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# Typical and Atypical Shoulder Impingement Syndrome: Diagnosis, Treatment, and Pitfalls

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## Abstract

*The cause of shoulder impingement syndrome usually is considered to be compression of the rotator cuff and subacromial bursa against the anterolateral aspect of the acromion. The typical symptom is anterolateral shoulder pain that worsens at night and with overhead activity. However, the pain may be caused by factors other than a hooked acromion. Atypical impingement syndrome most commonly results from an os acromiale, a subcoracoid disorder, acromioclavicular joint undersurface hypertrophy, a deconditioned rotator cuff, or scapular dyskinesis. The correct diagnosis is made through the patient history and physical examination, with appropriate diagnostic imaging. Nonsurgical treatment is successful for most types of impingement syndrome; if it is not successful, all structural causes of mechanical impingement must be corrected.*

**Instr Course Lect 2009;58:447-457.**

The anatomic area involved in typical impingement syndrome is defined by the subacromial space. Impingement of this area can lead to extrinsic compression of the rotator cuff, the bursa, the long head of the biceps tendon, or the subscapularis tendon. The superior border of the subacromial space is formed by the acromion, the coracoacromial (CA) ligament, and the acromioclavicular (AC) joint. The anterior border is formed by the CA ligament and the coracoid process, and the inferior border is the humeral head. Changes in any of these structures can lead to impingement syndrome (Table 1).

## Typical Impingement Syndrome

### *Classification*

The Neer impingement syndrome classification is commonly used.[1] Stage I involves edema or hemorrhagic change in the rotator cuff tendons. Stage II is characterized by tendon degeneration and fibrosis. Stage III involves further attenuation of the tendons with a structurally significant partial- or full-thickness rotator cuff tear.

### *Diagnosis*

#### *History and Physical Examination*

A patient with typical impingement syndrome often has sleep-disrupting anterolateral or lateral shoulder pain, which may be positional. Pain also may be present with overhead activity, and it often is associated with loss of motion or weakness. The onset of pain typically is insidious; in some patients, the onset is precipitated by a mild traumatic event or a heavy repetitive task such as painting or raking leaves. The medical history must include any endocrine disorder, a family history of rotator cuff disorder, a pending workers' compensation claim, or pending litigation. The physical examination must include visual assessment for skin lesions or discrepancy in shoulder height or scapular position.[2] Range of motion is assessed with attention to forward flexion, abduction, external rotation at the side and in abduction, and internal rotation up the spine. Internal rotation should be checked in the plane of the scapula if the patient participates in an overhead sports activity. The contralateral shoulder can be used to compare motion restriction, although significant restrictions in range of motion rarely are present with typical impingement syndrome. Tenderness to palpation is specific to the area of the anterior rotator cuff and bursa just anterolateral to the acromion. Tenderness over the AC joint or coracoid indicates the involvement of additional structures.

The patient may have increased pain with passive forward flexion in an internally rotated position (the Neer test) or with passive internal rotation in 90° of abduction (the Hawkins test). However, some pa-

**Table 1**
**Causes of Impingement Syndrome**

| |
|---|
| **Typical Impingement Syndrome** |
| • A type II or III acromion |
| • Subacromial spurs |
| • Osteoarthritic spurs of the AC joint |
| • A thickened or calcified CA ligament |
| **Atypical Impingement Syndrome** |
| • Os acromiale |
| • Shoulder instability |
| • Superior migration of the humeral head secondary to rotator cuff weakness |
| • Posterior capsular contractures (GIRD) |
| • Scapular dyskinesis |

GIRD = glenohumeral internal rotational deficiency

**Table 2**
**Differential Diagnosis of Typical Impingement Syndrome**

| |
|---|
| Calcific tendinitis |
| Gout |
| Adhesive capsulitis |
| Psoriatic arthritis |
| AC arthritis or osteolysis |
| Lyme disease |
| Glenohumeral arthritis |
| Lupus erythematosus |
| Septic arthritis |
| Osteonecrosis |
| Rheumatoid arthritis |
| Cervical radiculopathy |
| Thoracic outlet syndrome |
| Tumor |

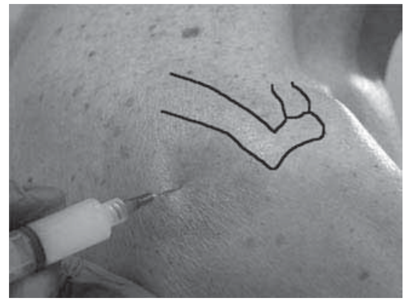


**Figure 1** Subacromial injection from a posterior approach.

tients have little pain with these tests. Strength testing of the elements of the rotator cuff typically reveals weakness in external rotation at the side (the infraspinatus) or in forward flexion in the plane of the scapula (the supraspinatus). A mismatch between passive and active range of motion suggests a significant decoupling of the rotator cuff.

Additional tests or an in-depth examination of the neck and upper spine may be needed to evaluate the patient's distal neurovascular status.

### *Imaging*

Plain radiography can be helpful in identifying the cause of impingement syndrome (especially an atypical cause such as an os acromiale). An AP view with the shoulder in 30° of external rotation, a standard axillary view, and an outlet Y view are most beneficial. The AP view reveals any AC joint pathology and allows evaluation of the glenohumeral joint. The axillary view shows the glenohumeral joint and the coracoid anatomy and is most useful for determining the presence of an os acromiale. The outlet Y view can be used to determine the type of acromion morphology (flat, curved, or hooked), although this classification has significant interobserver variance; nonsurgical treatment is less likely to be successful if the acromion is hooked or curved.[3]

Only plain radiography usually is necessary before nonsurgical treatment. However, a substantial mismatch between the active and passive ranges of motion or a traumatic onset of pain is an indication for further testing. MRI allows thorough evaluation of the soft tissues and bony structures around the shoulder. Ultrasonography and CT arthrography are less expensive, but specific expertise is required to conduct and interpret these tests.

### *Diagnostic Injection*

An injection of lidocaine into the subacromial space produces excellent relief of pain from typical impingement syndrome. Residual symptoms suggest the presence of a concomitant metabolic disorder or pathology in the AC joint, the glenohumeral joint, or the cervical spine (Table 2).

## *Nonsurgical Treatment*

Typical impingement syndrome can be successfully treated using nonsurgical techniques.[3] The goals of the first phase of treatment include resolution of limitations in the patient's range of motion and rehabilitation of weakness in the rotator cuff or periscapular muscles. The patient must avoid any inciting activity. Some patients respond well to the use of a nonsteroidal anti-inflammatory agent. If the symptoms persist, a therapeutic subacromial injection of lidocaine and a corticosteroid can be used. The injection is best done using a posterior approach and a 22-gauge, 1.5-inch needle with 9 cc of lidocaine and 1 cc of a corticosteroid (Figure 1). Although the long-term effect has not been established, therapeutic injection can resolve sleep-disrupting pain and allow the patient to continue a home-based physical therapy program.

## *Subacromial Decompression*

If a 3- to 6-month program of nonsurgical treatment does not resolve the symptoms, MRI or ultrasonography can be used to identify evidence of extrinsic impingement. A carefully planned subacromial decompression can then be done using an arthroscopic or open technique. Diagnostic glenohumeral arthroscopy is important for identifying and treating any intra-articular pathology such as a partial undersurface rotator cuff tear or early glenohumeral arthritis.

The goals of a subacromial decompression are to release the CA ligament; remove any thickened subacromial bursa; and achieve a flat, smooth acromial undersurface. Subacromial decompression has excellent long-term results.[4] If the patient's symptoms are not resolved with subacromial decompression, all potential causes must be considered. Failure of subacromial decompression can be classified as mechanical, physiologic, or psychosocial.

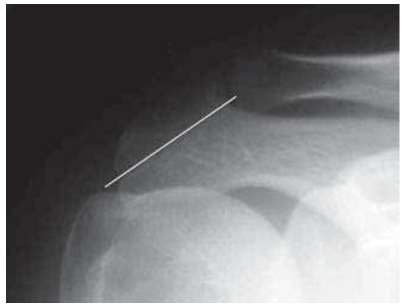


**Figure 2** AP radiograph showing significant residual lateral acromial tilt (*line*).

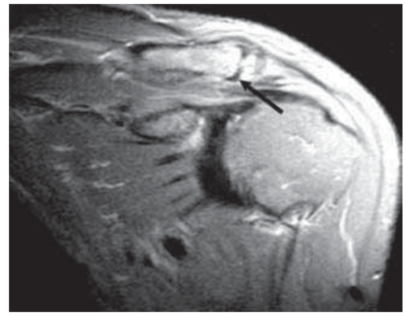


**Figure 3** MRI scan showing AC joint osteolysis *(arrow)*.

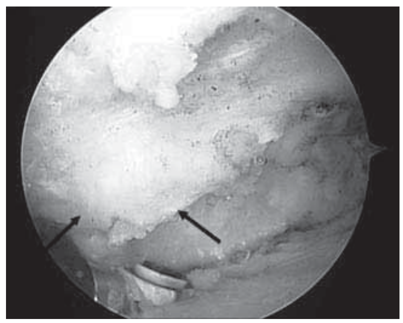


**Figure 4** Arthroscopic view from the posterior portal showing a retained AC spur *(arrows)*.

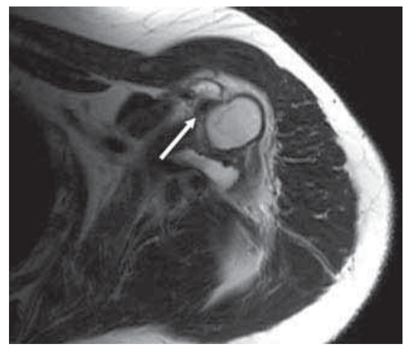


**Figure 5** Axillary MRI scan showing a dislocated long head of the biceps tendon *(arrow)*.

***Mechanical Issues***

Mechanical failure of subacromial decompression results from a technical error: failure to release the entire CA ligament or failure to correct a lateral or anterior acromial tilt (an acquired type III acromion). These anatomic structures can be seen on radiographs or MRI scans (Figure 2). A carefully planned revision subacromial decompression is required specifically to correct the pathologic anatomy.

The AC joint can be an isolated source of pain in patients with AC degenerative joint disease or osteolysis (Figure 3), and many patients with impingement syndrome have symptoms of an AC joint disorder. The patient's history and physical examination should lead to an accurate diagnosis of direct AC symptoms. Underlying AC joint osteophytes are a common cause of extrinsic impingement, and failure to remove undersurface AC osteophytes frequently leads to residual pain after a subacromial decompression (Figure 4). The osteophytes can easily be seen on plain radiographs or an MRI scan. A resection of the AC joint, with removal of osteophytes from the acromion and clavicle, often is successful in alleviating symptoms.

Pathology of one of the main structures of the subcoracoid space (the long head of the biceps tendon, the subscapularis tendon, or the coracoid process) can cause residual symptoms if not corrected during subacromial decompression. The long head of the biceps tendon can degenerate and become partially torn in the groove; if structurally intact, it can become subluxated or dislocated from the groove (Figure 5). The pathology can easily be seen during a diagnostic arthroscopic examination of the glenohumeral joint, and appropriate surgical débridement or biceps tenodesis can be done.

The subscapularis tendon rarely shows wear on its outer surface; most pathology is intra-articular. The tendon can be examined arthroscopically by elevating the patient's elbow away from the body with the forearm across the abdomen. This internal rotation exposes the insertion of the subscapularis and the anterior capsule as well as the neck of the humerus. From this position, a low-grade partial tear can be débrided, and a higher grade lesion can be repaired.

Extrinsic compression from the coracoid often is difficult to diagnose. However, a positive cross-body Hawkins test with residual anterior pain in the region of the coracoid suggests the presence of coracoid impingement. Measuring the coracoid indices on MRI or CT

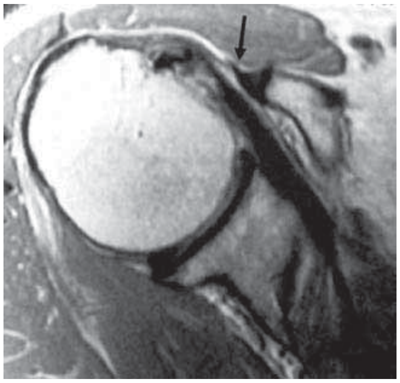


**Figure 6** Axial MRI showing lateral soft-tissue thickening *(arrow)* with resultant narrowed coracohumeral distance and subscapularis attenuation.

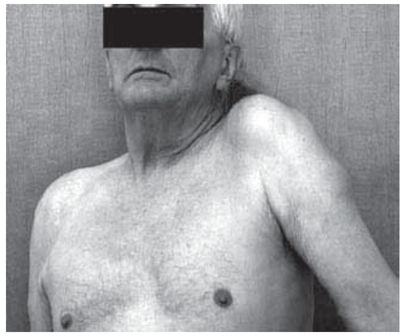


**Figure 7** A patient with superomedial shoulder instability.

also can be helpful.[5] However, coracoid decompression may be necessary if the index is normal because the faux humeri (lateral thickening of the conjoined tendon) is causing the extrinsic soft-tissue compression[6] (Figure 6).

Acromial fracture after an excessive resection is rare and extremely difficult to treat. If the deltoid attachment is intact, the unstable anterior acromial fragment can be resected arthroscopically. If the deltoid has been violated, excision of the fragment with a secondary deltoid repair can be attempted.

The CA arch must be maintained in a patient with a rotator cuff deficiency. Loss of the structural integrity of the arch leads to superomedial instability (Figure 7). Surgical treatment of superomedial instability is a reverse total shoulder replacement or a shoulder fusion.

### *Physiologic Issues*

Occasionally, the subacromial space fills with reactive scar in a bursal response to subacromial decompression. The etiology of this reaction is not well understood, but it may involve excessive use of thermal cautery or failure to remove bony debris. A subacromial injection typically leads to excellent relief of pain, but the patient's range of motion remains limited. A revision resection of the bursa and lysis of any subacromial adhesions often leads to excellent clinical recovery.

Reflex sympathetic dystrophy causes postsurgical pain that is more severe than expected, as well as reluctance to move the entire extremity. Often the patient has forearm and hand swelling as well as vasal reactive changes distally. If the diagnosis is made early, these dystrophic changes typically can be reversed by elbow and hand exercises and specific range-of-motion shoulder exercises.

Postsurgical adhesive capsulitis is not well understood. The symptoms are similar to those of idiopathic adhesive capsulitis. The condition appears to be more common in women of northern European heritage with fair skin and light hair color. Postsurgical adhesive capsulitis usually responds well to a gentle home-based physical therapy program.

A substantial number of postsurgical shoulder infections are caused by *Propionibacterium acnes*. The patients most at risk have skin currently or formerly affected by significant acne. The perisurgical antibiotic of choice for these patients is clindamycin.

Physical therapy is important for complete postsurgical recovery. However, recovery can be delayed by overly aggressive passive range-of-motion exercises or overly rapid increases in exercise repetitions and weights. A slow, gentle progression of physical therapy is preferable to a rapid progression, which can lead to significant setbacks and increased pain.

### *Psychosocial Issues*

The rate of failure of clinical improvement after subacromial decompression is significantly higher among patients involved in pending litigation or a workers' compensation claim. In these patients, a subacromial decompression often fails to relieve symptoms if the presurgical mechanical or structural findings are not clearly consistent with impingement syndrome or the clinical symptoms are not consistent with a diagnosis of impingement syndrome.

## Atypical Impingement Syndrome

### *Etiology*

Atypical impingement syndrome can have a structural or functional etiology. The structural form may involve an unstable os acromiale, most commonly a mesoacromion, or anterior shoulder pain secondary to coracoid impingement. The functional form can be caused by glenohumeral internal rotation deficit (GIRD), scapular malposition, or scapular dyskinesis.

### *Structural Impingement*

#### *Os Acromiale*

Os acromiale has been described as a fibrocartilaginous union of the acromial ossification centers; the reported incidence ranges from 1.3% to

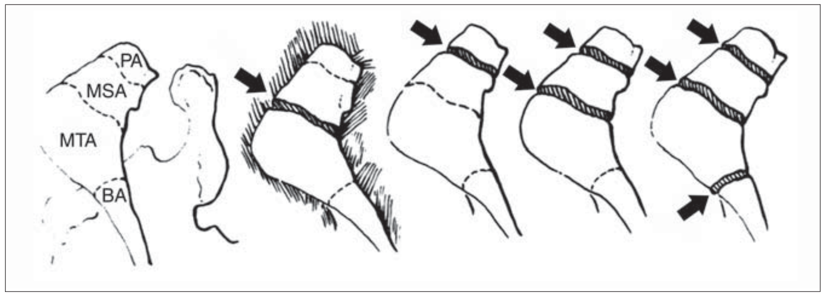



**Figure 8** Drawing of the centers of ossification. BA = base acromion, MSA = meso-acromion, MTA = meta-acromion, PA = preacromion. (Reproduced with permission from Mudge MK, Wood VE, Frykman GK: Rotator cuff tears associated with an os acromiale. *J Bone Joint Surg Am* 1984;66:427-429.)

**Figure 9** Axillary radiograph showing mesoacromial fragment.

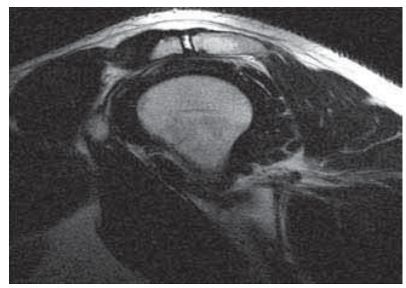


**Figure 10** Sagittal MRI scan showing fluid and edema at the mesoacromial site.

30% of the general population.[7-9] Two unrelated studies found an incidence of 8% in the Hamann-Todd Osteological Collection; approximately one third of the affected human skeletons had bilateral involvement.[8,9] These studies reported that an os acromiale was twice as common among African Americans or male specimens than among Caucasians or females. Another study found that as many as 62% of patients had bilateral involvement.[10]

The acromial apophysis develops from four separate centers of ossification: the base acromion, the meta-acromion, the mesoacromion, and the preacromion (Figure 8). Os acromiale almost always is mesoacromial; preacromial fragments occur infrequently, and meta-acromial involvement is rare. Complete union of all centers of ossification in the acromion may not occur until age 25 years.

The symptoms of an os acromiale frequently are similar to those of subacromial or outlet impingement syndrome; they can include difficulty in reaching over the head or fully extending the arm, nocturnal discomfort, and mechanical symptoms (especially with overhead or throwing activity).[11,12] The pain usually is localized over the superior aspect of the acromion, particularly if an unstable fragment is present. An unstable os acromiale may cause shoulder girdle weakness and generalized rotator cuff dysfunction. An os acromiale is unlikely to result from trauma.

The physical examination reveals positive impingement signs in conjunction with palpable tenderness over the os acromiale junction. The patient frequently has a painful arc of motion and difficulty in forward elevation and abduction. Gross motion of the os acromiale may be evident. The assessment of rotator cuff strength usually identifies generalized weakness in the anterosuperior cuff region. The impingement signs may be relieved by a diagnostic subacromial injection, but the localized tenderness over the os acromiale may not be relieved. Kurtz and associates[13] found that an injection into the os acromiale site may be more useful in diagnosing an unstable mesoacromion than the standard impingement test injection.

The initial imaging should include a radiographic series. The axillary view (Figure 9) is the most important because an os acromiale can be seen as a radiolucent line through the region of the acromion. The outlet view may reveal a radiolucent line at the junction between the anterior one third and posterior two thirds of the acromion. It is difficult to see an os acromiale on an AP view. MRI often reveals increased fluid and edema at the os acromiale site, indicating gross motion or instability (Figure 10). Hypertrophic changes also may appear on the superior or inferior surface at the interfragmentary junction. CT can reveal the smooth edges around the segments that suggest an os acromiale (Figure 11). The presence of an unstable os acromiale also can be revealed by technetium TC 99m bone scanning, which shows increased uptake in the region of the acromion.[14]

The initial treatment of a symptomatic os acromiale includes a physical therapy program focused on rotator cuff and scapular stabilization. A corticosteroid injection to the subacromial space and possibly

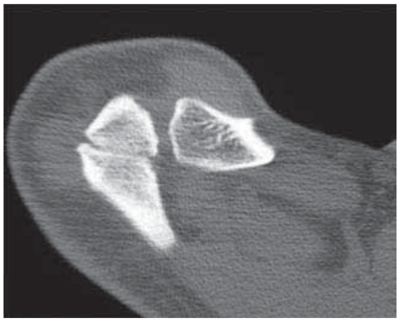


**Figure 11** Axial CT scan showing an os acromiale.

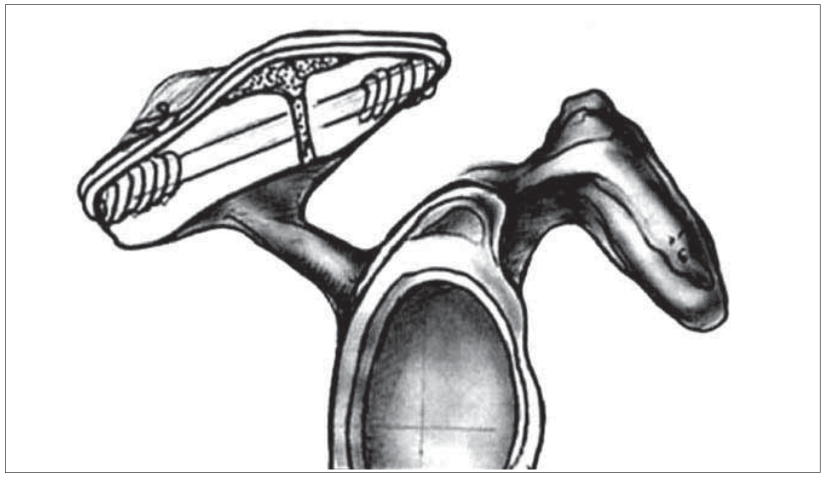


**Figure 12** Drawing showing open reduction and internal fixation of the mesoacromion. Parallel headless screw fixation is used with a tension band technique and bone graft augmentation. (Reproduced with permission from Ortiguera CJ, Buss DD: Surgical management of the symptomatic os acromiale. *J Shoulder Elbow Surg* 2002; 11:521-528.)

the os acromiale site may provide significant relief of symptoms and preclude the need for surgical intervention. In general, nonsurgical treatment should be used for 3 to 6 months before surgical treatment, although surgery may be considered sooner if the patient has a concomitant full-thickness rotator cuff tear. The reported frequency of associated rotator cuff pathology is as high as 50% in patients with an unstable os acromiale.[14,15]

The surgical options for treating an unstable os acromiale include open fragment excision, open reduction and internal fixation (with or without bone graft augmentation), arthroscopic subacromial decompression, and arthroscopic fragment excision. Open fragment excision usually is not recommended for an unstable mesoacromion. Multiple studies found that open fragment excision resulted in pain, weakness, and deltoid dehiscence or dysfunction.[14,16,17]

Open reduction and internal fixation is a successful method of treatment.[14,15,18-20] The principles associated with successful outcome and bony union include applying a rigid construct and limiting the extent of anterior deltoid takedown to preserve acromial vascularity. A transacromial approach was described as a method of preserving the acromial soft-tissue envelope and blood supply.[13] Cannulated screw fixation can be done with either a headless screw device or a partially threaded cannulated screw, with tension band augmentation using nonabsorbable suture or small-gauge wire (Figure 12). Bone graft augmentation often is useful, either as perpendicular bone grafting at the nonunion or grafting to the mesoacromial site using bone from the greater tuberosity. The use of demineralized bone matrix also has been described.[13] Tension band augmentation of the cannulated screw fixation stabilizes the bone graft. Cannulated screw and tension band constructs have achieved excellent union rates in conjunction with bone graft augmentation.[15]

Arthroscopic subacromial decompression for an unstable os acromiale was found to prevent the complications associated with open reduction and internal fixation, which include nonunion, the need for hardware removal, and deltoid dysfunction.[21,22] Studies reporting only arthroscopic decompression found that the results were less favorable than those of arthroscopic complete excision because partial excision of the os acromiale was inadequate for eliminating the site of pain.[21,22] Other studies reported excellent functional outcomes and pain relief when standard arthroscopic techniques were used for extensive fragment excision, while maintaining the integrity of the deltoid periosteum and CA ligament confluence.[23,24] Isokinetic testing demonstrated that rehabilitation after arthroscopic fragment excision had restored symmetric strength.[24]

The surgical treatment of an os acromiale is determined by the requirements of concomitant pathology. If there is no full-thickness rotator cuff tear, preacromial or mesoacromial fragments can be safely and effectively excised using an arthroscopic technique. The technical considerations include the necessity of avoiding disruption of the deltoid periosteum and maintaining the confluence of the CA ligament and deltoid fascia. If a partial-thickness or small or medium-size full-thickness tear is present, arthroscopic fragment excision can be combined with arthroscopic rotator cuff repair. A miniopen repair requires great care when the deltoid split is retracted because the deltoid periosteum can easily be disrupted, often resulting in a poor outcome.

Open reduction and internal fixation of an unstable mesoacromion should be strongly considered if a large rotator cuff tear is present, regardless of whether the tear can be repaired. The overall goal is to maintain the integrity of the CA arch. Cannulated screw fixation, with or without tension band wiring and bone graft augmentation, can achieve union and return of function. Transacromial approaches and deltoid takedown approaches have been described with successful results.[25] Meticulous repair of the deltoid with transosseous fixation should be considered if an anterior deltoid takedown is done. Open excision of the fragment must be avoided; the only exception is a salvage procedure after failed open reduction and internal fixation, often with concomitant hardware failure and os acromiale fragment comminution.

***Coracoid Impingement***

Patients with coracoid impingement frequently report anterior shoulder pain. The true etiology of coracoid impingement has not been described, although studies suggested that subcoracoid impingement and pain are caused by impingement of the subscapularis tendon between the lesser tuberosity and the coracoid process.[26-28] Despite the extensive attention to the contribution of the CA arch to subacromial impingement, Gerber and associates[28,29] described coracoid impingement as a primary contributor to anterior shoulder pain as well as resultant pathology of the rotator cuff and the long head of the biceps. Subsequent studies more accurately described anatomic variations of the coracoid and the contribution of coracoid impingement to rotator cuff pathology.[26,30-33]

A patient with coracoid impingement usually has anterior shoulder pain localized near the lateral aspect of the coracoid and the coracohumeral interval. The patient often has discomfort with cross-body activities such as driving or writing on a chalkboard or overhead activities such as throwing or tennis serving. The pain may radiate into the region of the biceps distally toward the elbow. Coracoid impingement can occur with typical outlet or subacromial impingement, and symptoms of those conditions may be present.

The physical examination reveals palpable tenderness in the region of the coracohumeral interval. The coracoid can be tender to palpation in the absence of pathology, and therefore coracoid tenderness alone does not indicate coracoid impingement. Palpation of the biceps within the groove proximally may produce discomfort. Subscapularis testing, including the belly press and lift-off tests, may cause pain, and manual muscle testing involving the subscapularis may reveal weakness. A Hawkins impingement test with a cross-body component frequently elicits localized pain in the anterior aspect of the shoulder near the coracoid region. Biceps tests, including the Speed test and Yergason active compression test, may be positive if the patient has associated biceps pathology.

CT is extremely useful for evaluating the bony elements of the coracoid and surrounding structures.[6,29,30] However, MRI is more sensitive and specific for evaluating adjacent soft-tissue lesions. Computerized digital measurement tools can be used in a clinical setting with CT or axial MRI studies to easily and consistently identify three simple measurements (Figure 13). The coracoid overlap is the perpendicular distance between the most prominent tip of the coracoid to the line representing the plane of the glenoid fossa. The coracoid index is measured in a similar fashion, but the point of reference is the base of the coracoid. The coracohumeral distance or coracohumeral interval is the shortest distance between the coracoid process and the subchondral bone of the humeral head. Although no specific parameters have been established for these measurements, anecdotal evidence suggests that a coracohumeral interval of less than 10 mm may create a predisposition to coracoid impingement. Coracoid overlap and a coracoid index of more than 15 to 20 mm also may contribute to progressive coracoid impingement.

The anterosuperior rotator cuff, including the subscapularis and the rotator cuff interval, should be evaluated to identify any concomitant pathology,[26,33,34] which will affect the choice of an open or arthroscopic surgical approach. The conjoined tendon frequently is thickened at its confluence with the CA ligament along the lateral aspect of the coracoid tip, and this condition may contribute to the soft-tissue component of coracoid impingement (Figure 6). A similarly thickened CA ligament is identified with subacromial impingement.

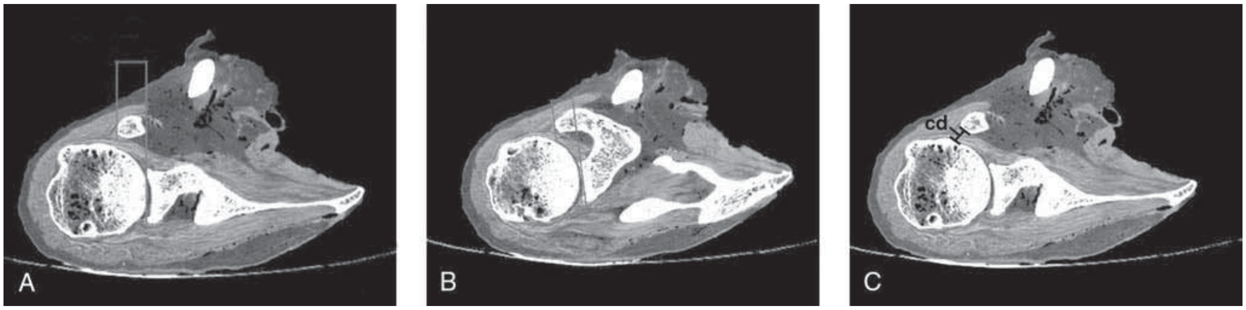



**Figure 13** Axial CT scans showing coracoid overlap **(A)**, coracoid index **(B)**, and coracohumeral distance—cd **(C)**. (Reproduced with permission from Kleist KD, Freehill MQ, Hamilton L, Buss DD, Fritts H: Computer tomography analysis of the coracoid process and anatomic structures of the shoulder after arthroscopic coracoid decompression: A cadaveric study. *J Shoulder Elbow Surg* 2007;16:245-250.)

The treatment of coracoid impingement syndrome is similar to that of subacromial impingement: avoidance of aggravating activities, including cross-body motions; and a standard rehabilitation program focusing on scapular stabilization, rotator cuff strengthening, and correction of any pectoralis major contracture. It is important to evaluate the scapula because scapular malposition can have a role in functional coracoid impingement. A subcoracoid injection can provide pain relief.

If the symptoms persist despite appropriate nonsurgical treatment, the lateral coracoid tip can be excised. Good results have been obtained using an open or arthroscopic subcoracoid decompression technique.[30,33,35] An arthroscopic approach carries little risk of injury to adjacent neurovascular and soft-tissue structures.[6,36] Presurgical imaging of the coracoid morphology can be useful in estimating the required extent of coracoid excision and determining whether an arthroscopic or open approach should be used. If resection of more than 10 mm of the coracoid tip is required, an open coracoid decompression allows the conjoined tendon to be approximated, with transosseous fixation into the residual coracoid. Transosseous fixation can prevent soft-tissue dehiscence in the area of the conjoined tendon and decrease the risk of subcoracoid soft-tissue adhesions and scarring.

Concomitant pathology, such as a subscapularis tear or long head of the biceps abnormality, often can be repaired using a standard deltopectoral approach, with a concurrent open coracoid decompression and conjoined tendon repair. A rotator cuff interval approach, as described by Kleist and associates[6] and Lo and associates,[33] can be used for arthroscopic coracoid decompression. The surgeon's preference is important in the choice of an arthroscopic or open approach.

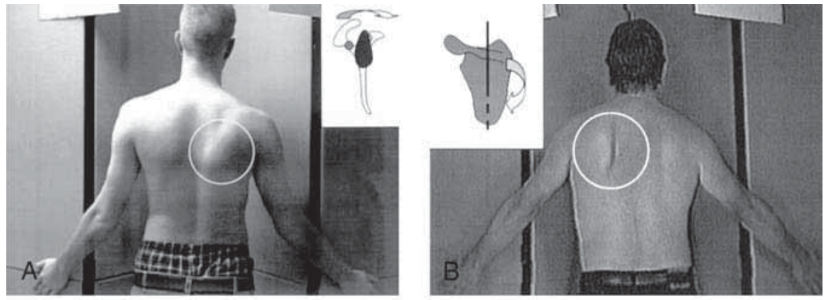
## Functional Impingement

### Glenohumeral Internal Rotation Deficit

Symptomatic functional impingement caused by a GIRD and scapular dysfunction most commonly occurs in athletes. A GIRD typically results in an altered range of motion, in which there is loss of internal rotation compared with the contralateral side. Several possible etiologies have been described, including microtrauma to the static and dynamic constraints of the glenohumeral joint from repetitive overhead throwing, contracture of the posteroinferior joint capsule, and osseous or bony adaptation of the proximal humerus to the effects of repetitive throwing and torsion.[37-40] The loss of internal rotation ultimately results from contracture and thickening of the posteroinferior portion of the glenohumeral joint capsule, which is the focus of stress during the deceleration phase of the overhead throwing motion. Two studies found that GIRD and subsequent mechanical alterations can lead to internal impingement and secondary subacromial impingement.[39,41] An overhead athlete with shoulder discomfort should be assessed for GIRD. Posterior capsular contracture can occur in a patient who has concomitant rotator cuff pathology or subacromial impingement or has undergone shoulder surgery. Prompt nonsurgical treatment often relieves symptoms and prevents further injury for a patient with functional impingement.

The physical examination findings for a patient with GIRD may be typical of subacromial or internal impingement, including positive impingement signs, a positive active compression test, and weakness of

**Figure 14** Assessment of internal rotation with patient in the side-lying (lateral decubitus) position.



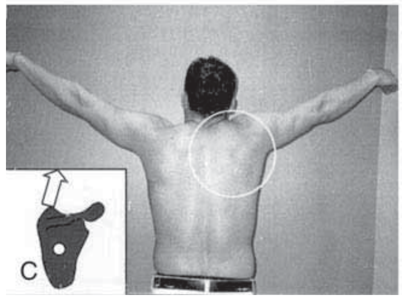




**Figure 15** A prominent inferomedial border dorsally at rest **(A)**, a prominent entire medial border dorsally at rest **(B)**, and a superior border elevated and scapula anteriorly displaced at rest **(C)**. (Reproduced with permission from Kibler WB, Uhl TL, Maddux JW, Brooks PV, Zeller B, McMullen J: Qualitative clinical evaluation of scapular dysfunction: A reliability study. *J Shoulder Elbow Surg* 2002;11:550-556.)

the rotator cuff on manual muscle testing. Internal and external rotation can be reliably assessed with the patient supine or in the side-lying (lateral decubitus) position. In the supine position, the patient's scapula is stabilized against the examination table, and the shoulder is brought into full external rotation with the arm off the side of the table and then into full internal rotation until a firm end point is identified. In the side-lying position, the scapula is stabilized, and the arm is brought into 90° of forward flexion while resting against the examination table, then externally rotated superiorly and inferiorly to assess the extent of internal rotation (Figure 14). This motion is compared with that of the contralateral side to determine the total arc of motion as well as internal and external rotation. In an asymptomatic overhead athlete, a GIRD typically is measured at 10° to 15°.[38,40,42] In contrast, throwing athletes with significant shoulder symptoms were found to have a GIRD of more than 20°, with continual shoulder pain and a type II superior labrum anterior and posterior lesion.[38,42,43]

A GIRD is initially treated with an aggressive program of posterior capsular stretching, in which standard stretching is combined with proprioceptive neuromuscular facilitation to stretch the rotator cuff contributor to the loss of internal rotation. This program is done in conjunction with a program for scapular stabilization, core stabilization, and rotator cuff strengthening. Nonsurgical treatment of a GIRD has an excellent success rate when begun early.[43,44] Continuing the stretching exercises for 2 to 3 years was found to offer long-term gains in internal rotation as well as decreased shoulder and elbow symptoms in major league pitchers.[43]

Posterior capsular tightness that is not resolved by nonsurgical treatment can be surgically treated. Successful outcomes were described after arthroscopic posterior capsular release in conjunction with débridement or repair of associated capsulolabral pathology.[39,41]

### *Scapular Dysfunction*

Scapular dysfunction and malposition are significant causes of ongoing shoulder girdle pain and dysfunction. As the ability to assess scapular motion and function has advanced, different alterations in scapular movement have been identified,[2,45] as well as specific treatment regimens. The form and pattern of scapular dyskinesis are dictated by alterations in the stabilizing functions of the upper and lower trapezius and the serratus muscles. The three common patterns of scapular dyskinesis are inferomedial, medial, and superomedial border prominence (Figure 15). Scapular dysfunction, including inflexibility of the pectoralis minor and generalized weakness and fatigue of the periscapular muscles, can result from inhibition secondary to pain in the shoulder girdle region.

Neurologic assessment should be routine for a patient with significant alteration in scapular function. Injury to the long thoracic or spinal accessory nerve can lead to paralysis of the serratus anterior or trapezius, respectively. Although nonsurgical treatment of scapular dysfunction frequently is successful, surgical intervention may be required if symptoms persist more than 12 to 24 months. A tendon transfer can be used to stabilize the scapula. Painful inhibition after injury to the glenohumeral joint or AC joint also can lead to an altered muscle activation pattern. Glenohumeral instability and rotator cuff injury can alter scapulohumeral rhythm, which improves after surgical treatment.[46]

All causative factors must be identified before scapular dysfunction is treated. Surgical intervention may be required to restore normal scapular kinematics after an injury to the glenohumeral joint, rotator cuff, or shoulder girdle suspensory areas, including the AC joint and the distal clavicle. Patients with specific muscle imbalance and inflexibility are treated using a rehabilitation program that includes posterior capsular stretching exercises for an associated GIRD and anterior stretching for any pectoralis minor contracture. Core strengthening exercises should be initiated in conjunction with general trunk flexibility exercises. In addition, closed-chain kinetic exercises, scapular stabilization exercises, and rotator cuff strengthening exercises should be progressively used in combination. The core strengthening and periscapular strengthening program usually should precede any distal rotator cuff program because scapular stability must be established before specific glenohumeral mechanics, to avoid exacerbating impingement-type symptoms.[47]

## Summary

Typical and atypical impingement syndromes are common causes of shoulder pain. An appropriate history and physical examination, with judicious use of diagnostic testing, allow an accurate diagnosis of a rotator cuff disorder. An appropriate treatment regimen can be prescribed after the type of impingement syndrome is determined.

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