motion contributes to loss of a stable platform for the shoulder in addition to jeopardizing the efficient transfer of power from the trunk and legs to the throwing shoulder.

Establishing the diagnosis of internal impingement can be a daunting task. Posterior shoulder pain in the young thrower should be a helpful clue. A positive relocation test in which shoulder pain is diminished with an anterior-to-posterior force applied with the arm maximally abducted and externally rotated is a common finding during the physical examination. Rather than testing instability, the anterior-to-posterior force may simply represent an "unlocking" of internally impinged tissue. Diagnostic testing in the form of plain radiographs, computed tomography (CT), or MRI continues to lack in both sensitivity and specificity. Using an MRI scan to make the diagnosis should be avoided as this contact is commonly witnessed in the asymptomatic thrower and as Walch and others have proposed, may simply represent a physiologic phenomenon.

Dynamic-assessment arthroscopy continues to be the gold standard in establishing the diagnosis of internal impingement. Viewing from a posterior portal with the shoulder in the ABER (abducted-externally rotated) position combined with extension, contact between the undersurface of the cuff and the posterior-superior glenoid and labrum is easily identified. Often, abnormalities of the posterior-superior labrum are present in the form of a "depression" or frayed labral tissue corresponding to the area of contact. In my experience, if the contact is easily visualized, one must entertain the possibility of capsular redundancy as a potential causative agent. Usually in the ABER position, capsular volume for arthroscopic visualization is diminished. In addition to assessing for internal impingement, arthroscopy allows for further diagnostic clarity to rule out labral, capsular or other contributing pathology.

The difficulty in establishing the diagnosis lies in the overlapping symptoms that can coexist. Although the primary problem might be a posterior-superior

type II SLAP with anterior "pseudolaxity" leading to an internal impingement injury, late secondary subacromial changes can also occur due to cuff fatigue and loss of humeral head containment, thereby making the physical examination difficult to assess and making the operative findings sometimes difficult to piece together. Despite the various etiologies, initial treatment should consist of a well-supervised conservative program emphasizing rotator cuff and scapular exercises, medication, appropriate activity modification and sport-specific technique adjustments.

Treatment

In those individuals unresponsive to a conservative program, surgery may be appropriate. In the "physiologic-to-pathologic" category, a limited debridement followed by another course of rehabilitation is an appropriate step. Although Sonnery-Cottet et al.[17] described residual pain in nearly 90% of his study group, nearly 80% of this elite class of tennis players were able to return to their sport. If anterior capsular laxity is present, either an open or arthroscopic stabilization is warranted.[18] For the posterior-superior type II SLAP injury, primary repair of the SLAP lesion is warranted. In most cases, the anterior "pseudolaxity" resolves with repair of the labral pathology.[12] For those individuals with persisting instability, treatment of the anterior-inferior capsular should be undertaken in the form of capsular plication or with the adjunctive use of thermal energy which has been recommended by some.[19] As noted earlier, humeral rotational osteotomy is the preferred treatment for loss of humeral retroversion while scapular dyskinesia is addressed through a supervised rehabilitation program.

Coracoid Impingement

By definition, coracoid impingement occurs when the subscapularis tendon is impinged between the lesser tuberosity and the coracoid tip. Possible causes have included trauma, postoperative (e.g., Bristow procedure) changes, anterior instability as well as an idiopathic cause.[20,21]

Patients with coracoid impingement present with anterior shoulder pain, and it can mimic or occur in combination with subacromial impingement findings. The most useful diagnostic maneuver is to elicit pain as the shoulder is passively forward flexed, adducted and internally rotated. This differs from the O'Brien test where active resistance is required in this position. A diagnostic and therapeutic injection into the subcoracoid space can be effective treatment as well as confirming the diagnosis. The coracoid index, a measurement of coracoid projection lateral to the tangential plane of the glenoid on an axial image, as well as the coracohumeral distance, the distance between the coracoid and the lesser tuberosity in maximal internal rotation (average 11 mm in normal *v* 5.5 mm in symptomatic) are 2 measurements which, although not pathognomonic, can help confirm the possibility of a coracoid impingement syndrome.

Treatment

If conservative measures are unsuccessful, a coracoidplasty is the next appropriate step. Using an open approach, the conjoint tendon is released, the tip of the coracoid debrided followed by reattachment of the conjoint tendon. An arthroscopic technique for coracoidplasty has been described,[22] and relies on an anterolateral portal and visualization of the CA ligament. The ligament is traced to its coracoid attachment and the tip debrided with care taken to avoid detachment or plexus injury.

"Secondary" Impingement

Unlike internal impingement, despite similar clinical conditions, the rotator cuff can fail on a tensile stress basis as opposed to direct mechanical fraying. Repetitive, high-energy eccentric loads can lead to cuff failure commonly encountered in circumstances often found in the throwing shoulder: anterior capsular insufficiency, multidirectional instability, labral pathology including SLAP, SLAC (superior labrum anterior capsule), and Bankart lesions, posterior capsular contractures resulting from trauma, postsurgical or functional causes, as well as scapular dysfunction or primary tendon degeneration with loss of humeral head depression. Suffice it to say that an articular-sided partial-thickness rotator cuff tear with an element of anterior capsular attenuation does not always result in internal impingement.

Physical findings are often nonspecific and difficult to interpret; however, ligamentous laxity with the potential for becoming pathologic is not uncommon. A positive sulcus sign, thumb-to-forearm ability, and a positive load and shift test especially in an inferior direction are risk factors. Posterior capsular tightness, and not always increased retroversion, can create a vector imbalance resulting in posterior-superior migration of the humeral head and secondary rotator cuff symptoms.[23]

Treatment

Much like the internal impingement lesion, the primary pathology must be identified and treated. If the partial-thickness tear approximates or exceeds 50%, the recommended course of treatment includes a formal rotator cuff repair, mini-open or arthroscopic. Payne et al.[24] noted that in patients with a partial rotator cuff tear, a normal subacromial space, increased translation, and an insidious onset, an acromioplasty was highly ineffective. Pain relief could be obtained with a decompression in those patients with a traumatic partial rotator cuff tear and subacromial changes although a return to sports was unlikely.

Primary Tensile Failure

Primary tensile failure occurs on a repetitive stress and eccentric load model in which the articular-sided fibers fail due to a poorer blood supply and a configuration less robust in resisting tensile forces.[25-29] Nakagawa et al.[30] studied this injury in 40 baseball players and determined that there was no causal relationship to motion loss, labral pathology, joint laxity, or symptomatic instability. They postulated a primary eccentric loading failure at the supraspinatus-infraspinatus interface, perhaps related to a stress-riser phenomenon.

Nakagawa has described the greater tuberosity notch sign that occurs at the superior and lateral border of the "bare spot" and consists of bone erosion most likely the result of chronic inflammation and granulation tissue. This finding may be misread as a remote Hill-Sachs lesion, and can be identified on plain radiographs, CT, or MRI studies. Furthermore, Nakagawa postulated that not only could the greater tuberosity notch sign reflect a partial-thickness articular-sided rotator cuff tear, the size of the notch sign also correlated with the size of the partial cuff tear.

Summary

The thrower's paradox consists of the need for extreme degrees of external rotation while maintaining capsular integrity. The mechanics of throwing are complex with significant forces stressing the shoulder joint on a repetitive basis. That injuries occur is not unexpected, and for the majority of rotator cuff injuries, a conservative, well-supervised rehabilitation program is usually successful.

The patient's history can be very revealing. Posterior shoulder pain in the young thrower should raise concerns of internal impingement. Subacromial decompressions in this population of throwers yield poor results. Although bony adaptive changes can account for some internal rotation loss compared with the opposite extremity, posterior capsular tightness is still a common finding in the younger throwing athlete and, if addressed with appropriate rehabilitation, responds well to a nonoperative program. Laxity, a physical finding, should not be confused with instability. Although one may predispose to the other, making the diagnosis of actual capsular insufficiency can be difficult because of its potential subtle nature. Scapular mechanics are often overlooked or missed and play a critical role in the transfer of power from legs and trunk to the throwing shoulder. Poor scapular function either resulting from neurologic compromise, or early fatigue and muscle imbalance, can and should be addressed early.

Diagnostic testing can be helpful, but can also be misleading because dynamic problems are often missed with static studies. Plain radiographs might show posterior osteophytes or bony changes suggestive of internal impingement lesions. Assessing the distance between coracoid and lesser tuberosity can help in establishing the diagnosis of coracoid impingement. MRI testing can often reveal contact between the undersurface of the cuff and the posterior-superior glenoid and labrum with the shoulder positioned in the ABER position. This finding is not diagnostic of internal impingement and is often found in the asymptomatic throwing shoulder. The addition of a contrast agent in the MRI study is highly recommended in the throwing athlete as the sensitivity and specificity for labral lesions and articular-sided rotator cuff tears is enhanced. CT scanning can assist in evaluating glenoid or humeral head retroversion, or lack thereof, as well as confirming any bony erosions associated with instability. The greater tuberosity notch sign should be recognized and its association with incomplete, significant partial rotator cuff tears recalled.

The challenge for the clinician is to be aware of the multitude of possible causes for rotator cuff pathology in the throwing athlete, to perform a thorough history and physical examination, to obtain appropriate adjunctive tests, and then to establish a working diagnosis. Overlapping symptoms can make this task a most difficult one. Should conservatives measures, which constitute the initial treatment, fail, dynamic-assessment arthroscopy should be undertaken with the specific goals of conclusively establishing the diagnosis and rendering definitive treatment of not only secondary pathology, but also any primary pathology, subtle or otherwise. If the clinician is armed with an over-

view of potential causes for rotator cuff disease in the throwing athlete, preoperative discussions of the treatment algorithm help avoid second surgeries, and permit the patient to have an active role in decision making as well.

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COMPREHENSIVE EVALUATION AND TREATMENT OF THE SHOULDER IN THE THROWING ATHLETE: BIOMECHANICS, PATHOMECHANICS, CLINICAL EVALUATION, AND DIAGNOSTIC TESTING

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In our evolutionary past, our hominid ancestors left the trees with a shoulder that was likely adapted to tree living and brachiation. With upright posture the upper extremity was freed from its ambulatory role and became an instrument for various uses. One of these uses, which was likely very important in the success of early hominids was throwing.[1-3] It is easy to believe that natural selection would favor the adaptation of throwing, as this skill would greatly assist in hunting and defense. Hominids who could throw accurately would be able to capture more food, and would be able to defend themselves and their offspring from predation. As such, it is reasonable to expect that the constellation of genes that promote the throwing skill would be more likely to be passed on to future generations.

Accuracy and speed when launching projectiles requires elaborate neural processing, which some believe may have led to the development of handedness and hominid language.[4-7] It can be argued that the

history of ballistics, from the sling to the bow, and with the advent of gunpowder, from cannons to intercontinental ballistic missiles, is a technological extension of our ability to throw.[8] It is clear that the throwing skill is an essential human trait and is worthy of study.

Historically, pathology in the thrower's shoulder has been poorly understood. The first treatise on injury in baseball players is A.H.P. Leuf's text "Hygiene for Base Ball Players" published in 1888.[9] Leuf was an amateur baseball player and served as the team physician for the National League Philadelphians, who later became the Phillies.[10] He divided the pathology seen in his players into that which affected the muscles, the ligaments, and the bones, and his assessment was clearly oriented toward the affected anatomy. His text recognized problems in the shoulders to be of paramount importance and recommended hot water, galvanism, and exercise for treatment, modalities surprisingly similar to what we currently use (now called hydrotherapy, electrical stimulation, and rehabilitation) (Fig 1).

The thrower's shoulder received little attention for the next 60 years until the 1950s when shoulder pain in throwers was generally divided into anterior pain or posterior pain, and given presumptive anatomy-based diagnoses, such as biceps tendonitis.[11-16] In the 1970s, C.S. Neer II revolutionized the approach to painful shoulders when he described the impingement syndrome and popularized anterior acromioplasty for its treatment.[17,18] Soon after, it was thought that shoulder pain in throwing athletes was caused by the impingement syndrome and acromioplasty and/or resection of the CA ligament became popular (although with limited success) methods of treatment.[19,20]

Neer is also credited with describing the concept of acquired laxity,[21] and recognized that athletes may have developed laxity through repetitive loading of the glenohumeral ligaments. Neer[21] and others[22] speculated that this acquired laxity may result from "microtrauma," although there is little evidence to support an injury and failed repair mechanism for the production of this laxity. Despite this, the concept that laxity may be the source of most shoulder pathology was not appreciated until Jobe[22-24] popularized the concept of "subtle instability and secondary impingement" in the 1980s. The essence of this concept is that rotator cuff pain resulted from overuse, as the cuff had to stabilize a glenohumeral joint with excessive laxity. Jobe performed instability operations on throwers with shoulder pain, and found improved success in returning the athletes to play.[25] More recently, newer concepts regarding the pathology in the thrower's shoulder, such as internal impingement and labral tears, have received more attention, in part due to an improved understanding of the kinematics of the throwing motion.

Biomechanics of Throwing

The throwing motion is a rapid sequence of events designed to send a projectile toward a target with speed and accuracy. Baseball pitching is clearly the most studied form of throwing. The pitching motion has been divided into different phases for analysis.

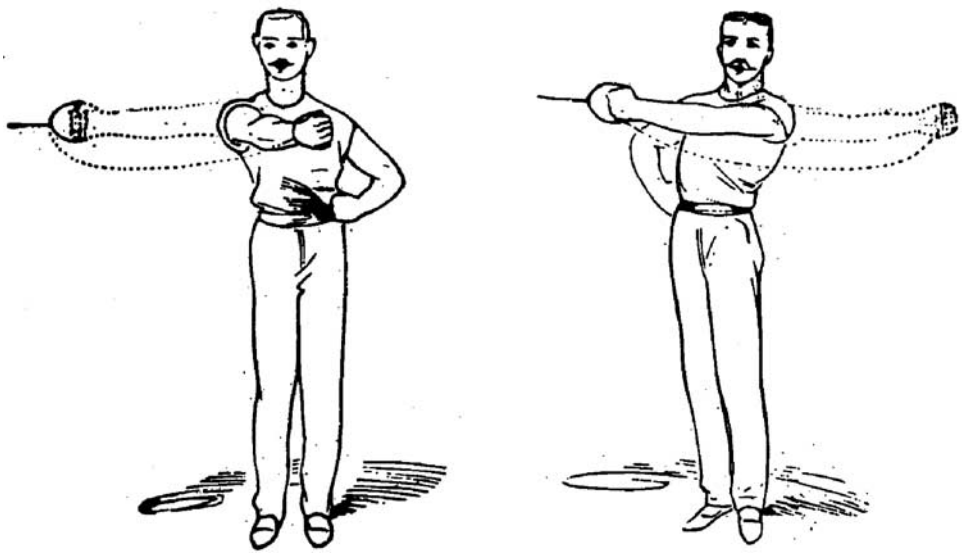

FIGURE 1. Two of many exercises prescribed for the treatment of shoulder injury in 1888. The role of rehabilitation for the treatment of baseball players' shoulder injuries was recognized over 100 years ago. The exercise regimen is surprisingly similar to that used today. (Reprinted from Leuf AHP. Hygiene for Base Ball Players.[9])

These phases include (1) windup, (2) early cocking, (3) late cocking, (4) early acceleration, (5) acceleration, (6) deceleration, and (7) the follow through (Fig 2).

Fleisig et al.[26] analyzed electromyographic muscle activity and throwing kinematics, and estimated the forces about the shoulder during the various phases of the throwing motion. They identified 2 critical moments during which the forces about the shoulder are the greatest, late cocking and early deceleration.

In the late cocking phase of throwing, the arm is found to be in 94° ± 21° of abduction, 11° ± 11° of horizontal adduction, and a remarkable 165° ± 11° of external rotation (Fig 3). The estimated forces about the joint include 250 ± 80 N of superior shear force, 310 ± 100 N of anterior force, and 480 ± 130 N of joint compression force.[26] The late cocking phase of throwing has been implicated in the development of type II SLAP lesion,[27-30] laxity in external rotation,[31] remodeling of the humerus,[32-35] and internal impingement.[36-40]

The other critical instant is the early deceleration phase of throwing[26] (Fig 4), in which the arm is positioned in 93° ± 10° of abduction, 6° ± 8° of horizontal adduction, and 64° ± 35° of external rotation. The forces about the shoulder, related to substantial rotator cuff muscle activity, include 1,090 ± 110 N joint compressive force to prevent glenohumeral joint distraction[41] and 100 ± 130 N inferior force.[26] This phase of throwing may be important in the development of type II SLAP lesions,[26,42] other labral pathologies,[26,43] and partial-thickness rotator cuff tears.[26,44]

FIGURE 3. Late cocking phase of throwing. Recognized as a critical instant for throwing by Fleisig,[26] the arm is abducted to 94° and reaches 165° of external rotation. The humeral head is subject to extremely high superior and anterior shear forces, as well as high rotational torque about the humeral shaft.

Pathomechanics of Throwing

How does the pathology in the shoulder of throwing athletes develop? It is important to realize that throwing is a rapid ballistic kinetic chain, and that to throw efficiently requires optimization of a coordinated effort from the toes to the fingertips. Conditions that affect the components lower in the chain may produce changes in distal links, and could conceivably lead to the development of pathology.[45,46]

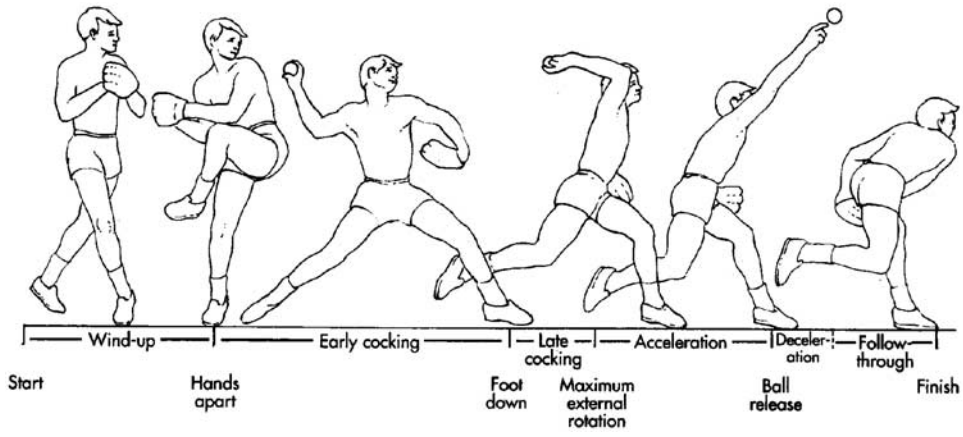

FIGURE 2. Phases of the throwing motion. (Reprinted with permission.[86])

Figure 4. Early deceleration phase of throwing. The second critical instant for throwing is characterized by 93° of arm abduction, 64° of external rotation, and extremely high joint compression forces, due to rotator cuff and other muscle activity.

Pathologic Laxity: Neer is credited as the first to recognize the laxity that develops in the athlete.[21] He described this form of laxity as "acquired," and thought it to be distinct from (although frequently seen with) traumatic or atraumatic instability. Neer postulated that acquired laxity developed from repetitive injury, and suggested that microtrauma may be important in its development. This concept has gained widespread acceptance[22,47] despite a lack of evidence to distinguish an injury with failed repair mechanism from a remodeling mechanism.

Glenohumeral joint laxity is probably the most studied and least understood component of pathology in the thrower's shoulder. The literature is clouded by confusion regarding the terms of laxity and instability. While these terms may be related, it is important to distinguish between them. Laxity does not equal instability. Laxity is excessive motion for a particular direction or rotation for a particular joint. It may be a normal adaptation for a given sport. Many authors reserve the term instability for a sensation that the humeral head is translating in the glenoid.[48] (As such, Jobe's nomenclature of "subtle instability" may have led to some confusion; a better term might have been "pathologic laxity.")

In general, most upper extremity athletic endeavors require repetitive motion of the shoulder at the limits of motion under high loads. It is important to realize that, because different sports load the shoulder differently, a *sport-specific laxity pattern* may develop. For example, in golfers, the swing of the club results in high tensile stress to the posterior capsule, which then could lead to posterior shoulder instability. Swimmers, who load the glenohumeral joint in a variety of patterns, develop a multidirectional laxity pattern. In throwers, the forces experienced by the shoulder are multiple, but a great deal of evidence suggests that external rotation of the humerus is extremely important. For example, it has been shown that the speed of the pitched ball correlates best with the amount of external rotation of the abducted arm.[49,50] As such, it can be inferred that the early phases of the throwing motion are trying to optimize external rotation of the abducted arm. Multiple studies have shown that throwing can lead to increases in humeral retroversion[32-35] and spiral humerus fractures.[51,52] It is likely that the inferior glenohumeral ligament, which is the primary restraint to external rotation of the humerus in the throwing position,[31] may also see high stress and could develop laxity in external rotation.

In fact, examinations of asymptomatic throwers have demonstrated that throwing athletes have increased external rotation and decreased internal rotation of the abducted arm.[33-35,47,53,54] It is thought by some that these changes result from laxity in the inferior glenohumeral ligament and contractures of the posterior capsule.[31,45,47,55,56] Pitchers, interestingly, have been found to have an increased sulcus sign,[47] which may be related to laxity in the coracohumeral ligament, another restraint to external rotation of the abducted arm.[31,57,58] This altered range of motion is certainly related to the changes in humeral torsion that develop with throwing.[32-35] While some have suggested that humeral torsion predominates over laxity for the physical findings in throwers,[34,35] others believe that laxity of the inferior glenohumeral ligament is an important part of the pathology[33,47,53] and recommend capsule plication as a part of the surgical treatment of throwing athletes.[25,59,60] Furthermore, some have suggested that the contractures of the posterior capsular are the cause of pathology in the thrower's shoulder[55,56] and have recommended release of the posterior capsule in some throwers (C.D. Morgan, personal communication). Clearly this remains an area in need of more study.

While some laxity may be essential to compete in high-level sports, excessive laxity may be responsible for other pathologies in the shoulder. This athlete may have pain and have damage to the labrum and/or

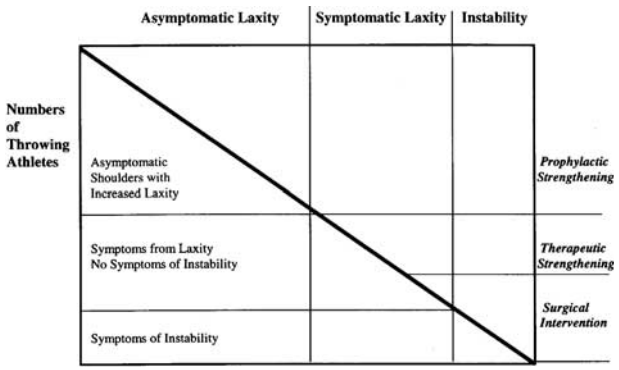

FIGURE 5. Laxity spectrum in the thrower's shoulder. Most throwers will have asymptomatic but detectable laxity in external rotation. A smaller number of throwers will develop excessive laxity that could lead to a variety of pathologies in the shoulder, most of which are treated with therapeutic strengthening and rehabilitation. A subset of this group of throwers with pathologic laxity will have symptoms refractory to rehabilitation, and will require surgery. Finally, a small population of throwers will have excessive laxity to the point of developing symptoms of instability, and will generally require surgical treatment.

rotator cuff, which are related to the excessive laxity in the glenohumeral ligaments, but this athlete will not have *symptoms* of instability. This pathologic laxity is the "subtle instability" described by Jobe.[22-24] At a higher degree of damage, the excessive laxity may be enough to produce symptom*s* of instability. In fact, one can think of laxity falling along a spectrum in the thrower's shoulder (Fig 5).

Superior Labral Lesions: Although many superior labral lesions have been identified in the thrower's shoulder, the most common is the type II SLAP lesion in which the labrum and biceps anchor are avulsed from the superior glenoid.[61] Andrews suggested that traction on the biceps was likely responsible for the development of these lesions during the deceleration phase of throwing,[26,42] but recent biomechanical studies and observations made during arthroscopy suggest that the extreme external rotation seen in the thrower's shoulder may be the position in which type II SLAP lesions are more likely to occur.[28-30] 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.[27] Further support that external rotation contributes to the generation of type II SLAP lesions comes from studies that show improved outcomes of type II SLAP lesion repairs are improved when thermal or suture plication of the inferior glenohumeral ligament is performed.[59,60]

Partial-Thickness Rotator Cuff Tears: Articular-surface partial-thickness rotator cuff tears have been recognized as a common pathology in the thrower's shoulder since the advent of arthroscopy.[44] These tears are frequently seen in conjunction with degenerative tears of the posterosuperior labrum, and interestingly, when the arm is brought into the position of abduction and maximal external rotation, these 2 pathologic areas come into contact.[36-40] This contact, which likely occurs during throwing (and may occur in normal individuals), has been called internal impingement,[37-39] and it is generally thought that the rotator cuff is compressed between the greater tuberosity and the posterosuperior glenoid and labrum.[36] The articular surface of the rotator cuff is thought to fail under compressive load.[36]

How these structures come into contact and develop pathology is a matter of debate, particularly when these structures could make contact in normal, healthy shoulders.[36] Nevertheless, some authors suggest that the anterior capsule becomes lax, which allows the humerus to move into increased horizontal adduction.[37,38] Others suggest that excessive external rotation may be the cause.[39,40] A third potential cause is a loss of obligate translations. Harryman and others have shown that when the normal arm is abducted and externally rotated, the humeral head moves posteriorly in the glenoid as the anteroinferior capsule is tightened.[62,63] This phenomenon was named "obligate translation," and may be lost in patients with traumatic anterior instability,[64] and could conceivably be lost in throwers with pathologic laxity. When the obligate translation is lost, the humeral head remains perched anteriorly in the glenoid facilitating the contact between the greater tuberosity and the posterosuperior glenoid (Fig 6). Although each explanation is possible, it is likely that all have a role in internal impingement, as laxity in the inferior glenohumeral ligament could produce each of these mechanisms. It is interesting to note that reducing the laxity in the inferior glenohumeral ligament seems to significantly improve the outcome in throwers with internal impingement.[59,60]

Alternatively, this articular surface of the rotator cuff may fail under tensile, not compressive, loading. It has been recognized that the supraspinatus and infraspinatus generate extremely high force as they eccentrically contract to decelerate the thrower's arm, preventing shoulder distraction.[26,41] This eccentric loading during the deceleration phase of throwing has been proposed as a mechanism for the development of partial-thickness rotator cuff tears.[44] The tendon of the supraspinatus is a complicated interwoven structure and, while the bursal surface appears more tendinous

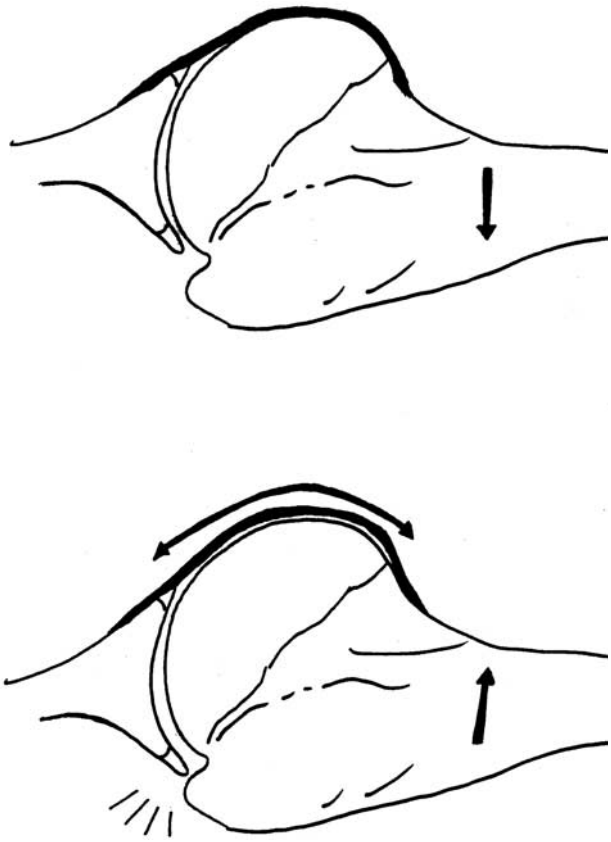

Figure 6. Obligate translation and internal impingement. External rotation of the abducted arm should result in tightening of the inferior glenohumeral ligament, which would normally direct the humeral head posterior in the glenoid. If the inferior glenohumeral ligament is lax, the humeral head will stay anterior in the glenoid allowing for contact between the posterior superior glenoid and the greater tuberosity, producing internal impingement.

in appearance and in its biomechanical properties, the articular surface is structurally more like the glenohumeral joint capsule[65] and biomechanically may be more likely to fail under tensile loading.[66] It is interesting to note that an extension of collagen fibers from the coracohumeral ligament reinforce the region of the rotator cuff where articular surface partial-thickness rotator cuff tears occur.[65] The coracohumeral ligament is another important restraint to external rotation of the abducted shoulder.[31] It is conceivable then that these partial-thickness rotator cuff tears may represent tensile failure of the superior part of the capsule as a result of excessive laxity in external rotation during the late cocking phase of throwing. At this time, the mechanism behind the development of these tears remains unknown.

Tight Posterior Capsule: Another finding in the thrower's shoulder thought by some to be related to the development of pathology is a tight posterior capsule.[45,55,56,67,68] As mentioned previously, asymptomatic throwers exhibit decreased internal rotation of the abducted arm. Although this is clearly related to increased humeral retroversion, a process thought to be related to bone remodeling in young throwers, some believe the posterior glenohumeral joint capsule may be contracted, limiting motion.[45,55,56] The effects of a tight posterior capsule are thought to include the development of anterior impingement symptoms due to a yo-yo effect with arm elevation[45,69] (Fig 7). Morgan believes that a tight posterior capsule may produce superior humeral head migration leading to superior labral tears.[55,56] There is evidence to suggest that a tight posterior capsule is a risk factor for the development of pathology in a thrower's shoulder.[45,56] Many have suggested stretching of a tight posterior capsule is an essential component of the rehabilitation in throwers.[45,67,68] Some authors have recommended surgical release of a tight posterior capsule[70] (C. Morgan, personal communication). How this region of the capsule becomes contracted, and the effects of a tight posterior capsule remain areas of interest and study.

Evaluation of the Throwing Athlete

History: The evaluation of the throwing athlete begins with a thorough history. Particularly relevant questions include the age of the patient, years throwing, the hand dominance, the level of competition, a medical history and review of systems, a history of pre-existing shoulder problems, and a history of other orthopaedic conditions.

The age of the patient is relevant to determine if the patient's physes are open and if the presenting shoulder pain is related to physeal injury.[71] Older pitchers are more likely to experience rotator cuff pathology, whereas younger pitchers are more likely going to

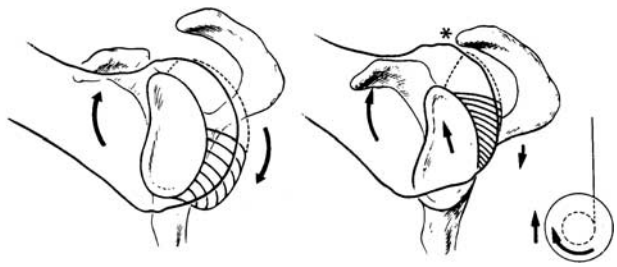

Figure 7. Tight posterior capsule and anterior impingement. A tight posterior capsule could result in anterior translation of the humeral head, as the humeral head rides up on the posterior capsule like a yo-yo. This would accentuate rotator cuff impingement. (Reprinted with permission.[69])

have problems with laxity. Pitchers in the middle of their careers may exhibit both. The years throwing and the level of competition are helpful to understand the expectations of the thrower, and predict the duration before a recovery can be expected. A history of other shoulder or other orthopaedic conditions is important when remembering the kinetic chain concept,[45,46] and realizing that the lower extremity or spine problem may have led to altered throwing mechanics, which are the cause of the shoulder pain.

When assessing the shoulder, specific complaints should be addressed. Is pain the major concern? The location of the pain can help identify particular anatomic structures that may be involved. Is the patient experiencing weakness or fatigue? Some throwing athletes may have symptoms of looseness in the shoulder with symptoms of instability. A feeling of popping or catching may suggest labral pathology or instability. In addition, it is helpful to know if the symptoms developed acutely or chronically. Acute injuries are more likely to require surgical intervention, whereas symptoms that developed gradually without an inciting episode may be more likely to respond to nonoperative treatment.

A profile of the patient's symptoms is also important to ascertain. When during the pitch do the symptoms occur? Pain during cocking suggests internal impingement, laxity, and/or instability. Pain that occurs after ball release and during deceleration suggests rotator cuff pathology. When during the game do the symptoms occur, early or late innings? What is the location and duration of symptoms? In addition, related symptoms distal from the shoulder should be sought. Does the patient have a dead arm or paresthesias, which may be indicators of instability? The profile of the thrower's symptoms can often predict the response to nonoperative treatment. In general, symptoms that occur later in a game or after repeated bouts of throwing suggest rotator cuff fatigue, which may respond well to rest and rehabilitation.

A discussion with the thrower's coach can be very helpful. An understanding of the player's pitch counts will lead to an understanding of the fatigue. How has the pitcher's form changed? Does he drop his elbow when throwing (a frequently cited sign of shoulder pathology)? Do these changes develop in later innings? Does the thrower show other signs of fatigue, such as relying on pitches other than the fastball in the later innings?

Physical Examination: Inspection of the asymptomatic throwing athlete at rest will typically reveal asymmetry. The dominant arm in a seasoned thrower is typically hypertrophied, and one may find symmetry or very subtle atrophy in chronic shoulder conditions. Some pitchers with shoulder pathology will hold the scapula in a depressed and protracted position.

Palpation for pain can help identify the structures that have been injured, and can be used to distinguish disorders of the subacromial space or supraspinatus, long head of the biceps, and teres major tendons. Range of motion, both glenohumeral and scapulothoracic, should be evaluated. Scapulothoracic motion should be smooth and symmetrical. Painful crepitus with scapulothoracic motion may suggest the presence of an inflamed scapulothoracic bursa. Rotation of the abducted arm typically demonstrates increased external rotation and decreased internal rotation of the dominant arm. In asymptomatic throwers, the total range of rotation should be the same for both shoulders, with the dominant arm range shifted toward increased external rotation and decreased internal rotation. Limitations in internal rotation beyond the normal-but-shifted range, may place athletes at risk for the development of shoulder problems, and one may wish to emphasize posterior capsule stretching in this athlete.

Glenohumeral joint translations may be evaluated for anterior, posterior, and inferior directions. This should be done in multiple positions, with the athlete standing, sitting, and lying supine. While increased laxity in the dominant arm may not necessarily be the source of pathology, the patient should be asked if these maneuvers reproduce his symptoms, which may be very helpful in identifying the presence and direction of glenohumeral instability.

Strength testing of the rotator cuff, deltoid, and periscapular muscles should be performed. The infraspinatus and teres muscles are evaluated with resisted external rotation of the arm at the side. The supraspinatus can be evaluated with resisted abduction with the arm held abducted 30° in the plane of the scapula and the thumbs pointed toward the ground. The subscapularis is evaluated with the lift-off test[72] or the belly-press test. Any pain elicited during strength testing will help identify the source of the patient's symptoms.

Provocative tests are particularly helpful in finding the source of pain in a thrower's shoulder, and should include the Neer and Hawkins impingement tests for evaluating the subacromial space and supraspinatus. The apprehension and relocation tests[24] are very sensitive when fear or apprehension is produced, but less helpful when pain is produced.[73] If the abducted arm

is rapidly externally rotated, pain in the posterior shoulder may suggest the presence of internal impingement. A variety of provocative tests for lesions of the superior labrum have been described. In general, these tests should be performed in throwers. While these tests may be sensitive for finding labral tears, none of the tests has shown great specificity, and as a result, pain during these tests may indicate labral or other pathology.

Diagnostic Testing: Radiographs are helpful to show bony pathology and typically include a thrower's shoulder series[74]: anteroposterior views with the arm held in internal (helpful for identifying Hill-Sachs lesions) and external rotation, a scapular outlet view to assess acromial morphology, and an axillary lateral or West Point view[75] to identify bony Bankart lesions. Other helpful views include a Stryker notch view[76] to observe the Bennett's Lesion (an exostosis on the posterior glenoid).[14] Other views can be used when the physical examination would suggest them helpful, i.e., AC joint views for AC joint pathology.

CT and ultrasound have specific, limited use in evaluating the thrower's shoulder. CT is especially helpful in fractures or to help determine the extent of bone loss in cases of shoulder instability. Ultrasound is helpful in determining the extent and severity of rotator cuff tendinosis, but is highly reader specific, and may not be available at most hospitals.

Other than radiographs, MRI is the ancillary procedure of choice for most of the conditions seen in the thrower's shoulder. The MRI is helpful to see all of the soft tissue structures and, with T2 and fat-suppressed imaging, can identify regions of pathologic interest. MRI has been shown to be particularly useful for rotator cuff pathology[77-80] and injury to the glenoid labrum.[81-83] It is important to note that MRI scans of asymptomatic throwing athletes may also show pathologic changes,[84] and as such, the findings on the MRI should be used to support a diagnosis made primarily by the history and physical examination. To detect intra-articular pathology, such as labral tears, the sensitivity of MRI can be augmented by the intraarticular injection of saline or gadolinium.[85] However, MRI arthrography is still under investigation and is not widely available at this time.

Summary

Although humans have been throwing for over a million years, we are only now beginning to understand the biomechanics and pathomechanics in the thrower's shoulder. An understanding of these topics will invariably lead to improved diagnostic efforts, better treatment, and improved strategies for prevention of injuries in the thrower's shoulder. When examining a thrower, a careful history will usually ascertain the problem, and a thorough physical examination with imaging will typically confirm the diagnosis, and set the course for treatment.

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MISCELLANEOUS LESIONS OF THE SHOULDER OF THE THROWING ATHLETE

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In patients who engage in overhead sports, particularly baseball players, the presenting complaints are usually the same: pain, weakness, inability to create ball velocity, and inability to perform to their full potential. In a majority of these athletes, the symptom complex is typical of rotator cuff tendinitis, instability, or superior labrum pathology. The symptoms associated with those conditions also can reflect a wide variety of less common conditions. In the overhead athlete, the "uncommon" lesion can present more often than in nonthrowing populations, and the treating physician should be aware of the less frequent lesions in these patients. This awareness will allow the provider to make an accurate diagnosis at initial presentation and will allow further investigation in the patient who does not improve with traditional therapies. For example, the "dead arm syndrome" of pitchers as described by Rowe[1] is associated with subtle instability of the shoulder, but the accompanying symptoms of tingling, numbness and fatigue could also reflect nerve entrapments, vascular lesions or thoracic outlet syndrome. The authors review the less common diagnosis that should be kept when evaluating and treating the throwing and overhead sport athlete. This article is not meant to be an exhaustive review, but rather a reminder of the potential lesions that can occur, how to make the diagnosis, and to review current treatment recommendations.

Synovial Cysts

Synovial cysts are a good example of the unusual conditions that may be seen in athletes but which have few localizing signs to help with the diagnosis. Synovial cysts can occur in almost any location around the shoulder, but the symptoms are typically a vague ache. In some cases, compression of the suprascapular nerve at the spinoglenoid notch or the suprascapular notch can cause atrophy and weakness of either the infraspinatus alone or of the supraspinatus and infraspinatus respectively.[2-6] It is recommended that athletes be examined so that their posterior thorax can be evaluated for atrophy, and that resisted external rotation be evaluated in all throwers. However, unless there is nerve entrapment, there is no one finding on physical examination that is pathognomonic for these lesions.

Many of these lesions are discovered at the time of MRI, and MRI is the diagnostic modality of choice for evaluating these lesions (Fig 8). Gadolinium enhancement is not typically necessary.[6,7] It is important not to mistake a normal extension of the joint capsule beneath the coracoid, which is called the subcoracoid recess, for a synovial cyst.

Treatment of these lesions initially should be nonoperative unless there is clear recent onset of nerve compression. Most of these lesions are found incidentally and do not require surgery, but the symptomatic patient should be followed closely for signs of nerve

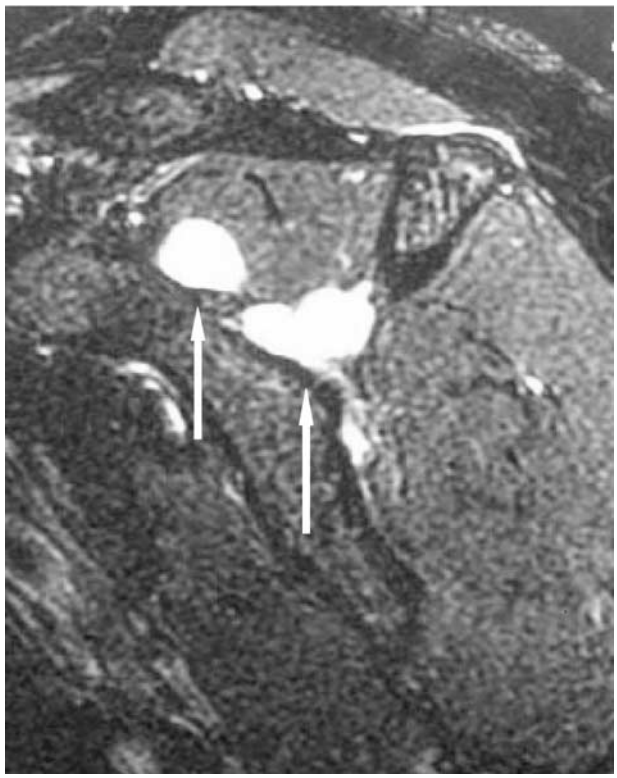

Figure 8. T1-weighted MRI sagittal image of a left shoulder showing a ganglion cyst located in the spinoglenoid notch (arrows).

compression. In the symptomatic patient, there are many options which reflect the vagary of the literature on this topic.[8-10] Aspiration under CT or MRI guidance has been reported.[11] Hawkins et al. reported an 18% failure rate for aspirating the cyst, and in those aspirated, 48% had a recurrence, although 54% of the aspirated patients were satisfied with the outcome.[12]

The surgical options are myriad, and the critical issues in approaching these lesions are the comfort level of the surgeon with the procedure and whether it is felt that there is a labral injury associated with the cyst. Arthroscopy of the shoulder to evaluate the posterior and superior labrum, where some physicians suggest the cyst communicates with the labrum tear, has been shown to be effective in decompressing the cyst and offers the opportunity to repair the labrum.[3,6,13,14] The only difficulty with this technique is that not all synovial cysts are associated with labral tears, and not all will be seen to decompress into the joint. Arthroscopy can puncture the cyst and make it difficult to find later if an arthrotomy needs to be performed. To avoid this difficulty open procedures may be performed before arthroscopy. Open procedures carry the assurance that the cyst will be located in most instances, and arthroscopy can be carried out after definitive resection of the cyst.

Arthroscopy of the spinoglenoid notch for evaluation of the cyst has been reported.[15] The anatomy in this area is complicated by the proximity of the neurovascular bundle and the variability of the spinoglenoid ligament.[16,17] The relative rarity of this lesion may preclude extensive experience with this technique.

Nerve Injuries

Nerve injuries to the shoulder region of the overhead athlete can occur without the presence of synovial cysts or other space occupying lesions.[18] The most commonly seen injuries include suprascapular nerve and long thoracic nerve lesions. Spinal accessory nerve lesions are particularly uncommon, but their initial presentation may be similar to long thoracic nerve injuries.

The exact etiology of suprascapular nerve injury in the overhead athlete is not known.[19-22] Typically the infraspinatus branch of the suprascapular nerve is involved and the supraspinatus muscle is spared. This entity is quite common in volleyball players, with studies showing an incidence of up to 20% in professional volleyball players.[22] In a majority of volleyball players it is asymptomatic, and they can participate in their sport with no limitations.[23] Most individuals with an isolated infraspinatus muscle atrophy due to this nerve injury can perform their activities of daily living with no detriment. This lesion is also common in baseball players, but the exact incidence is not known.

Infraspinatus nerve palsy in athletes will typically present like tendinitis of the shoulder with pain and weakness. There are few historical features that will hint of the diagnosis, and it often will be made by observing infraspinatus atrophy and weakness to strength testing for external rotation with the arm at the side. A complete neurologic evaluation is recommended to make sure that there are no signs of a cervical disc or other neurologic disorders. The initial treatment in patients in this nerve entrapment should be to decrease the pain and inflammation with medication, relative rest, and physical therapy. Electromyography can confirm the diagnosis, and MRI is recommended to rule out a synovial cyst, torn rotator cuff, or other abnormalities. A large percentage of patients treated nonoperatively will become asymptomatic and not require surgery, despite having con-

tinued muscle atrophy and some weakness. These patients should be followed-up to confirm that there is no progression of the lesion or of new lesions.

Operative treatment of this lesion remains controversial, although most feel that surgery should be reserved for those with continued pain and inability to perform their sport after nonoperative treatment has failed. One reason for this sentiment is that the exact etiology of this lesion is unknown. It has been postulated to be a traction injury of the nerve as it makes the turn around the scapular spine from the supraspinatus fossa to the infraspinatus fossa. The surgery involves open decompression of the nerve at the notch along with release of the spinoglenoid ligament and removal of bone at the base of the scapular spine if needed. The second reason that surgery should be reserved for symptomatic cases is that while the results of surgery can be beneficial in up to 87% of cases, complete recovery of the nerve is seen in only 50%.[24,25] A review of the literature suggests that surgery can resolve symptoms in a majority of patients who are symptomatic, but only about half of patients with this lesion who present with problems will need surgery.

Winging of the scapula of athletes can occur for a variety of reasons, both traumatic and atraumatic.[26-30] Overhead athletes who have pain in the shoulder frequently will have alterations of their scapulothoracic rhythm with elevation of the arm, and this can be due to a variety of etiologies. This abnormal rhythm may produce scapular winging, and a careful neurolgoical evaluation is recommended in these cases. Kibler has described three types of scapular dyskinesia, but the exact diagnostic and therapeutic value of this schema needs further study.[31] Electromyography can distinguish a neurologic etiology of the winging from fatigue of the serratus anterior or other causes of winging. In cases of bilateral winging, rare diagnosis such as fascio-scapular-humeral (FSH) dystrophy should be entertained (Fig 9).

Another cause of winging in overhead athletes is trapezius palsy due to spinal accessory nerve injury.[32] We have seen only one case in a professional baseball player which occurred with no trauma. Trapezius palsy can result in winging which has subtle differences from long thoracic nerve palsies, and the diagnosis will be obvious in cases where there is atrophy of the trapezius muscle. The etiology of this lesion in athletes is believed to be due to traction, and nonoperative treatment with recovery of the nerve typically occurs over several months.

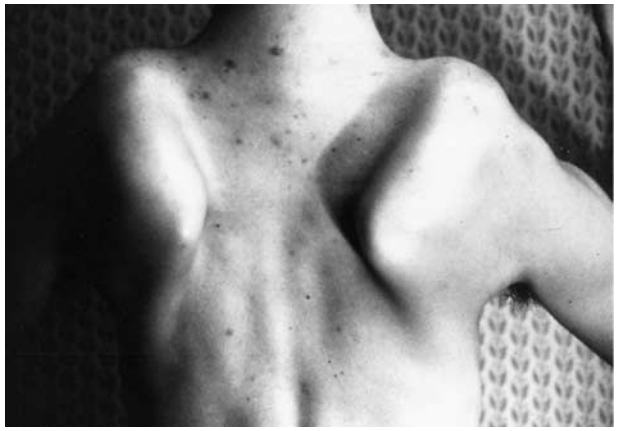

FIGURE 9. A case of bilateral winging due to fascio-scapular-humeral (FSH) dystrophy in an athlete.

Vascular Lesions

Vascular lesions of the upper extremity of throwing athletes are rare and include injuries to the venous and arterial systems. The largest series of these injuries was 34 patients and arterial lesions appear to be more common than venous lesions.[33,34] Vascular lesions have been described in baseball players, volleyball, tennis, cycling, marksmanship, and kayaking.[34] Arterial lesions include compression of the subclavian artery at the level of the anterior scalene muscle, of the axillary artery at the level of the pectoralis minor, or compression of the posterior humeral circumflex artery (PHCA) in the quadrilateral space, which is commonly called "quadrilateral space syndrome."[33-37] Aneurysms of the arterial tree around the shoulder include the subclavian or axillary artery and numerous tributaries including frequently the PHCA.[36,37] Embolic occlusion of the axillary artery in throwing athletes has also been described, but the exact etiology of these clots is unknown.[33]

Symptoms of arterial compression often are initially vague and nonspecific, but later complaints of coolness of the hand or digits should increase suspicion of a vascular lesion. Parasthesias can be present, but there will frequently be no specific dermatomal or peripheral nerve distribution. The patient may also complain of cold intolerance. On examination, the patient may have a cool extremity or digits, and there may or may not be present pallor of the extremity, sluggish capillary refill and punctuate skin lesions typical of arterial emboli.[34-37] Pulses may or may not be diminished depending on the type and severity of the lesion, and auscultation for bruits is recommended

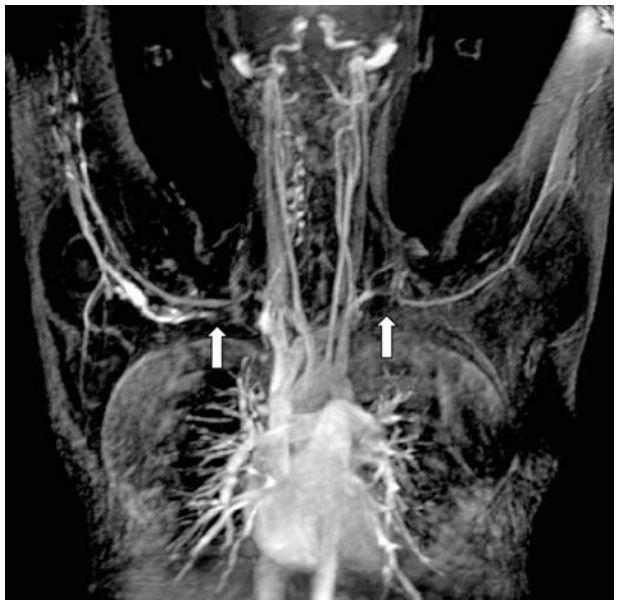

Figure 10. MR arteriogram administrating gadolinium that shows loss of flow in the subclavian artery (thin arrows) and vein (thick arrows) bilaterally, in a patient with symptoms of bilateral thoracic outlet syndrome.

but rarely will be positive. The heart should be examined carefully as a potential source of emboli or clots, and other causes of vascular abnormalities such as Reynaud's Syndrome should be considered. Likewise, digital ischemia has been reported in athletes due to repetitive trauma, and careful examination of the digits is important.[36,38-40]

In patients where an arterial lesion is suspected, testing should progress at a pace consistent with the severity and chronicity of the symptoms. Doppler ultrasound can be beneficial as a first test, but its use as a dynamic test should be limited. Using duplex scans of the upper extremity in 92 extremities of normal professional pitchers, minor pitchers, and normal controls, Rohrer et al. found compression of the axillary artery in 83% of the extremities with the arm in a throwing position.[33] Plethysmography is a good initial screening tool, and MRI of the arterial tree also provides more information.[41] Our philosophy is to involve a vascular surgery consultation early in the work-up so as to obtain the best test. The gold standard for the diagnosis of arterial lesions is an arteriogram, and this is the test of choice for preoperative planning[37] (Fig 10). Treatment depends on the lesion, its chronicity, and the severity of the symptoms.

Quadrilateral space syndrome is a vascular condition in overhead athletes that remains controversial.[42-45] First described by Cahill and Palmer in 1983, the syndrome involves compression of the PHCA or axillary nerve in the quadrilateral space.[42] Symptoms are nonspecific and include posterior shoulder pain and discomfort. It has been suggested that on examination point tenderness over the quadrilateral space may aid in the diagnosis, but this is also an inconsistent finding.[45] Weakness in abduction, atrophy of the deltoid, or paresthesias in the distribution of the axillary nerve may aid in the diagnosis. Electromyography is rarely positive.[45] In most cases, the diagnosis of quadrilateral space syndrome can only be made with clinical suspicion. Unfortunately the only way to make the diagnosis is with arteriogram, and even then occlusion of the PHCA with abduction and external rotation of the arm may be a normal variant.[46] Surgical intervention was shown to be successful by Cahill when fibrous bands are found restricting the PHCA or axillary nerve.[42] Quadrilateral space syndrome is a difficult diagnosis to make and surgical intervention for this condition alone requires careful consultation with the patient and his or her family.

Venous lesions can also occur in the upper extremity of overhead athletes, and the most common condition is compression of the subclavian vein at the thoracic outlet or compression of the axillary vein anterior to the shoulder.[47-50] Subclavian venous thrombosis (SVT) has been described in baseball, football, swimming, rock climbing, and wind surfing.[34] In patients where the compression is intermittent the diagnosis can be difficult to make reliably. In patients with thrombus formation the diagnosis may be more obvious. The initial symptoms typically are swelling and pain or both.[34,47] Paresthesias may be present and a sense of coolness may be present. A bluish or mottled appearance to the extremity is usually present if a thrombus is present. Collateral veins may become more prominent as time or the lesion progresses.

The best initial screening test is a Doppler ultrasound of the extremities.[38,51] If a clot is suspected then the next best test is a venogram of the upper extremity.[41,52] The evaluation should also include blood studies to rule out a hypercoagulable state. The treatment of SVT and axillary thrombi depends on many factors, but catheter directed thrombolysis is usually the first step in symptomatic patients with clots. Vascular consultation is recommended for these lesions to aid in the evaluation and to assist in long term anticoagulation. Surgery may be indicated in those patients with recurrent clots or who have persistent thoracic outlet symptoms.[34,47] In the series of 12 athletes with SVT

reported by Arko et al., 8 (67%) required eventual thoracic outlet decompression and subclavian vein venolyis despite treatment with anticoagulation.[34] In their series all patients recovered without subsequent symptoms.

AC Injuries

AC injuries in overhead athletes are uncommon as most overhead sports involve low to moderate levels of contact. Most physicians agree that grade I and grade II AC separations do not require surgical treatment. Grade III lesions remain controversial despite randomized trials that indicate nonoperative treatment is successful in most cases.[53-55] The reason for this is the perception by some physicians that the patient with a grade III AC separation experiences fatigue and loss of performance. This argument is extrapolated to the throwing athlete where it is presumed that an abnormal AC joint leads to altered scapulothoracic mechanics, thereby altering the throwing motion and affecting performance.

Unfortunately there is not much guidance in the literature regarding the ideal treatment of a throwing or overhead athlete who sustains a grade III AC separation. McFarland et al.[56] performed a survey of physicians of professional baseball teams and only 32 lesions had been seen by this group of physicians. When queried about treatment of a hypothetical starting pitcher who sustained a grade III AC separation, 69% reported they would treat the lesion nonoperatively and 31% would treat it operatively. If treated surgically, a majority would use a Weaver-Dunn procedure reinforced with high-strength suture between the clavicle and the acromion. It should be noted that this study did not examine patients and relied on physician opinion only.

Bennett Lesions

A Bennett lesion is an exostosis of the posterior-inferior glenoid in throwing athletes first described by one of the first baseball physicians, Dr. Bennett.[57] While the exact cause is unknown, Bennett postulated that it was a traction lesion due to pull on the posterior capsule or to traction from the triceps tendon.[58-60] Others have suggested that it may be due to contact of the humeral head to the labrum or posterior-superior labrum.[51,62,63]

Imaging studies have shown that this calcification is definitely extra-articular and that it does not involve the triceps tendon.[62-64] The lesions can be seen on axillary radiographs but they are best imaged with CT

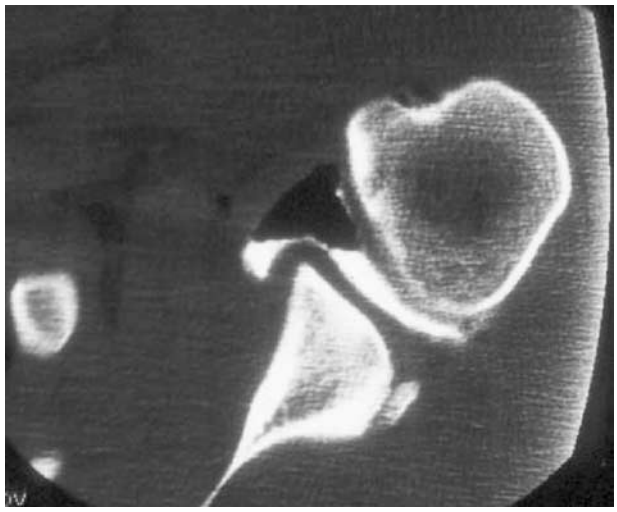

FIGURE 11. CT arthrogram of right shoulder in a throwing athlete showing a posterior Bennett lesion.

(Fig 11). Only one study has attempted to define the incidence of these lesions in symptomatic baseball players.[65] Of 100 players studied, only one third had Bennett lesions visible on plain radiographs.[65] Other studies have found an association between these lesions and posterior labrum tears and partial tears of the infraspinatus.[62,63] The location of the lesion is where the capsule attaches to the posterior scapular neck. It typically cannot be seen arthroscopically without some release of the posterior capsule.[62,63]

The exact place of this lesion in the constellation of findings in the throwing shoulder is unknown. Its relationship to posterior shoulder pain in the throwing athlete is also unknown. Consequently, surgical procedures which address the Bennett lesion suffer from the lack of a clear indication for surgery and typically other pathologies are addressed at the same time as the Bennett lesion.[60,61,66] Ferrari et al.[63] operated on 7 professional and collegiate throwers over 6 years, and at surgery they found posterior labral damage in 6 and partial cuff damage in 6. None of the Bennett lesions was debrided, and all but 1 player returned to his previous level of play. Meister et al.[66] reported on arthroscopic debridement of the Bennett lesion in 11 of 22 throwing athletes with Bennett lesions who where evaluated arthroscopically. Overall only 55% of the throwers returned to their previous level of throwing. We currently do not recommend debridement of Bennett lesions unless the lesion is addressed incidentally to other posterior shoulder pathologies.

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