

Symptomatic Internal Impingement of the Shoulder in Overhead Athletes

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Abstract: The term “internal impingement” describes the normal physiological contact that occurs between the posterosuperior glenoid and the greater tuberosity in positions of hyperabduction and external rotation. This physiological contact can become symptomatic when repeated overhead motion results in partial articular-sided posterosuperior rotator cuff tears and lesions of the posterosuperior glenoid labrum. The precise pathophysiology involved with the development of symptomatic internal impingement has been debated extensively over the past few decades. However, current literature suggests that symptomatic internal impingement may result from a combination of multiple factors involving repetitive overhead activity, physiological remodeling of the throwing shoulder, posterior capsule contracture, and scapular dyskinesia, among other factors. These can all lead to scapulohumeral hyperangulation and associated pathologic findings. The purpose of this article is to review the relevant anatomy, pathophysiology, diagnosis, and management of symptomatic internal impingement through a critical review of current evidence.

Key Words: shoulder injury, overhead athletes, throwing athletes, rotator cuff, internal impingement, posterosuperior impingement

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Internal impingement is a normal physiological phenomenon in which the greater tuberosity comes into contact with the posterosuperior glenoid rim in positions of abduction and external rotation. Although its mechanistic function is to prevent excessive external rotation, repeated bouts of abduction, and external rotation, as which occurs with repetitive throwing activities, can lead to symptomatic internal impingement in which both the posterosuperior labrum and posterosuperior rotator cuff become “pinched” between the greater tuberosity and the glenoid rim. This interposition can result in articular-sided posterosuperior cuff tears along with posterior or posterolateral labral tears that result in posterior shoulder pain in the abducted and externally rotated shoulder.

The precise biomechanics involved with the development of symptomatic internal impingement have been widely debated over the past 2 decades. Some researchers

have concluded that acquired anterior instability is causative,^{1–5} whereas others have refuted this notion, citing more recent studies that have found no correlation between symptomatic internal impingement and anterior glenohumeral translation.^{6–8} Current thinking suggests that symptomatic internal impingement is most likely multifactorial, involving a combination of physiological remodeling of the shoulder, posterior capsular contracture, and scapular dyskinesia (SICK scapula) that can all lead to a glenohumeral internal rotation deficit (GIRD), humeral hyperangulation, and resulting pathologic findings such as posterosuperior glenoid impingement lesions and superior labral anterior to posterior (SLAP) tears.^{9–11}

The purpose of this article is to review the pathoanatomic features of symptomatic internal impingement and the pertinent concepts involved with diagnosis and management of this condition.

HISTORICAL PERSPECTIVE

In 1959, Bennett¹² was the first to describe the posteroinferior glenoid exostosis that commonly occurs in overhead athletes with posterior shoulder pain. This finding, commonly known as the “Bennett lesion,” was initially thought to result from traction by the posterior band of the inferior glenohumeral ligament (IGHL) complex and the triceps origin. Although more recent studies have concluded that the triceps tendon is not involved with the development of the Bennett lesion,^{13,14} the presence of posterior contracture of the IGHL complex and capsule in patients with symptomatic internal impingement is well-known.^{9,15} Bennett¹² was also the first to note the presence of articular-sided posterosuperior rotator cuff tearing in these same athletes.

Bennett’s¹² work spurred the development of other studies that further explored the etiology of posterior shoulder pain in overhead athletes. In 1977, Lombardo et al¹⁶ reported 4 cases of posterior shoulder pain related to posteroinferior glenoid exostoses (so called “Bennett lesions,” Fig. 1) and discussed their potential etiologies, including repetitive microtrauma and “wringing” of the capsule in positions of hyperexternal rotation. Although the authors were unable to determine whether capsular traction primarily occurred during the late cocking or follow-through stages of the throwing motion, they were the first investigators to acknowledge a possible pathomechanistic cause for symptomatic internal impingement and the development of Bennett lesions.

In 1985, Andrews et al¹⁷ reported that articular-sided posterosuperior cuff tears were significantly associated with the presence of SLAP lesions in overhead athletes with posterior shoulder pain. Later, Jobe¹⁸ and Walch et al^{19,20} described the impingement mechanism in which the undersurface of the rotator cuff, at the interval between the supraspinatus and infraspinatus, becomes entrapped

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FIGURE 1. AP radiograph demonstrating a Bennett lesion (yellow arrow) in an overhead athlete with posterior shoulder pain.

between the glenoid labrum and greater tuberosity in positions of abduction and external rotation (Fig. 2). Although these landmark studies substantially enhanced our knowledge regarding the precise mechanism in which internal impingement occurs, the pathoanatomic and biomechanical factors involved with the gradual progression of internal impingement from a physiological state to pathologic state has been heavily debated.

PATHOPHYSIOLOGY AND BIOMECHANICS

Interestingly, although the study by Walch et al¹⁹ excluded patients with anterior instability, more recent authors have suggested that anterior glenohumeral laxity plays a primary role in the development of symptomatic internal impingement largely due to the preponderance of throwing athletes that present with signs of instability.^{1,5,17,21,22} The theory that increased anterior humeral translation in the abducted and externally rotated position as a result of gradual anterior capsular attenuation would facilitate posterosuperior cuff entrapment between the posterosuperior glenoid and greater tuberosity was widely accepted and supported for a number of years.

More recently, Sonnery-Cottet et al⁸ reported on a series of 25 tennis players with lesions consistent with symptomatic internal impingement; however, none of these players displayed anterior glenohumeral laxity or instability. Others have reported that the increasing severity of anterior instability is actually protective against internal impingement and the development of associated pathologic lesions.^{3,19,23,24} In addition, Burkhart²⁵ suggested that the loss of physiological internal impingement is actually pathologic since this would facilitate hyperexternal rotation and the development of SLAP tears. Therefore, pathomechanisms other than increased anterior glenohumeral translation were eventually sought.

In 2003, Burkhart et al⁹ contended that a loss of internal rotation (GIRD) initiates a series of anatomic

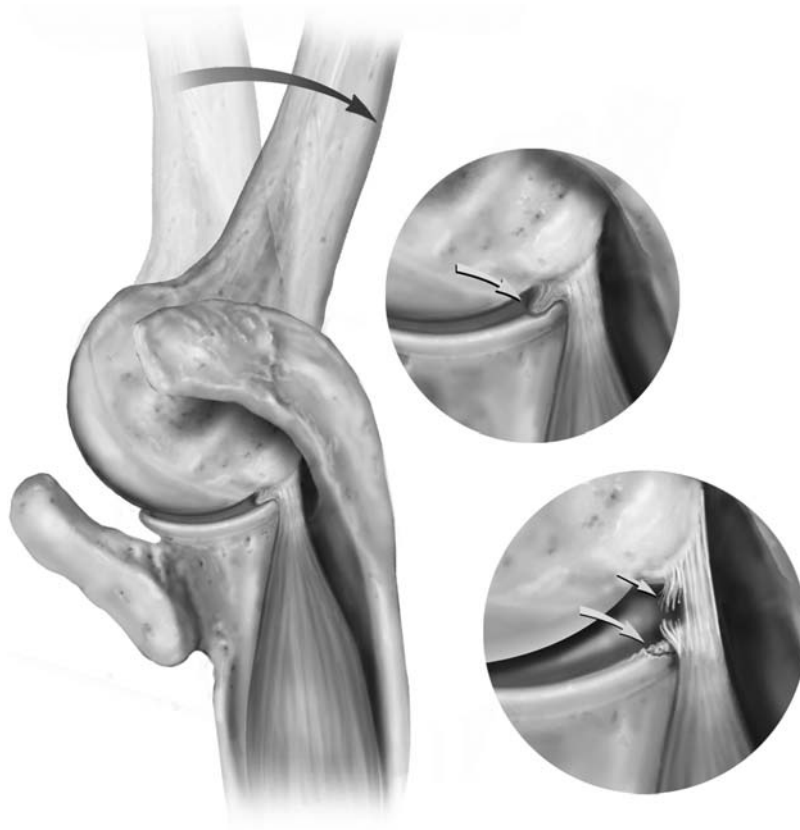


FIGURE 2. Illustration of the pathomechanism behind the development of symptomatic internal impingement. Jobe²¹ and Walch et al^{19,20} postulated that the posterosuperior cuff and labrum can become entrapped between the greater tuberosity and the posterosuperior glenoid resulting in partial-thickness articular-sided rotator cuff tears and posterosuperior labral tearing. Arrows indicate the resulting pathologic lesions associated with symptomatic internal impingement.

changes that can lead to pathologic lesions in the throwing shoulder. The first of these changes involves contracture of the posterior IGHL complex and posteroinferior capsule from reactive scarring that essentially shifts the point of glenohumeral articulation and center of rotation posterosuperiorly. This is supported by a biomechanical cadaveric study by Harryman et al²⁶ in which posterosuperior migration of the humeral head was observed after posterior capsular tightening. Posterosuperior humeral head positioning allows the humeral head to translate anteriorly without subluxating or dislocating, possibly explaining the perceived anterior laxity that has been reported by others.^{1,5,17,21,22}

This posterosuperior position also allows for a greater degree of external rotation before physiological internal impingement can occur, elevating the potential for the development of pathologic lesions. For example, increasing degrees of external rotation alters the path of the long head of the biceps (LHB) tendon such that an increased torsional load is applied to the superior labrum in a posterior direction. This so called “peel-back mechanism” is thought to be responsible for the significant rate of SLAP tears seen in throwing athletes (Fig. 3).^{9,28,29} Burkhart et al¹⁰ argue that these SLAP tears contribute to “pseudolaxity”—a common condition seen in both symptomatic and asymptomatic throwers in which features of anterior laxity are present without clinical instability, resulting in perceived instability in these athletes. Posterosuperior humeral head positioning and the prevalence of SLAP tears in overhead athletes may explain the previous notion that anterior instability played a primary role in the development of symptomatic internal impingement.

Included in the series of anatomic changes described by Burkhart et al⁹⁻¹¹ is scapular malposition as a result of stress and fatigue in the throwing shoulder.¹¹ Scapular malposition results in prominence of the inferomedial border, coracoid tenderness and scapular dyskinesia, also called the “SICK scapula syndrome.” This overuse syndrome results in scapular protraction and glenoid malposition that facilitates humeral hyperexternal rotation and elevates the potential for SLAP tears and symptomatic internal impingement in throwing athletes. In their study of 64 patients with posterosuperior labral tears, 95% of patients displayed increased scapular internal rotation and/

or scapular protraction resulting in a prominence of the medial scapular border.¹¹ Increased internal rotation of the scapula has also been found in a biomechanical study to result in increased posterosuperior glenohumeral contact pressure, which is hypothesized to be involved with the development of posterosuperior labral tearing and articular-sided rotator cuff tears.³⁰

Others have investigated the role of osseous remodeling in throwing athletes. Specifically, retroversion of the humeral head and the glenoid has been shown to be potentially involved with the development of hyperexternal rotation and resulting pathologic lesions.^{19,31} Walch et al¹⁹ was the first to notice the increased humeral retroversion that occurred in their series of throwing athletes. Later, Crockett et al³¹ found increases in humeral and glenoid retroversion in the throwing shoulders of professional baseball pitchers compared with their nondominant shoulders. Osseous remodeling may therefore be an adaptation that occurs in younger athletes as abnormal stresses are placed upon open physes. When the athlete reaches skeletal maturity, the proximal humeral and glenoid physes close in retroverted positions, facilitating scapulohumeral hyperangulation. However, a study by Sweitzer et al³² noted that acquired glenoid retroversion may actually be a protective adaptation that prevents a loss in the total arc of motion—the resulting hyperexternal rotation compensates for the loss of internal rotation, thus maintaining the total arc of motion in overhead athletes.

Although all of these anatomic factors are likely involved in the eventual development of symptomatic internal impingement, debate still exists regarding how and when the posterosuperior rotator cuff becomes entrapped between the greater tuberosity and posterosuperior glenoid rim. Many authors suggest that repeated impingement episodes during the late cocking and early acceleration phases of the throwing motion causes the pathologic lesions found at diagnostic arthroscopy.^{18,33-35} Others contend that the increased traction forces placed upon the cuff tendon during the deceleration phase of the throwing motion play a key role in the development of posterosuperior glenoid impingement lesions.¹⁷ This has been disputed since increased tension also occurs in the subscapularis tendon in positions of hyperextension and abduction; however, pathologic lesions are rarely found in

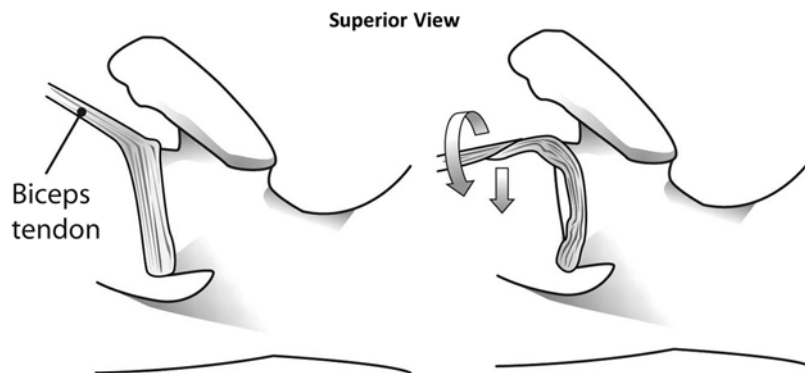


FIGURE 3. Illustration depicting the peel-back mechanism. Increased torsional strain is placed on the biceps anchor with increasing degrees of humeral external rotation thus increasing the potential for superior labral tearing and biceps anchor detachment. The straight arrow indicates the direction of pull on the biceps anchor during the throwing motion. The curved arrow indicates torsional strain placed on the biceps anchor in positions of hyperabduction and external rotation. (Adapted from Braun et al²⁷ with permission.) Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

the subscapularis tendon in relation to the throwing shoulder.⁵

Owing to the large body of evidence suggesting various factors that may be involved with the development of symptomatic internal impingement and its corresponding pathologic lesions, it is most likely a complex, multifactorial process that has yet to be completely elucidated. Further research is needed to more clearly demonstrate the association between the various pathophysiological adaptations in the throwing shoulder with the pathologic lesions seen in patients with symptomatic internal impingement.

EPIDEMIOLOGY

The incidence of symptomatic internal impingement is unknown due to the variety of associated pathologic lesions, diagnostic difficulty and incomplete reporting of the condition. Most cases involve younger patients (< 40 y old) that participate in activities requiring repetitive external rotation and abduction. While throwing activities, such as baseball, are classically associated with symptomatic internal impingement, it is important to note that the condition can occur in athletes and nonathletes alike. In one series, Jobe²¹ treated 11 patients with symptomatic internal impingement; however, 6 of these patients were not overhead athletes. Although nonathletes may also develop the condition, the majority of those with symptomatic internal impingement are throwing athletes.

CLINICAL PRESENTATION

The clinical presentation of patients with symptomatic internal impingement is often nonspecific and unyielding. Most athletes will present with chronic, diffuse, posterior shoulder pain that is exacerbated by activities requiring abduction and external rotation. In the series reported by Jobe,²¹ it was found that while all 11 patients injured their shoulders during some sort of abduction/external rotation activity, those who were nonathletes were more likely to report acute posterior shoulder pain rather than the chronic, diffuse pain commonly reported in throwing athletes.

If the patient is an overhead throwing athlete, they will also typically complain of a gradual decrease in throwing velocity, accuracy and overall throwing performance over a period of months.^{36,37} Davidson et al²³ reported that symptomatic internal impingement was most common cause of rotator cuff lesions in young overhead athletes; thus, the presence of classic rotator cuff disease should be ruled out in all patients with possible symptomatic internal impingement.^{23,38} Symptoms of instability such as clicking and subluxation may also be present; however, the presence of both anterior instability and symptomatic internal impingement is less common than was once thought.^{3,23,39}

PHYSICAL EXAMINATION

In the overhead athlete suspected to have symptomatic internal impingement, a complete shoulder examination is necessary due to the wide range of pathologies that are commonly associated with this condition. Palpation of the glenohumeral joint should first be performed since posterior joint line tenderness is often present in these patients. Active and passive range of motion should be assessed along with provocative testing for rotator cuff lesions, SLAP tears, subacromial impingement and instability. In addition, maintenance of the total arc of shoulder motion

should be assessed with a goniometer, taking into account the potential presence of humeral retroversion.^{40,41} Specifically, a loss of internal rotation of 30 to 40 degrees with a corresponding decrease in the total arc of motion compared with the contralateral shoulder is generally considered to be pathologic (Fig. 4).⁴² However, Wilk et al⁴³ found that an internal rotation deficit of > 5 degrees was a predisposing factor for future injury in professional baseball pitchers. Dynamic examination of the scapula is also necessary to detect scapular dyskinesis, which is thought to be intimately related with both symptomatic internal impingement and outlet impingement.³⁶

Few physical examination maneuvers can properly reproduce the pain experienced by the patient with symptomatic internal impingement and is therefore difficult to diagnose. However, Meister et al³⁴ developed a test (called the “posterior impingement sign”) that may specifically detect posterior labral lesions and posterosuperior articular-sided rotator cuff tears. This technique involves placing the shoulder in an abducted and externally rotated position, similar to the late cocking phase of the throwing motion, and noting the presence of deep posterior shoulder pain when the shoulder is brought into maximal external rotation. In a series of 69 overhead athletes with an average age of 22.7 years, the sensitivity of the test was found to be 76% with a specificity of 85%. Of note, the sensitivity and specificity of the test improved to 95% and 100%, respectively, when patients with contact injuries were excluded from the analysis.³⁴

Some authors suggest that the Jobe relocation test may also be useful to identify patients with symptomatic internal impingement.^{5,21,23} This test involves placing the arm in 90 degrees of abduction with maximal external rotation, mimicking the late cocking phase of the throwing motion. With an anteriorly directed force on the proximal humerus, the patient with symptomatic internal impingement will complain of posterior shoulder pain that is relieved when a posteriorly-directed force is applied. The reproduction of pain with this maneuver is thought to result from either cuff and labral impingement between the glenoid rim and the greater tuberosity or the increased tension placed upon the previously damaged tissues. The development of pain in

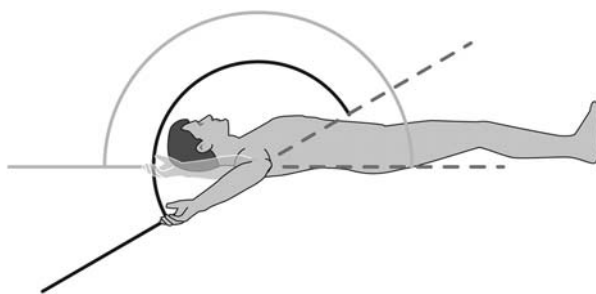


FIGURE 4. Illustration of the total arc of motion in an overhead throwing athlete. Note that the total arc of motion is unchanged (approximately 180 degrees) despite the decreased internal rotation and hyperexternal rotation. A decrease in the total arc of rotation of 30 to 40 degrees, especially with loss of internal rotation, is considered to be pathologic.⁴² (Adapted from Braun et al²⁷ with permission.) Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

this position must be differentiated from apprehension which is most commonly seen in patients with anterior glenohumeral instability.

IMAGING

Radiographic Evaluation

Patients suspected of having symptomatic internal impingement should receive standard anteroposterior (AP), axillary, scapular Y, West Point and Stryker notch films to fully evaluate the shoulder for concomitant lesions such as Bennett lesions,^{12,44} osteochondral lesions of the posterosuperior humeral head (cystic “geodes”),²⁰ sclerosis of the greater tuberosity⁴⁵ and/or erosion or spurring of the posterosuperior glenoid rim.⁴⁶

Wright et al⁴⁴ used a “modified Bennett view” to evaluate asymptomatic shoulders in a series of Major League baseball players. In this view, the arm was abducted 45 degrees with the x-ray beam angled 5 degrees cephalad in an effort to identify bony excrescences near the inferior glenoid rim. In their study, inferior glenoid osteophytes occurred in 60% of the asymptomatic throwing shoulders, leading the authors to conclude that this finding may not represent a true symptomatic lesion, but rather a common radiographic feature that occurs in throwing athletes.

At diagnostic arthroscopy, Paley et al⁵ discovered osteochondral lesions of the posterosuperior humeral head (so called “cystic geodes”⁴⁷ on preoperative AP radiographs) near the articular-sided insertion of the supraspinatus tendon in their series of overhead athletes with symptomatic internal impingement. Walch et al¹⁹ also noted the presence of these lesions in 8 of 17 patients with symptomatic internal impingement who underwent diagnostic arthroscopy. However, although present in many patients, the use of standard radiographs to identify these osteochondral lesions is difficult, if not impossible, in the majority of cases.

Sclerotic and/or cystic changes of the greater tuberosity are common radiographic findings that can occur in up to half of patients presenting with symptomatic internal impingement.^{20,35} However, Wright et al⁴⁵ radiographically evaluated 57 asymptomatic Major League baseball players and found that 39% of the players had cystic changes of the greater tuberosity. These findings suggest that sclerosis and/or cystic changes of the greater tuberosity, although common in patients with symptomatic internal impingement, are unlikely to be the cause posterior shoulder pain in overhead athletes.

Erosion of the posterosuperior glenoid rim may also become apparent on radiographs as a result of its repeated contact with the greater tuberosity, especially in throwers who have yet to reach skeletal maturity. However, this radiographic finding has been inconsistent in those with symptomatic internal impingement, occurring in approximately one third of patients in at least 1 study.³⁵

MRI Evaluation

Magnetic resonance imaging (MRI) (with or without contrast) is considered the diagnostic gold standard in young throwers with persistent posterior shoulder pain. In patients with signs and symptoms of symptomatic internal impingement, MRI evaluation most commonly demonstrates articular-sided rotator cuff tears at the supraspinatus-infraspinatus interval and lesions of the posterosuperior glenoid labrum. Several studies have confirmed the diagnostic efficacy

of noncontrast MRI in the detection of these lesions in overhead athletes.^{23,48,49} Along with these findings, cystic changes of the posterosuperior humeral head have been identified in addition to Bennett lesions and posterior capsular thickening at the level of the posterior band of the IGHL.⁵⁰

However, as many studies have shown, it is important to recognize that abnormal findings on MRI may not necessitate treatment since many MRI findings do not correlate with clinical symptoms.^{24,51,52} Therefore, integration of all information from the patient encounter is necessary to not only arrive at an accurate diagnosis, but to also formulate an effective treatment plan.

CT Scanning

Computed tomography (CT) scanning is rarely indicated for the evaluation of patients with symptomatic internal impingement because most symptomatic lesions involve soft tissue structures that are best viewed with MRI. However, CT scans can be used on occasion for the measurement of humeral and glenoid version that is thought to occur as a result of abnormal physeal strain in skeletally immature throwing athletes in addition to soft tissue adaptations.^{15,53} Three-dimensional CT scanning (and MRI scanning on one occasion⁵⁴) has also been reported to be useful in the precise measurement of potential osseous adaptations that may occur in the shoulder of overhead athletes.⁵⁵

PREVENTION PROGRAMS

Implementation of prevention programs in throwing athletes is the most efficacious manner by which the incidence and prevalence of symptomatic internal impingement can be reduced.

As several studies have shown that a decreased range of motion is associated with an increased risk of injury in overhead throwing athletes,^{35,36} the maintenance of total arc of motion is necessary to help prevent overuse injuries such as symptomatic internal impingement. As such, the combined total arc of motion should be equal bilaterally despite a loss of internal rotation and greater external rotation in the dominant arm.⁵⁷ To achieve this, passive stretching, such as the horizontal adduction stretch to prevent posterior capsular contracture, should be performed to maintain range of motion with an emphasis on the avoidance of “overaggressive” stretching in an attempt to improve mobility.⁴¹ Baseball players and other throwing athletes should be monitored regularly over the course of the season to detect range of motion loss, especially in those experiencing a decline in velocity and/or accuracy.

It is also necessary to maintain adequate strength of periscapular and glenohumeral muscles both during and in between competitive seasons.^{41,58,59} Electromyographic studies have suggested that the glenohumeral external rotators and scapular retractors are especially important in maintaining dynamic stability of the shoulder girdle during the throwing motion.^{60–62} Loss of this dynamic stability can lead to range of motion loss, scapular dyskinesis and subsequent pathologic lesions such as SLAP tears and symptomatic internal impingement. One component of dynamic stability that is often overlooked is the concept of neuromuscular control, including both efferent and afferent stimulation, which is necessary to help prevent injuries in overhead throwing athletes.⁶³ Thus, every prevention program should include neuromuscular stimulation with the

primary goal of achieving simultaneous contraction of opposing muscles to maintain dynamic stability. There are many neuromuscular drills described elsewhere, such as plyometrics, which can be catered to each individual athlete.^{59,64,65}

TREATMENT OPTIONS

Nonoperative Management

Typically, conservative management is recognized as the treatment of choice in patients with atraumatic shoulder injuries, particularly for those patients with symptomatic internal impingement. Most patients benefit from rest, cryotherapy and oral anti-inflammatory medications in addition to a structured, supervised physical therapy regimen. When GIRD is noted on physical examination, physical therapy should include posterior capsular stretching along with subscapularis and periscapular muscle strengthening.^{9,11,63} In a study by Burkhart et al,¹¹ symptomatic high-level tennis players were prospectively divided into 2 groups: 1 group performed daily posterior stretching (the “sleeper” stretch, Fig. 5) while the other group did not stretch. The group that performed the stretching exercises showed improved internal rotation capacity and total arc of motion along with decreased pain compared with the control group. Aldridge et al⁶⁷ evaluated the effects of a 12-week posterior capsule stretching program in a series of asymptomatic collegiate baseball players. In their study, the dominant arm showed a significant increase in internal rotation and total range of motion after completion of the daily stretching program. Others have found that posterior capsular stretching also increases acromiohumeral distance, which may prevent bursal-sided partial-thickness rotator cuff tears in throwing athletes.⁶⁶

In addition to stretching therapy, periscapular muscle strengthening may help to decrease upward rotation and internal rotation of the scapula, which are factors often implicated in the development of symptomatic internal impingement and SLAP tears in throwing athletes. Burkhart et al¹¹ treated 96 overhead athletes with isolated SICK scapula syndrome with periscapular muscle



FIGURE 5. Demonstration of the “sleeper stretch” that is purported to stretch the posterior capsule, improve range of motion, and help restore scapular kinematics.^{11,66} (Adapted from Braun et al²⁷ with permission.) Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

strengthening. In their study, the rate of return to throwing activities was 100% after approximately 4 months of scapular rehabilitation with a marked decrease in symptoms. In addition, Tyler et al³⁹ found that the combination of posterior capsular stretching and periscapular muscle strengthening increased internal rotation capacity and reduced pain associated with symptomatic internal impingement in the majority of patients.

After the resolution of pain and adequate posterior capsular stretching, subscapularis strengthening and periscapular muscular strengthening has taken place, a supervised throwing regimen of gradually increasing intensity may begin with a heightened emphasis on proper throwing mechanics. It is especially important to maintain the humerus within the scapular plane to prevent scapulohumeral hyperangulation and the recurrence of symptoms.

Operative Management

The indications for surgical intervention most often includes the failure of conservative management to resolve signs and symptoms of symptomatic internal impingement or the inability to return to competition despite 4-6 months of structured rehabilitation. In rare cases, the presence of larger rotator cuff tears or symptomatic type SLAP lesions may be an indication for early operative treatment.

Because physical examination findings in the alert patient with symptomatic internal impingement are unreliable, the findings of bilateral examination under anesthesia (EUA) should direct the final therapeutic procedure. It is especially important to identify any signs of instability that may be present such that concomitant pathologies that may be encountered during diagnostic arthroscopy can be anticipated.

Rotator Cuff Tears

Several treatment options have been proposed for the treatment of rotator cuff tears associated with symptomatic internal impingement. Depending on the findings at diagnostic arthroscopy, the partial-thickness tear can be either debrided or repaired. Many surgeons advocate the “50-50 rule” for both athletes and nonathletes alike: tears that involve < 50% of the tendon thickness should undergo debridement while tears that involve > 50% of the tendon thickness should undergo rotator cuff repair.⁶⁸ Others have also suggested that partial-thickness rotator cuff tears should simply be debrided since return to throwing after repair can be very challenging in high-level athletes.^{8,69}

Sonnery-Cottet et al⁸ followed 28 tennis players with partial-thickness articular-sided tendon avulsions (PASTA lesions) that were treated with debridement alone for approximately 46 months. In their study, 23/28 players (82%) were satisfied with the result of surgery and 22/28 players (79%) returned to their previous level of competition; however, 20/22 players (82%) who returned to sport reported persistent residual pain with competition. Reynolds et al⁶⁹ reported on a series of 82 elite pitchers who underwent debridement for small partial-thickness rotator cuff tears. Although 67/82 of players (82%) returned to play, 30/67 players (45%) were unable to return to their previous level of competition. Other authors have demonstrated success with repair of the partial-thickness defect by either completing the tear and repairing it with a single- or double-row technique^{52,70,71} or by performing a PASTA-type repair in which a side-to-side sutures are placed

followed by abrasion of the tendon insertion site on the greater tuberosity.^{35,72} Bursectomy can also be performed in throwing athletes when there is evidence of subacromial inflammation; however, the addition of anterior acromioplasty in patients with symptomatic internal impingement is generally not recommended owing to unsatisfactory clinical outcomes.^{35,73}

Labral Tears

Posterosuperior labral tears are a common finding in patients with signs and symptoms of symptomatic internal impingement. Although arthroscopic debridement of posterosuperior labral tears in overhead athletes is a widely accepted treatment method, several studies have shown unsatisfactory clinical results. Payne and Altchek⁷⁴ performed arthroscopic debridement in 41 overhead athletes with symptomatic internal impingement. In their study, patients who underwent debridement for posterior labral tears rarely returned to their preoperative level of competition. Similarly, Meister et al¹⁴ reported that only 55% of overhead athletes (10/18) returned to competition after debridement of posterosuperior labral and rotator cuff tears. Despite these results, posterior labral repair in overhead athletes is rarely indicated unless there is unequivocal evidence of posterior glenohumeral instability.

In addition to posterosuperior labral fraying and tearing, some authors have reported the concomitant presence of anterior labral lesions along with SLAP tears in many of these patients.^{5,17,35} In fact, Burkhart et al¹⁰ argued that the SLAP lesion is the most common pathologic finding in symptomatic overhead athletes. Thus, the superior labrum should always be assessed even when the presence of a clinically significant SLAP tear is not suspected based on information obtained from the history, physical examination and subsequent imaging studies. When a significant SLAP tear is present, repair is typically performed before considering operative management of anterior instability as the SLAP lesion itself may be responsible for glenohumeral microinstability or “pseudolaxity,” which is often detected by EUA.

Several studies have reported the clinical outcomes after repair of type II SLAP lesions in overhead athletes.^{75–77} In a retrospective review by Neuman et al,⁷⁶ 30 overhead athletes who underwent repair of symptomatic type II SLAP lesions were followed for a mean of 3.5 years. Of the 30 athletes, the overall satisfaction rate was reported to be 93.3%; however, clinical outcome scores were significantly worse in athletes who participated in baseball or softball. Park et al⁷⁷ followed 24 overhead athletes who underwent type II SLAP repair for a mean of 45.8 months. In their study, only 12 (50%) athletes returned to overhead sports, the majority of whom were not baseball players. In addition, the investigators found that labral integrity, as determined by postoperative CT arthrography, did not improve the rate of return to overhead sports. Neri et al⁷⁵ and van Kleunen et al⁴⁷ also found that the presence of partial-thickness rotator cuff tear in addition to the SLAP lesion significantly affected the rate of return to preoperative level of overhead competition. These studies suggest that return to previous level of competition after SLAP repair, especially those with concomitant partial-thickness rotator cuff tears, is very difficult to achieve even in the most elite athletes. In a few studies, primary biceps tenodesis has been found to be a reliable alternative to biceps reinsertion with satisfactory outcomes.^{78,79} However,

further studies need to be conducted to more clearly define the potential role of primary biceps tenodesis in overhead athletes with type II SLAP tears.

Anterior Instability

Although the precise role of anterior laxity and/or instability in the development of symptomatic internal impingement has not been determined, many studies cite unrecognized microinstability as a possible mechanism of failure of debridement alone in the treatment of rotator cuff and labral defects.^{14,80} Payne and Altchek⁷⁴ reported only a 25% rate of return to pre-morbid level of competition and a 37% satisfaction rate in a series of patients with anterior instability and concomitant internal impingement lesions that were treated with debridement alone. Paley et al⁵ suggested performing capsulolabral reconstruction in addition to debridement to improve outcomes. Several other studies have found a much higher rate of return to sport and satisfaction after surgically addressing the anterior capsule.^{2,81–83} Levitz et al⁸³ compared the rate of return to play in patients with symptomatic internal impingement treated with debridement alone or in combination with anterior thermal capsular shrinkage. In the group that underwent debridement alone, the rate of return to previous level of competition was 67% compared with 90% in the group that also received thermal capsulorrhaphy. Although most studies advocate suture capsulorrhaphy due to the high rate of chondrolysis associated with thermal capsulorrhaphy,^{84–87} this study highlights the importance of addressing anterior capsular laxity in patients with symptomatic internal impingement.

Posterior Capsule Contracture

Burkhart et al^{9–11,25} contend that contracture and thickening of the posterior band of the IGHL is responsible for initiating the cascade of events that eventually lead to GIRD, symptomatic internal impingement, and SLAP tears in overhead athletes. Although physical therapy is most often successful, arthroscopic capsular release is indicated when supervised physical therapy, including “sleeper stretches,” fails to eliminate GIRD and symptoms associated with symptomatic internal impingement.

Bennett Lesions

The surgical management of Bennett lesions is still controversial. Although some authors argue that operative management is unnecessary,¹³ others suggest that when the lesion is present in combination with posterior shoulder pain, debridement should be performed.^{14,62} Yoneda et al⁸⁰ reported on a series of 16 patients with symptomatic internal impingement who, in combination with other soft tissue procedures, underwent arthroscopic removal of the Bennett lesion (so called “Bennett-plasty”). In their study, 88% of patients were satisfied with the procedure after a minimum 1-year follow-up period. Meister et al¹⁴ found satisfactory results after resection of Bennett lesions in 11 patients with large enthesophytes (>100 mm²). Despite these results, many surgeons choose not to address the Bennett lesion since performing such procedures has not been shown to improve outcomes.

Osteochondral Lesions of the Humeral Head

During diagnostic arthroscopy, the humeral head should be placed through a large range of motion to identify any osteochondral lesions of the humeral head. Most

commonly, these lesions occur on the posterosuperior aspect of the humeral near the insertion site of the supraspinatus and/or infraspinatus tendons. At least one study found that most lesions were < 1 cm in diameter with grade I or II Outerbridge changes.⁵ Although debridement can be performed, these lesions often do not require treatment.³⁵ Currently, there are no studies that have documented an improvement in outcomes after debridement or microfracture of these small chondral defects.

Osseous Adaptations

Crockett et al³¹ originally described the humeral head and glenoid retroversion that can occur in young baseball players who eventually develop symptomatic internal impingement. Rarely, derotational osteotomies can be performed as a salvage procedure after failure of previous arthroscopic management. In a study by Riand et al,⁸⁸ 20 patients underwent derotational osteotomy of the proximal humerus along with myorrhaphy of the subscapularis muscle for the treatment of internal impingement. Sixteen of the 20 athletes (80%) returned to their previous sport at, or slightly below, their preinjury level of competition.

Surgical Rehabilitation

Specific rehabilitation protocols after surgical intervention of the throwing athlete's shoulder depends on the specific pathologic lesions that were encountered and the preferences of the treating surgeon. After the repaired tissues have healed, it is necessary to begin a rehabilitation program emphasizing range of motion, strength, endurance, dynamic stability, and neuromuscular control.⁴¹ The reader is directed to review the manuscript by Reinold et al⁴¹ for more detailed information on specific rehabilitation protocols for the throwing athlete.

SUMMARY

Symptomatic internal impingement is a complex disorder, most commonly found in throwing athletes, that can result in articular-sided rotator cuff tears, labral tearing, SLAP lesions, and scapular dyskinesis. Patients typically present with diffuse, nonspecific posterior shoulder pain with unyielding physical examination findings. Noncontrast MRI is currently the gold standard imaging modality for the diagnosis of articular-sided rotator cuff tears, labral lesions, and other findings associated with symptomatic internal impingement. First-line treatment for the disorder is physical therapy, emphasizing posterior capsular stretching along with subscapularis and periscapular muscle strengthening. If non-operative management fails to reduce or eliminate symptoms, operative intervention can be considered in selected patients.

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