**Fall**

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**PHYT 875.965: ADVANCED ORTHOPEDIC ASSESSMENT AND INTERVENTION**

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**Fall**

Review of Common Shoulder Pathologies in the Overhead Athlete

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Index

Introduction …………………………………………………………………………………………………………............ 2

I. Superior Labrum Anterior Posterior (SLAP) Tear…………………………………………………........... 2

1. SLAP Tear Mechanism………………………………………………………………………........... 3
2. SLAP Tear Diagnostic Techniques…………………………………………………………….. 3
3. SLAP Tear Treatment…………………………………………………………………………..........4

II. Rotator Cuff Pathology…………………………………………………………………………………………......... 5

1. Rotator Cuff Pathology Mechanisms…………………………………………………………...5
2. Rotator Cuff Pathology Diagnostic Techniques…………………………………………....7
3. Rotator Cuff Treatment……………………………………………………………………………...8

III. Internal Impingement………………………………………………………………………………………………..9

1. Internal Impingement Mechanisms…………………………………………………………..10
2. Internal Impingement Diagnostic Techniques………………………….........................11
3. Internal Impingement Treatment……………………………………………………………..12

IV. Glenohumeral Internal Rotation Deficit (GIRD)…………………………………………………………12

1. GIRD Mechanisms…………………………………………………………………………………...13
2. GIRD Diagnostic Techniques……………………………………………………………………13
3. GIRD Treatment………………………………………………………………………………………14

Conclusion……………………………………………………………………………………………………………………14

**Introduction**

The purpose of this paper is to review common shoulder conditions in overhead athletes. The paper will review the mechanisms, diagnostic techniques and treatments for four common pathologies as well as suggest general prevention techniques. Repetitive use shoulder injuries are common in volleyball, baseball, swimming, handball, tennis, javelin and softball but can be seen in any activity where repeated overhead motion is required. Estimates show that 44-75% of overhead athletes have a history of shoulder pain and 12% have a history of substantial shoulder problems1 (substantial shoulder problems defined as injury resulting in moderate to severe reduction in training volume or performance).

Overhead athletic activity imposes multidirectional forces and high tensile loads on the shoulder complex.2 During throwing and overhead athletic movement the glenohumeral joint experiences increased stress as forces from the lower extremities and trunk are transferred to the upper extremity to produce torque and velocity.3 These large forces are necessary for high level athletic performance but also largely contribute to the development of shoulder pathology. Repetitive overhead activities can result in bony, capsular, muscular and ligamentous adaptations that increase glenohumeral external rotation while limiting internal rotation.2 These changes can result in superior labrum anterior to posterior (SLAP) tears, rotator cuff tears, internal impingement, and glenohumeral internal rotation deficit (GIRD).2 This paper will explore each of these pathologies in greater depth. Scapular dyskinesis is commonly associated with these conditions as well and will be briefly discussed.

**I. SLAP Tear**

The glenoid labrum is a fibrocartilaginous structure encircling the glenoid and serving to deepen the glenoid fossa while increasing stability of the glenohumeral joint. The long head of the biceps inserts proximally into the superior portion of the labrum, which is also the weakest attachment point of the labrum on the glenoid. This anatomical fault predisposes the labrum to SLAP lesions. SLAP lesions have four classifications: type I is described as fraying and degeneration of the labrum with a normal biceps tendon, type II is described as labral and biceps detachment from the supraglenoid fossa, type III is described as a bucket handle tear with an intact biceps tendon, and type IV is described as a bucket handle tear that extends into the biceps tendon.7

i. Mechanism of Injury

Overhead athletes stress the labrum excessively in the late-cocking phase of the throwing motion2 (glenohumeral abduction and maximal external rotation). This position can impinge the labrum between the head of the humerus and glenoid, the compressive and sheer forces exerted on the labrum may result in SLAP lesions.4 Superior posterior migration of the humeral head caused by posterior capsular tissue shortening can also predispose athletes to SLAP lesions.4,24,30 Increased maximal glenohumeral external rotation and large stabilizing forces exerted by the biceps in late cocking phase can result in excessive strain at the long head biceps insertion resulting in a “peel-back” lesion of the labrum.5 During the follow through phase of the throwing motion the massive deceleration force of the biceps may result in a SLAP lesion. SLAP lesions can also be caused traumatically by a FOOSH type injury or a glenohumeral traction injury.7

ii. Diagnostic Techniques

MRI is the gold standard for diagnosing SLAP lesions. The reliability of accurate diagnosis with MRI has been disputed however it has been shown that a magnetic resonance arthrogram with intra-articular gadolinium injection has 89% sensitivity and 91% specificity in diagnosing labral lesions.8

 Oh et al. demonstrated that the diagnostic cluster of O’Brien test (active compression test), labral crank (compression-rotation test) and Bicep Load II test had 95% specificity with a positive likelihood ratio of 4.4.10 Oh et al. also found that the Whipple test had a sensitivity of 65% and the Yerganson test had a specificity of 87%, both of which were the largest values of the tests included in the study.10 A type II SLAP lesion will present differently than other types because a type II lesion will include biceps detachment, thus test that create tension in the long head of the biceps should exacerbate symptoms more and may help differentiate a type II lesion from other types.10 An additional test that has shown utility in the diagnosis of SLAP lesions is the SLAPrehension test. To perform this test the patient should be seated with their arm positioned in 90° abduction, elbow extended, shoulder internally rotated and forearm pronated. The patient is then asked to horizontally adduct against resistance from that position and again with the forearm supinated and shoulder externally rotated. Pain with the first position and relief with the second indicates a SLAP lesion.7 Conversely, Cook et al. showed poor utility in the use of clinical tests for diagnosing labral lesions and expressed the need for imaging.11

It is common for overhead athletes with a SLAP lesion to report pain at the posterior superior glenohumeral joint line in the late-cocking phase of throwing motion.2,9 Decreased throwing velocity, catching, locking, snapping, instability and limitations in overhead activities are also common symptoms.4

iii. Treatment

 Non-operative management of SLAP lesions is commonly unsuccessful, however individuals with type I lesions may respond to conservative treatment that consists of restoration of normal scapulothoracic motion and glenohumeral internal rotation, posterior capsule lengthening, and scapular muscle strengthening along with NSAIDs and cessation of overhead activities.9 This treatment can then be progressed to include core strengthening, plyometrics and sport specific overhead activities9 under supervision.

 In most cases surgical intervention is required to repair the damaged tissue and suture the labrum to the superior glenoid.9 Post-operative rehabilitation will be dependent on the type and degree of injury. Generally, the patient will be immobilized for 2-3 weeks, followed by a focus on motion restoration (limiting external rotation) and strengthening, then a progression to sport related activity similar to that of conventional treatment.9 Dodsen recommends sport related activity be withheld for at least 4 months after surgery to repair a SLAP tear.9

 Ide et al. found 90% favorable results in overhead athletes who underwent surgical repair of type II SLAP lesions 41 months after surgery, noting that individuals with non-traumatic mechanisms had less favorable results.12 This suggests that over-use injuries may be less responsive to surgical intervention.

**II. Rotator Cuff Pathology**

 The rotator cuff is comprised of the supraspinatus, infraspinatus, subscapularis and teres minor muscles; the long head of the biceps is also commonly included when considering rotator cuff pathology. These muscles work in conjunction to stabilize and control motion of the shoulder girdle. Studies have shown that the muscles of the rotator cuff work as the primary glenohumeral stabilizers during overhead athletic movement to counteract the massive forces being generated through the upper extremity.12 In the overhead throwing motion eccentric rotator cuff control is required to maintain stability and compression of the glenohumeral joint, however this action imposes large tensile and shear forces that can damage the musculotendinous unit.2

Tears of the rotator cuff are categorized as partial or full thickness, the former being more common in overhead athletes.2 Partial tears can be further classified as bursal sided or articular sided tears. Rotator cuff pathology can be difficult to assess in overhead athletes because they commonly occur in conjunction with other injuries and asymptomatic athletes commonly present with rotator cuff abnormalities.13

i. Mechanism of Injury

 Rotator cuff tears are commonly caused by acute trauma, over use and chronic degenerative changes.7 The common mechanisms of rotator cuff tears in overhead athletes include tensile overload, primary extrinsic impingement and internal impingement.2 The most common location of rotator cuff injury in the overhead athlete is the posterior portion of the supraspinatus and the anterior portion of the infraspinatus.2 Tensile overloading occurs during the deceleration phase of the throwing motion. The accumulation of tendon microtrauma caused by repetitive eccentric tensile force results in tissue failure.12 In the eccentric phase of throwing demand is placed on the posterior superior portion of the rotator cuff, thus the reason for injury to the anterior infraspinatus and posterior supraspinatus tendons.14 Repetitive overhead athletic movements have also been shown to alter patterns of vascularity in rotator cuff tendons, weakening the structures and increasing the risk of injury.13

Subacromial impingement can result in rotator cuff pathology, although this is less common in the athletic population. As the acceleration phase of the throwing motion begins the glenohumeral joint is in maximal abduction and external rotation, as the arm begins to forcefully accelerate forward the subacromial bursa and cuff tendons will be compressed below the subacromial arch and coracoacromial ligament. Repeated compressive stress can cause bursal inflammation and degenerative changes to the tendon.13 This can be exacerbated by mechanical dysfunction as a result of scapular dyskinesis and weak rotator cuff musculature.13

Internal impingement occurs when the rotator cuff contacts the superior glenoid and labrum during the late cocking phase of throwing.15 In the maximally abducted and externally rotated position the rotator cuff and posterior superior labrum are compressed between the greater tuberosity posterior superior glenoid, this repeated compressive stress can cause simultaneous SLAP lesion and partial thickness rotator cuff tear.13 Contracture of the posterior capsule can exacerbate this mechanism.4 Internal impingement will be investigated in greater depth in section III of this paper.

ii. Diagnosis

As previously mentioned rotator cuff injuries in overhead athletes rarely occur in isolation making diagnosis of the injury difficult. Diagnostic imaging should be utilized if a rotator cuff tear is expected, this imaging should include radiographs, MRI and/or ultrasound. MRI is considered the gold standard for diagnosing athletes with rotator cuff pathology. With the inclusion of intra-articular contrast MRI has been shown to be 84% specific and 96% sensitive in diagnosing partial-thickness rotator cuff tears.2  Placing the arm in an abducted and externally rotated position mimicking the late cocking phase of the throwing motion has been shown to increase the diagnostic accuracy of MRI in throwing athletes.17 Ultrasonography has also been shown to be effective in the diagnosis of partial thickness tears as well, with evidence of 94% sensitivity and 93% specificity.2

 Bak et al. demonstrated that the clinical diagnostic cluster of the external rotation lag sign test, empty can test, and active abduction less than 90 degrees has a specificity of 65% and sensitivity of 54%; Bak also showed that replacing the lag sign test with the Hawkins-Kennedy test improved the sensitivity of the cluster to 72%.18 Other commonly accepted clinical tests used to diagnose rotator cuff pathology include the drop arm test (supraspinatus), lift arm test (subscapularis), external rotation resistance test (infraspinatus) and Yerganson test (long head of biceps); all of which have been shown to have a high level of specificity.7

Taking an appropriate history is an integral part of diagnosing rotator cuff pathology in overhead athletes. Generally these individuals will lack a clear mechanism of injury but rather they will experience progressively worsening pain that is exacerbated by overhead activities16 therefore identifying specific aggravating symptoms can help make diagnoses and devise treatment strategies. Other common complaints are lateral shoulder pain radiating into the deltoid and decreased throwing velocity.13

iii. Treatment

Conservative treatment is typically the initial strategy for rotator cuff pathology. If conservative treatment has been exhausted and is proven ineffective then surgical intervention should be considered.13  Treatment should be individualized based on the extent of the injury, impairment to the athlete, nature of the injury (traumatic or atraumatic) and other clinical findings.

Euler et al. suggest a four-phase approach to conservative treatment of rotator cuff tears. Phase I consists of rest, ice and NSAIDs to control pain and inflammation as well as light range of motion activity to maintain glenohumeral range.19 Glenohumeral internal rotation deficit is commonly seen in this population and will be investigated further in section IV of this paper. Phase II of Euler’s approach should begin when pain and inflammation are controlled, in this phase physical therapy intervention should target maximizing range of motion, strengthening rotator cuff (with a focus on external rotation7) and scapular musculature (with a focus on scapular retractors7), and incorporating closed chain neuromuscular control exercises.19 Phase III should begin when upper extremity strength is optimized and range of motion is normalized. This phase should include a progression of neuromuscular control exercises to include plyometrics, intensive core stability training and correction of faulty throwing mechanics.19 Finally, phase IV is a gradual return to sport.19 It has also been suggested that scapular dyskinesis should be assessed and addressed in this population.13

 Surgical repair of a full-thickness rotator cuff tear in an overhead athlete is treated similarly to the general population, evidence suggests a transosseous-equivalent double-row repair is most appropriate to maximize strength, prevent stiffness and decrease recovery times.2 The repair of full thickness tears has shown poor outcomes in overhead athletes.13 However, there is a report of 6 professional baseball pitchers who had surgical repair of full-thickness rotator cuff tears, 5 of whom returned to their pre-injury level for at least one season.2

There are discrepancies in the literature regarding surgical intervention for partial-thickness tears. Convention says that any rotator cuff lesion with depth greater than 50% should be surgically repaired.13 Rudzki and Shaffer argue that the supraphysiologic demands of the throwing motion may threaten the repair, therefore repairs should only be done if the depth of the lesion is 75% or greater.20 Lesions on the articular side should be debrided if they are not deep enough to warrant repaire.2 As previously mentioned, posterior supraspinatus and anterior infraspinatus partial-thickness tears are most common in overhead athletes, a study investigating overhead athletes who underwent debridement of partial thickness supraspinatus tears found that 85% of patients were able to return to their previous level of competition.21 Generally, surgical outcomes correlate with the severity of the injury. There is little evidence to guide specific timing of post-surgical rotator cuff rehabilitation.22 Patients are commonly immobilized (in an abducted and externally rotated position to promote blood flow) for a period following the surgery, however early passive motion has been shown to improve range of motion in the early stages.22 Following immobilization a progression similar to what Euler19 suggests should be utilized. The progression of return to sport activities should be conservatively projected to begin about 6 months after surgery.22

**III.** **Internal Impingement**

As previously mentioned, internal impingement (or posterior superior glenoid impingement) is most commonly seen in throwing and overhead athletes. It is a chronic pathologic condition that occurs due to repetitive maximal external rotation and abduction in the late-cocking phase of throwing or other overhead athletic movements.27 In this position the supraspinatus, anterior infraspinatus and posterior superior labrum are compressed between the greater tuberosity of the humerus and the posterior superior glenoid.15,24,27. Internal impingement is classified in three stages. In stage I the athlete will report decreased performance in overhead activity and vague discomfort in the late-cocking position.24 In stage II the athlete can localize pain in the posterior aspect of the shoulder during late-cocking, and in stage III the athlete reports persistent symptoms after completing a course of physical therapy intervention.24

i. Mechanism of injury

Halbretch et al. conducted an MRI study looking at throwing and non-throwing shoulders and concluded that contact between the posterior superior glenoid and greater tuberosity is a normal occurrence in the late-cocked position, however repetitive contact in this position may cause pathologic changes to the tissue.15 This contact is intensified in overhead athletes who commonly display excessive external rotation and repetitively load this position during the throwing motion.24 These factors cause impingement that can result in rotator cuff tears, type II SLAP lesions, bony changes of the humeral head or posterior superior glenoid and inferior glenohumeral ligament pathology.24,30 Heyworth et al. argue that anterior instability may be the most significant factor in internal impingement due to resultant abnormal anterior humeral head translation in the late-cocking phase27, however that theory was disputed by Halbretch et al. who found no difference in translation of throwing and non-throwing shoulders of baseball pitchers.15 It has also been theorized that posterior inferior capsular contracture and fibrosis causes internal impingement.24 It is believed that the alteration in posterior inferior tissue structure is caused by repetitive microtrauma resulting from the follow through phase throwing motion.24 Excessive external rotation can also result in elongation of the inferior glenohumeral ligaments and result in excessive anterior translation of the humeral head.30 Abnormally large amounts of glenoid anteversion or abnormally low amounts of humeral head retroversion may predispose athletes to internal impingement, however athletes with normal values have also been shown to develop the condition.27 The development of internal impingement is also commonly seen in conjunction with scapular dyskinesia and glenohumeral internal rotation deficit.2

ii. Diagnosis

 Dissimilarly to SLAP lesions and rotator cuff tears, MRI findings do not necessarily correlate with pain and decreased functionality and therefore cannot be used in isolation to diagnose internal impingement. Studies have found abnormal MRI results in the shoulders of asymptomatic overhead athletes and these findings did not increase the risk of developing symptoms of internal impingement at a 5 year follow-up.23,24 However, MRI findings of humeral head cysts, articular side rotator cuff tears, posterior superior labral lesions and thickening of the inferior glenohumeral ligaments may indicate internal impingement if the patient history and clinical findings correlate.2,24

Clinically, most athletes will report decreased throwing accuracy and velocity along with chronic diffuse or localized posterior joint line pain in the late-cocking.27 Athletes may also report difficulty “getting loose.”31 Symptoms of internal impingement are similar to those of atraumatic rotator cuff pathology, in which case imaging can help make a correct diagnosis. Young athletes with these complaints may be more indicative of internal impingement because atraumatic rotator cuff injuries are uncommon in the younger population however internal impingement can lead to rotator cuff pathology.27

The posterior impingement sign has been demonstrated to have 77.5% sensitivity and 85% specificity in diagnosing internal impingement.28 This test is completed with the athlete in supine and the shoulder in 90°-110° abduction, 10°-15° extension (or horizontal abduction) and maximal external rotation, a positive test will result in posterior joint line pain in that position.28 Diagnostic accuracy of this test is improved if symptoms onset is insidious. Anterior instability and posterior joint line pain in the apprehension-relocation test (relief of symptoms with posteriorly directed force on the humeral head) is also a sign of internal impingement.27 The location of pain during this test differentiates findings between internal impingement from general instability. Athletes with internal impingement will also commonly show internal rotation deficits with the shoulder abducted to 90°.31 Glenohumeral internal rotation deficit and scapular dyskinesis are also commonly seen in conjunction with internal impingement.27

iii. Treatment

 In most cases conservative treatment is successful when internal impingement is addressed early. Conservative treatment should initially consist of rest, ice and NSAIDs. Athletes with localized posterior joint line pain should rest 4-6 weeks. 31 When the athlete is asymptomatic posterior capsule stretching (sleeper stretch, cross body horizontal adduction stretch, ect.30) and strengthening of external rotators and scapular musculature has been shown to improve symptoms.2,15,27,29 Addressing scapular dyskinesis27 and faulty movement patterns will also improve symptoms. After athletes are asymptomatic they should complete a progressive activity specific program before returning to competition.2 Similar treatments have also shown good results for glenohumeral internal rotation deficit and scapular dyskinesis.27,31 A four phase treatment approach similar to that proposed by Euler19 for rotator cuff pathology is suggested for athletes with internal impingement.

When conservative treatment fails surgical procedures may be required to address capsular laxity, torn rotator cuff or labral tissue, or bony lesions of the humeral head or glenoid. Posterior capsular release may also be indicated in athletes who do not respond to posterior capsular lengthening intervention.27 Generally, surgical intervention should be reserved to address pathologic lesions that correspond to patient symptoms.27

**IV. Glenohumeral Internal Rotation Deficit**

 GIRD is described as a deficit in internal rotation and total arc of motion in the dominant arm.32 Deficits greater than 30° – 40° in glenohumeral internal rotation accompanied by excessive external rotation are commonly seen.27 GIRD in itself is not considered an injury, however an internal rotation deficits of 18° or more (when compared to the non-dominant side) increases the risk of injury by 1.9 times.34 The alteration in range of motion leads to altered shoulder mechanics which puts excessive stress on dynamic stabilizers during the overhead activities.2 GIRD is commonly seen in conjunction with internal impingement and scapular dyskinesis.

i. Mechanism

 There is no clear mechanism for GIRD, though it is believed to be caused by shortening of posterior tissues (posterior capsule, posterior deltoid, infraspinatus and teres minor), bony and ligamentous adaptations, inflammation and scar tissue formation.2,32,33 Changes in tissue are the result of repetitive overhead activity.32 Posterior capsular thickening related to GIRD is thought to be the result of repeated stress during the deceleration phase of the throwing motion.33 Humeral retroversion may also play a role in capsular hypertrophy, however this adaptation (caused by torsional loading in young athletes) also benefits athletes by increasing ball velocity.33 Alterations in range of motion result in biomechanical changes in the athletes throwing motion or overhead activity as well as changes in glenohumeral arthrokinematics that predispose the athlete to injury.33 GIRD has been shown to be one of the largest factors in atraumatic shoulder and elbow injury and is a detriment to performance in the overhead athletic population.33

ii. Diagnosis

GIRD can be diagnosed by assessing glenohumeral rotation with the athlete in supine and erect with the shoulder abducted to 90°.33 As previously mentioned, deficits greater than 30° – 40° of internal rotation are indicative of GIRD, however some authors suggest deficits greater than 25° are enough to warrant a diagnosis of GIRD.33 Other studies suggest a deficit of 18° compared to the non-dominant arm is indicative of GIRD.34 Tests for subacromial impingement are commonly positive in individuals with GIRD due to increased joint compression.29 Athletes with GIRD will commonly complain of diffuse shoulder pain and pain at night.30 It is important to note that a difference of a 10°-15° of glenohumeral internal rotation is expected in asymptomatic overhead athletes when comparing sides.33

iii. Treatment

Posterior capsular stretching has been shown to resolve symptoms related to GIRD and internal impingement..27,29 One study showed that 90% of throwers reduced GIRD to acceptable levels following 2 weeks or posterior capsular stretching (sleeper stretch),27 however other studies have shown the cross body horizontal adduction stretch is superior to the sleeper stretch for resolving symptoms.27 Further, passive (therapist assisted) cross body stretching may be superior to active cross body stretching and the sleeper stretch.32  Evidence has also been shown in support of overhead triceps stretching for the resolution of GIRD.32 A combination of the cross body stretch, sleeper stretch and overhead triceps stretching are suggested to address GIRD.32 Internal rotation stretching with a towel behind the back has also shown to improve GIRD as well.33 It is suggested that stretching exercises be completed daily for best results, especially during the competitive season.33

 Individuals who do not show improvement in range of motion following 4-6 weeks of intervention should be considered for surgery to release the posterior capsular.33 However, in most cases athletes will respond to conservative treatment.

**Conclusion**

Overhead athletic activity exposes the shoulder to supraphysiologic ranges of motion and requires massive amounts of force transmission through the shoulder. Repeated overhead demands make it common for these athletes to develop overuse injuries. Shoulder injuries in the overhead athlete seldom occur in isolation and are commonly multifactorial which increases diagnostic and treatment difficulty. Taking an adequate clinical history, integrating imaging findings and possessing clinical the skill to differentially diagnose these conditions will help the clinician choose appropriate intervention strategies to expedite the recovery process for these athletes.

Another common shoulder condition in overhead athletes that was not specifically investigated in this paper is scapular dyskinesis (synonymously known by the acronym SICK). SICK stands for scapular malposition, inferior medial border prominence, coracoid pain and malposition, and dyskinesis of scapular movement.35 Scapular dyskinesis has been shown to be present in 61% of overhead athletes and 67%-100% of those with shoulder injury.37 Scapular dyskinesis increases the risk of injury in the overhead athlete and is a closely associated with all four conditions investigated in this paper. Burkhart et al. demonstrated that 100% of athletes (n=96) with scapular dyskinesis were able to return to their pre-injury throwing level within 4 months after following a scapular muscular strengthening rehabilitation program.30 Similar programs should be considered in symptomatic overhead athletes with any of the conditions mentioned in this paper who present with SICK signs.

A shift in rehabilitative and prehabilitative efforts from isolated shoulder exercise to the involvement of the entire kinetic chain is gaining popularity in the literature.35 The kinetic chain theory postulates that during overhead athletic activity force is generated through the lower extremities and core and transmitted to the upper extremity through the shoulder, therefore a “weak link” at any point in the chain can lead to altered biomechanics and predispose an athlete to injury.35 Following this theory, as well as rehabilitation strategies proposed in phase III of Euler’s19 program, kinetic chain exercise including dynamic core stabilization and plyometrics should be included in all overhead rehab and prehab programs.35

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