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Athletic Shoulder I

-Pathophysiology of Throwing-

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Although overuse of the throwing shoulder can contribute significantly to injury, many difficulties begin with improper mechanics and poor conditioning. Common problems in pitching mechanics that can lead to injury begin with the foot plant. Hyperextension of the knee while planting the striding leg and landing on the heel cause a sudden deceleration of the body, which results in undue counterforce on the throwing arm.

The planted foot should always point toward home plate. Placing the striding foot outside the target (toward the first-base side for a right-handed pitcher) and wide to the torso results in opening up too soon . In this instance, pelvic rotation occurs too early, creating increased stress across the anterior shoulder and elbow. Planting the foot toward the third-base of home plate slows down rotation of the torso, taking from the body's momentum and forcing the throw to be delivered entirely by the arm.

The throwing motion should be a smooth acceleration and deceleration of the center of gravity toward the target. This fluid motion should be maintained regardless of the type and velocity of pitch being thrown.

Classification of Injury

Injury from throwing can occur to any of a number of structures contributing to the dynamic or static restraint of the shoulder. Isolated injury to the rotator cuff, labrum, and capsule can occur, but far more frequently, injury to one structure is secondary to the breakdown of another.

Classification of Shoulder Instability

I. Primary disease (overuse syndromes)
Primary tendinitis (rotator cuff/biceps)
Tensile rotator cuff failure
SLAP lesion
Subacromial impingement
Bennett's lesion
II. Primary instability
A. Secondary to repetitive microtrauma
Secondary impingement
Labral tears
Fraying
SLAP lesion
B. Secondary to generalized ligamentous laxity
III. Acute traumatic instability
IV. Posterosuperior glenoid impingement

I. Primary disease.

Primary disease refers to injury that occurs in the throwing shoulder that can be attributed to normal, but excessive, forces and extreme motion seen in all throwers. The stresses across the joint during the throwing motion in the stable shoulder are great enough to result in damage to the static and dynamic restraints even without significant underlying glenohumeral instability. In some athletes with articular or periarticular injury, assessment of glenohumeral laxity may reveal only minimal-to-no asymmetric laxity.

Rotator cuff / Biceps Superolabral Complex

As the supraspinatus, infraspinatus, and teres minor muscles fire in the late cocking phase of a throw, rotator cuff muscle function becomes significant as the superior and posterior cuff muscles move the shoulder to the point of maximum external rotation. Those same muscles fire violently during the deceleration phase, as significant posterior shear and compressive loads are recorded across the joint.

The area of insertion of the supraspinatus muscle has been shown to be watershed area of

diminished blood flow that is particularly susceptible to repetitive overload stresses. The normal processes of aging have also been shown to be a major contributing factor to the failure of the rotator cuff. The repetitive stresses of throwing may speed up normal degeneration. Therefore, repetitive stressful loading of the rotator cuff as the cuff muscles attempt to resist distraction, horizontal adduction, and internal rotation of the shoulder during arm deceleration can result secondary to fatigue in an acute inflammatory response in the early stages and in tendon failure in the late stages.

During the late cocking phase, biceps muscle firing is moderate. However, during deceleration, biceps contraction is particularly strong as it contracts to both decelerate elbow extension and act with the rotator cuff to resist glenohumeral distraction. A study that evaluated the role of the biceps muscle and superior labrum in anterior instability of the shoulder suggests that the biceps muscle is essential to limiting torsional forces to the shoulder in the abducted, external rotated position.

In the position of extreme external rotation, the biceps muscle may have two functions. It is primarily an internal rotator to the humerus. Its secondary function is to resist distraction and compress the humeral head against the glenoid. Biomechanical studies showed that anterosuperior and posterosuperior labral strain are greatest in shoulder abduction. Biceps tendon strain is greatest in shoulder adduction.

Biceps muscles load may increase as a results of excessive throwing and poor mechanics. With proper pitching mechanics, maximum elbow flexion torque occurs before maximum shoulder compressive force. With improper mechanics, these two loads may occur closer together in time, requiring greater maximum force by the biceps muscles. Loss of the biceps muscle anchor with complete avulsion of the superolabral complex with the arm in the cocked position may reduce torsional rigidity as much as 38%. As a result, strain in the inferior glenohumeral complex may increase as much as 100%. Therefore, initial failure of the biceps-superolabral complex may contribute to late failure of the anterior glenohumeral ligaments.

Andrews et al. have introduced the concept of the grinding factor as a potential cause of labral damage in the stable throwing shoulder. This factor results from translation of the humeral head during arm acceleration and deceleration. Humeral head displacement combined with compression and internal rotation during deceleration cause the humeral head to grind on the labrum. Tear at the base of the biceps tendon as well as at the anterosuperior portion of the labrum are commonly seen.

Subacromial impingement may be a factor contributing to primary disease of the rotator cuff and biceps tendon. The shoulder is repeatedly positioned at 100 of abduction and, with every throw, moves from external horizontal abduction to a position of horizontal adduction and internal rotation. During the arm deceleration phase, a large inferior force and adduction torque is produced. With weakness in the rotator cuff muscles, fatigue, or improper mechanics, an inability to generate needed forces can lead to superior migration of the humeral head and subacromial impingement. Additionally, loss of internal rotation that can occur over an athlete's career, partly from contracture of the posterior capsule, has been shown to result in anterior and superior migration of the humeral head. Superior migration of the humerus causes impingement of the greater tuberosity, rotator cuff muscles, or biceps muscle against the inferior surface of the acromion or coracoacromial ligament.

(2) Bennett's lesion

In 1941, Bennett was the first to describe the presence of a bony exostosis on the posteroinferior border of the glenoid fossa. Bennett thought that this was a symptomatic lesion that caused irritation of the capsule and the synovial membrane and, at times, irritation of the axillary nerve, referring pain to the deltoid muscle. Most recently, it has been accepted that the exostosis probably occurs from traction of the posterior band of the inferior glenohumeral ligament during deceleration. In the symptomatic shoulder, the exostosis is often associated with tearing of the posterior labrum and posterior rotator cuff. However, a large enough isolated lesion referred to the deltoid muscle secondary to entrapment of the axillary nerve.

II. Primary Instability

Secondary to Microtrauma

The late cocking and early acceleration phase of the throwing cycle place significantly shear across the anterior aspect of the shoulder during normal throwing. Rotation of the torso after foot plant has been reported to generate an anterior shear at 400 N. Over time, secondary to poor mechanics (opening up in late cocking), overthrowing, or weakness the anterior capsule sees increasing loads unshielded by equal contributions of the surrounding musculature. The anterior capsule then fatigues and fails, resulting in increased anterior laxity of the glenohumeral joint.

Failure of the anterior capsule leads to increased anterior translation in the most stressful

phase of the throwing cycle. Manifestations of this increased laxity include secondary rotator cuff tendinitis, or subacromial impingement, anterior labral fraying from increased translation in deceleration, SLAP lesions, and posterior glenohumeral impingement. Distinguishing lesions that occur secondary to instability from those that result from primary forces on the shoulder during throwing is paramount in the ultimate treatment of this group, as correction of the observed lesion without correction of the instability will eventually lead to recurrence.

Secondary to generalized ligamentous laxity

Persons with this type of instability exhibit signs of generalized ligamentous laxity. These athletes exhibit bilateral, symmetric increase in shoulder laxity. The microtrauma of throwing, couples with a baseline level of increased symmetric laxity, puts this athlete at risk for developing damage to the intra- and periarticular structures of the shoulder. As a result of their increased baseline level of laxity, many of these athletes will throw with the most velocity and ball movement but will have the greatest risk of long-term injury.

Arthroscopic examination of the glenohumeral joint of athletes with this type of instability reveals a hypoplastic glenoid labrum and an increased joint volume. Abnormalities of the labral complex and rotator cuff are often seen.

Acute traumatic instability

This cause of instability is seen least in the overhead throwing athlete. A specific acute traumatic event leading to unidirectional pattern of instability is characteristic. Complaint of pain while throwing may be present, but instability is often the primary complaint. An athlete who attempts to continue to throw after this injury and does not develop overt primary instability may often manifest difficulties secondary damage to the structures, the rotator cuff and the superior and posterior labral complexes.

Posterosuperior glenohumeral impingement

In overhead throwing sports, repeated extreme movements of glenohumeral abduction and external rotation result in contact of the superior and posterior glenoid rim with the supra- and infraspinatus muscles and posterior humeral head. The lower loads of throwing do not result in fracture but can produce undersurface tearing of the rotator cuff, tearing of posterior or superior labrum, and changes on the posterior humeral head (expanded bare area and cyst

formation).

In normal stable shoulder, contact of the tuberosity with glenoid is possible. There may be factors that contribute to a more pathologically significant contact in the athlete in overhand throwing sports. Increased impingement may result from increased anterior capsular laxity. A loss of normal posterior translation in the late cocking and early acceleration phase of throwing may result in impingement of the undersurface of the rotator cuff rather than the posterior humeral head. Repetition of this contact may be responsible for tearing of the undersurface of rotator cuff and posterosuperior glenoid labrum. Increased capsular laxity, resulting in increased anterior translation as well as increased external rotation can further increase the degree of internal contact. As the arm horizontally adducts and internally rotates during acceleration and deceleration, further grinding and contact of the greater tuberosity may be responsible for significant tears of superior labral complexes.

Some authors have suggested that differences in humeral version may be responsible for more refractory cases of internal impingement. Decreased humeral retroversion in the dominant shoulder in maximum external rotation may results in increased contact of the posterior humeral structures with the glenoid.

The thrower with internal impingement will most often complain of pain in the posterosuperior aspect of the shoulder in the late cocking and early acceleration phase of throwing. An inability to fully rotate the shoulder secondary to posterior pain will cause a loss of velocity. An early ball release results in loss of control.

Scapulothoracic joint

Scapula provides for a stable base in the glenohumeral articulation, retraction and protraction of the shoulder complex along the scapular wall, elevation of the acromion, a base for muscle attachment, and a link to the transfer of forces from the trunk to the arm in the normal throwing motion.

The appropriate force couples for scapular stabilization include the upper and lower portions of the trapezius muscle and rhomboid muscles, paired with the serratus anterior muscle. The appropriate force couples for acromial elevation are the lower trapezius and serratus muscles paired with the upper trapezius and rhomboid muscles. Balanced function of the muscle groups is essential to the efficient function during the kinetic chain of events.

Injury/Dysfunction of scapula

The most common causes of dysfunction are direct trauma to the scapular musculature and indirect injury from repetitive microtrauma. The serratus anterior and lower trapezius muscles are the most sensitive to this inhibitory effect. Their dysfunction becomes evident early in abnormalities of the glenohumeral joint.

Less commonly, nerve injury, specifically to the long thoracic nerve or spinal accessory nerve, can result in scapular dysfunction.

Loss of glenohumeral motion can result in scapular dysfunction. Particularly in the throwing athlete, tight posterior capsule and musculature leads to increased protraction of the scapular in cocking and follow-through phases. The increased protraction results in more anterior and inferior movement of the scapula, closing down the subacromial arch, which leads to decreased clearance of the rotator cuff and increased impingement.

Lack of full scapular retraction causes loss of a stable cocking point, dissipating the flow of energy from the torso and trunk to the arm. A 20% decrease in energy delivered from the trunk to the arm necessitates a 34% increase in rotational velocity at the shoulder to deliver the same amount of resultant force. Loss of coordinated retraction/protraction also results in relative glenoid anteversion, which leads to the loss of the normal bony buttress to resist anterior translation of the humeral head. With relative loss of this bony buttress, increased shear is felt across the anterior soft tissue structures and leads to injury.

Lacks of appropriate acromial elevation in the cocking and follow-through phases can result in impingement problems. Inhibition or fatigue of the lower trapezius and serratus anterior muscles can lead to relative closure of the coracoacromial arch. Relative loss of the arch space can arch space can result in primary impingement or contribute to the problems of secondary impingement in cases of concomitant instability.