Internal Impingement: A Review on a Common Cause of Shoulder Pain in Throwers

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Abstract: Internal impingement refers to a condition classically occurring in younger, active overhead athletes. This has been postulated to occur due to the many. The repetitive nature of the overhead athlete’s activities under extreme loading conditions at the limits of functional shoulder motion result in tremendous strain. Over time, this results in both soft tissue and osseous adaptations to the normal morphology of the glenohumeral joint. Internal impingement occurs when these morphological changes lead to abnormal contact between the undersurface of the rotator cuff tendons and the posterior margin of the glenoid, resulting in a painful shoulder.

Keywords: internal impingement; shoulder; throwers

Introduction

Internal impingement refers to a condition classically occurring in younger, active overhead athletes. This has been postulated to occur due to the many. The repetitive nature of the overhead athlete’s activities under extreme loading conditions at the limits of functional shoulder motion result in tremendous strain. Over time, this results in both soft tissue and osseous adaptations to the normal morphology of the glenohumeral joint. Internal impingement occurs when these morphological changes lead to abnormal contact between the undersurface of the rotator cuff tendons and the posterior margin of the glenoid, resulting in a painful shoulder. This pathologic contact is distinct from asymptomatic contact between the posterior glenoid and the rotator cuff, and may result in injury to the labrum or rotator cuff. Thus, this entity is defined by the pathologic biomechanics of repetitive throwing rather than the constellation of various pathologies (rotator cuff tears, labral tears, anterior laxity) that the patient may present with. This injury is distinct from the subacromial impingement originally described by Neer et al. In addition, Jobe et al described instability-associated impingement in athletes, and other authors have improved our understanding of the etiology of pain in the overhead athlete. This article reviews the literature and current understanding of internal impingement, including the limitations of available evidence and treatment strategies.

Historical Perspective

In 1959, Bennett first described posterior shoulder pain in the throwing arms of professional baseball players, attributing the symptoms to inflammation of the posterior shoulder capsule and the insertion point of the triceps tendon. The pain was associated with a radiographic finding called the Bennett lesion, which refers to exostosis at the postero-inferior glenoid rim. In 1977, Lombardo et al further described an open approach to the treatment of posterior shoulder pain in throwers, finding both ossification of the posterior capsule and increased fibrosis of the surrounding tissues in the affected shoulder. This provided visual evidence of the Bennett lesion, but did not offer further insight into possible treatment options for the pain.

In 1985, Andrews et al reported on the debridement of partial supraspinatus tears in overhead athletes that were discovered on arthroscopic examination. On follow-up, 85% of the patients returned to premorbid athletic condition. The authors concluded that the debridement process aided in the healing of the partial rotator cuff, but they did not offer a mechanism of injury. Instead, Andrews et al observed that the pain occurred primarily during throwing, and suggested that the repeated stresses placed on the shoulder during the throwing motion caused the injury.
Posterior shoulder pain was first associated with posterosuperior glenoid impingement and anterior instability of the shoulder in 1989.11 Anterior capsulolabral reconstruction with appropriate rehabilitation was performed in 25 patients presenting with this constellation of symptoms, with excellent results reported in 68% of patients, and good results reported in 24%.12 In this subset of patients, Jobe et al12 noted the failure of subacromial decompression in throwers with symptoms of impingement, further distinguishing this condition from that of classical subacromial impingement. The authors offered a mechanism for these pathologies, believing that anterior instability of the shoulder led to capsular stretch resulting in the symptoms of impingement. The authors also noted injuries to the superior and inferior labrum, rotator cuff tendons, greater tuberosity, inferior glenohumeral ligament, and superior glenoid bone.

In 1991, Walch et al13 reported single case of a young thrower who developed a partial tear on the deep surface of the supraspinatus tendon against the posterosuperior glenoid.13 With the arm abducted and externally rotated, the patient developed pain, and the partial tear was visualized by arthroscopy. Walch et al1 then examined 17 patients with undersurface rotator cuff tears treated by arthroscopy, and provided the first clinical evidence to support internal impingement as a cause of shoulder pain.

Biomechanics
The forces applied to the shoulder by the overhead athlete can lead to a variety of pathologic changes in the labrum, biceps, rotator cuff, capsule, and glenoid. Asymptomatic contact between the rotator cuff tendons and the posterosuperior glenoid has been demonstrated in cadaveric studies, thus contact alone may not be enough to cause the disease.3,4 The mechanics of the different phases of throwing have been studied extensively, and poor throwing technique has been shown to result in shoulder disease.14,15 Muscle fatigue and imbalance can also affect mechanics, leading to humeral hyperextension in the late-cocking phase of throwing, a condition in which the rotator cuff cannot completely resist the large acceleration forces. This may lead to damage of the posterior capsulolabral structures.3 During the follow-through phase of throwing, rapid deceleration of the arm can lead to abrasions of the rotator cuff on the posterosuperior glenoid and scapular dysfunction, resulting in symptoms known as “dead arm” syndrome.16,17 This condition is further permitted by the development of anterior capsular microinstability, although some believe that the anterior laxity found in internal impingement is actually a pseudolaxity, noting a functional lengthening of the anterior capsule.18 The “peel-back” mechanism also has been proposed, in which the posterosuperior capsular contraction leads to translation of the humeral head. The biceps and labrum may then “peel off” of the glenoid, implicating a hyper-twist mechanism because of the large sheer forces.

Pathophysiology
Internal impingement can present as a constellation of pathologic processes, including partial- or full-thickness rotator cuff tears; anterior or posterior capsular injury; labral tears; glenoid chondral erosion; chondromalacia of the posterosuperior humeral head; and biceps lesions. Moreover, the absence of these lesions does not exclude a diagnosis of internal impingement. Rather, the changes that occur as a result of pitching lead to excess stress being placed on several of the stabilizing structures of the glenohumeral joint. These changes can occur in the overhead athlete as a result of shoulder stress from the throwing motion. Repetitive overhead activity at high velocity allows for adaptations in shoulder morphology. In the throwing arms of baseball pitchers, a maximal internal rotation of 7000° per second has been observed, which is the fastest of all overhead athletes.19 The throwing mechanics have been extensively detailed, and the distraction force acting on the posterior-inferior capsule has been shown to be > 750 N during the follow-through phase.20

As a result of these forces acting on the shoulder, adaptation occurs within the joint. In the shoulders of elite overhead athletes, there is increased glenohumeral external rotation, anterior capsular laxity, and increased humeral head and glenoid retroversion.4,5,12,21-23 In addition, the associated posterior capsular contracture that is often seen in overhead athletes results in a glenohumeral internal rotation deficit (GIRD). Glenohumeral internal rotation deficit has been documented in overhead athletes with superior labral anterior to posterior (SLAP) II tears, as proven by arthroscopy.18 The association between GIRD and the Bennett lesion is unclear. In one study of professional baseball pitchers, the authors noted a 22% prevalence of Bennett lesions in 55 asymptomatic pitchers, demonstrating that the lesion is a common finding, but failing to define its role in internal impingement of the shoulder. Because of this high rate of asymptomatic Bennett lesions and past failure of treatment options targeting the lesion, patients with posterior shoulder...
pain and an identified Bennett lesion should be worked up for concomitant pathology.

Other studies have investigated the kinematics of throwing, shoulder rotation, and adaptation of the joint using cadavers. To examine the effects of capsular changes in overhead athletes, Grossman et al.\(^2\) used a cadaveric model of overhead athletes, and concluded that the shoulder allowed for greater external rotation and increased anterior capsular laxity in response to posterior capsular contraction. In a small prospective study of patients with internal impingement, Tirman et al.\(^3\) noted anterior instability. However, the question remains whether this finding should be identified as a pathologic or an adaptive process, resulting in instability or subluxation.

Injury to the rotator cuff tendon can also present with symptoms of internal impingement. Partial-thickness, articular-sided rotator cuff tears have been described. The likely mechanism of this injury is due to repetitive microtrauma of the rotator cuff, particularly during the deceleration phase of the throwing motion in combination with capsular laxity.\(^7\)\(^8\)\(^9\)\(^10\)\(^11\)\(^12\)\(^13\)\(^14\)\(^15\)

Several studies of overhead athletes demonstrate the high rate of concurrence of articular-sided rotator cuff tears, with the symptoms and the presence of such tears as a key finding in the diagnosis of internal impingement.\(^1\)\(^2\)\(^3\)\(^4\)\(^5\)\(^6\) However, the importance of these deep-surface rotator cuff tears has been refuted, as Connor et al.\(^7\) reported that 40% of asymptomatic dominant shoulders in 20 elite overhead athletes demonstrated either partial-or full-thickness tears on magnetic resonance imaging (MRI) compared with no detection of such lesions in the nondominant shoulders. With this knowledge of the prevalence of asymptomatic tears, Yamanaka and Matsumoto\(^8\) examined the natural history of partial rotator cuff tears in 40 older patients by arthrography, reporting that 20% of the tears healed or decreased in size, 52.5% increased in size, and 27.5% progressed to full-thickness tears.\(^9\) Again, the study population had a mean age of 61 years, and to date, we know of no studies characterizing the natural history of partial rotator cuff tears in young, active overhead athletes.

Injuries to the superior labrum are well described in the literature; however, there is no clear evidence on whether the lesion is diagnostic or essential for the occurrence of internal impingement. Walch et al.\(^10\) described a 71% incidence of posterosuperior labral lesions in 17 patients with internal impingement. Similarly, Paley et al.\(^11\) used arthroscopy to identify posterosuperior labral fraying in 88% of 41 overhead athletes with internal impingement. Kaplan et al.\(^12\) conducted a retrospective review of MRI scans of 9 symptomatic patients, and all were found to have posterosuperior labral lesions. In a larger prospective cohort study of 376 patients undergoing arthroscopic shoulder surgery, 74% of those with internal impingement also had type II SLAP lesion, leading the authors to conclude that internal impingement may lead to labral tears. However, in a study of 10 college baseball athletes, Halbrecht\(^13\) identified 3 superior labral tears in the throwing shoulders of the athletes, but this result had no clinical correlation to instability on examination.

### Clinical and Radiographic Assessment

The assessment of a patient with possible internal impingement should begin with a detailed history. The patient may describe the onset of posterior shoulder pain, particularly during the late-cocking phase of throwing, when the arm is in 90° of abduction and full external rotation. The pain is common in overhead athletes such as pitchers and tennis players. Jobe\(^14\) developed a classification scheme to further distinguish between the varying severities of internal impingement (Table 1). Muscular asymmetry and differing degrees of laxity are common in the overhead athlete when comparing the dominant and nondominant shoulders. As described previously, internal impingement can lead to increased global laxity and anterior translation of the glenohumeral joint. Myers et al.\(^15\) examined the range of motion of the glenohumeral joint in throwers with pathologic internal impingement and found that these athletes had 10° to 15° of internal rotation deficit and increased posterior shoulder tightness compared with controls. This evidence was supported by a study of collegiate baseball players with shoulder pain who had a smaller total arc of motion and an internal rotation deficit in the affected dominant shoulder when compared with both the nondominant and asymptomatic control players’ shoulders.\(^16\)

The glenohumeral joint may have subtle instability in internal impingement. However, it can be extremely difficult
for even the most experienced physician to distinguish between physiologic asymmetric laxity of the joint and pathological microinstability. This is because virtually all throwers have some laxity. To maximize velocity, the shoulder requires more external rotation in the late-cocking phase of throwing. This allows for greater arc of acceleration during the wind-up. This is accomplished by some of the aforementioned morphologic adaptations, but anterior laxity is virtually a prerequisite. Thus, most high-level pitchers will have some laxity. However, the important factor to distinguish is when this laxity leads to instability or clinical symptoms. Some signs of the pathologic process include a so-called “dead arm,” the feeling of shoulder and arm weakness after throwing, and a subjective sense of slipping of the shoulder. These historic events can be further evaluated using the posterior impingement test, in which the patient’s shoulder is placed in 90° to 100° of abduction, 110° to 115° of extension, and then moved into maximal external rotation; a positive test reproduces the athlete’s pain (Figure 1).40,41 This test has been found to be even more sensitive when performed on athletes with noncontact injuries.33

The table below illustrates the clinical classification of internal impingement by Jobe.

### Table 1: Jobe Clinical Classification of Internal Impingement

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptoms</th>
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<tr>
<td>Stage I: early</td>
<td>Shoulder stiffness and a prolonged warm-up period; discomfort in throwers occurs in the late-cocking and early acceleration phases of throwing; no pain is reported with activities of daily living.</td>
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<tr>
<td>Stage II: intermediate</td>
<td>Pain localized to the posterior shoulder in the late-cocking and early acceleration phases of throwing; pain with activities of daily living and instability are unusual.</td>
</tr>
<tr>
<td>Stage III: advanced</td>
<td>Similar to those in stage II who have been refractory to nonoperative treatment modalities.</td>
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The next step in diagnostic evaluation of a patient with posterior shoulder pain is to obtain standard radiographs of the affected arm. These should consist of internal and external anteroposterior, scapular Y, and axillary or West Point views, with particular attention to gross malalignment, although subtle findings are more likely. The Bennett lesion can often be identified on axillary or West Point views.5,42 Attention should also be paid to the base of the greater tuberosity in the anteroposterior external rotation view because this radiograph can demonstrate sclerosis at the base of the tuberosity.

The next modality useful for diagnosis of internal impingement is MRI. Both sensitivities and specificities of > 95% have been shown for MRI in detection of rotator cuff and labral tears.43,44 Magnetic resonance imaging is particularly useful for the diagnosis of intrasubstance tears, which are not easily visualized on arthroscopy. In patients with internal impingement, MRI may show evidence of tears, especially on the articular surface of the rotator cuff and the intersection of the infraspinatus and supraspinatus tendon insertions to the humeral head. The tear is often not full thickness, but a small percentage of the size of the tendon. As tears become smaller, the detection of such lesions becomes more difficult using standard MRI. Injection of gadolinium into the joint can aid in diagnosis and increase the rate of detection to 84% for small tears under 25% of the total rotator cuff diameter.33

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**Figure 1.** Posterior impingement sign. This is performed with the patient supine and the affected arm in 90–100° of abduction, maximal external rotation, and 10° of forward flexion.
Risks involved in this procedure include an intra-articular injection, pain, and reaction to the contrast agent; however, noncontrast or high-resolution MRI can be effectively used to diagnose superior labral lesions without these associated risks. In patients with clinically or operatively diagnosed internal impingement, imaging studies have shown undersurface tears of the supraspinatus and infraspinatus tendons as well as cystic changes of the posterior humeral head with evidence of posterosuperior labral pathology.

A patient presenting with posterior shoulder pain due to internal impingement may present with this constellation of findings. In addition, the Bennett lesion may be demonstrable by imaging as mature periosteal bone formation at the scapular attachment of the posterior capsule (Figure 2). Moderate-to-severe posterior capsular contracture may be present at the level of the posterior band of the inferior glenohumeral ligament. Pressure on the posterior glenoid with the arm abducted and externally rotated may lead to remodeling of the glenoid, including subchondral depression and large osteophyte formation. The resulting anatomical changes can narrow the spinoglenoid notch near the suprascapular neurovascular bundle. The clinician must be aware of the prevalence of asymptomatic lesions on MRI. In a study of the throwing shoulders of college baseball pitchers, 4 of 10 pitchers had abnormal signal changes in the rotator cuff, and more importantly, no correlation between imaging and physical examination or symptoms was appreciated. This study is reinforced by others who have demonstrated partial cuff tears in completely asymptomatic patients, thus stressing the need for clinical correlation between imaging, history, and physical examination.

Computed tomography (CT) scans are rarely indicated for imaging patients with internal impingement of the shoulder because the condition is mainly one of soft tissue changes that are better identified by MRI. However, CT is the gold standard for the diagnosis of humeral and glenoid version, a condition that is important in both internal impingement and rotator cuff tears. Newer, 3-dimensional reconstructions of CT data have also proven useful for identifying osseous anatomy and adaptations.

**Treatment Strategy**

Conservative management of internal impingement is an appropriate initial approach, particularly in patients who do not report an acute traumatic event. In early disease, the pain can be poorly localized, and the shoulder is stiff, which should be treated with rest and nonsteroidal anti-inflammatory drugs. If the pain is more localized to the posterior shoulder, treatment options include avoidance of throwing for at least 4 to 6 weeks with appropriate physical therapy. The natural history of the condition was studied in 39 professional baseball pitchers who entered spring training with GIRD. Over the course of the season, 60% of the athletes developed shoulder injuries that prevented them from pitching. Posterior shoulder stretching exercises can be employed as both a therapeutic and protective regimen. Daily stretching routines in major league pitchers resulted in no innings lost to injury. Daily posterior capsular stretching exercises in elite tennis players using the "sleeper stretch" resulted in an increase in internal rotation and total rotation of the shoulder, as well as 38% less incidence of shoulder injury (Figure 3). However, caution must be taken when stretching the anterior capsule and inferior glenohumeral ligament because this may result in an exacerbation of anterior laxity, which could be pathologic. Four distinct phases of rehabilitation have been described (ranging from diminishing symptoms to plyometric strengthening), with the aim to improve anterior laxity by strengthening the dynamic shoulder stabilizers. The importance of proper mechanics in young throwers is not to be overlooked as a preventative measure to the development of shoulder pain in athletes.
Injections of the shoulder have often been used as diagnostic tool for the Bennett lesion, typically using a local anesthetic. The pain may be relieved with throwing, but may not be completely eliminated, suggesting the presence of concurrent pathology. To our knowledge, there have been no studies on the role of injections in internal impingement. Clinicians must exercise caution, especially in young, active patients, and must weigh the benefit of limiting the inflammatory cascade with the risk of permanent tendon damage.

Partial tears of the rotator cuff, as diagnosed by MRI, can occur with an adjacent tear and abrasion of the labrum; this constellation of findings is called a kissing lesion (Figure 4). Surgically, this can be managed by debridement or repair, with or without simultaneous acromioplasty. A general rule followed by surgeons is that tears > 50% of the diameter require surgical repair. Following primary repair, both full- and partial-thickness rotator cuff tears perform and heal similarly on clinical follow-up. Another method described for partial tears is to mobilize the soft tissues by completing the tear and performing a 2-row repair.

The shoulder pathology in overhead athletes is not limited to the rotator cuff; patients may also present with posterosuperior labral detachment, degenerative labral changes, fraying and tenosynovitis of the biceps, and grades II to IV changes in the humeral head. Studies have demonstrated an unsatisfactory outcome with this procedure in patients with posterior labral tears with increased anterior glenohumeral translation who underwent arthroscopic debridement alone. Similarly poor results were noted in patients with known Bennett lesions who received arthroscopic removal of the lesion and rotator cuff debridement. The role of the Bennett lesion in both the etiology and treatment of internal impingement is not entirely clear. Earlier studies showed success with debridement of the lesion; however, a prospective cohort study of 16 baseball players with pain at the posterior shoulder relieved by local injection demonstrated that pain was improved or resolved after operative removal, although only 11 of the 16 players resumed previous activity. Of those who with unsatisfactory outcomes, additional lesions were identified in the shoulder as the likely cause of symptoms.

The subtle anterior laxity that develops in the affected shoulder of patients with internal impingement has been identified as a cause of treatment failure. Jobe, Andrews and Dugas, and Montgomery and Jobe recommend operative management of this joint laxity through anterior capsulolabral reconstruction, believing this may be the cause of the pathomechanics leading to further injury. Altchek and Dines developed a less invasive procedure for capsular repair after concern for the morbidity of an open approach, aiming to restore capsular tension without functional range of motion limitations.
Most of the surgical options described are aimed at treating the soft tissue abnormalities associated with internal impingement, though some have investigated procedures focused on the bones of the shoulder. For example, Riand et al. described a derotational osteotomy with myorraphy of the subscapularis muscle as a treatment option. In these patients, the persistent pain after articular debridement was believed to be caused by increased humeral retroversion and prevented the patients from returning to their throwing sports. After the osteotomy, 55% of the patients were able to resume their sport at full level.

**Summary**

From the authors’ perspective, the literature from the past 2 decades has demonstrated increasing awareness and knowledge of the causes of posterior shoulder pain and internal impingement of the shoulder. More data and long-term follow-up studies can be expected, especially investigating the different treatment options. To date, we know of no prospective studies can be expected, especially investigating the different outcomes. Looking to the future, further studies into the role of the Bennett lesion, glenoid version, and a standardized classification system of these shoulder injuries should help to improve our understanding of internal impingement the overhead athlete.

**Conflict of Interest Statement**

Steven Behrens, MD, Jeffrey Compas, BA, Matthew E. Deren, BS, and Mark Drakos, MD disclose no conflicts of interest.

**References**

Behrens et al