Mobility and Stability Adaptations in the Shoulder of the Overhead Athlete: A Theoretical and Evidence-Based Perspective

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Mobility and Stability Adaptations in the Shoulder of the Overhead Athlete

A Theoretical and Evidence-Based Perspective

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Abstract

Overhead athletes require a delicate balance of shoulder mobility and stability in order to meet the functional demands of their respective sport. Altered shoulder mobility has been reported in overhead athletes and is thought to develop secondary to adaptive structural changes to the joint resulting from the extreme physiological demands of overhead activity. Researchers have speculated as to whether these structural adaptations compromise shoulder stability, thus exposing the overhead athlete to shoulder injury. Debate continues as to whether these altered mobility patterns arise from soft-tissue or osseous adaptations within and around the shoulder. Researchers have used quantitative techniques in an attempt to better characterize these structural adaptations in the shoulders of overhead athletes. Throwing athletes have been shown to display altered rotational range of motion (ROM) patterns in the dominant shoulder that favour increased external rotation and limited internal rotation ROM. Throwers also show a loss of horizontal or cross-body adduction in the throwing shoulder when compared with the non-throwing shoulder. This posterior shoulder immobility in the throwing shoulder is thought by some researchers to be associated with reactive scarring or contracture of the periscapular soft-tissue structures (e.g. posterior capsule and/or cuff musculature); however, evidence of reactive scarring or contractures of the posterior-inferior capsule or cuff musculature from anatomic or noninvasive imaging studies is lacking. Conversely, translational ROM (laxity) has been consistently shown to be symmetric between dominant and non-dominant shoulders of overhead athletes.

From a skeletal perspective, throwing shoulders are shown to have more humeral retroversion when compared with the non-throwing shoulder. Alterations in humeral retroversion are thought to develop over time in young pre-adolescent throwers when the proximal humeral epiphysis is not yet completely fused. Even though the evidence is inconclusive at the present time, there is more compelling evidence that leads us to believe that altered shoulder mobility in the overhead-throwing athlete is more strongly associated with adaptive changes in proximal humeral anatomy (i.e. retroversion) than to structural changes in the articular and periarticular soft tissue structures. In addition, this retroversion is thought to account for the observed shift in the arc of rotational ROM in overhead athletes. However, in some athletes, capsulo-ligamentous adaptations such as anterior-inferior stretching or posterior-inferior contracture may become superimposed upon the osseous changes. This may ultimately lead to pathological manifestations such as secondary impingement, type II superior labrum from anterior to posterior (SLAP) lesions and/or internal (glenoid) impingement.

Overuse injuries in the overhead athlete are a common and perplexing clinical problem in sports medicine and, therefore, it is imperative for sports medicine clinicians to have a thorough understanding of the short- and long-term effects of overhead activity on the shoulder complex. It is our intention that the information presented will serve as a guide for clinicians who treat the shoulders of overhead athletes.

1. The Overhead-Throwing Shoulder

Overhead athletes require a delicate balance of shoulder mobility and stability in order to meet the functional demands of their respective sport.[1,2] Overhead athletes include, but are not limited to, throwers, swimmers, tennis, water-polo and volleyball players. Altered mobility patterns have been consistently reported in the dominant shoulder of elite baseball pitchers,[3-11] team handball and tennis players,[12-14] water-polo players,[15] as well as in the shoulders of competitive swimmers.[16-19] Shoulder mobility in the overhead athlete has been found to be both excessive (hypermobile) and limited (hypomobile) compared with shoulders that are not exposed to overhead sports, and researchers have debated for years whether altered shoulder mobility in the overhead athlete is inherent, which in turn may pre-select an athlete to an overhead sport, or acquired through adaptive change to joint structures. Acquired hypermobility and hypomobility is thought to develop secondary to structural changes to the glenohumeral joint capsule, ligaments, glenoid labrum, rotator cuff musculature or osseous structures from long-term exposure to overhead activity.[20,21] In addition, researchers have speculated as to whether these structural adaptations compromise shoulder stability, thus exposing the overhead athlete to shoulder injury.[22-26]

Overhead activities such as swimming, throwing, spiking or serving have mobility, stability and functional requirements that are unique to their individual sport. Shoulder hypermobility in swimmers is considered by many to be advantageous given that increased shoulder mobility has been directly correlated with greater stroke length, swimming speed and overall swimming performance,[19,27] while hypermobility in throwers is thought to allow for greater arm cocking and ball velocity upon release.[7,28-30] The inherent contradiction for overhead athletes is the fact that the shoulder must be loose or hypermobile enough to perform overhead activity, yet stable enough to prevent the joint from 'giving way' or subluxating. Wilk and Arrigo[30] have referred to this illogicality as the 'throwers paradox'. Researchers theorize that inherent/congenital or acquired mobility patterns as seen in these overhead athletes play a causal role in the development of shoulder overuse injuries.

From a functional standpoint, baseball, tennis and handball players require repetitive overhead motions that are discontinuous and ballistic in nature. In these activities, the arm is forcefully propelled forward from maximal to near maximal external rotation to internal rotation and requires the posterior rotator cuff musculature to act eccentrically in order to decelerate or 'brake' the arm as it internally rotates and horizontally adducts across the body. On the other hand, freestyle swimming requires a more continuous and repetitive bilateral overhead motion, while submerged in water, where the arms are used to propel the body forward during the 'pull through' phase.[18,31] During the corresponding 'recovery' phase, the arm is lifted out of the water and brought over the body in preparation for hand entry and the next stroke cycle. This type of activity produces less stress and eccentric loading to the joint; however, the continuous nature of the freestyle technique permits less opportunity for muscular recovery and a greater risk of fatigue-induced microtrauma to the joint.[18,31] Water polo represents a unique combination of both forceful throwing and swimming. Therefore, the forces imparted upon the shoulders of water-polo players include the forceful unilateral stresses observed in overhead throwing as well as the more continuous bilateral forces observed in swimmers.[15]

Overuse injuries in the overhead athlete are a common and perplexing clinical problem in sports medicine and, therefore, it is imperative for sports medicine clinicians to have a thorough understanding of the short- and long-term effects of overhead activity on the shoulder complex.[32-34] The shoulder complex involves integrated movements occurring simultaneously at four articulations: glenohumeral, scapulothoracic, acromioclavicular and sternoclavicular, with impairment in one articulation likely causing overall shoulder dysfunction. This article will present information on shoulder mobility and stability requirements of overhead athletes followed by identifying and describing a select number of adaptive changes and corresponding shoulder overuse pathologies as seen in overhead athletes based on the most recent theories and evidence available in the literature. It is our intention that the information presented will serve as a guide for clinicians who treat the shoulders of overhead athletes.

2. Shoulder Mechanics

2.1 Shoulder Mobility

2.1.1 Glenohumeral Joint

Shoulder mobility is characterized by the magnitude of rotational and translational range of motion (ROM). Rotational motion about the long axis of the humerus or humeral 'spin' is referred to kinematically as internal and external rotation, while angular rotation or 'rolling' of the humeral head on the glenoid occurs during elevation or abduction/adduction depending on the anatomic plane.[35] Translatory motion is linear in nature and is described as a 'sliding' or 'gliding' of the humeral head on the glenoid fossa.[35] Rotational ROM is referred to as 'physiological' motion in that the athlete's movements occur voluntarily, while translational ROM is referred to as 'accessory' in that these motions occur involuntarily and yet are necessary in order for the overhead athlete to obtain full ROM.[35] Translatory motion has been often referred to in the literature as joint laxity. Operationally, shoulder laxity has been defined as 'the magnitude of humeral head displacement on the glenoid resulting from the application of a small force'.[36] When defining translational laxity, reference should also be made to humeral position and direction of the applied force.[36] Researchers are beginning to understand a great deal more regarding the 'normal' range of shoulder laxity in overhead athletes with researchers showing a wide spectrum of laxity to be normal.[8,37] A minimum amount of humeral head translation (laxity) is required at the glenohumeral joint for humeral rotation to occur.[38] Glenohumeral translation associated with humeral rotation has been termed 'coupled' or 'obligate' motion.[39,40] For example, external rotation of the shoulder results in posterior humeral head translation.[41]

Rotational ROM in overhead athletes has been well characterized in the literature and is measured clinically in either passive and/or active modes, while translational ROM is typically measured passively with the athlete's shoulder relaxed. Both forms of motion can be measured objectively using instruments aimed at measuring angular or linear displacement as a function of an applied manual or instrumented force. Rotational ROM of the shoulder is typically measured objectively using a goniometer, while translation is measured using an arthrometer. Both instruments are available commercially for use in the clinical or research laboratory.

Baseball pitchers, tennis and handball players have been repeatedly found to have an increased range of external rotation with a corresponding decreased range of internal rotation in their throwing shoulder compared with the contralateral shoulder when assessed at 90° of abduction.[2-12,14,42] The increased range of external rotation is commonly referred to as external rotation gain (ERG) and the decreased range of internal rotation is commonly referred to as glenohumeral internal rotation deficit (GIRD). In the shoulders of competitive swimmers, this same rotational alteration has been observed when compared with the shoulders of nonswimming controls.[16] Water-polo players demonstrate a unique mobility adaptation profile of greater throwing shoulder external rotation with symmetric internal rotation, which may be due to their predominantly unilateral overhead-throwing motion coupled with their bilateral overhead swimming motion.[15] The magnitude of the ERG has been reported to be in the range of 5–12° and the ERG is usually offset by a symmetrical loss of internal rotation (range = 8–15°),[2-5,7,9-11,42] except in water-polo players who have demonstrated symmetric internal rotation.[15]

Interestingly, the total arc of rotational ROM (external + internal rotation) is not significantly different between the throwing and non-throwing shoulder.[2-11] It appears that the total arc of rotation in the overhead-throwing shoulder adapts by shifting 'backward', favouring more external rotation at the expense of internal rotation. Wilk et al.[21] has referred to this rotational arc shift phenomenon as the 'total motion concept'. For throwing sports, a greater range of external rotation allows for more arm cocking, therefore providing a greater ball velocity during the acceleration to the ball-release phase of the throw.[7]

2.1.2 Scapulothoracic Joint

Scapular kinematics have been a major area of investigation in recent years.[43-61] Scapular motions are a direct result of and may also contribute to the dynamic activity of the shoulder articulations, as well as the angular accelerations of the humerus and the flexibility of the surrounding musculature. The scapula exhibits five degrees of freedom (three rotations and two translations) with no motion occurring in isolation.[52,55] These scapular motions occur with respect to the thorax and are generally described as upward/downward rotation, anterior/posterior tilt, internal/external rotation, anterior/posterior translation and superior/inferior translation.[50,53]

Upward/downward rotation of the scapula occurs about an axis perpendicular to the scapular plane with upward rotation occurring when the inferior angle moves laterally and superiorly. This motion accounts for the 2 **:** 1 ratio of the shoulder. This ratio was originally termed by Inman et al.[62] as a 'scapulohumeral rhythm' and is described as 2° of glenohumeral elevation for every 1° of scapulothoracic upward rotation. Anterior/posterior tilt occurs about an axis parallel to the spine of the scapula with posterior tilt depicting the superior scapular border moving posteriorly away from the thoracic cage. Internal/external rotation occurs about a vertical axis with external rotation taking place when the lateral scapular border moves posteriorly away from the thoracic cage.

The scapulothoracic joint is a free-floating articulation and therefore the only connections of the scapula to the thorax are at the sternoclavicular and acromioclavicular joints.[53] Because of the clavicle's connection between these two articulations, the distance between the joints remains constant, thus allowing the scapula two additional degrees of freedom (anterior/posterior translation and superior/inferior translation). Because of the rigid point of fixation via the clavicle, true medial/lateral translation of the scapula is not possible. However, anterior/posterior translation of the scapula, via the clavicle, is possible. The composite motions of anterior translation and internal rotation are frequently referred to as scapular protraction and the composite motions of posterior translation and external rotation as scapular retraction. Protraction/retraction is defined as the medial-lateral scapular movement around the thorax, while superior/inferior translation is described by superior-inferior movement of the scapula on the thorax.[63]

Numerous investigations utilising various 2- and 3-dimensional (3-D) technology have attempted to describe the degrees of rotations and sequence of scapular rotations during arm tasks in different planes of movement.[43,46,48,53,61,64] Based on these data, researchers have generally accepted the theory that the scapula demonstrates a pattern of upward rotation, external rotation and posterior tilt during humeral elevation tasks.[61] During scapular plane humeral elevation, McClure et al.[53] reported the scapula to have approximately 50° of upward rotation, 30° of posterior tilt, 24° of external rotation, 21° of posterior translation (retraction) and 10° of superior translation (elevation).

Throwing athletes, without a history of shoulder injury, have been reported to have several scapular adaptations in their dominant shoulders during a humeral elevation task.[58] Such adaptations include increased amounts of upward rotation,[42,65] internal rotation and retraction when compared with non-throwing subjects.[58] However, because of restrictions in 3-D research equipment, there are currently no empirical data regarding scapular kinematics during the actual throwing or swimming motion. Subsequently, this lack of information has added to the confusion surrounding proper scapular kinematics among various overhead athletic activities.

2.2 Shoulder Stability

2.2.1 Glenohumeral Joint

Shoulder stability in overhead athletes is controlled by active and passive restraining mechanisms that serve to maintain sport-specific glenohumeral joint mechanics.[66] Because of the repetitive nature of overhead activities and the extreme functional demands on the overhead-throwing shoulder, stability can become compromised secondary to mechanical damage, muscular fatigue and neuromuscular control deficits. Pitchers have been found to generate arm velocities >7000 °/sec and rotational torques >70 Nm with shear forces in the range of 300–400 N and compressive forces >1000 N.[67,68] Elite swimmers regularly train 10–12 months of the year, practising 1–2 times per day, 5–7 days per week. Daily distance may vary between 7315–18 288 m (8000–20 000 yards) per day. This translates to up to 16 000 shoulder revolutions per day with the majority of revolutions being done repetitively without rest or recovery.[16-18]

During overhead activities, the scapular stabilizing and rotator cuff muscles function in a balanced manner to maintain a centred relationship between the humeral head and the glenoid fossa.[69] This dynamic stability mechanism is crucial to glenohumeral joint stability during the mid-ranges of arm movement.[70] At the extremes of motion, glenohumeral joint stability is provided mainly via passive bony and soft-tissue restraints.

There is an inherent lack of bony stability at the glenohumeral joint. The surface area of the glenoid fossa is one-third to one-quarter that of the humeral head.[69,71] This relationship has been likened to that of a golf ball on a tee. However, the bony geometry of the glenohumeral joint still plays an important roll in passive stability.[51,72] To compensate for the relatively shallow glenoid fossa, a fibrocartilage ring (called the glenoid labrum) encompasses the entire glenoid rim.[73] The glenoid labrum effectively deepens the glenoid concavity by as much as 50%.[74] By deepening the glenoid socket and providing a soft-tissue rim around the glenoid the labrum serves to limit glenohumeral joint translation.[75]

The glenohumeral joint capsule extends from the labrum and glenoid rim to the neck of the humerus.[76] The capsule envelops the entire humeral head and creates a sealed space within the glenohumeral joint that has been referred to as a 'soft-tissue socket'.[77] The capsule is reinforced by the ligaments of the glenohumeral joint that can be observed as functional thickenings of the capsule.[78] The glenohumeral joint ligaments function collectively with the labrum and capsule to maintain a centred humeral head and limit excessive translation.[73]

The soft-tissue components that contribute to passive glenohumeral joint stability have received considerable attention in the clinical and research literature.[79] Passive joint stability has been often referred to in the literature as passive glenohumeral stiffness. Passive stiffness is a reflection of the static structures resisting humeral head displacement from the glenoid, and is characterized by the joint's ability to provide static stability while simultaneously controlling arthrokinematic behaviour during upper extremity movement. Passive glenohumeral stiffness is quantified experimentally as the amount of force (N) required to displace the humeral head by a given amount (mm).[80,81] Instrumented arthrometers, like the LigMaster™ **1** (Sport Tech, Inc., Charlottesville, VA, USA), are capable of quantifying passive joint stiffness by calculating the slope of the force-displacement curve. Passive glenohumeral stiffness measures provide information concerning the structural and mechanical properties of the joint, and are considered to be clinically important when assessing joint stability.[80] A stiffer joint is able to withstand greater force for a given displacement without disrupting the integrity of the capsulo-ligamentous restraints, and therefore may decrease the risk of injury.[82,83]

During throwing, the anterior band of the anterior-inferior glenohumeral ligament complex maintains joint stability by resisting anterior-inferior humeral head displacement from the glenoid. In order to withstand the repeated stresses of pitching, greater than normal levels of passive stiffness would be beneficial for the pitcher by resisting joint displacement forces during pitching, and thus acting as a prophylaxis against injury. As long as the dynamic musculature of the shoulder complex is able to maintain the humeral head centred within the glenoid, the joint will remain stable.[84] However, if inherent or acquired pathological alterations have significantly diminished the contribution from the passive joint restraints, the dynamic stabilizing muscles may not be able to compensate and maintain proper humeroscapular balance.[85]

2.2.2 Scapulothoracic Joint

The broad flat body of the scapula rests cohesively with the thoracic cage and has the atypical joint characteristic of lacking direct ligamentous attachment. In fact, other than the corresponding musculature, the scapula's only attachment to the thorax comes from it's articulation with the distal clavicle and the subsequent ligamentous structures.

Primary stability of the scapula on the thoracic cage occurs via numerous muscular attachments. Kibler[51] has classified these dynamic stabilizers into three categories. The first group is primarily responsible for upward/downward rotation and stability and consists of the upper, middle and lower trapezius, rhomboids, serratus anterior and levator scapulae. The second group consists of extrinsic muscles including the deltoids, pectoralis minor, biceps and triceps. And the third group consists of the intrinsic muscles, such as the subscapularis, infraspinatus, teres minor and supraspinatus.

Co-contractions of several periscapular muscles create force couples to further increase the stability and function of the scapulothoracic articulation. For example, the balance of forces created by co-contractions of the upper and lower trapezius allow for smooth upward rotation of the scapula and is known as an 'anatomical force couple'.[86] The upward pull caused by contraction of the upper trapezius is negated or 'balanced' by the downward pull caused by a simultaneous contraction of the lower trapezius, thereby allowing both muscles to aid in scapular

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1 The use of trade names is for product identification purposes only and does not imply endorsement.

upward rotation without any scapular elevation or depression. Another force couple critical in stability and the production of scapular upward rotation consists of all three portions of the trapezius (upper, middle, lower) and the serratus anterior. While both the trapezius and the serratus anterior are responsible for scapular upward rotation, the trapezius subsequently causes scapular adduction, while the serratus anterior causes scapular abduction.[87] Therefore, co-contraction of the two muscles causes a balance between their respective adduction and abduction movements allowing for stabilization of the scapula on the thorax while still producing upward rotation.

In the following sections, we will present and discuss several theories that have received considerable attention in the published literature. Acquired mobility and stability patterns are theorized to occur from some form of structural adaptation to the shoulder resulting from the extreme physiological demands of overhead activity. We will describe these theories that are thought to play a causal role in the development of altered shoulder mobility patterns and pathologies based on the evidence available.

3. Acquired Hyperlaxity

3.1 Theory

The theory of acquired anterior hyperlaxity in overhead athletes describes a gradual stretching out of the anterior capsulo-ligamentous restraints producing a lax and mechanically unstable shoulder. Published reports have indicated that elite swimmers posses inherently lax shoulders and may acquire further capsular laxity as a result of the extreme physical demands placed on the shoulder during swimming.[16-18] Subacromial impingement in swimmers is thought to develop secondary to capsular hyperlaxity;[16,18,19] however, the two conditions have never been empirically linked.

Shoulder instability has long been recognized as an aetiology for the symptomatic shoulder. However, the identification of subtle shoulder instability as the primary pathology in overhead throwers was first reported by FW Jobe and associates in the late 1980s and early 1990s.[25,26,88,89] Jobe et al.[90] observed what they described as a subtle anterior instability in the dominant shoulder of throwers during surgical repair for chronic impingement-related symptoms. Based upon poor functional outcomes in throwers treated with subacromical decompression surgeries,[91] Jobe and colleagues[88] developed their theory of acquired hyperlaxity.

It was a short time later that CM Jobe[92] and Walch et al.[93] expanded FW Jobe's theory by suggesting that anterior hyperlaxity results in the subsequent development of internal impingement in throwers. Many of the extreme arm positions inherent in overhead activity, such as the late-cocking stage of throwing, involve extreme glenohumeral external rotation, abduction and horizontal extension.[94] In this position, the humeral head has been shown to contact the undersurface of the supraspinatus tendon in the posterior-superior glenoid region.[93]

On the anterior-inferior side of the joint, the primary static stabilizing structure to anterior humeral translation, the anterior band of the inferior glenohumeral ligament complex, is under maximal strain during abduction and external rotation.[95,96] Stretching of the capsulo-ligamentous restraints as a result of this chronic strain is thought by FW Jobe and others to result in subtle anterior humeral head translation (micro-instability) and postero-superior labral pathology.[25,90,92,97] The combination of micro-instability and labral tearing is thought to produce a corresponding gain in external rotation ROM or ERG in the dominant arm of overhead athletes.[25,94] The hypermobility associated with this increase in anterior capsular laxity is also thought to result in secondary impingement of the rotator cuff tendons in the subacromial space.[88,98]

3.2 Evidence

Despite widespread acceptance in the sports medicine community, no definitive study in humans using objective, quantitative measures of glenohumeral joint translation has confirmed the theory of acquired hyperlaxity in the overhead-throwing athlete. Published data do exist that support the presence of excessive laxity and ERG in non-throwing shoulders with pathological damage to the anterior-inferior joint capsule.[99-101] Warner et al.[100] assessed ROM and laxity in patients with unilateral shoulder instability resulting from a traumatic dislocation and found significantly greater joint laxity and external rotation in the injured shoulder compared with the uninjured shoulder. Grossman et al.[99] induced anterior hyperlaxity in a cadaveric model and identified increased anterior translation and ERG in shoulders with controlled stretching to the anterior-inferior capsule. Mihata et al.[101] assessed ROM and laxity following a non-destructive 30% external rotation stretch in a cadaveric model and found a corresponding 30% increase in the length of the inferior glenohumeral ligament, 30% increase in anterior, inferior and coupled anterior-posterior translation, and a 30% ERG. This is one of the few experimental studies available that demonstrates a link between ERG and anterior hyperlaxity. The findings from Warner et al.[100] may not be applicable to overhead athletes since hyperlaxity and rotation gains (e.g. ERG) in overhead-throwing athletes are theorized to develop from an overuse atraumatic mechanism.

There are numerous published studies with data to support the presence of ERG in the dominant shoulder of overhead athletes; however, ERG has yet to be linked experimentally to the development of micro-instability and anterior hyperlaxity in overhead athletes. While rotational ROM can be easily assessed using standard objective techniques such as goniometry, translational ROM (laxity) has been traditionally assessed with manual laxity tests that use a qualitative grading system to gauge the magnitude of translation. The high degree of observer dependence and subjectivity with these tests has shown them to lack repeatability and precision.[100,102,103] However, newly developed instrumentation has recently become available that can reliably and accurately quantify translational motion in the shoulder (figure 1).[3,4,8,17,100,102-104] Thus far, these instrumented tests have provided the best evidence for supporting or refuting the acquired hyperlaxity theory in overhead athletes.

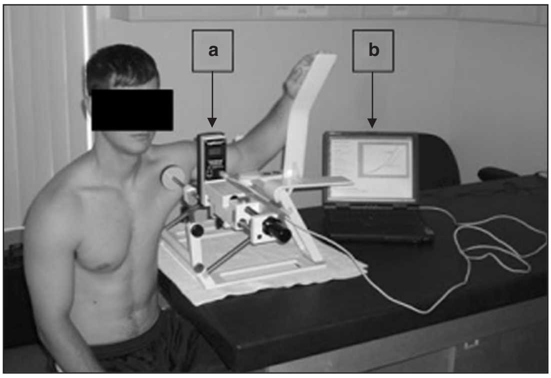

Fig. 1. Instrumented stress examination technique with the arm stabilized in the functional overhead position of 90° abduction and 60° of external rotation. The stress device (**a**) is interfaced with a laptop computer (**b**) for processing and displaying the force-displacement response data for each trial in a graphic and/or numeric format.

Ellenbecker et al.[8] and Borsa et al.[3,4,17] assessed humeral translation in overhead athletes using similar instrumentation and test procedures. Both researchers used arthrometric techniques designed to measure force-induced changes within the joint while the shoulder was positioned in the functional overhead position. Ellenbecker et al.[8] used stress radiography and Borsa et al.[4,17] used stress sonography to objectively measure humeral translation in a group of professional baseball pitchers and elite swimmers. Ellenbecker et al.[8] found no significant differences in anterior translation between the throwing and non-throwing shoulders of 20 professional baseball pitchers. Similarly, Borsa et al.[4,17] did not find significant differences in anterior translation between the throwing and non-throwing shoulders of 43 asymptomatic professional baseball pitchers and no significant differences in anterior translation between the shoulders of 42 collegiate swimmers and 44 age-matched nonswimming control subjects.

Instrumented tests of glenohumeral stiffness have also shown the anterior-inferior joint to be stable when resisting anterior-directed forces. Borsa et al.[3] and Crawford and Sauers[105] both used the LigMaster™ to quantify glenohumeral joint laxity and stiffness in baseball pitchers. Borsa et al.[3] measured the passive resistance or stiffness of the glenohumeral joint in 34 asymptomatic professional baseball pitchers and Crawford and Sauers[105] measured anterior and posterior laxity and stiffness in 22 asymptomatic high-school baseball pitchers. In the test position of abduction and external rotation, anterior-directed forces tested the structural integrity of the anterior-inferior capsulo-ligamentous restraints. In both studies, bilateral comparisons found no significant differences in passive anterior joint stiffness between the throwing and nonthrowing shoulder.[3,105] Any significant capsular attenuation would have been displayed graphically as a decreased stiffness response while the joint was progressively stressed. Testing in the functional throwing position of 90° abduction and 90° external rotation actually demonstrated a decrease in anterior laxity and corresponding increase in anterior stiffness compared with testing in 90° abduction and neutral rotation.[105] This finding actually supports the concept that the anterior inferior glenohumeral ligament complex provides greater joint stability in the functional throwing position.

Sethi et al.[106] utilized an instrumented manual laxity examination to compare anterior-posterior laxity in a mixed population of asymptomatic professional and division I college baseball pitchers and position players. Pitchers demonstrated significantly greater (~4 mm) anterior-posterior translation and ERG in their throwing shoulder compared with their non-throwing shoulder. Position players demonstrated symmetric external rotation ROM between sides. Side-to-side asymmetry of >3 mm anterior-posterior translation was observed in 12 of 20 professional pitchers, 10 of 17 college pitchers and only 1 of 19 position players. Currently, this is the only *in vivo* study that the authors are aware of that has quantitatively recorded greater laxity and ERG in the throwing shoulder of baseball pitchers. Bigliani et al.[1] found a higher prevalence of the sulcus sign in professional baseball pitchers (61%) compared with position players (47%). However, there was no significant difference in the presence of a sulcus sign between the throwing and non-throwing shoulders in pitchers or position players suggesting that this sample of pitchers may have simply been more mobile in general. Neither the data from Sethi et al.[106] or Bigliani et al.[1] demonstrate an increase in isolated anterior laxity compared with the non-throwing shoulder.

Kinematic studies analysing coupled motion of the shoulder dynamically during pitching have not been able to accurately track translational motion. This has limited our understanding of translational motion during throwing especially in the late-cocking phase. However, cadaveric[39,40,99,107] and *in vivo*[41] studies have been able to measure coupled motion during humeral elevation. These studies[39-41,99,107] were able to quantify humeral translation during active and passive humeral elevation and rotation. In ligament-intact shoulders, researchers were able to show that the humeral head translates between 1 and 5 mm posteriorly when the humerus is elevated and externally rotated. During the cocking phase of the pitching cycle, while the arm is abducted and moving into external rotation and horizontal abduction, obligate tightening of the anterior capsule and inferior glenohumeral ligament is reported to force the humeral head posteriorly and slightly inferior especially during late cocking.[39,99] Similarly, Borsa et al.[4] were able to show that professional baseball pitchers have an average of 5.94 mm posterior translation in their dominant shoulder when assessed for laxity in the functional position of abduction and external rotation. The posterior translation findings from Borsa et al.[4] are consistent with the findings of Harryman,[39] Baeyens[41] and Howell,[107] with the throwing shoulder in the abducted and externally rotated position. This may explain why pitchers in the study of Borsa et al.[4] were found to have almost a millimetre more posterior translation in their throwing shoulder then the non-throwing shoulder. The increased posterior translation may be an adaptive response of the posterior capsule providing the throwing shoulder an ‘allowance’ for greater external rotation during late cocking. Greater external rotation in the throwing shoulder has been linked to increases in angular velocity of the humerus during follow through and at ball release.[41]

The posterior capsule is the thinnest portion of the glenohumeral capsule,[108] while the antero-inferior capsule and inferior glenohumeral ligament has been described as the thickest and strongest portion.[78,109] This may account for the asymmetries found between the anterior and posterior translation measures in the study of Borsa et al.[4] With the shoulder in the abducted and externally rotated position, the thick antero-inferior ligaments successfully resist anterior humeral head translation, while the posterior capsule affords less resistance to posterior displacement during posterior loading.[78,99] In addition, when the arm is in abduction and moving into external rotation, the posterior capsule and the posterior band of the IGHLC resist inferior displacement.[78] This inferior resistance may contribute to posterior displacement of the humeral head when the arm is in this position. The minimal anterior displacement found by Borsa et al.[4,17] in the abducted and externally rotated position, indicates that the primary static restraints to anterior humeral translation are very much intact in the shoulder of the overhead athlete.

3.3 Summary

In the functional test position of abduction and external rotation, Ellenbecker et al.,[8] Borsa et al.[4,17] and Crawford and Sauers[105] all found minimal anterior translation in several populations of overhead athletes. These findings suggest that the anterior-inferior restraints are indeed intact and stable in nonpathological overhead-throwing shoulders, and therefore are not stretched out as postulated by Jobe et al.[25] However, recent data from Sethi et al.[106] provide evidence supporting the link between increased shoulder laxity and ERG. Currently, we are unaware of any study that has quantitatively evaluated anterior laxity in overhead-throwing athletes with shoulder pathology. Therefore, the available evidence in human subjects is equivocal regarding the theory of acquired hyperlaxity in the nonpathological dominant shoulder of overhead athletes.

4. Posterior Shoulder Immobility

4.1 Theory

In the last several years, the posterior joint capsule and cuff musculature in overhead athletes has received considerable attention. Overhead-throwing athletes frequently complain of posterior shoulder pain and tightness.[22-24,110,111] In 1985, Pappas et al.[112] was the first to hypothesize that posterior shoulder immobility results from repetitive microtrauma leading to the development of fibrotic scar tissue to the posterior capsule. Contracture of the posterior joint structures has been proposed as a major contributor to the loss of glenohumeral internal humeral rotation and cross-body or horizontal adduction in the overhead-throwing athlete.[22-24,110,111] Currently, it is unclear which posterior structure actually undergoes contracture with most clinical commentaries implicating either the posterior capsule or posterior cuff musculature as the culprit. Researchers theorize that a tight posterior-inferior capsule or cuff musculature results in arthrokinematic alterations resulting in secondary damage to joint structures.[22-24,99,113] The major proponents of this theory are Burkhart and Morgan[114,115] who first began describing the role of posterior capsule tightness in the development of superior labrum from anterior to posterior (SLAP) lesions in the late 1990s. The theory of Burkhart and Morgan is based on personal opinion and clinical observation and is not supported by any scientific data. According to their ‘peel-back’ mechanism theory, an acquired contracture of the posterior capsule results in a posterior-superior shift in the humeral head during the late-cocking phase of throwing.[23,24,114] Collectively, the twisting of the biceps in this position and the tension from the humeral shift place the posterior-superior labrum on traction, eventually pulling it off of the superior glenoid. This results in the development of a type II posterior SLAP lesion. Burkhart et al.[23,24,114] theorize that this SLAP lesion causes an anterior ‘pseudolaxity’ that is not the result of anterior-inferior capsular stretching. Based on their clinical experience, Burkhart et al.[114,116] advocate posterior capsule release and SLAP repair in symptomatic overhead-throwing athletes rather than FW Jobe’s[90] anterior capsulolabral reconstruction. This controversy highlights our lack of a definitive understanding of the mechanisms underlying shoulder pathology in the overhead-throwing athlete.[22]

4.2 Evidence

There is a substantial body of evidence to support the development of posterior shoulder immobility in response to long-term overhead activity; however, a direct cause for the immobility is not known at the present time. Overhead athletes with posterior shoulder immobility present clinically as having less than normal ranges of glenohumeral internal rotation and horizontal adduction, and these conditions can be assessed using primarily two clinical measurement techniques: (i) passive internal humeral rotation at 90° of abduction (figure 2); and (ii) passive humeral horizontal adduction (figure 3). Several investigators[9,15,42,65,100,110,111] have used the horizontal adduction method for quantifying posterior

Fig. 2. Passive internal rotation range of motion of the shoulder with the athlete in the supine position and the arm placed at 90° of abduction. The examiner stabilizes the scapula, rotates the joint until a discernable end feel is felt at which time a measurement is taken using a goniometer.

shoulder immobility; however, it is not exactly clear which structures (tight posterior capsule or cuff musculature) cause the motion restriction. This method of cross-body adduction with the scapula retracted and the arm in 90° of abduction has been validated as a means of assessing posterior shoulder immobility in intercollegiate baseball pitchers and patients with impingement syndrome.[9,100,110,111] Coincidentally, Myers et al.[9] and Tyler et al.[110] both found internal rotation and horizontal adduction to be interrelated when performed on shoulders with a known impairment. Their data were able to consistently show that every 1 cm deficit in cross-body adduction equated to a loss of 4–5° of internal rotation.[9,110]

Subsequent studies have reported a ≥2.0 cm difference between the throwing and non-throwing shoulders of overhead athletes who also demonstrate GIRD.[42,117,118] This magnitude of difference has been shown to be statistically significant in a larger series of professional baseball pitchers;[117] however, this same difference was not significant in a separate study that may have been under-powered.[42] Interestingly, horizontal adduction has been demonstrated to be symmetric in water-polo players who also demonstrate ERG, but no GIRD.[15] Recently, Myers et al.[9] found GIRD and decreased horizontal adduction in a group of baseball players with and without internal impingement. Myers et al.[9] was the first to show through controlled experimentation that throwers diagnosed with internal impingement have significantly more GIRD and less horizontal adduction in the throwing shoulder compared with throwers without the documented impairment. The data from Myers et al.[9] suggest that posterior shoulder immobility may be a contributing factor in the development of shoulder pathology such as internal impingement. Debate still continues with respect to which posterior shoulder structure (capsule or rotator cuff) is altered and therefore directly responsible for the posterior shoulder immobility.

No published data exist that directly quantify soft-tissue contracture *in vivo*; however, Burkhart et al.[24] have reported posterior capsule contracture from observations during surgical intervention in 124 throwers with type II SLAP lesions. Burkhart et al.[24] reported that all of these patients had a >25° loss of internal rotation in the involved shoulder compared with the non-involved shoulder. Myers et al.[9] also noted a similar amount of GIRD in throwers with internal impingement (19.7°), indicating a possible pathological threshold range for GIRD. Most studies performed in nonpathological

[redacted]

Fig. 3. Horizontal abduction of the humerus is also measured with a goniometer. With the athlete supine, the examiner stabilizes the scapula with one hand and moves the humerus across the body.

throwing shoulders found GIRD to be on the average around 10 ± 2° (or 12–17%) compared with the non-throwing shoulder,[3-5,7,8,10,11,13,14,42] whereas GIRD in the pathological shoulder of throwers was found to be in the range of 19.7–25° when compared with the non-dominant shoulder.[9,24] Therefore, it can be surmised that GIRD ≥30% when compared bilaterally may be a contributing factor that leads to pathological changes to shoulder joint structures. Until this crucial piece of the puzzle is discovered, the root of posterior shoulder immobility will remain speculative.

Several cadaveric studies have evaluated the effects of simulated posterior capsule contracture. The first study to evaluate surgically induced posterior capsular contracture was reported by Harryman et al.,[39] who demonstrated significantly increased anterior superior humeral head displacement with passive flexion and significantly increased anterior translation with cross-body adduction. They did not evaluate humeral motion with abduction and external rotation or during internal rotation. Anderson et al.[119] surgically induced a posterior capsule contracture and reported a significant decrease in internal rotation ROM at 0° and 90° of elevation and found that coupled anterior-posterior translation was significantly displaced anteriorly an average of 7 mm. Grossman et al.[99] evaluated translation in the late-cocking phase of throwing before and after a surgically created 10 mm posterior capsular plication. They reported a significant decrease in internal rotation ROM (8.8 ± 2.3°) and a nonsignificant trend towards a more posterior-superior humeral head position moving from 0° to 90° of external rotation at 90° of abduction.[99] In addition, Grossman et al.[99] reported no significant differences in anterior, posterior, superior, or inferior laxity in response to a 20-N force application.

An alternative method of measuring posterior shoulder tightness in overhead athletes is posterior translation as measured in the functional overhead position using an instrumented arthrometer.[4] If a tightened or contracted posterior capsule is the cause of GIRD, then it could be hypothesized that posterior translation in the dominant shoulder would be deficient or significantly restricted when compared with the nondominant shoulder because of the soft-tissue contracture. Borsa et al.[4] assessed shoulder ROM and laxity in a group of asymptomatic professional baseball pitchers and found internal rotation in the throwing shoulder to be limited by 9.7° compared with the nonthrowing shoulder; however, posterior translation measures showed a mean of 5.94 mm in the throwing shoulder compared with 4.82 mm in the nonthrowing shoulder. Therefore, pitchers had, on average, 1.12 mm more posterior laxity in the throwing shoulder compared with the nonthrowing shoulder.[4] In addition, posterior translation in the throwing shoulder was more than double that of anterior translation (5.94 vs 2.62 mm) indicating a directional asymmetry with respect to capsular laxity. A similar study by Crawford and Sauers[105] in high-school baseball pitchers reported no significant difference in posterior laxity or stiffness between the throwing and non-throwing shoulders at 90° of abduction and neutral rotation in response to a 150-N force. Collectively, the findings from Borsa et al.[4] and Crawford and Sauers[105] do not conform to the theory of posterior capsule contracture and may indicate that the deficits in internal rotation may originate from sources other than the posterior capsule.

4.3 Summary

There is compelling evidence for posterior shoulder immobility in the dominant shoulder of overhead-throwing athletes; however, the exact cause for the limited mobility remains unclear. Several studies suggest that the posterior capsule progressively tightens, while others speculate that the posterior cuff musculature tightens as a result of repetitive eccentric overload stress to the cuff during arm deceleration after ball release. Regardless, throwers with GIRD ≥19° have been shown to have pathological changes to joint structures when compared with throwers with ≤19° of GIRD. From studies in overhead-throwing athletes, it appears that there is a pathological threshold by which GIRD will likely produce pathological changes to the joint.

5. Retroversion

5.1 Theory

Recent scientific evidence has attributed the altered mobility patterns as seen in the throwing shoulder to adaptive changes in the bony architecture of the glenohumeral joint.[2,10,11,14] Opposing muscle forces imparted on the humeral head during repetitive overhead throwing are thought to result in osseous adaptations of the proximal growth plate rather then the periarticular soft-tissue structures surrounding the glenohumeral joint.[2,10,11] Adaptive osseous changes include increased humeral and glenoid retroversion. Researchers suggest that this retroversion and subsequent motion adaptation develops during youth baseball participation, probably between 12 and 16 years, while growth plates are 'open'.[2,10,11] Repetitive stress to the proximal physis from throwing is thought to induce an adaptive remodelling response that favours humeral retroversion.[10]

5.2 Evidence

Increased humeral and glenoid retroversion in the throwing shoulder of baseball pitchers and handball players has been shown in several recent studies.[2,10,11,14] Reagan et al.[11] and Osbahr et al.[10] used radiographic analysis and Crockett et al.[2] used CT scans to identify osseous changes in the form of increased humeral head and/or glenoid retroversion in the dominant shoulder of elite baseball pitchers. Crockett et al.[2] and Reagan et al.[11] speculate that humeral retroversion is primarily responsible for the rotational asymmetries, ERG and GIRD, found in these athletes. Humeral retroversion enables the arm to externally rotate to a greater extent and internally rotate to a lesser extent before being constrained by the capsulo-ligamentous restraints. Researchers also speculate that humeral retroversion acts as a controlling mechanism for the shift in internal and external rotation.[2,10,11,14] Osbahr et al.[10] reported a significant correlation between the side-to-side difference in retroversion of the humerus compared with the side-to-side difference in external rotation at 90° of abduction.

In addition to providing a mechanism for increased external rotation, retroversion may act as a prophylaxis to injury. A retroverted humeral head is thought to spare the glenohumeral joint from capsular tension, thereby preserving the joint's static stabilizing mechanism. Osbahr et al.[10] theorizes that during the late-cocking phase of pitching, the restraining capsular tissues would stretch less per degree of external rotation, thus reducing strain on the anterior-inferior capsule,[10] and sparing the joint from repeated microtrauma. Similarly, Pieper[14] noted that in a group (n = 51) of team handball players the subgroup (n = 13) that did not display humeral retroversion in their dominant shoulders complained of chronic shoulder pain. Pieper[14] concluded that the chronic shoulder pain was a direct result of the players' lack of adaptive change to the proximal humeral epiphysis. Pieper[14] further concluded that the lack of retroversion caused the anterior-inferior capsular structures to come under repetitive stress during external rotation.

Support for this theory can be found from the recent findings of Borsa et al.[3,4] and Ellenbecker et al.,[8] showing positive rotational range-of-motion shifts (ERG and GIRD), while also showing no significant side-to-side differences in glenohumeral translation or capsular stiffness between the throwing and nonthrowing shoulders of professional baseball pitchers. Translational and rotational ROM is reported to be related[99] and, therefore, if periarticular soft-tissue adaptations were the cause of the shift in rotational ROM then it could be expected that translational motion would also be affected. For example, greater anterior translation would be indicative of acquired hyperlaxity or stretching of the anterior-inferior capsular structures and less posterior translation would indicate posterior shoulder tightening or contracture due to reactive scarring of the posterior soft-tissue structures.

5.3 Summary

Osseous adaptations in the form of humeral and glenoid retroversion have been shown in the dominant shoulder of throwing athletes. It has been speculated that retroversion acts a controlling mechanism for overhead activity such as throwing and prevents excessive strain on the capsule-ligamentous structures. Even though evidence does exist for osseous adaptations, more research needs to be done in order to draw direct connections between altered mobility and adaptive structural changes in the dominant shoulder of overhead athletes.

6. Scapular Dyskinesis

6.1 Theory

Scapular dyskinesis is a commonly used term among clinicians describing abnormal scapular kinematics. This dyskinesis may range from alterations in any one or a combination of two or more of the five degrees of freedom of the scapula as it rotates and translates on the thoracic cage. Due to the repetitive and violent nature of the throwing, serving and spiking motion, these athletes are required to produce precise scapular function in order to create the large amounts of shoulder rotation, force and repeated overhead elevation of the arm, while remaining at a competitive and injury-free level.[97,120] Thus, scapular dyskinesis has been implicated as having a cause-and-effect relationship with decreased performance and several shoulder pathologies.[23,121-123]

Kibler et al.[124] have provided an extensive description of several factors of scapular dyskinesis that may prove detrimental to both performance and injury development in the throwing athlete. Kibler et al.[124] described these ‘shoulders at risk’ as having one of three different types of scapular dyskinesis. A type I scapula presents with inferior-medial border prominence, type II have a medial-only scapular border prominence, and type III have superior-medial scapular border prominence.

During scapular internal rotation, the medial scapular border tilts posteriorly off of the thoracic cage, thus the medial component recognized clinically by Burkhart et al.[23] may be an increase in scapular internal rotation. Several clinicians have described the occurrence of increased scapular protraction (internal rotation, anterior translation and anterior tilt) among symptomatic throwing athletes.[51,121] As previously stated, the large forces placed on the posterior shoulder during the deceleration phase of the throwing motion may result in contracture of the posterior soft-tissue structures and a subsequent loss of glenohumeral horizontal adduction (cross body arm movement).[51,112,121] However, the follow-through phase of the throwing motion requires a significant amount of glenohumeral horizontal adduction. Therefore, these athletes may adapt for this loss of cross body motion with an acquired increase in scapular protraction (internal rotation, anterior translation and anterior tilt) in an effort to reach the follow-through position.

The medial border prominence, which may also be present at rest, increases along with a decrease in upward rotation as the arm is pulled back into the late-cocking phase of the throwing motion. This decrease in retraction is thought to restrict the athlete from achieving the fully cocked position.[51] Therefore, the acceleration phase may be altered resulting in a loss of arm acceleration speed and ball velocity. From the point of acceleration through deceleration of the humerus, increases in scapular protraction would tend to pull the scapula in an anterior and inferior direction as the scapula moves around the thoracic cage.[51] Kibler[51] hypothesized that these abnormalities could result in detrimental concavity/compression alterations between the humerus and the glenoid. This alteration in scapular coordination may result in the anterior portion of the glenohumeral joint to ‘open up’, also known as glenoid antetilting (posterior edge of glenoid moves anteriorly),[121] particularly during the late-cocking position, causing an increase in anterior translation of the humerus. This anteversion would place increased stress on the anterior stabilizing structures of the glenohumeral articulation, thereby increasing the risk of pathology.

A decrease in scapular upward rotation among throwing athletes may also result in subsequent shoulder pathology, such as subacromial impingement. If scapular upward rotation during the late cocking and acceleration phase of a throw is decreased, the rotator cuff cannot pass freely underneath the lateral acromion of the scapula.[51] Similarly, if the inferior angle of the scapula is prominent during clinical evaluation, due to increased anterior scapular tilt, the anterior acromion may fail to adequately elevate and the humerus may intrude into the subacromial space. These decreases in acromial elevation may allow the humerus to encroach into the subacromial space and potentially lead to soft-tissue damage.[123] In overhead athletes, weakness and/or fatigue of periscapular musculature during repetitive overhead movements may make such athletes particularly susceptible to scapular dyskinesis and the ensuing pathology.[118] Clinically, pitchers experiencing fatigue or pain due to subacromial compression from lack of acromial elevation may present with a dropped elbow during throwing in an attempt to increase the subacromial space and alleviate pain and compression.

6.2 Evidence

Proper scapular kinematics requires good periscapular strength and neuromuscular coordination. Ludewig and Cook[123] reported subacromial impingement subjects to have decreased serratus anterior activity and subsequent lost upward scapular rotation during a humeral elevation task compared with a control group. Laudner et al.[118] reported a moderate to good correlation between decreased lower trapezius strength and decreased scapular upward rotation among a group of baseball players. Ebaugh et al.[46] reported that upper and lower trapezius and serratus anterior muscles play a critical role in the development of scapular upward rotation especially during the mid-ranges of arm elevation. Unfortunately, it remains unclear whether the decreased strength and neuromuscular coordination cause the lost upward rotation or does this scapular dyskinesis decrease the stable base of support of the attaching muscles, thereby decreasing the muscles efficiency to produce force.

Any loss in upward rotation may predispose individuals to the development of shoulder pathology. Ludewig and Cook[123] reported subacromial impingement subjects to have decreased upward scapular rotation during a humeral elevation task compared with a control group. Using a Moire topography technique, Warner et al.[125] reported subacromial impingement patients to have an increase in medial scapular border prominence and also stated that 54% of these patients had scapular dyskinesis. These findings have also been clinically observed in subacromial impingement and rotator cuff lesion patients.[121] Tyler et al.[110] noted lost glenohumeral horizontal adduction attributed to posterior shoulder contracture among subacromial impingement patients. This finding was supported by the clinical finding and theory of Burkhart et al.[121] that posterior shoulder contractures increase scapular protraction as an adaptive response to the loss of glenohumeral horizontal adduction, resulting in increased medial border prominence, which was identified among throwing athletes diagnosed with subacromial impingement. However, this increase in scapular protraction was not found among subjects diagnosed with internal impingement[122] and subsequent posterior shoulder tightness,[9] adding to the confusion regarding this relationship. Despite these conflicting results, increased scapular protraction (anterior tilting, anterior translation and internal rotation) would tend to position the acromion (anterior and inferior) in a closer proximity to the humeral head, thereby decreasing the subacromial space and increasing the risk of impingement of the associated soft-tissue structures.[51] Increased anterior scapular tilt, a component of protraction, also places overhead athletes in a predisposition to the development of pathology. This dyskinesis would result in an increased prominence of the inferior scapular angle and has been confirmed among subacromial impingement patients during both laboratory testing[123] and clinical observations.[121]

Supplementary causes of scapular dyskinesis and the occurrence of subacromial impingement include several posture abnormalities. Forwarded head has been associated with increased anterior scapular tilt and decreased scapular upward rotation among such patients.[126] This cervical lordosis is believed to increase the stretch on the levator scapulae, thus tending to not only pull the scapula into a more anteriorly tilted position, but also in resisting upward rotation of the scapula.[126] Similarly, excessive thoracic kyphosis has been reported to increase anterior scapular tilt.[109,127] Increased scapular protraction can be observed among patients with rounded or forward shoulders.[128] From a lateral view and with a plum line extending from the ear lobe, the acromion process of these individuals is positioned anteriorly to the line.[128] These postural abnormalities present with various scapular position and orientation patterns that ultimately place the overhead athlete into a vulnerable position for development of subacromial impingement.

Although scapular dyskinesis plays a large role in subacromial impingement symptoms, other pathologies have also shown a strong association with these scapular alterations. Burkhart et al.[121] clinically observed medial-inferior scapular border prominence among throwers with SLAP lesions in their dominant shoulders. Thus, the medial-inferior component recognized clinically may be an increase in scapular protraction, which has been described as a combination of internal rotation (medial prominence) and anterior tilt (inferior prominence).[129] Using a 3-D tracking device, Laudner et al.[122] identified increased scapular posterior tilt and elevation among a group of baseball players diagnosed with pathological internal impingement compared with a group of baseball players without a history of shoulder pathology. Laudner et al.[122] hypothesized that the increased scapular elevation may be an adaptation by the throwing athlete experiencing pathological internal impingement to avoid contact of the humeral head with the superior portion of the glenoid labrum. The increased scapular posterior tilt among this pathological group was stated to support previous clinical observations by Burkhart et al.,[121] that throwers with impingement and rotator cuff lesions present with increased superior scapular prominence. Warner et al.[125] reported that 32% of shoulder instability patients presented with scapular dyskinesis.

6.3 Summary

Proper scapular kinematics is critical in the function of periscapular musculature as well as the congruency between the glenoid fossa and the humeral head. Even the slightest discrepancy in scapular motion can prove detrimental for athletes who repetitively elevate their arms overhead, leading to chronic damage of the supporting soft tissue and bone. Whether scapular dyskinesis is the cause or the result of the pathology remains unclear. Regardless, periscapular strengthening and stretching of the surrounding soft tissue should be addressed as both a preventative measure and during the rehabilitation of the aforementioned pathologies.

7. Clinical Implications: Where Does the Evidence Lead Us?

There is substantive evidence that clearly shows altered mobility patterns in the shoulder(s) of overhead-throwing athletes. It has been debated whether these altered mobility patterns arise from soft-tissue or osseous adaptive structural changes. From a skeletal perspective, there is compelling radiographic evidence showing that throwing shoulders develop humeral retroversion. Humeral retroversion is thought to develop over time in young pre-adolescent throwers when the proximal humeral epiphysis is not yet completely fused.[2,10,11] Early reports indicate that the retroversion is a normal adaptive remodelling response of the osseous tissue to throwing and is, therefore, not pathological in these young athletes.[2,10] Researchers have speculated that this active remodelling may be a source of the soreness in the shoulders of adolescent throwers.[10]

Objective data have consistently shown anterior laxity and rotational ROM to be symmetrical between sides in nonpathological shoulders even though the throwing shoulder undergoes a gain in external rotation (ERG) and a symmetrical loss of internal rotation (GIRD). Throwers also show a loss of horizontal or cross-body adduction in the throwing shoulder when compared with the nonthrowing shoulder. This posterior shoulder immobility in the throwing shoulder is thought by some researchers to be associated with reactive scarring or contracture of the periscapular soft-tissue structures (e.g. posterior capsule and/or cuff musculature); however, evidence of reactive scarring or contractures of the posterior-inferior capsule or cuff musculature from anatomical or noninvasive imaging studies is lacking. In most throwing shoulders, this immobility does not appear to be problematic, although empirical data suggest that excessive posterior immobility (30% GIRD) is linked to the development of shoulder pathology.[9,24]

There is limited evidence that supports the daily use of posterior shoulder stretching as a means to limit the development of posterior shoulder immobility and reduce the incidence of shoulder injury. Kibler et al.[13] compared shoulder range of motion and injury incidence between tennis players who performed daily posterior shoulder stretches versus a group of tennis players who did not participate in the stretching programme (control group). Kibler et al.[13] found that the stretching group had significantly more shoulder ROM and less incidence of shoulder injury than the control group. Therefore, given the prevalence of posterior shoulder immobility in throwers it would be prudent to advocate posterior shoulder stretching in these athletes as a means to prevent reactive scarring, limited mobility (e.g. GIRD) and soft-tissue pathology.[9,24]

Even though the evidence is inconclusive at the present time, there is more compelling evidence that leads us to believe that altered shoulder mobility in the overhead-throwing athlete is more strongly associated with adaptive changes in proximal humeral anatomy (i.e. retroversion) than to structural changes in the articular and periarticular soft-tissue structures. In addition, this retroversion is thought to account for the observed shift in the arc of rotational ROM in overhead athletes. However, in some athletes, capsulo-ligamentous adaptations such as anterior-inferior stretching or posterior-inferior contracture may become superimposed upon the osseous changes. This may ultimately lead to pathological manifestations such as secondary impingement, type II SLAP lesions and/or internal (glenoid) impingement.

8. Conclusions

Researchers have used an array of quantitative techniques in an attempt to better characterize altered mobility patterns in the shoulders of overhead athletes. Throwing athletes have been shown to display altered rotational ROM patterns in the dominant shoulder that favour increased external rotation and limited internal rotation ROM. Throwers also show a loss of horizontal or cross-body adduction in the throwing shoulder when compared with the non-throwing shoulder. This posterior shoulder immobility in the throwing shoulder is thought by some researchers to be associated with reactive scarring or contracture of the periscapular soft-tissue structures (e.g. posterior capsule and/or cuff musculature); however, evidence of reactive scarring or contractures of the posterior-inferior capsule or cuff musculature from anatomic or noninvasive imaging studies is lacking. Conversely, translational ROM (laxity) has been consistently shown to be symmetric between dominant and non-dominant shoulders of overhead athletes. This finding of bilateral symmetry in laxity suggests that the capsular restraints are intact and not ‘stretched-out’ as the acquired hyperlaxity theory indicates.

From a skeletal perspective, throwing shoulders are shown to develop more humeral retroversion when compared with the non-throwing shoulder. Even though the evidence is inconclusive at the present time, there is more compelling evidence that leads us to believe that altered shoulder mobility in the overhead-throwing athlete is more strongly associated with adaptive changes in proximal humeral anatomy (i.e. retroversion) than to structural changes in the articular and periarticular soft-tissue structures. In addition, this retroversion is thought to account for the observed shift in the arc of rotational ROM in overhead athletes. However, in some athletes, capsulo-ligamentous adaptations such as anterior-inferior stretching or posterior-inferior contracture may become superimposed upon the osseous changes. This may ultimately lead to pathological manifestations such as secondary impingement, type II SLAP lesions, and/or internal (glenoid) impingement. Future research should be directed at identifying the source(s) of these altered mobility patterns, especially posterior shoulder immobility. This will aid in the development of therapeutic strategies designed to prevent or ameliorate those structural and functional adaptations that may negatively affect shoulder health.

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