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Prevention of the Most Common Shoulder Injuries in the Overhead Athlete

Introduction:

Overhead (OH) athletes are at high risk of experiencing shoulder injuries, particularly in throwing or hitting activities in sports such as baseball, tennis, volleyball and handball.[1] The shoulder faces high loads and forces at extremely high angular velocities during serving, smashing and pitching, increasing risk of injury.[1,2] The incidence of shoulder injury in OH sport is identified to be between 0.2/1000 hours and 1.8/1000 hours.[3] Internal impingement is identified as the most common cause of shoulder pain in the OH throwing athlete.[4] Additionally, posterosuperior rotator cuff (RC) tears represent one of the hallmarks of internal impingement.[4] Thus, these two pathologies will be the focus of this paper. Such injuries can have a significant impact on the athlete's playing career and can limit their ability to participate in competitive sports. Due to the high prevalence and burden associated with these injuries, injury prevention is a vital component of the OH athlete's training program.[3] Physical therapists (PTs) play a vital role in the rehabilitation and prevention of shoulder injuries. It is imperative that PTs have a good understanding of the anatomy of the shoulder complex, the biomechanics of the OH pitching and serving motion, pathomechanics and risk factors associated with shoulder pathologies, as well as diagnostic screening tools, evidence-based interventions and prevention strategies that address internal impingement and RC tears, in order to prevent shoulder injuries and re-injuries and enhance performance for successful results during the athlete's season.

Anatomy and Biomechanics:

The shoulder joint is also known as the glenohumeral (GH) joint and is a multiaxial, ball and socket, synovial joint with the most extensive range of motion (ROM) in the human body.[5,6] The GH joint is the major articulation of the shoulder girdle and involves the humeral head with the glenoid cavity of the scapula.[5] In the anatomic position, the humeral head faces medially, slightly posteriorly and superiorly, while the glenoid cavity faces laterally, anteriorly and superiorly.[5] The glenoid labrum, which is the ring of fibrocartilage, serves to deepen the glenoid cavity about 50% to partially compensate for this incongruity and provide passive support.[5] The GH joint capsule is thin and lax to allow for full ROM.[6] In addition, the GH capsule is reinforced by the coracohumeral and superior GH ligament that limit inferior translation during the first 30° of abduction. The middle GH ligament also provides reinforcement as it is the primary restraint to anterior translation in 45-75° of abduction and limits external rotation (ER) between 45-90° of abduction.[6] The inferior GH ligament consists of an anterior and posterior band, and an axillary pouch in between that acts like a 'sling.'[6] The anterior band tightens during ER and the posterior band tightens during internal rotation (IR), forming a barrier to anterior and posterior dislocation, respectively.[6]

In addition, the GH joint gains active support from musculature, with the two main dynamic GH stabilizers being the deltoid and the RC muscles.[6] The RC muscles function as humeral rotators and depressors and assist with shoulder elevation while creating a force couple by compressing and stiffening the GH joint to counterbalance the deltoid's superior shear force and prevent impingement. [6,7] The supraspinatus is the primary abductor of the humerus, the infraspinatus and teres minor are the primary external rotators of the GH joint and the subscapularis' is the primary internal rotator of the GH joint.[6,7] An injury to any of the RC muscles can affect GH joint stability and disrupt normal shoulder kinematics.[7] Moreover, the static and dynamic stabilizing structures allow for extreme degrees of motion in multiple planes of the body that predisposes the GH joint to instability events.[7] Figure 1 provides a graphical representation of the structures associated with the GH joint.[5]

Regarding vasculature, the posterior humeral circumflex and suprascapular arteries provide blood supply to the infraspinatus and teres minor, the anterior humeral circumflex artery provides blood supply to the subscapularis and the thoracoacromial artery, that anastomoses with two circumflex arteries, provide blood supply to the supraspinatus.[5] It is important to note that the supraspinatus and infraspinatus are hypovascular, making those sites more vulnerable to lesions.[5]

The three other articulations of the shoulder complex are the sternoclavicular (SC), acromioclavicular (AC) and scapulothoracic joints.[6] It is important that the PT has a basic understanding of all these joints as movements of these joints is required to achieve full shoulder ROM.[5] The AC joint attaches the acromion process of the scapula to the lateral end of the clavicle, and functions to transmit and distribute forces from the shoulder to the axial structures, permit rotation of the scapula and clavicle to achieve full shoulder ROM and prevent superior dislocation of the clavicle on the acromion.[6] Additionally, it is supported by the superior and inferior AC ligaments that restrain anteroposterior translation of the clavicle and the coracoclavicular ligaments, the conoid and trapezoid, that restrain superior and posterior displacement of the clavicle.[6] The SC joint attaches the appendicular skeleton to the axial skeleton, and functions to stabilize the closed kinetic chain of the shoulder complex and absorb and distribute forces.[6] Like the AC joint, it relies on ligaments for its strength, including the anterior and posterior SC ligaments, the interclavicular ligament and the costoclavicular ligament, which is the main ligament that maintains the integrity of the SC joint.[6] The scapulothoracic joint is considered a functional joint rather than a true anatomical joint since the thorax and the scapula are connected via musculature with no bony articulation.[6] The scapulothoracic joint functions to add ROM for the arm and acts as a base of support for the humerus.[6] It is important that PTs assess the scapulothoracic joint during evaluation as a stable scapula is required for the shoulder to function correctly and to minimize risk of shoulder injury.[6]

The pitching motion in baseball consists of six phases (Fig 2).[8] The throwing motion is initiated via the wind-up phase, which is from initial movement to maximum knee lift of the stride leg.[8] The goal of this phase is to position the body to optimally generate the forces and power required to achieve high velocity.[9] The pitcher keeps his center of gravity (COG) over his back leg, in order to have maximum momentum when forward motion is initiated.[8,9] If the pitcher's COG and momentum fall forward prematurely there will be a disruption in the kinetic chain, which will result in increased shoulder force to propel the ball at high velocity.[9] During this phase the RC muscles have their lowest activity, thus shoulder forces and torques are also low and injuries are very rare.[8] The second phase is the stride phase, which begins as the leg reaches maximum height and ends at stride foot contact.[9] The goal of the stride leg is to increase the distance over which angular/linear trunk acceleration occurs to allow for increased energy production and transfer to the upper extremity.[9] The supraspinatus has its highest activity during this phase since it works to abduct the shoulder, as well as compress and stabilize the GH joint.[8] Evidence suggests that optimal stride length should be approximately 75-90% of the pitcher's body height and the lead foot should land in line with the stance foot pointing toward home plate, in slight IR, in order to achieve high velocities.[10] The third phase, which is the cocking phase begins at lead foot contact and ends at maximum shoulder ER.[8] After lead foot contact a peak pelvis angular velocity, followed by a peak upper torso angular velocity is generated that causes the arm to lag behind the upper torso and forces the throwing shoulder into horizontal abduction.[11] This results in high tensile stress within the anterior shoulder structures, compression of the posterior RC and labrum and shoulder ER that reaches as high as 170-190° –

far beyond normal ranges (Fig 3).[4,8,11] The RC muscles provide a compressive force of 550-770 N to resist GH distraction and increase GH stability.[9] Phase four is known as the arm acceleration phase that begins at maximum shoulder ER and ends at ball release.[8] The trunk moves into flexion, the scapula protracts and the humerus undergoes horizontal adduction and vigorous IR.[8] Specifically, the GH internal rotators have their highest activity during this phase and generate rapid IR, with a ROM of ~80° from maximum ER to ball release, in only 30-50 msec.[8] Arm deceleration is the fifth phase that begins at ball release and ends at maximum shoulder IR. [8] The goal of this phase is to dissipate the excess kinetic energy and reduce the risk of shoulder injury.[8] The greatest amount of joint loading is generated at the shoulder during this phase including excessive posterior (400 N) and inferior shear forces (300 N), elevated compressive forces (>1000 N) and adduction torques.[9] The posterior shoulder musculature contract eccentrically to decelerate horizonal adduction and IR, and aid in resisting shoulder distraction and anterior subluxation forces. [8] Additionally, the teres minor, which is commonly the site of tenderness in pitchers, exhibits its maximum activity during this phase. The last phase of the pitching cycle is known as the follow-through phase where the body continues to move forward as the RC muscles decelerate the shoulder until motion has ceased.[8] This phase consists of decreased joint loading and minimal forces, thus injuries during this phase are rare.[9]

The tennis serve biomechanics are similar to the baseball pitching biomechanics described above, however, there are some significant differences that the PT should be aware of. These include planes of motion, the nondominant arm tossing the tennis ball, the altered lever arm secondary to the tennis racket, the trajectory of forces produced and released, the technical components of the serve and the different types of serve (spin, speed etc.)[12] The tennis serve can be examined via an 8-stage model that consists of 3 distinct phases: preparation, acceleration and follow-through phase.[12] A more detailed description and graphical representation of each phase and stage is provided in figure 4.[12]

Pathomechanics and Risk Factors:

Internal or posterosuperior impingement is characterized by repetitive or excessive contact of the undersurface of the RC between the greater tuberosity of the humeral head and the posterosuperior aspect of the glenoid labrum in the abduction and maximum external rotation (ABER) position.[4] This occurs during the transition from late cocking and early acceleration, where the greatest forces and angular velocities on the shoulder occur secondary to the sudden transition from ER to IR, resulting in pathologic impingement of the RC tendons (supraspinatus/infraspinatus).[4] Figure 5 provides a graphical representation of this mechanism. Additionally, during the arm cocking phase horizontal abduction and anterior force peak result in compression/impingement of the RC and labrum that can lead to pathologic posterior impingement.[11]

During the stride phase, an open foot position will cause the pelvis to rotate prematurely whereas an excessive closed foot position can limit pelvis rotation.[10,13] Specifically, the stride foot should be in a closed position at an angle approximately 15° away from the center of the mound, in order to minimize the anterior force placed on the shoulder during the cocking phase and decrease risk of injury (Fig 6). [10,13,14] Early increased shoulder ER during this phase (average shoulder rotation being 53° ± 26° at the point of stride foot contact) can also increase forces at the shoulder and elbow, specifically increasing the anterior shoulder force during the cocking phase.[10] During the cocking phase, an increase of shoulder horizontal abduction range (average being 23° ± 12°) has been associated with higher velocity pitching, as well as shoulder injury.[10]

Additionally, early onset of trunk rotation has been found to increase internal rotation torques on the shoulder, which can also increase risk of injury.[10]

Furthermore, OH athletes perform repetitive high stress, high velocity throwing actions that can result to specific osseous and soft tissue adaptations.[15] Adaptive changes that can lead to internal impingement include anterior GH instability caused by repetitive stretching of the anterior GH capsule, which allows for increased anterior humeral head translation.[16] In addition, tightness of the posterior GH capsule can lead to glenohumeral internal rotation deficit (GIRD), which may compromise the 'sling' function of the inferior GH ligament, increasing risk of impingement.[17] Burkhart et al., defines GIRD as a loss of greater than 20° of IR in the throwing shoulder relative to the nonthrowing shoulder.[17] Nonetheless, a recent study by Wilk et al., proposed that ER insufficiency (<5° difference in ER between throwing and nonthrowing arms) rather than GIRD is associated with shoulder injury, therefore this is also something PTs should consider during the athlete's evaluation.[1] The literature suggests that the prevailing theory regarding the etiology of internal impingement is shoulder muscle fatigue from excessive throwing or deconditioning.[4] This ultimately results in hyperangulation in the alignment of the humerus and scapular during acceleration, where the humeral head translates anteriorly increasing tensile stress and contact of the RC undersurface with the glenoid border.[4] Fatigue or muscle weakness, specifically of the scapular retractors, decreases the force production of the RC muscles, which also causes abnormal positioning of the GH joint.[15,16] Moreover, lack of neuromuscular control and muscle imbalances also contributes to scapular dyskinesis, which increases risk of internal impingement and RC tear.[15]

Posterosuperior RC tears are the most common tears in the OH athlete and are referred to as "tears of necessity" as they typically occur to accommodate the maximal ER during the OH motion.[4] In the OH athlete, these tears commonly result from chronic repetitive trauma, specifically from the eccentric contraction during the deceleration phase of the throwing motion, which is further supported by a correlation between innings pitched and incidence of RC tears.[4,7] Nevertheless, an acute tensile overload is also a potential etiology.[4] Contributing factors include GIRD and internal impingement.[4] Rotator cuff tears can be classified as either partial-thickness of full-thickness disruptions.[7] Figure 7 represents an MR arthrogram of a partial thickness tear of the posterior supraspinatus tendon, which is a frequently seen in the OH athlete.[4] Moreover, evidence suggests that injury risk increases with age, as well as level and volume of play, placing the elite adult athlete who trains and competes at high levels at a higher risk.[2,3]

Diagnostic Screening Tools:

Internal impingement has a similar presentation to numerous shoulder pathologies, including RC tears, capsular pathologies, subacromial impingement, superior labrum anterior to posterior (SLAP) lesion, biceps tendon lesion, scapular dysfunction and GH instability, which it is most frequently confused with.[5] A thorough history is a key component to the identification of internal impingement, however, it cannot be used alone for diagnosis as symptoms tend to vary from patient to patient.[16] Thus, a thorough and complete examination of the shoulder complex is vital for accurate diagnosis. Moreover, it is important that other shoulder pathologies are ruled out before initiating the rehabilitation program to ensure safe and optimal outcomes. Common signs and symptoms of internal impingement in the OH athlete include: diffuse posterior shoulder pain particularly during the late cocking phase; decrease in throwing velocity; increase in anterior laxity or anterior instability, with decreased dorsal GH glide and posterior capsule tightness; muscle asymmetry between the dominant and non-dominant shoulder; abnormal scapulothoracic rhythm; decreased GH IR and increased GH ER (10-15°); RC weakness, as well as weakness of the middle/lower trapezius, rhomboids and serratus anterior muscles.[18] It is important to note that there are no cluster of tests that help identify internal impingement and tests for other shoulder pathologies may be positive or negative due to the variable clinical presentation of internal impingement.[18] The Posterior Internal Impingement test has been shown to be very accurate in ruling out posterior RC tears.[5,16] A positive test is demonstrated by deep posterior shoulder pain with the arm in 90-110° of abduction and 10-15° of extension and maximal ER.[5,16] Another useful test used to identify internal impingement is the Relocation test where a positive test is demonstrated with posterior shoulder pain that is relieved with a posterior directed force on the proximal humerus.[18]

Clinical presentation of RC injuries varies by type and mechanism of injury, which is why a thorough history that includes arm position at the time of injury, in addition to a physical examination is vital for an accurate diagnosis of a RC injury.[7] Common signs and symptoms of OH athletes with RC pathology include: GIRD; pain with OH activity; RC muscles weakness; pain at night; tenderness over the greater tuberosity; loss of shoulder active ROM with passive ROM preserved; positive painful arc; shoulder stiffness.[7] Special tests specific for supraspinatus and infraspinatus RC tears include: ER lag sign, Drop arm test and Empty-Can test.[19] A prospective study by Bak et al., identified a cluster of 3 tests to diagnose RC tears that includes: Empty-Can test, ER lag sign and Active abduction less than 90°.[20] Nonetheless, this cluster has low sensitivity and specificity (54 and 65, respectively), which emphasizes the importance of a through and complete assessment of the athlete.[20] Furthermore, the cervical spine and elbow should be ruled out as a possible referral source for the shoulder pain.

Magnetic resonance imaging (MRI) is the gold standard for assessing the integrity of the RC tendon and the musculature.[7] MRI is not useful in providing diagnostic information for internal impingement, however, it aids in assessing complications associated with internal impingement, such as a RC tear or labral injury.[15]Additionally, MRI's of patients with internal impingement commonly include mature periosteal bone formation at the posterior scapular attachment and posterior capsular contracture at the site of the posterior band of the inferior GH ligament.[18,21]

Prevention Program:

Three key risk factors have been identified that form the basis for recommendations for the prevention of injury/re-injury and return to play following injury. These include: GIRD, RC strength deficits and imbalance, specifically weakness in the external rotators, and scapular dyskinesis, including position and movement.[2] The cut-off values for IR ROM vary in the literature, ranging from 18-25°.[2] Thus, side-to-side difference in IR ROM should be less than 18° and the difference in total range of motion (IR and ER) should be no more than 5° to assure maximal protection of the athlete.[1,2] Additionally, athletes with increased humeral retroversion are more susceptible to ROM deficits and shoulder injuries, thus closer monitoring of these athletes is warranted.[2] Goniometric measures of shoulder IR and ER should be performed with the patient in supine, the shoulder abducted 90° and the elbow flexed 90°.[2] Evidence suggests that posterior shoulder tightness has a big influence on shoulder kinematics, thus improving posterior shoulder flexibility is advised as a preventive intervention.[2] This can successfully be achieved via the cross-body stretch and/or sleeper stretch (Fig 8) when performed daily for 3 repetitions of 30 sec.[2] Shoulder joint mobilizations, including dorsal and caudal humeral glides performed by a PT also lead to similar results.[2] Evidence suggests that both techniques demonstrate a statistically significant increase in IR ROM in OH athletes that aids in preventing injury and decreasing shoulder pain, with no difference in mobility or shoulder functional outcomes identified between the two interventions.[22]

OH athletes typically exhibit a decrease in strength of their external rotators, which results in muscle imbalance of the RC muscles.[2] Cut-off values to identify a healthy shoulder versus a shoulder at risk for injury include isokinetic ER/IR ratio of 66% or an isometric ER/IR ratio of 75%, with a 10% RC strength increase in the dominant shoulder compared to the non-dominant shoulder.[2] The literature has recently shifted its focus to eccentric RC muscle strength, specifically of the external rotators due to its important role in deceleration during the OH motion and its correlation with shoulder injury.[2] Hand held dynamometers (HHD) have a higher sensitivity, as well as intra- and inter- reliability compared to manual muscle testing (MMT) in identifying RC strength deficits, thus are preferred for evaluation.[2] Specifically, HHD measurements of eccentric external rotator strength demonstrate excellent intratester (ICC=0.88) and good intertester (ICC=0.71) reliability, and good concurrent validity compared to an isokinetic device. This procedure is done with the athlete seated, where the PT supports the athlete's arm, as the athlete brings their shoulder from 90° abduction and 90° ER to 90° abduction and 0° ER, in order to load the external rotators eccentrically (Fig 9).[2] Currently, no cut-off values are identified, however, the PT can compare side-to-side differences and use their clinical judgement to identify limitations. When prescribing eccentric ER strengthening exercises, it is important to individualize the magnitude, frequency and duration depending on the athlete's level, injury status and their season, as well as make the exercise sport-specific to enhance outcomes. Exercises that aim to increase eccentric strength of the external rotators include: side-lying eccentric ER with a dumbbell; standing, resistance band "Y's," where the athlete pulls down with the opposite arm until the desired resistance is met and then slowly lowers the shoulder as they IR the shoulder with the thumb pointing down; supine resistance band 90-90 eccentric ER (Fig 10); prone 90-90 plyometric ball release/catch (Fig 11).[2]

Scapular dyskinesis is identified via visual observation, thus it is important that the PT is knowledgeable and confident in identifying abnormalities.[2] Scapular symmetry should be evaluated at rest and with motion, however, some degree of scapular asymmetry has been identified as normal in some OH athletes, which is something the PT should take into consideration.[2] In addition, scapular inter- and intra- muscular balance should be assessed, which can also be done via an HHD. Evidence suggests that in healthy individuals, isokinetic ratio of protraction/retraction is 100%, with small changes seen in OH athletes that favor the protractors.[2] Additionally, an increase of 10% in scapular muscle strength is recommended on the dominant shoulder.[2] The preventive program aims at restoring flexibility, specifically of the pectoralis minor, levator scapulae, rhomboid and posterior shoulder structures, and increasing scapular muscle performance, including muscle control, strength and balance.[2] Exercises to balance upper, middle and lower trapezius muscle activity include: side-lying forward flexion and side-lying ER, prone horizontal abduction with ER and prone extension in the neutral position (Fig 12).[23,24] To address the serratus anterior, exercises include: resistance band “bear-hugs,” “bear-hugs” with maximum flexion, and push-up plus, progressed from performed prone on elbows to performed with the feet elevated, in order to increase scapular and RC muscle recruitment (Fig 13).[15,23] To address the rhomboid muscles exercises include: standing resistance band low and high rows.[23]

The PT should also pay close attention to the biomechanics of the OH motion specific to the athlete’s sport, as poor technique is a major risk factor for shoulder injury.[11] This can be analyzed in more detail via video recording.[11] A videotaped analysis will also provide the athlete with visual feedback for more optimal correction of their technique. It is important that the PT has a good understanding of the biomechanics specific to the athlete's sport for optimal evaluation.[11] Regarding baseball pitchers, the PT should pay close attention to the lead foot angle, trunk rotation, amount and timing of shoulder ER and horizontal abduction.[10] Fatigue is another major risk factor for shoulder injury.[13] As the athlete fatigues they rely more on their arm than their lower extremity, which results in technical flaws, a decrease in motor control and a decrease in ball velocity, ultimately increasing risk of shoulder injury.[13] The PT and/or coach can detect fatigue via observation of the athlete's mechanics and performance and modify the athlete's training parameters as needed. Lastly, equipment is another factor that the PT needs to consider during evaluation. Modern tennis rackets are designed to be stiffer than older ones.[25] While this helps develop more power in the tennis serve, it has also been associated with an increase in overuse upper extremity injury rates.[25]

Conclusions:

In sum, it is important that the PT has a good understanding of the anatomy, biomechanics, pathomechanics and risk factors identified in this paper, as well as be knowledgeable of diagnostic screening tools and measures to identify athletes at risk of shoulder internal impingement and RC tear. While a standardized protocol for the prevention of shoulder injuries in OH athletes has yet to be identified, research supports the utility of a comprehensive prevention program that addresses posterior shoulder tightness and GIRD, deficits in RC muscle strength and scapular dyskinesia, including deficits in scapular muscle strength, balance and control.[1,2,10,11,15,23,24] Such a program is consistent with the mechanism of internal impingement and RC tears that are very common in OH athletes.[3] Given the high incidence of shoulder injuries in OH athletes, a prevention program that addresses the risk factors identified should be implemented as part of the athlete's and/or team's training program, in order to prevent injury and ultimately enhance performance. Future research should aim at evaluating shoulder injury prevention programs that are specific to each overhead sport, specifically how they relate to decreasing the risk factors identified and reducing shoulder injury rates.

Figure 1: Lateral aspect of the internal surface of the right GH joint. (Image obtained from David J. Magee, 2013)[5]

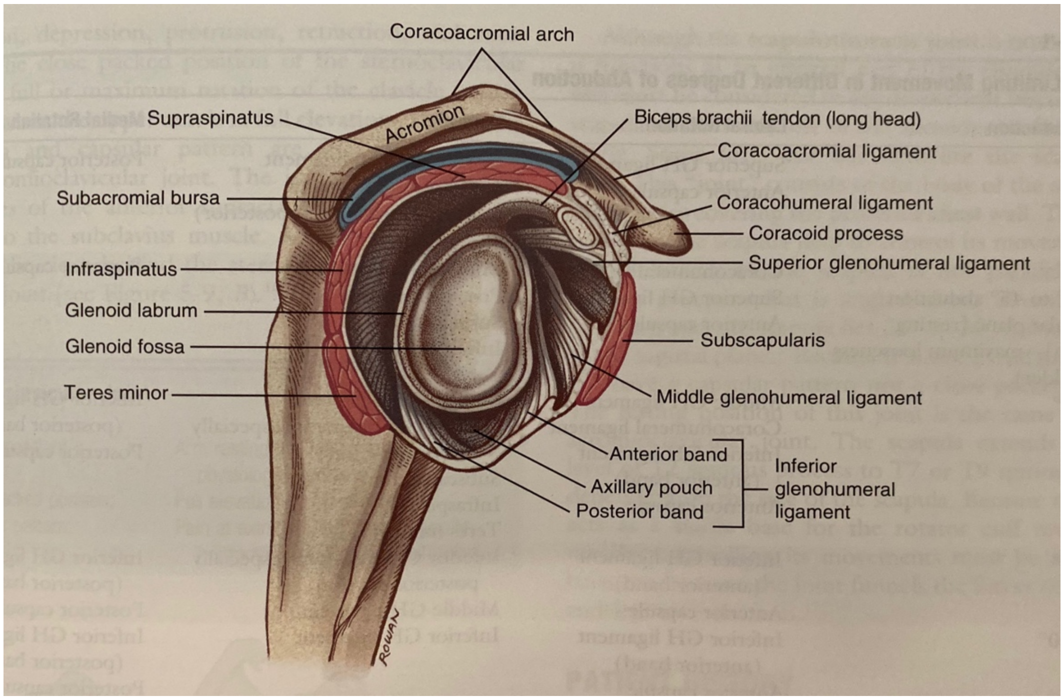


Figure 2: A schematic representation of the six stages in the OH pitching motion. (Image obtained from Escamilla et al., 2009)[8]

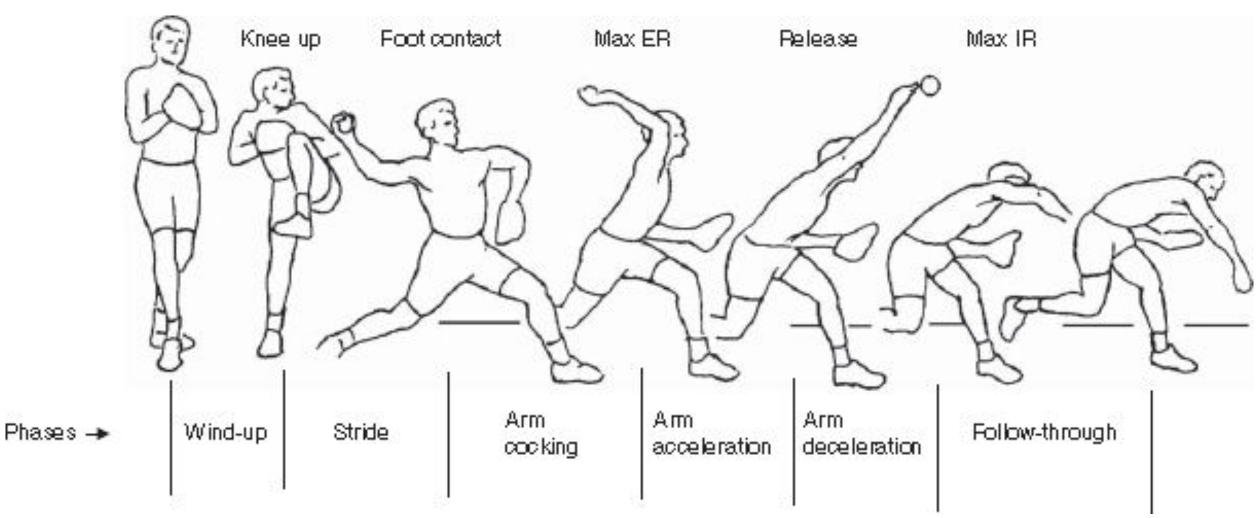


Figure 3: A graphic representation of the difference in the total shoulder arc of motion between OH athletes and non-throwers. The normal total arc of monition in non-throwers is ~180° with approximately equal degrees of ER and IR. OH athletes develop adaptive osseous and soft tissue changes as described above, that permit increased maximum ER, in order to achieve higher velocities. (Image obtained from Lin et al., 2008)[4]

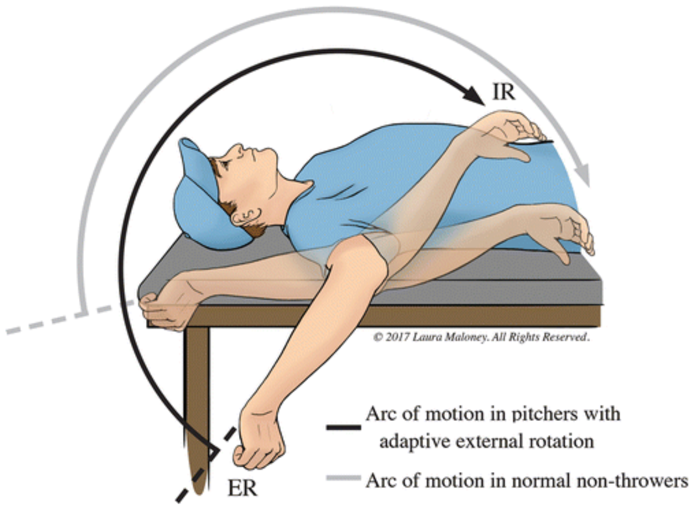
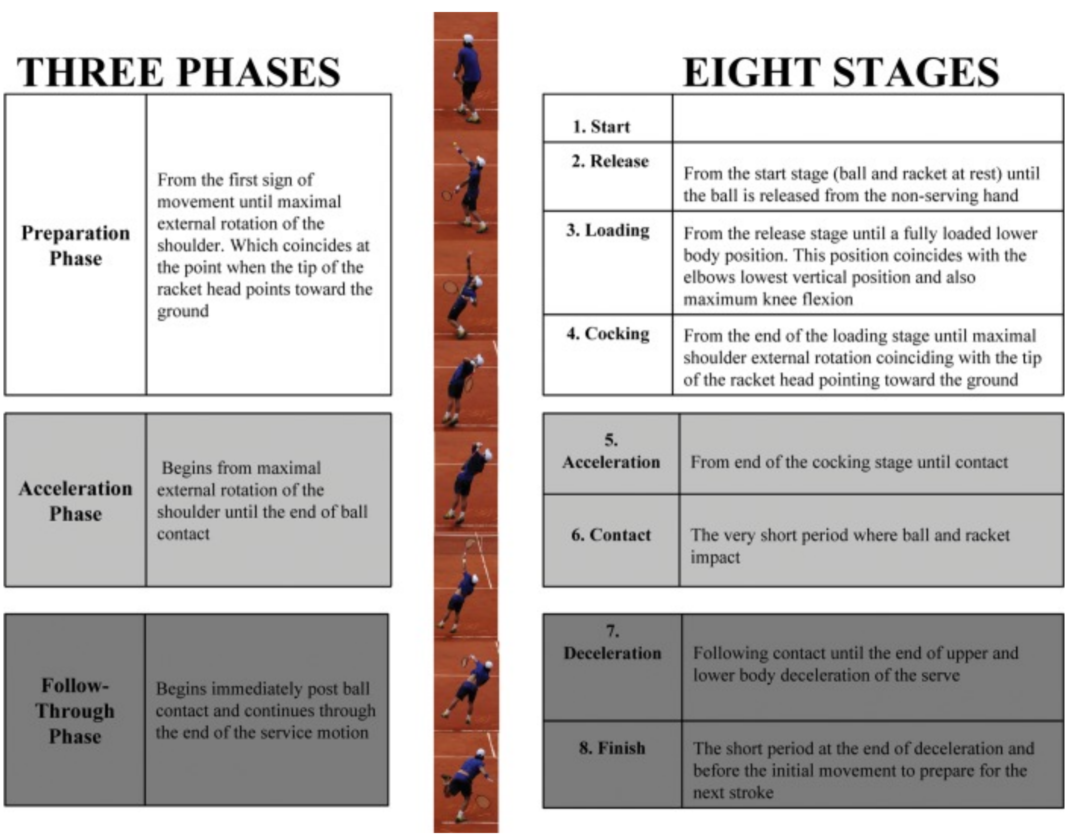


Figure 4: A schematic and descriptive representation of the 8-stage model of the tennis serve. (Image obtained from Kovacs et al., 2011)[12]



THREE PHASES

Preparation Phase	From the first sign of movement until maximal external rotation of the shoulder. Which coincides at the point when the tip of the racket head points toward the ground
Acceleration Phase	Begins from maximal external rotation of the shoulder until the end of ball contact
Follow-Through Phase	Begins immediately post ball contact and continues through the end of the service motion

EIGHT STAGES

1. Start	
2. Release	From the start stage (ball and racket at rest) until the ball is released from the non-serving hand
3. Loading	From the release stage until a fully loaded lower body position. This position coincides with the elbows lowest vertical position and also maximum knee flexion
4. Cocking	From the end of the loading stage until maximal shoulder external rotation coinciding with the tip of the racket head pointing toward the ground
5. Acceleration	From end of the cocking stage until contact
6. Contact	The very short period where ball and racket impact
7. Deceleration	Following contact until the end of upper and lower body deceleration of the serve
8. Finish	The short period at the end of deceleration and before the initial movement to prepare for the next stroke

Figure 5: Internal impingement of the undersurface of the RC against the posterior aspect of the glenoid labrum in the ABER position. (Image obtained from David J. Magee, 2013)[5]

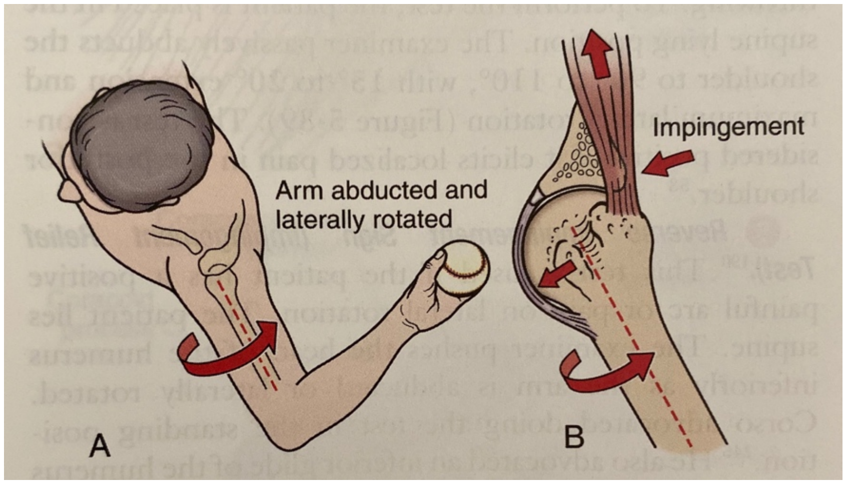


Figure 6: A graphical representation of the proper body alignment during the stride phase of the OH pitching motion. Note that the stride foot should be angled in a closed position, at approximately 15° away from the center of the mound (Θ), to prevent excessive shoulder anterior forces and decrease risk of injury. (Image obtained from Fleisig GS, 1994)[14]

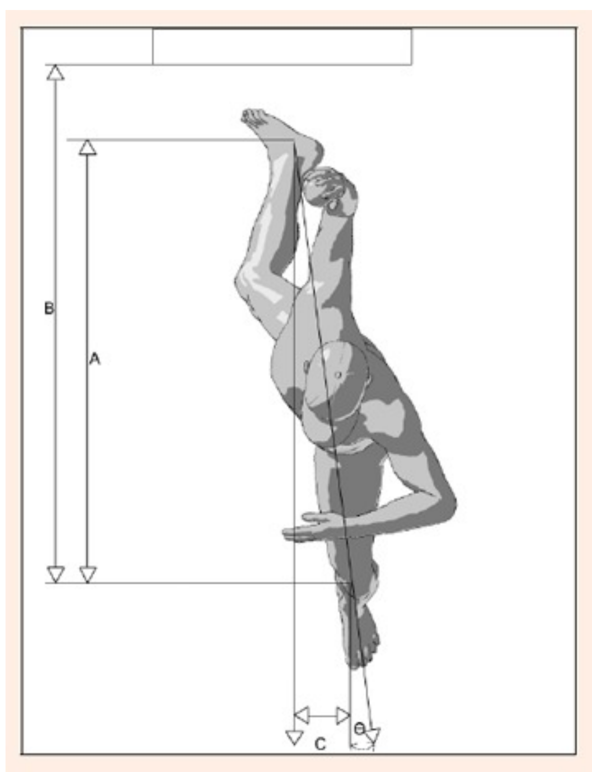


Figure 7: An MR arthrogram from a 35-year-old semiprofessional baseball pitcher demonstrating a partial-thickness posterior supraspinatus tendon tear (arrow) and a posterosuperior labral tear (arrowhead). These are typical findings for internal impingement. (Image obtained from Lin et al., 2008)[4]

Figure 8: Image 'A' represents the cross-body stretch that can also be performed in standing. Image 'B' represents the sleeper stretch. The goal of both exercises is to increase posterior shoulder flexibility and prevent GIRD, in order to decrease the risk of shoulder injury. (Images obtained from Cools et al., 2015)[2]

A. 

B. 

Figure 9: A graphic representation of the eccentric testing protocol utilizing an HHD.
The PT supports the arm of the tester, who brings the shoulder from 90° abduction and 90° ER to 90° abduction and 0° ER to load the external rotators eccentrically. (Image obtained from Cools et al., 2015)[2]

Figure 10: A graphic representation of the supine resistance band 90-90 eccentric ER exercise in the abducted position. Note how this exercise loads the muscles eccentrically by accentuating the eccentric phase and avoiding the concentric phase. (Image obtained from Cools et al., 2015)[2]

Figure 11: A graphic representation of the prone 90-90 plyometric ball release and catch exercise. This exercise can be performed in a slow manner for absolute strength or in a fast manner for endurance and plyometric capacity. (Image obtained from Cools et al., 2015)[2]

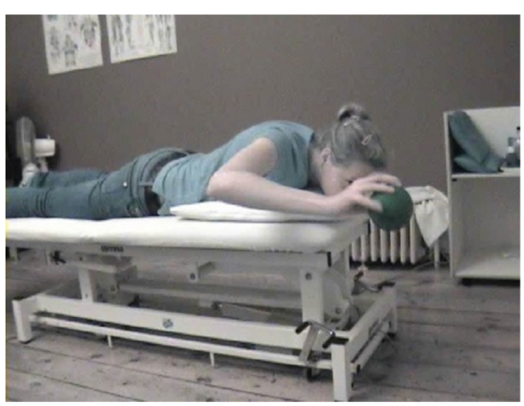


Figure 12: Graphical representations of exercise to balance upper, middle and lower trapezius muscle activity: 1) prone horizontal abduction with ER (thumb up); 2) side-lying forward flexion; 3) side-lying ER; 4) prone extension in the neutral position. (Images obtained from De Mey et al., 2012)[24]

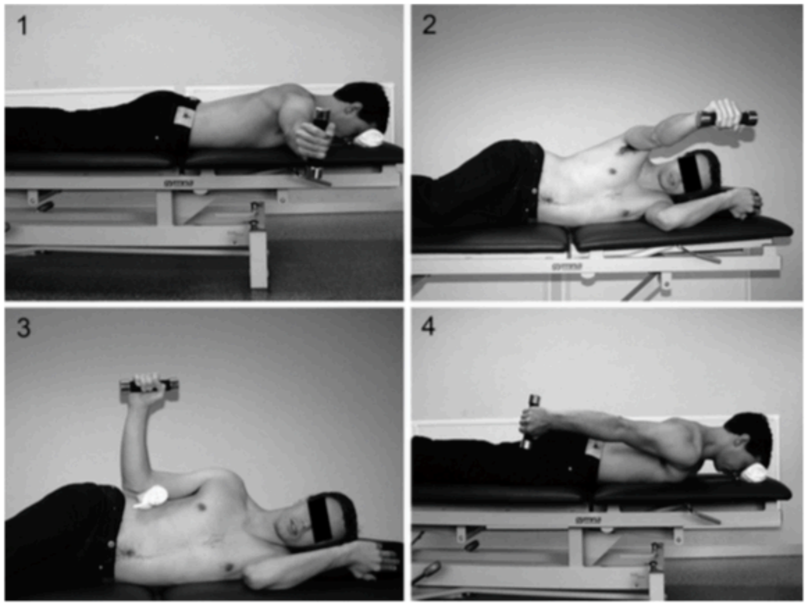
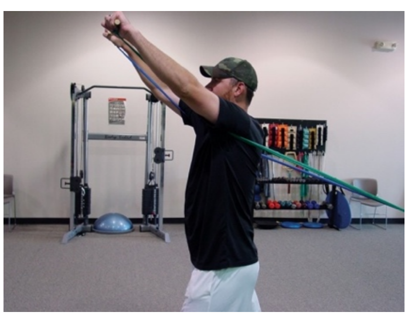
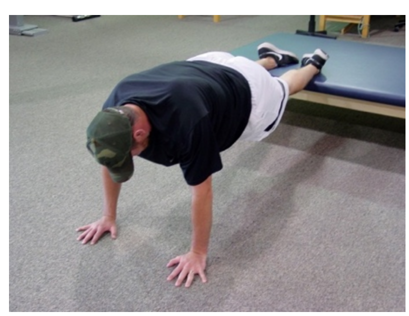


Figure 13: Graphical representations of exercises that address the serratus anterior, which is a vital component of normal scapulohumeral rhythm: 1) resistance band "bear-hug's with maximum flexion; 2. A) push-up plus performed prone on elbows that is further progressed to B) push-up plus performed with feet elevated, in order to increase scapular and RC muscle recruitment (Image obtained from Manske et al., 2013)[15]

1. 

2. A)  B) 

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