# Internal Impingement in the Shoulder of the Overhand Athlete: Pathophysiology, Diagnosis, and Treatment

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## ABSTRACT

**The etiology of rotator cuff disease is multifactorial in nature. The process by which the articular surface of the rotator cuff can become diseased secondary to direct abutment against the glenoid rim and labrum has been termed internal impingement. Damage to the undersurface of the rotator cuff can occur from contact at the extremes of shoulder motion and can increase secondary to adaptive changes in bone and soft tissue. Diagnosis is achieved in most instances by a thorough physical examination. Adjunctive tests, particularly magnetic resonance imaging, can increase the accuracy of the diagnosis. If this disease is recognized early, nonoperative intervention may be successful. When nonoperative treatment fails, the use of arthroscopy for the treatment of torn rotator cuff and labral tissue and capsular laxity may be indicated to resolve symptoms and restore the premorbid level of function.**


The etiology of rotator cuff (RTC) disease has been debated for decades. Each of the numerous theories that postulate the causes of pain, inflammation, and tears to the RTC has contributed information on the pathogenesis of this disease. The shoulder in the throwing or overhand athlete has become an area of particular focus. Recent dedication to unraveling the pathomechanics and pathophysiology of the disease processes that plague the overhand athlete has brought about a greater understanding of shoulder function that can be usefully extrapolated in the treatment of all individuals.

The articular surface of the RTC and greater tuberosity has been shown to come in contact with the posterior and superior glenoid rim and labrum at the extremes of shoulder abduction and external rotation.[1-4] Walch and colleagues[5] have provided clinical evidence that this contact, particularly in the overhand-athlete population, can result in damage to the articular surface of the RTC and glenoid labrum. The internal impingement occurring on the articular surface of the RTC has provided a tenable explanation for the posterior shoulder pain commonly experienced by the overhand athlete.

## HISTORICAL PERSPECTIVE

Bennett, in 1941, first recognized the development of chronic posterior shoulder pain as career threatening in the thrower. His initial description of the development of bony deposit and calcification of the posteroinferior glenoid rim and capsule led to the designation of this lesion as a Bennett's lesion (Figure 1). However, he credited the development of pain and the pain associated with this lesion to be secondary to repetitive traction on the posterior capsule and triceps tendon and to irritation of the axillary nerve from the bony deposit.[6,7]

In 1977, Lombardo and colleagues[4] provided a description of successful open treatment of posterior shoulder lesions in 4 throwing athletes who complained of pain in the posterior aspect of the shoulder in the late cocking phase of throwing. Posterior capsule ossification and fibrous tissue formation were noted in each case. All were successfully treated with open debridement of the bone and soft tissue and posterior capsule repair. In the absence of arthroscopy, concomitant changes on the humeral head and RTC were not recognized. However, the

Figure 1. Bennett's lesion. Exostosis (arrow) formed on the posteroinferior glenoid rim.

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authors did hypothesize that the impingement of the humeral head upon the posterior capsule and rim of the glenoid during cocking could account for the development of the signs and symptoms encountered in the athletes.[4]

Andrews and colleagues,[8] in 1985, reported on a series of athletes with confirmed tears to the articular surface of the RTC. All underwent arthroscopic debridement of their lesions. In addition to tears of the RTC, all individuals had tearing of some portion of the glenoid labrum (either anterosuperior or posterosuperior). The suggested cause in these individuals was attritional, secondary to the repetitive tension overload of the RTC. The early establishment of arthroscopy as a viable therapeutic and diagnostic modality in the shoulder can be credited to the successful outcomes of the individuals in this series.[8]

In 1989 Jobe and colleagues[9] presented a classification system relating instability in the throwing shoulder to the development of RTC disease. The relocation test was suggested as a sensitive means of eliciting occult anterior instability that leads to rotator cuff disease.[9] According to Jobe,[10] shoulder pain during the apprehension maneuver that resolves with a posteriorly directed force on the humeral head is a positive test (Figure 2); anterior shoulder pain occurs secondary to subacromial impingement and posterior pain secondary to internal impingement.[11] Posterior pain that resolved from the posteriorly directed force was thought to result from decompression of the posterior "kissing lesion."[11]

However, it was Walch and colleagues[5] who first presented, in 1992, clinical evidence to support the development of intra-articular sided tears to the RTC as a direct consequence of internal impingement. The clinical link between posterior shoulder pain, partial RTC tears, and internal impingement was established by his series of 17 individuals with undersurface tearing of the rotator cuff treated with arthroscopic debridement.

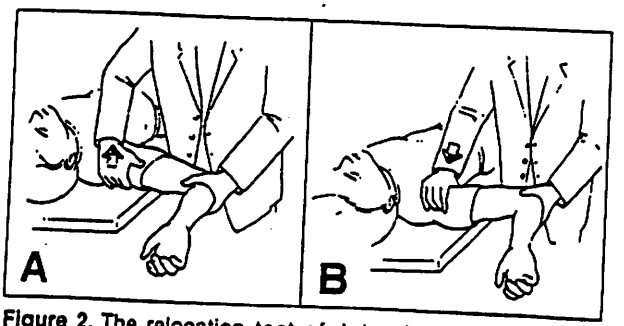


Figure 2. The relocation test of Jobe. (A) The shoulder in abduction and maximum external rotation. (B) Posteriorly directed force on the humeral head eliminates symptoms of pain and apprehension. Reprinted with permission from Jobe et al.[10]

## PATHOPHYSIOLOGY

The glenohumeral joint allows for motion in all planes; however, its geometry limits motions that occur in simple arcs. Full elevation cannot take place with only coronal plane abduction with the arm maintained in an internally rotated position. Attempts at elevation with the arm internally rotated leads to abutment of the greater tuberosity against the superior glenoid.[1] External rotation during abduction moves the greater tuberosity posteriorly, eliminating initial contact of the greater tuberosity against the superior glenoid rim and allowing for full abduction.[1]

Correlation between internal impingement and the development of RTC pathology was first recognized in throwers. The observation that repair of anterior instability in the thrower reduced RTC symptoms, coupled with new anatomic, magnetic resonance imaging (MRI) and arthroscopic data, reenforced the correlation between instability and RTC disease.[2,12-14]

Jobe[13] was responsible for a cadaveric study that provided evidence of distortion of the posterior superior labrum and the inner fibers of the RTC in a glenohumeral joint fixed in 60∞of abduction and maximum external rotation. This study was primarily responsible for proving the contact of these structures in nonthrowing individuals without evidence of instability.

In an MRI study that evaluated the impingement sign with the arm in full elevation and internal rotation, one individual was observed in the 90∞ abducted, externally rotated position. All images demonstrated deformation of the soft-tissue structures against the glenoid rim. In addition, all subjects experienced increasing discomfort after holding these positions for prolonged periods in the MRI gantry.[2,15]

Four structures remain at risk with the arm in the abducted, externally rotated position through direct contact with each other: the greater tuberosity, supraspinatus tendon, superior labrum, and superior glenoid bone. The inferior glenohumeral ligament (IGHL)is at risk through tension overload (Figure 3).[16]

In the late cocking phase of throwing, the glenohumeral joint is in about 60° of abduction, maximum external rotation, and slight horizontal extension. Passive restraint against this position of extreme rotation is provided by the anterior portion of the IGHL. Active restraint is provided by a competent subscapularis. Fatigue of the subscapularis and/or failure of the anterior IGHL can result in painful posterior glenohumeral impingement even prior to the detection of significant instability (Figure 4).[5]

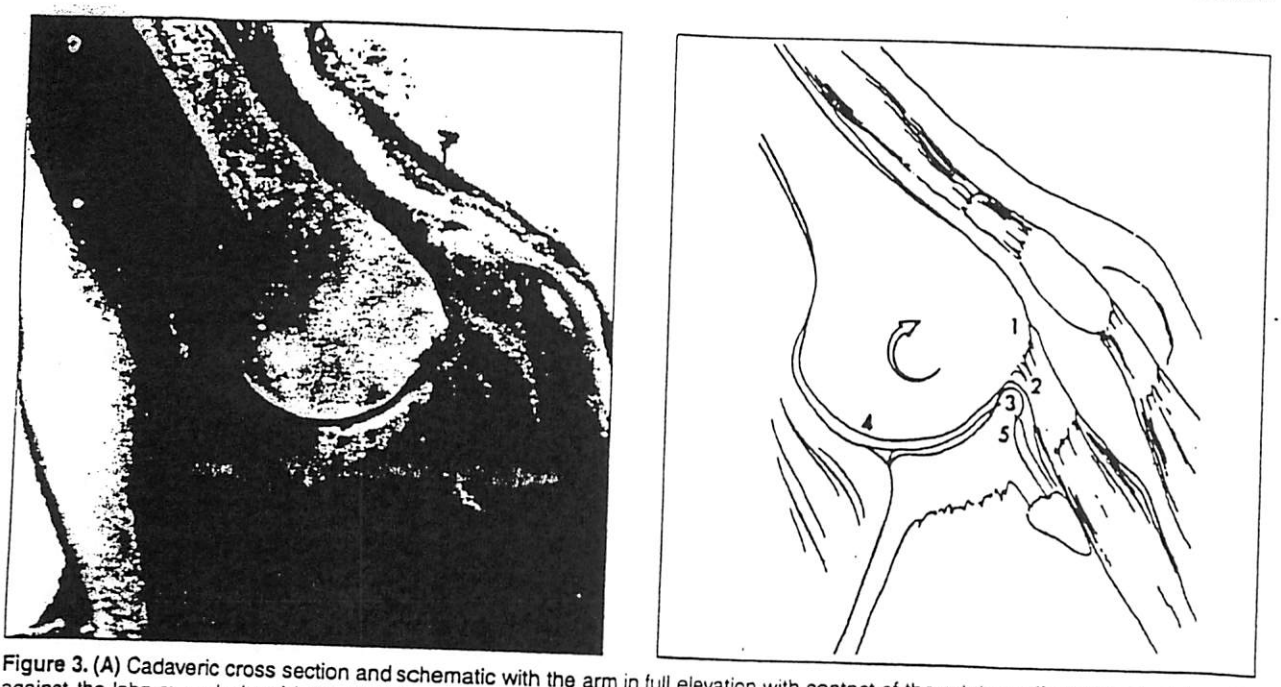


**Figure 3.** (A) Cadaveric cross section and schematic with the arm in full elevation with contact of the rotator cuff and greater tuberosity against the labrum and glenoid rim. (B) Diagrammatic representation: 1 = greater tuberosity; 2 = rotator cuff tendon; 3 = superior labrum; 4 = inferior glenohumeral ligament; and 5 = superior glenoid bone. Reprinted with permission from Jobe.[14]

Although almost any individual may develop internal impingement given development of sufficient laxity of the anterior capsule, weakness of the RTC musculature, repetitive use of the shoulder in extremes of rotation, and certain anatomical factors also may put some individuals at risk. Walch and Colleagues[15,17,18] suggested that decreased retroversion in the proximal humerus might be a risk factor for the refractory nature of symptoms in some of those treated arthroscopically. Additional factors that may contribute to the development of impingement include excessive glenoid anteversion and strength deficits in the periscapular musculature. Both excessive protraction and decreased elevation of the scapula in the cocking phase and increased glenoid retroversion may lead to increases in the compression between the posterior glenoid and humeral structures.

In the acute setting the same structures are also at risk. Codman[19] suggested a mechanism of injury of the greater tuberosity that results in an isolated displaced fracture from shearing of the tuberosity over the superior glenoid rim. Jobe[14] has also presented an expanded spectrum of the disease process by reporting cases of acute RTC tears, superior labral tears, tuberosity fractures, and anterior Bankart lesions from injury with the shoulder in the elevated position.

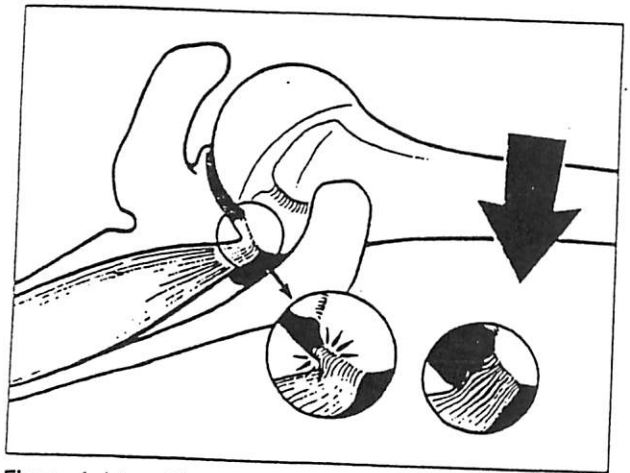

**Figure 4.** Internal impingement in the late cocking phase of throwing with entrapment of the articular surface of the rotator cuff. Reprinted with permission from Walch et al.[5]

## EVALUATION

### History

A typical pattern of symptoms may be elicited from the individual experiencing onset of symptomatic internal impingement. Jobe's classification of glenoid impingement in the thrower describes three stages of disease progression.[2]

Stage I disease is present in individuals who present at the earliest onset of symptoms. These athletes will complain of stiffness and a prolonged difficult period of warm-up. They often complain of discomfort in the late cocking and early acceleration phases of their motion but are unable to localize their pain. Pain is not present with activities of daily living (ADL). Stage II and III disease are both characterized by the clear localization of pain in the posterior aspect of the shoulder in the late

cocking and early acceleration phases of the throwing motion. Pain with ADL is unusual, as are complaints of instability. Individuals who have been refractory to nonoperative measures of treatment are considered to have Stage III disease.

### Examination

Observation may reveal asymmetry in muscular development with hypertrophy of the dominant side of the torso and extremity. Localized tenderness over the posterior cuff and capsule may be best elicited with the arm draped across the chest in an adducted position. Differences in range of motion between the dominant and nondominant extremity in the thrower are often seen. A measured increase in external rotation in the 90° abducted position may be 10°–15°or greater. A symmetrical decrease in internal rotation in the same position in the dominant versus the nondominant extremity is typical.

Shoulder stability testing may or may not reveal any increase in laxity. The shoulder in the throwing athlete, however, may present with global laxity or a grade increase in anterior translation in the dominant extremity. With isolated internal impingement, Neer's impingement sign is usually negative.

In the intermediate and late stages of internal impingement, the relocation test of Jobe and the posterior impingement sign are usually positive. The posterior impingement sign places the shoulder in the late cocking position of the throwing motion. With the shoulder 90°– 100° abducted, extended 110°– 15°, and maximally externally rotated, recreation of pain in the posterior aspect of the shoulder is greater than 90% sensitive for the detection of tears to the posterior labrum and/or RTC (Figure 5).[20,21] With the shoulder in this position, the relocation test is performed by applying a posteriorly directed force to the humeral head. A positive relocation test, reduction in symptoms with reduction of the humeral head, further confirms the diagnosis.[11,13]

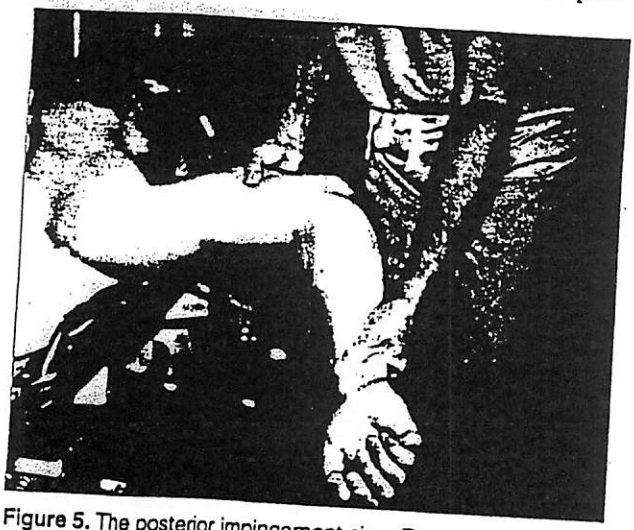

**Figure 5.** The posterior impingement sign. Recreation of posterior shoulder pain by positioning the shoulder in the late cocking position.

### Radiography

MRI has evolved into an extremely useful tool in the diagnosis of the spectrum of this disease. Evidence of increased sclerosis at the base of the greater tuberosity in the externally rotated anteroposterior view may be the only indication of disease on routine radiographs.[22] In our clinic, MRI arthrography with gadolinium enhancement can successfully detect irregularity in the undersurface of the RTC and tears less than 25% the thickness of the cuff in more than 90% of cases.[23] Others have shown that evaluation of the labrum with similar success can be achieved by using MRI without the use of arthrographic technique.[24]

Computed tomography scanning can be used to measure proximal humeral retroversion to an accuracy of 2°– 3°.[5,17,18] Measurements of retroversion from plain radiographs are accurate to within 5°.[17,25]

### Additional Studies

Numerous studies have been performed that establish normal parameters of isokinetic strength in the throwing shoulder. Strength ratios on internal to external and abduction to adduction strength have been established for varied test speeds (Table). Abnormalities in ratios or overall deficiencies in strength can be responsible for the development of the disorder.[25,26]

Additional useful information may be gained through evaluation of the biomechanics of an individual's throwing motion. Common abnormalities in throwing mechanics can be responsible for the development of a spectrum of maladies in the overhand athlete.

## TREATMENT

The differential diagnosis for posterior shoulder pain in the throwing athlete can be lengthy and

**Table. Isokinetic Strength Profiles in the Throwing Athlete (Unilateral Muscle Ratios)**

| | Reported Ratio (%) | Goal (%) |
|---|---|---|
| ER/IR | 62 – 66 | 66 – 75 |
| ABD/ADD | 78 – 84 | 66 – 72 |
| ABD/ER | 62 – 66 | 64 – 69 |

ER/IR = ratio of external to internal strength; ABD/ADD = ratio of abduction to adduction strength; ABD/ER = ratio of abduction to external strength.

include evaluation for strains of the posterior cuff, deltoid and rhomboids, suprascapular or axillary nerve entrapments associated with or without a ganglion cyst of the spinoglenoid notch, and acute capsular strains. However, once the initial diagnosis is established, both nonoperative and operative approaches may be indicated in treating the disorder.

## Nonoperative Treatment

Early phases of the disorder should be treated nonoperatively. These individuals have poorly localized discomfort and complain primarily of stiffness and loss of velocity. A 2- to 4-week rest from throwing accompanied by an intensive period of RTC and scapular strengthening is recommended. As initial complaints resolve, a return to throwing program can be initiated with an expeditious return to the premorbid level of throwing.

Individuals who present with more localized posterior complaints also may be treated initially with nonoperative management. Characteristically, examination reveals a positive posterior impingement sign and relocation test. Most throwers also will reveal some indication of asymmetrical capsular laxity. These athletes usually require a period of rest from throwing of at least 1 month. In conjunction, a rehabilitation program that stresses strengthening of both the RTC and periscapular musculature is recommended.

Throwers who present with initial symptoms of greater than 3 months' duration or who have failed an initial course of therapy require further evaluation with MRI arthrography. MRI of the throwing shoulder is most useful when correlated with symptoms. Caution must be used when interpreting the scan and using this information too early in the rehabilitation period. Halbrecht and colleagues[27] reported that up to 40% of all throwers may exhibit changes in the RTC upon MRI of the dominant shoulder. Therefore, such scans should not be the only basis for abandonment of nonoperative measures of treatment.

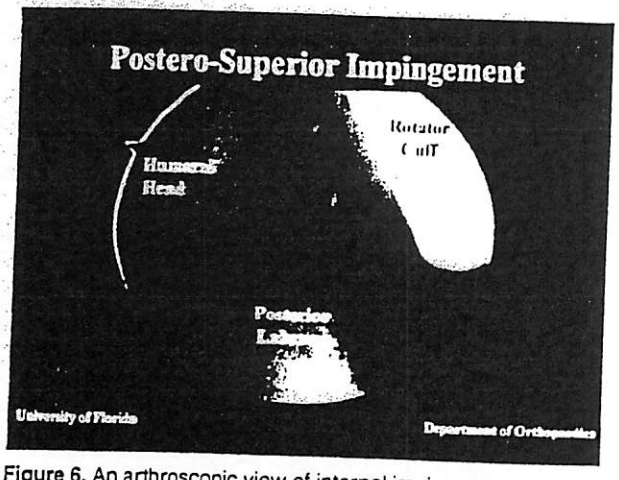


Figure 6. An arthroscopic view of internal impingement.

## Operative Treatment

Operative intervention may be indicated in individuals who have failed a conservative management course of at least 3 months' duration and who have examination and MRI findings consistent with internal impingement. Three approaches have been taken in the treatment of significant symptomatic articular-sided RTC and labral lesions in the throwing athlete.

The first approach involves arthroscopy of the glenohumeral joint with debridement of the rotator cuff and labral pathologic lesions. A standard posterior shoulder portal is made with the arthroscope with either beach chair or lateral decubitus positioning. Confirmation of impingement can be made by removing the shoulder from traction and, while viewing through an anterior portal, rotating the shoulder into an externally rotated and 90° abducted position (Figure 6). Examination of the subacromial space is usually unrevealing.

Treatment of impingement with isolated debridement of the RTC and labrum has had varied success. Failures with this technique may be secondary to unrecognized pathologic laxity of the shoulder capsule. The failure to recognize pathologic laxity with concomitant damage to the articular structures will usually result in failure of the initial surgical procedure.[8,28]

Jobe[29] has published extensively on the merits of his anterior capsulolabral reconstruction (ACLR) to treat subtle instabilities in the thrower (Figure 7). His technique of capsular advancement has led to success rates of 85% in the overhand athlete.[13,30] Arthroscopic evaluation of the shoulder with treatment of tears to the RTC and labrum followed by an open capsular shift procedure may increase the chances of returning an athlete to the premorbid level of activity. Altchek[30] also has described a less aggressive method of capsular plication through a horizontal capsular incision to address subtle insta-

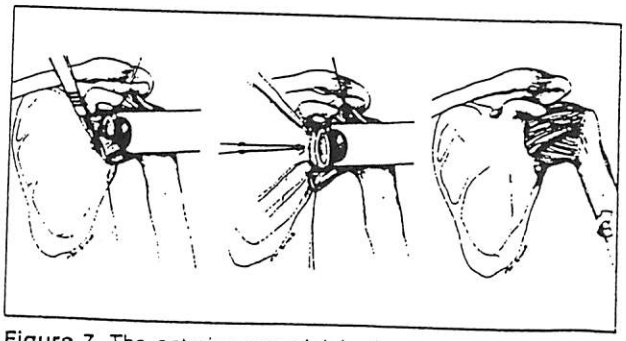

Figure 7. The anterior capsulolabral reconstruction of Jobe. Reprinted with permission from Altchek and Dines.[30]

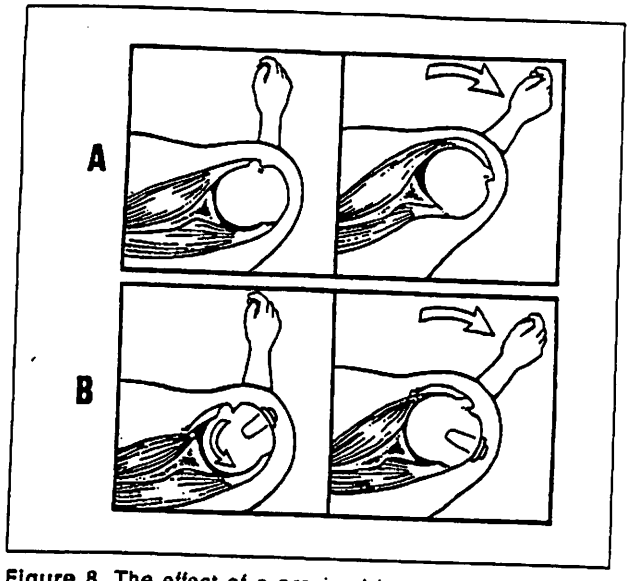


**Figure 8.** The effect of a proximal humeral derotational osteotomy on reduction of internal impingement. Reprinted with permission from Riand et al.[18]

bilities in the overhand athlete.[30] With either technique, rehabilitation throwing is usually begun at 16 weeks, but an athlete may not reach his or her preinjury level of function for up to a year.

Most recently, an arthroscopic approach to reduction of capsular volume has used thermal treatment of the joint capsule. The effect of thermal energy on the glenohumeral joint capsule has been well established.[31–33] However, to date, clinical follow-up has been relatively short term and little has been published to support the clinical efficacy of this newer method of treatment.

In a study based on a population of European overhand athletes, Riand and colleagues[18] suggested that differences in humeral version contribute to the development of internal impingement. Individuals who have failed arthroscopic debridement of their lesions may have measurable decreases in humeral retroversion that increase internal impingement in the externally rotated shoulder. Successful treatment of 14 individuals was achieved with proximal humeral derotational osteotomy (Figure 8).[18]

## CONCLUSIONS

Not all damage to the RTC occurs secondary to subacromial impingement. Internal impingement with articular sided compression of the RTC against the posterior and superior glenoid labrum are at least as responsible for both acute and attritional tears of the RTC.

In the overhand-athlete population, onset of symptoms may be characterized in the earliest phases by complaints of stiffness followed by more localized complaints of posterior shoulder pain. The posterior impingement sign, relocation test of Jobe, and MRI arthrography are useful in the evaluation of the disorder.

When nonoperative management fails, surgical intervention may be required. An associated capsular procedure, either open or arthroscopic, may improve the overall outcome in those individuals undergoing arthroscopy and debridement of their tears. Whether increased success is a result of limitation of shoulder translation or a slight decrease in external rotation is not clearly understood. Most likely, both slight limitation of rotation and translation are important factors in the return of the athlete to his or her premorbid level of activity.

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