Current Concepts Review

Internal Impingement of the Shoulder in the Overhead Athlete

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- Internal impingement of the shoulder refers to a constellation of pathologic conditions, including, but not limited to, articular-sided rotator cuff tears, labral tears, biceps tendinitis, anterior instability, internal rotation deficit, and scapular dysfunction.
- Physiologic adaptations to throwing include increased external rotation, increased humeral and glenoid retroversion, and anterior laxity, all of which may predispose an individual to internal impingement.
- Nonoperative treatment should always be attempted first, with a focus on increasing the range of motion and improving scapular function.
- When an operative intervention is chosen, it is important to address microinstability in order to have a good outcome and prevent failure.

The term *internal impingement* describes pathologic contact between the margin of the glenoid and the side of the rotator cuff that faces the articular surfaces of the shoulder[1,2]. This entity classically presents in younger, active overhead athletes. Contact between the glenoid and the rotator cuff has been described in asymptomatic shoulders without evidence of pathologic change[3,4]. However, an overhead athlete's shoulder typically performs repetitive activities at the limits of the functional arc of motion under extreme loading conditions. Over time, these conditions have been shown to result in both osseous and soft-tissue adaptations[5-10]. In this population, the contact between the posterosuperior aspect of the labrum and the surface of the rotator cuff facing the glenoid may become pathologic, resulting in injury to the labrum and the rotator cuff. This condition is defined as internal impingement.

Over the past decade, several biomechanical and clinical studies have enhanced our understanding of the pathophysiology of shoulder pain in the overhead athlete. The classic description of subacromial impingement by Neer et al. and the definition of instability-associated impingement in athletes by Jobe et al. have been combined with the contributions of Walch and others to improve our understanding of internal impingement[2,7,11-15]. The purpose of this review is to clarify the current body of evidence, identify its limitations, and discuss approaches to treating this disorder.

Historical Background

The etiology of posterior shoulder pain has been debated for decades. In 1959, Bennett described posterior shoulder pain in professional baseball pitchers and proposed that it was due to inflammation of the posterior aspect of the shoulder capsule from repetitive triceps traction[16]. Radiographic evidence of this lesion had been reported in 1941[17], and the so-called Bennett lesion became classically defined as an exostosis at the posteroinferior glenoid rim.

In 1977, Lombardo et al. described posterior shoulder pain in overhead athletes during the late cocking phase of throwing[18]. In their initial treatment of this problem through an open approach, they noted ossification of the posterior aspect of the capsule as well as excess fibrous tissue. In 1985, Andrews et al. reported on thirty-six athletes, primarily pitchers, with a partial tear of the supraspinatus portion of the rotator cuff[19]. The predominant symptom among these patients was pain during throwing and often it was poorly lo-

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calized. The authors surmised that the tears were a result of the tremendous repeated stresses placed on the throwing shoulder; however, they did not propose a specific mechanism.

In 1989, Jobe et al. described posterosuperior glenoid impingement and associated anterior instability in overhead athletes[11]. They reported on the limited success of subacromial decompression in this population and, in later studies, presented their results of anterior capsulolabral reconstruction[2,7,11,20,21]. On the basis of their success with that procedure in this population, they theorized that anterior instability due to capsular stretch may be the cause of impingement-like symptoms in the overhead athlete. They also noted that overhead athletes with posterosuperior glenoid impingement had an associated injury to one or more of the following structures: the superior or inferior aspect of the labrum, rotator cuff tendon, greater tuberosity, inferior glenohumeral ligament, and superior glenoid bone.

In 1991, Walch et al. reported on impingement occurring between the deep side of the supraspinatus tendon and the posterosuperior edge of the glenoid cavity in a young thrower[15]. This impingement occurred when the arm was abducted and externally rotated, and it was described as being associated with a partial-thickness articular surface tear of the deep side of the rotator cuff as visualized with arthroscopy. In a subsequent series, Walch et al. reported on seventeen patients in whom an undersurface tear of the rotator cuff had been treated with arthroscopic débridement[1]. This study provided the first clinical evidence to support the concept of internal impingement.

Anatomic Changes and Associated Pathologic Conditions

Overhead athletes' successful performance is related in part to the adaptive changes that result in response to their repetitive overhead activities. Baseball pitchers have been shown to have a maximum internal rotation velocity of upwards of 7000°/sec, which is the fastest motion in all overhead athletes[22]. Furthermore, velocity can be optimized by expanding the arc of rotation (increasing external rotation in the late cocking phase of throwing). To this end, several adaptations occur in throwing athletes, including increased glenohumeral external rotation, increased humeral head and glenoid retroversion, and anterior capsular laxity[4,5,8,9,13,20]. As a result, several compensatory and potentially pathologic anatomic changes may occur—specifically, anterior instability and a posterior capsular contracture, which produce a glenohumeral internal rotation deficit. Each of these variants has been implicated in the pathogenesis of internal impingement.

In the context of internal impingement, there is a spectrum of associated pathologic conditions, including partial and full-thickness rotator cuff tears, anterior and posterior capsular injury, labral tears, glenoid chondral erosion, chondromalacia of the posterosuperior aspect of the humeral head, and biceps lesions[3,6,7,18,20,23-31]. Each of these entities may exist alone or as a concomitant pathologic condition. In a series of thirty-six overhead athletes treated arthroscopically for refractory shoulder pain, there was a high prevalence of associated labral tears (100%), partial tears of the long head of the biceps (17%), and biceps tendinitis (8%)[32]. Other authors have described Bankart or anterior capsulolabral lesions in association with internal impingement, with one retrospective study demonstrating anterior labral fraying in 36% of forty-one professional overhead athletes[2,4].

In order to attain a supraphysiologic range of motion, overhead athletes have undergone adaptations in the glenohumeral joint. These changes are manifested by a stretching of the capsular structures and a remodeling of the osseous architecture of the glenohumeral joint. Investigators have noted very large distraction forces of upwards of 750 N that are absorbed by the posteroinferior aspect of the capsule in the follow-through phase of throwing[33]. The magnitude of these stresses causes capsular remodeling, leading to a posterior capsular contracture and a subsequent glenohumeral internal rotation deficit. In a series of arthroscopically proven type-II SLAP (superior labrum anterior and posterior) lesions in overhead athletes, a substantial glenohumeral internal rotation deficit was found in all eighty-one affected shoulders[24,34,35]. Glenohumeral internal rotation deficits have been found in association with the Bennett lesion, which is a posterior extra-articular ossification associated with a scarred capsule. In a recent study of fifty-five asymptomatic professional baseball pitchers, Wright and Paletta reported that 22% had a Bennett lesion[10]. They concluded that the Bennett lesion is a relatively common finding, and its importance within the realm of internal impingement has been questioned. Therefore, it has been strongly recommended that physicians thoroughly search for concomitant pathologic conditions when evaluating posterior shoulder pain in a patient with a Bennett lesion.

In a study of a cadaver shoulder model, Grossman et al. found that a simulated posterior capsular contracture resulted in a significant decrease in internal rotation ($p < 0.05$)[36]. The authors concluded that this condition translates the humeral head posterosuperiorly in the cocking phase of throwing and may be a potential etiology of posterosuperior labral lesions. Additional evidence was offered by Huffman et al., who similarly used a cadaver model to demonstrate posterior humeral translation in a maximally externally rotated shoulder[37]. This suggests that the physiologic adaptation that occurs in a pitcher's throwing shoulder also alters the normal throwing kinematics.

To account for this posterior contracture and increased external rotation, the anterior capsule has commensurate laxity. In a retrospective case series, Tirman et al. reported that six of eight patients with internal impingement had evidence of anterior instability[38]. However, determining the difference between adaptive laxity and pathologic laxity resulting in anterior instability or subluxation is a challenging task. Furthermore, failure to address anterior capsulolabral injuries has been implicated in many of the poor results of the treatment of this disease[28,30,32,39,40].

Partial-thickness articular-sided rotator cuff tears in association with internal impingement have been well described

in the literature[27-31]. Repetitive microtrauma from intratendinous strain with eccentric contraction of the rotator cuff during the deceleration phase of throwing in combination with subtle capsular laxity are likely prominent factors in the pathogenesis of articular surface partial-thickness tears[41]. All of seventeen overhead athletes with shoulder pain in the study by Walch et al. had a partial-thickness rotator cuff tear[1]. Paley et al. described articular-sided rotator cuff fraying as a key finding in patients with internal impingement and reported that it occurred in 93% of forty-one symptomatic throwers[4]. Other authors have described rotator cuff magnetic resonance imaging signal change or abnormality in the context of internal impingement[29]. However, it is important to recognize that a partial-thickness rotator cuff tear in the dominant arm was reported in 40% of twenty asymptomatic elite athletes[42]. Thus, the question arises: When do these tears or presumed tendinosis become symptomatic and when do they progress in size? Yamanaka and Matsumoto reported on forty patients with a partial-thickness articular-sided tear of the supraspinatus tendon observed with arthrography over a one-year period[43]. They found that 20% of the tears either healed or decreased in size, 53% increased in size, and 28% progressed to full-thickness tears. This was an older patient population (average age, sixty-one years). To our knowledge, no one has evaluated the natural history of partial-thickness articular-sided tears in a population of overhead athletes.

Superior labral lesions have been well described in the literature on internal impingement; however, the true incidence is difficult to ascertain on the basis of the limited sample sizes of the studies published to date. Walch et al. presented a case series of seventeen patients with internal impingement in which twelve had a posterosuperior labral lesion[1]. Paley et al. reported a finding of posterosuperior labral fraying on arthroscopy in 88% of forty-one symptomatic overhead athletes[4]. In a retrospective review of the magnetic resonance images and arthroscopic findings in nine throwing athletes, Kaplan et al. found that all patients had a posterosuperior labral lesion[26]. Halbrecht et al. reported that three of ten asymptomatic college baseball players had a superior labral tear with an adjacent paralabral cyst[6]. In a recent prospective series of 376 patients undergoing shoulder arthroscopy, 74% (277) demonstrated internal impingement in flexion, which had a significant association with type-II SLAP lesions ($p < 0.05$)[44]. Internal impingement is often defined, in part, by a posterosuperior labral lesion. It remains unclear if this associated entity is either diagnostic of internal impingement or essential for its occurrence.

Pathomechanics

Cadaver and arthroscopic studies have shown that contact between the rotator cuff and the posterosuperior aspect of the glenoid occurs in asymptomatic individuals[3,4]. However, it has also been established that the repetitive nature of the forces generated by overhead athletes may lead to pathologic changes in the rotator cuff, labrum, biceps, capsule, and glenoid. The mechanics of throwing have been extensively studied, and isokinetic strength profiles have been established for external/internal rotation, abduction/adduction, and abduction/external rotation parameters. Poor throwing techniques and deviation from these ratios may be responsible for the disease within the rotator cuff, labrum, biceps, capsule, and glenoid[45,46].

Some authors have contended that muscle fatigue and imbalance lead to abnormal mechanics of the shoulder—specifically, fatigue-related humeral hyperextension that occurs during the late cocking phase when the rotator cuff muscles cannot completely resist the large acceleration forces generated while pitching. Edelson and Teitz postulated that this repetitive motion may also cause a progressive delamination of the posterior capsulolabral structures by the rotator cuff[3]. In essence, the excessive violent deceleration of the arm in follow-through may cause an abrasive degeneration of the rotator cuff on the posterosuperior aspect of the glenoid. In combination with scapular dysfunction, the disease may manifest as the so-called dead-arm syndrome[47,48]. Moreover, microinstability due to anterior capsular laxity allows this process to continue.

This theory has been refuted by several authors. Burkhart and Morgan proposed that the posterior capsular contracture produces a functional lengthening of the anterior aspect of the capsule[24]. This causes a pseudolaxity rather than a pathologic instability. These authors described a so-called peel-back mechanism in which a contracted posteroinferior aspect of the capsule can lead to pathologic translation of the humeral head and a subsequent SLAP tear. They proposed that the glenohumeral internal rotation deficit caused by the posterior contracture results in a superior and posterior translation of the contact point of the humeral head with the glenoid when the shoulder is in abduction and external rotation. This reduces the cam effect of the humeral head and permits supraphysiologic external rotation and increases peel-back forces. Moreover, Burkhart and Morgan proposed that the pathologic condition in the rotator cuff and the labrum was not an abrasive phenomenon, but rather was a result of a hypertwist mechanism with large shear stresses leading to a fatigue failure of both the rotator cuff and the biceps tendon insertion point of the labrum. Burkhart et al. later went on to assert that internal impingement is not a pathologic condition, but rather a natural restraint to hyperexternal rotation[24,34,35,49]. Despite these insights, there is controversy over the exact pathomechanics of this clinical entity.

Clinical Assessment

A thorough history is a particularly important element in the diagnosis of internal impingement. Very often, the patient reports posterior shoulder pain, particularly in the late cocking phase of throwing. In addition to pitchers and throwers, overhead athletes such as tennis players may have internal impingement. Jobe proposed a classification scheme based on the clinical presentation[7] (Table I).

Overhead athletes, and throwers in particular, often have muscular asymmetry between the dominant and the nondominant shoulder as well as increased laxity of the dominant

TABLE I Jobe's Clinical Classification of Internal Impingement[7]

Stage	Symptoms
I: Early	Shoulder stiffness as well as 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
II: Intermediate	Pain localized to the posterior aspect of the shoulder in the late cocking and early acceleration phases of throwing; pain with activities of daily living and instability are unusual
III: Advanced	Similar to those in Stage II but refractory to nonoperative treatment modalities

shoulder. A patient with isolated internal impingement may have an increase in global laxity or an increase in anterior translation alone. Typically, the dominant shoulder has 10° to 15° more external rotation, 10° to 15° less internal rotation, and greater muscular development than the nondominant shoulder[8]. However, in a recent study, Myers et al. found that a group of eleven throwing athletes with pathologic internal impingement had a greater internal rotation deficit and a larger degree of posterior shoulder tightness when compared with a control group of eleven asymptomatic throwing athletes[8]. This finding was supported by Ruotolo et al., who showed a decrease in the total arc of motion of the dominant arms of collegiate baseball pitchers compared with that in asymptomatic controls[50].

Patients with internal impingement may have subtle instability. They often have asymmetric laxity, but distinguishing pathologic microinstability from physiologic laxity in a thrower can be challenging for even an experienced physician. Several signs that may suggest the presence of this pathologic entity include symptoms such as a dead arm, shoulder weakness after throwing, and a subjective sense of slipping of the shoulder. This sensation frequently occurs without a frank dislocation or subluxation. Apprehension in the provocative position is also a frequent finding. This can be assessed with use of the posterior impingement test, which is performed by placement of the shoulder in 90° to 100° of abduction and 10° of forward flexion followed by maximal external rotation of the arm[31,51] (Fig. 1); reproduction of pain in the posterior aspect of the shoulder is considered a positive result. The sensitivity of this test for the detection of posterior labrum tears and/or tears of the rotator cuff has been reported to be >75%. However, the sensitivity rises to >94% in patients who present with a noncontact injury[29]. Translation in the scapular plane should also be evaluated but may be difficult to assess. As a part of standard arthroscopy, an examination with the patient under anesthesia is performed to allow an accurate measure of instability.

The impingement sign described by Neer[12] is typically absent, while the test described by Jobe[7] is often positive. The Jobe relocation test is performed with the patient supine with the arm in 90° to 100° of abduction and maximal external rotation, and pain is reproduced as the arm is pulled anteriorly.

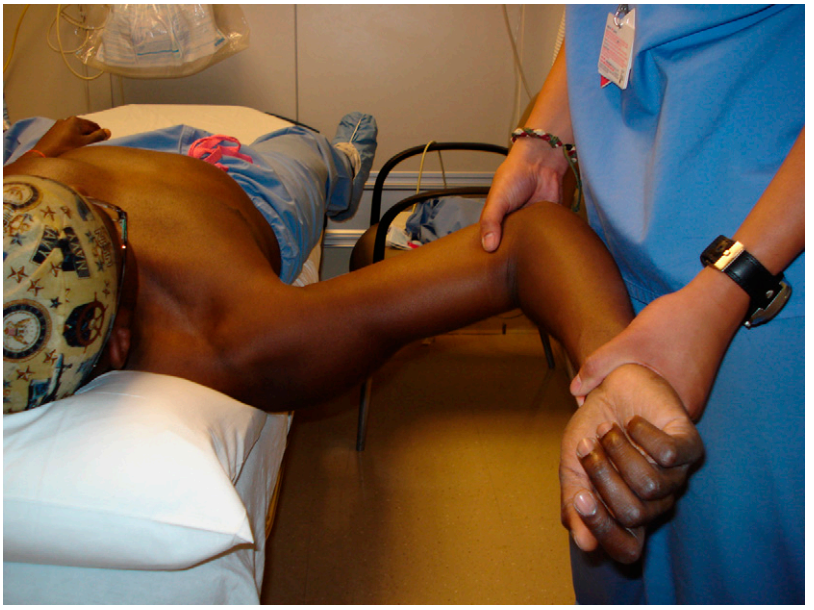

Fig. 1

The posterior impingement sign is elicited with the patient supine and the affected arm in 90° to 100° of abduction, maximal external rotation, and 10° of forward flexion.

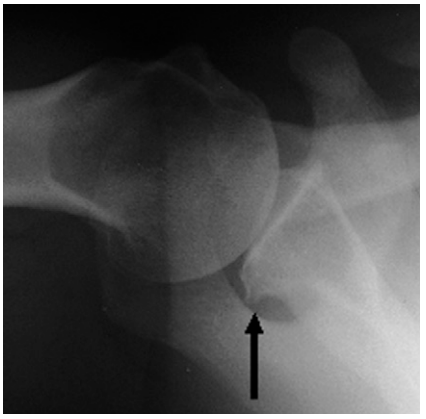

Fig. 2
Axillary radiograph demonstrating the Bennett lesion (arrow) on the posterior aspect of the glenoid.

However, when a posteriorly directed force is applied to the humeral head, the pain resolves. A positive test was speculated to be indicative of decompression of a so-called kissing lesion between the rotator cuff and the glenoid[2]. During examination of patients with internal impingement, it is important to recognize that the impingement often occurs in the setting of concomitant pathologic conditions of the shoulder. SLAP lesions, cysts, bursal-sided rotator cuff tears, and tendinitis are also common in overhead athletes and may present simultaneously.

Radiographic Assessment and Diagnostic Imaging

Standard radiographs including internal and external rotation anteroposterior, scapular Y, axillary, and West Point views are important for the thorough evaluation of shoulder pain. Gross malalignment as well as other subtle findings may be identified. The Bennett lesion, an ossification on the posteroinferior aspect of the glenoid rim, can be seen on axillary or West Point views[16,52] (Fig. 2). One may also see increased sclerosis at the base of the greater tuberosity on an anteroposterior external rotation view.

Magnetic resonance imaging has been used frequently to diagnose pathologic conditions of the shoulder. Its sensitivity and specificity for the detection of labral tears and rotator cuff disease are on the order of ≥95%[53,54]. Magnetic resonance imaging has the advantage of being able to detect intrasubstance tears that may be difficult to visualize with arthroscopy. The findings of magnetic resonance imaging of patients with internal impingement are usually more subtle. The tear is located on the articular side of the rotator cuff, typically at the intersection of the infraspinatus and supraspinatus insertions onto the humeral head (Fig. 3), and may involve only a small portion of the total cross section of the tendon. However, investigators have shown that magnetic resonance imaging enhanced with gadolinium can detect tears involving <25% of the cuff with 84% sensitivity[29]. Other investigators have shown that non-contrast magnetic resonance imaging can predict labral lesions with sensitivities on the order of 98% and have proposed that high-resolution non-contrast magnetic resonance imaging can accurately demonstrate superior labral lesions to aid in preoperative assessment without the risks or morbidity (pain, infection, or reaction to the contrast agent) associated with injection of contrast medium[54]. Recently, Giaroli et al. demonstrated that magnetic resonance imaging of patients with clinically and operatively diagnosed internal impingement shows undersurface tears of the supraspinatus or infraspinatus tendon and cystic changes in the posterior aspect of the humeral head associated with posterosuperior labral pathology[55]. Giaroli et al. asserted that this constellation of findings is diagnostic of internal impingement, and this observation was supported by the work of Kaplan et al.[26].

Additional findings on magnetic resonance imaging of patients with internal impingement include mature periosteal bone formation at the scapular attachment of the posterior aspect of the capsule (the Bennett lesion) and moderate-to-severe posterior capsular contracture at the level of the posterior band of the inferior glenohumeral ligament (Figs. 4 and 5). The chronic pressure placed on the posterosuperior aspect of the glenoid with the arm abducted and externally rotated also creates a characteristic remodeling of the glenoid, yielding subchondral depression, often with a large osteophyte (Fig. 6). In severe cases, this may result in narrowing of the spinoglenoid notch adjacent to the suprascapular neurovascular bundle.

Magnetic resonance imaging may also reveal lesions that are asymptomatic. Halbrecht et al. attempted to correlate the findings of physical examination with those of magnetic res-

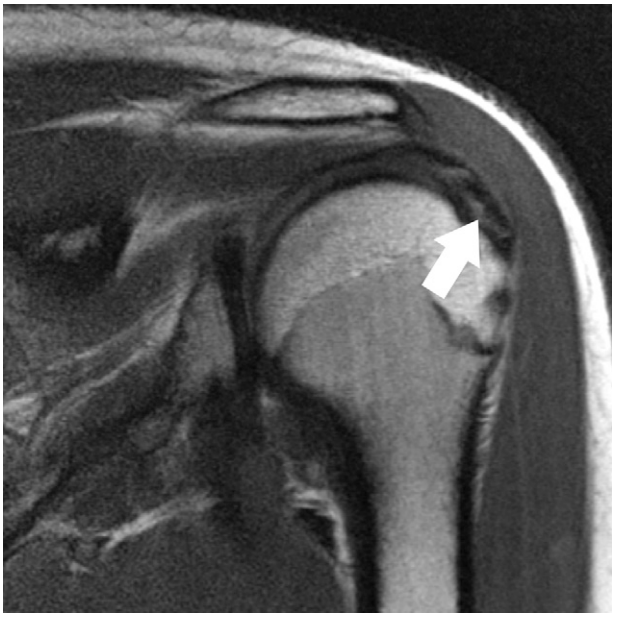

Fig. 3
Oblique coronal fast-spin-echo magnetic resonance image demonstrating a partial-thickness tear (arrow) of the anterior fibers of the infraspinatus tendon.

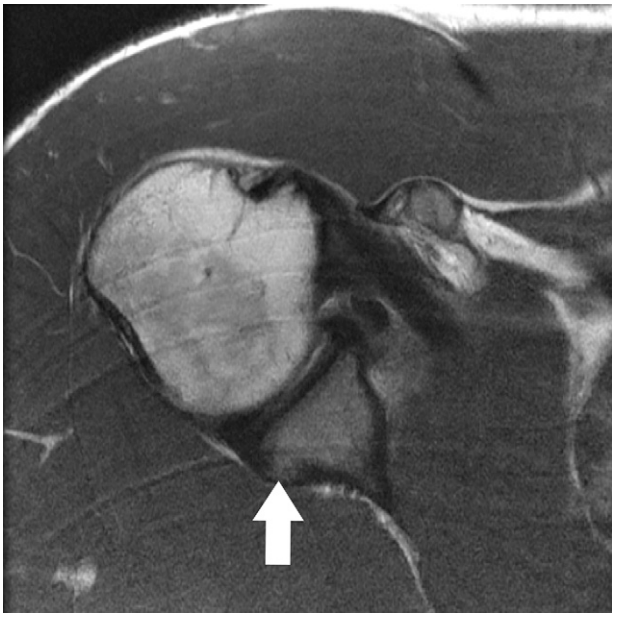

Fig. 4

Axial fast-spin-echo magnetic resonance image demonstrating mature periosteal bone formation at the scapular attachment of the posterior aspect of the capsule, consistent with a Bennett lesion (arrow).

onance imaging of the dominant and nondominant shoulders of asymptomatic college baseball pitchers[6]. Four of ten throwing shoulders had abnormal signal change in the rotator cuff tendons; however, there was no correlation between the positive magnetic resonance imaging findings and the findings on physical examination. Several other studies have also shown partial-thickness rotator cuff tears in asymptomatic shoulders[41,42,56]. These data emphasize the importance of clinical correlation with the findings of the radiographic evaluation.

Computed tomography is rarely used for the evaluation of internal impingement of the shoulder. This is because the variety of soft-tissue lesions, such as those in the rotator cuff, biceps, and labrum, encountered in association with internal impingement of the shoulder is better visualized with magnetic resonance imaging. However, computed tomography is the so-called gold standard for measuring both humeral version and glenoid version, which have been implicated in the etiology of internal impingement and rotator cuff tears[57]. In a study by Osbahr et al., there was a significant correlation between retroversion of the humerus and external rotation of the dominant arm of pitchers ($p < 0.05$)[9]. The authors concluded that rotational changes in the throwing shoulder are due to osseous as well as soft-tissue adaptations. These humeral changes can be measured accurately with use of computed tomography or a semi-axial radiograph as described by Söderlund et al.[58]. In addition, Kwon et al. recently described the accuracy of three-dimensional computed tomography reconstruction images of the glenoid as a means of better understanding its osseous anatomy and possible adaptations[59]. We are not aware of any studies comparing glenoid version between symptomatic and asymptomatic throwers.

Nonoperative Management

Once a diagnosis of internal impingement has been made, the physician should first recommend a trial of nonoperative management. According to Jobe, patients with early stages of the disease report poorly localized pain with stiffness[7]. These patients are nearly always treated conservatively with nonsteroidal anti-inflammatory drugs and rest. Patients with more localized posterior shoulder pain require longer periods of activity cessation—four to six weeks or more. These patients may also benefit from physical therapy. In one study, thirty-nine professional baseball pitchers identified in spring training as having a glenohumeral internal rotation deficit were followed over the course of a season[34]. Sixty percent of the study cohort ultimately sustained shoulder injuries that prevented them from pitching. Because of this study and others, a posterior shoulder-stretching regimen that may be therapeutic as well as protective against future injury has been advocated[34,35,46,49,60]. In a two-year prospective study of high-level tennis players, a group performing daily posterior capsular stretching with use of the so-called sleeper stretch (Fig. 7) and other modalities was compared with a control group who did not stretch[49]. The authors found that the treatment group had increases in internal rotation and total rotation as well as a 38% decrease in the prevalence of shoulder problems. This finding was supported by another study, of major-league baseball pitchers who underwent a daily stretching regimen and lost no innings because of shoulder injury[34]. Caution should be exercised with regard to stretching the anterior aspect of the capsule and the inferior glenohumeral ligament complex as it may exacerbate anterior laxity.

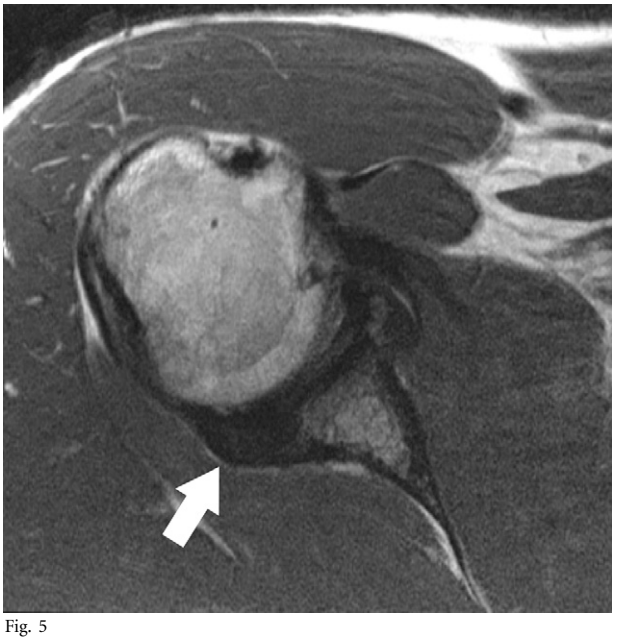

Fig. 5

Axial fast-spin-echo magnetic resonance image demonstrating hypertrophic remodeling of the posterior band of the inferior glenohumeral ligament (arrow) and the presence of a moderate posterior capsular contracture.

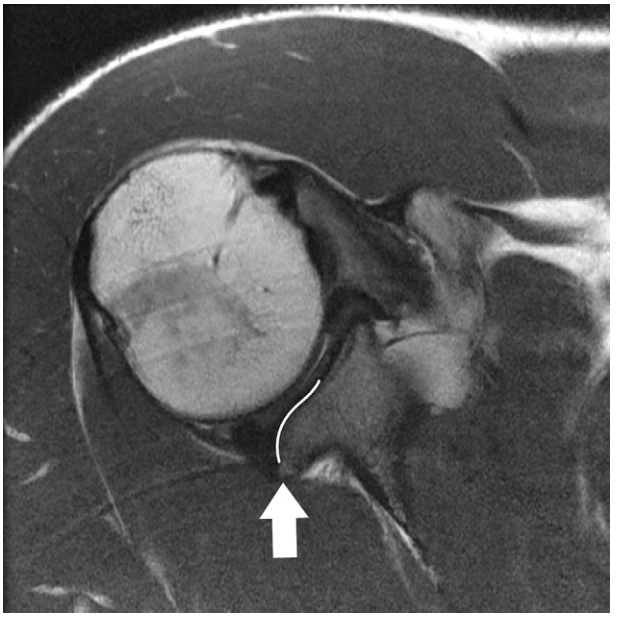

Fig. 6

Axial fast-spin-echo magnetic resonance image demonstrating deformation of the posterosuperior aspect of the glenoid (line), with a subchondral osseous depression and remodeling (arrow), consistent with the presence of internal impingement.

Wilk et al. reviewed the modalities currently used for rehabilitation of overhead athletes[46]. They described four phases of rehabilitation, which progressed from diminishing symptoms to a plyometric strengthening program. The authors postulated that excessive anterior laxity is a potential cause of internal impingement and, therefore, strengthening of the dynamic shoulder stabilizers may improve shoulder kinematics. Proper throwing mechanics, particularly by younger throwers, should be enforced.

Injections into the shoulder and particularly into Bennett lesions have been described[61]. However, these injections have been primarily used as local anesthetics and have served as diagnostic tools rather than therapeutic modalities. Moreover, in several of the patients, throwing pain decreased but was not eliminated, suggesting that concomitant pathologic conditions were present and the Bennett lesion may not have been the predominant pain generator. We are not aware of any data on the results of therapeutic injections in the setting of internal impingement, and one should be wary about using this intervention in younger, active patients. The goal of limiting the inflammatory cascade should be tempered by the potential risk of permanent tendon damage.

Operative Management

The spectrum of pathology observed in this disease lends itself to multiple treatment options. In one study, a partial tear of the articular surface of the rotator cuff was observed in >80% of forty-one professional overhead athletes with a clinical diagnosis of internal impingement[4] (Fig. 8). This tear is usually associated with an adjacent tear or abrasion of the labrum and is referred to as the "kissing lesion." Several treatment methods have been described. The tear can be débrided or repaired with or without an acromioplasty. Some authors have had mixed results with débridement alone. Andrews et al. described arthroscopic débridement in a young, active population of thirty-six patients, including twenty-three pitchers, with a partial tear of the supraspinatus tendon[19]. Eighty-five percent returned satisfactorily to their premorbid level of athletic activity. The authors postulated that the débridement stimulated tendon-healing. These results were supported by a recent study by Sonnery-Cottet et al.[40], in which twenty-eight young, active tennis players with internal impingement were treated with arthroscopic débridement of a partial-thickness tear of the supraspinatus and glenoid lesions. Twenty-two (79%) of these patients returned to playing tennis; however, twenty (91%) of the twenty-two reported some persistent pain with activity.

Many surgeons have determined that, as a general rule, lesions involving >50% of the thickness of the tendon are best treated with rotator cuff repair. Park et al. reported that patients with a partial-thickness repair had clinical results similar to those of patients with a full-thickness repair[62]. The authors did assert that the results at six months postoperatively were better in patients with a bursal-sided partial-thickness rotator cuff tear. Some authors have even supported the completion of larger tears to facilitate soft-tissue mobilization[63], and several have recommended completion of the tear followed by a two-row arthroscopic suture repair[63-65].

In addition to rotator cuff tears, posterosuperior labral detachment, fraying or degenerative labral changes, fraying or tenosynovitis of the biceps, and Grade-II or III changes in the humeral head have all been reported in patients with a clinical diagnosis of internal impingement[1,4,7,19,66]. Payne and Altchek evaluated forty-one young athletes who had similar pathologic conditions of the shoulder[67]. These patients had a normal-appearing subacromial space and often increased anterior glenohumeral translation with a posterior labral tear. Overall, the group did poorly after arthroscopic débridement of the

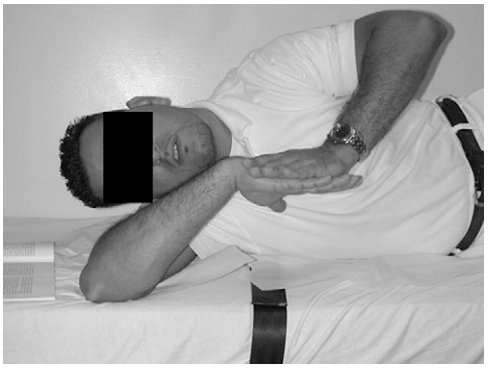

Fig. 7

A patient performing the sleeper stretch to improve a posterior capsular contracture.

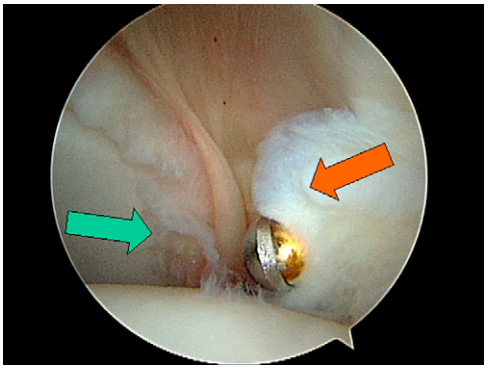

Fig. 8

Arthroscopic view of the so-called kissing lesion. Note the abrasion of the undersurface of the rotator cuff (orange arrow) and the posterosuperior aspect of the labrum (green arrow) where abnormal contact has taken place.

tear, with only 38% having a satisfactory result and only 25% returning to sports. Meister et al. reported similar results in their group of twenty-two throwing athletes with internal impingement[30]. The patients underwent débridement of the rotator cuff and labral tear, and eleven patients had arthroscopic removal of a Bennett lesion as well. At an average of six years postoperatively, only 55% of the throwers returned to their premorbid level of throwing. Although it was not significant, the authors noted a trend toward worse results in association with large osteophytes (>100 mm^2).

The Bennett lesion has been targeted as a potential cause of internal impingement. Several studies have demonstrated an association among the Bennett lesion, posterior shoulder pain, and internal impingement, with varied success reported following débridement of the Bennett lesion[30,61]. The results of these studies and a critical analysis of the previous literature bring into question the exact role of the Bennett lesion in the etiology of internal impingement and posterior shoulder pain. Yoneda et al. reported on sixteen baseball players who had undergone arthroscopic removal of a Bennett lesion[61]. In this prospective cohort study, each patient had a Bennett lesion, posterior shoulder pain, tenderness at the posterior aspect of the glenohumeral joint, and relief of pain following a local injection. The pain either disappeared or decreased after the operation in every patient, but only eleven of the sixteen were able to resume their previous level of activity. In this latter group, additional lesions identified on preoperative imaging tests at the time of the arthroscopic inspection were also treated. In at least one patient who had a reoperation, capsular laxity that was not addressed at the index procedure was determined to be an underlying cause of the failure.

Several of the aforementioned studies have pointed to either unrecognized or subtle anterior laxity as a cause of failure of débridement alone. Andrews and Dugas advocated the operative treatment of subtle anterior laxity associated with internal impingement[32]. Jobe believed that excessive anterior laxity provides the means for abnormal shoulder mechanics[2]. In a retrospective study of twenty-five athletes treated with anterior stabilization, they reported a 92% rate of good and excellent results at an average of thirty-nine months postoperatively. They subsequently recommended anterior capsulolabral reconstruction to treat anterior laxity in skilled overhead athletes. In another study, seventeen (68%) of twenty-five athletes resumed their prior level of function after being treated with that open procedure[7]. Montgomery and Jobe demonstrated the effectiveness of anterior capsulolabral reconstruction in thirty-two consecutive athletes[39]. All patients had either anterior subluxation or dislocation. Postoperatively, 81% returned to the same level of competition, and the rate of good-to-excellent results was 97%. The increased morbidity associated with an open approach led Altchek and Dines to develop a less invasive procedure, in which a horizontal capsular incision is made and then plicated[66]. The goal is to restore capsular tension without limiting the functional range of motion.

The focus in the treatment of this disease has been on soft-tissue interventions. Few surgeons have attempted osseous procedures to correct or manipulate the osseous adaptations that may be integral to the pathogenesis of the disease. Riand et al. reported on derotational osteotomies for the treatment of internal impingement[68]. The prevailing idea was that increased humeral retroversion was the etiology in patients who continued to have pain after articular débridement for the treatment of posterosuperior impingement syndrome. These patients were unable to resume sports activity, and a derotational humeral osteotomy with a myorrhaphy of the subscapularis muscle was performed. Eleven (55%) of twenty patients resumed sports activity at the same level, and an additional five resumed sports activity but at a lower level.

Authors' Perspective

Over the past decade, the concept of internal impingement has continued to evolve, and the frequency with which it is recognized continues to increase. Now that the awareness of internal impingement as a pathologic entity has increased, more long-term results should become available to help guide treatment. To our knowledge, there have been no prospective studies comparing therapeutic modalities for the treatment of internal impingement. Much of the research that has been published is retrospective, based on clinical follow-up studies in which a particular treatment algorithm was used but not compared with another regimen. This is further complicated by the spectrum of pathologic conditions of the shoulder associated with this disease, which makes it difficult to compare patient groups. Moreover, because this entity has been described only relatively recently, there are few reports with long-term data from which any conclusions can be drawn.

Anecdotally, we have also noted an increase in glenoid retroversion in throwers with internal impingement, as originally reported by Crockett et al.[5]. They noted that throwers had an increase in glenoid version in the dominant arm as compared with that in the nondominant arm, whereas non-throwers did

not. In our opinion, when the throwing motion becomes pathologic there is an overall change in the shape of the glenoid, particularly the posterior part. As the glenoid remodels and the posterior aspect of the capsule contracts, the posterior part of the glenoid also becomes more prominent. This is emphasized by the presence of a Bennett lesion. As this portion of the glenoid becomes more prominent, it increases the likelihood of undersurface contact of the rotator cuff with the posterior glenoid margin. We hypothesize that this shape change is a substantial contributor to limited internal rotation and may lead to internal impingement. What remains unclear is the effect that this retroversion has on the natural history of the throwing shoulder. Regardless of whether the effective glenoid retroversion is protective or deleterious, it may be a target of therapy in the future.

Repetitive throwing by overhead athletes, and particularly pitchers, leads to both soft-tissue and osseous adaptations. These may include exostoses, capsular laxity, increased humeral retroversion, scapular muscle imbalance, and rotator cuff tendinitis. It should be noted that many of these changes represent the biologic response to the physical stimulus, which allows these athletes to achieve a supraphysiologic range of motion to perform at a high level. Arthroscopic débridement alone has had poor results in patients with an undersurface rotator cuff lesion, increased anterior glenohumeral translation, and a posterior labral lesion[69]. We speculate that this may be due to microinstability that was not addressed at the time of the surgery. However, distinguishing pathologic instability from the throwing shoulder's adaptive laxity is a challenge for even the most experienced shoulder surgeon. As is true for many other pathologic conditions of the shoulder, nonoperative measures to address the symptoms are an appropriate initial approach. However, when conservative interventions fail, surgery may be the most effective option to alleviate symptoms and return the players to their preinjury activity level.

It is of utmost importance that associated comorbidities (e.g., capsular laxity or contracture) also be addressed at the time of surgery to prevent a poor result. Areas for future research include clarification of the relevance of the Bennett lesion, the role of glenoid version in the development of internal impingement, and the development of a standard classification system with which similar pathologic entities can be effectively studied and compared. ■

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