

## **Clinical abnormalities that affect the rotator cuff: A guide for MR interpretation**

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Rotator cuff disease can be related to a number of factors that include degenerative, vascular, traumatic and mechanical causes. The most common cause of rotator cuff pathology is primary degeneration of the rotator cuff tendons with wear and aging. External and internal impingement, microinstability of the shoulder, trauma, overuse associated with athletic and occupational activities, underlying systemic disorders that weaken the tendon such as diabetes, renal and collagen vascular disease, steroid use and smoking contribute to rotator cuff pathology. Identification of the problem and the origin of the disease can often be augmented by the use of magnetic resonance imaging (MRI). It is important to be aware of these causes because they may not be apparent by just identifying the pathology seen on MRI. When finding these lesions on an MRI, the interpreting radiologist and referring clinician should correlate them with symptoms, keeping in mind that there can be a discrepancy between MRI findings and symptoms related to rotator cuff disease. The clinician and the radiologist also need to associate various combinations of abnormalities together and connect them with the etiology.

Tendinosis, partial and full thickness tears of the rotator cuff are common, especially as people get older. The supraspinatus tendon is most often involved, followed by the infraspinatus and subscapularis tendons. The teres minor is rarely abnormal, and when it is torn, it is often related to posterior shoulder instability. Rotator cuff defects can be seen in 30% of asymptomatic persons over the age of 60 years (1) and 65% of asymptomatic persons over 70 years of age (2). Tears can be seen in professional throwing athletes without any pain or decrease in performance (3). Many tears do not interfere with normal shoulder activity (4). Yet, there are many symptomatic patients with rotator cuff tears who benefit from therapeutic intervention by arthroscopic or open repair. MRI aids in the assessment of the tendons and muscles for clinical decision-making. It can show which tendons are involved, the type and size of the tear, and if there is retraction or fraying of the tendon ends. Muscle atrophy and fatty infiltration may play a big role in determining if surgery should be attempted. Other findings, such as labral and biceps tendon tears in association with rotator cuff tears are also of crucial interest to the referring clinician.

### *Shoulder Impingement*

Shoulder impingement is a common cause of pain in adults. It is divided into external and internal categories. External impingement is produced by structural changes outside of the joint and includes primary and subcoracoid impingement. Internal impingement also called secondary extrinsic impingement is caused by rotator cuff and capsular abnormalities and is divided into postero-superior, anterosuperior, and anterior forms.

### *Primary Extrinsic impingement*

The primary extrinsic impingement syndrome is a common, progressively painful compression of the supraspinatus tendon, subacromial-subdeltoid bursa, and long head of the biceps tendon between the humeral head and the coracoacromial arch. The coracoacromial arch is comprised of the undersurface of the anterior third of the acromion, the coracoacromial ligament, the anterior third of the coracoid process, the acromioclavicular (AC) joint, and the distal clavicle. The pain occurs when the

arm is raised into a position of abduction and external rotation or is elevated forward and internally rotated ((5). Hypovascularity in the supraspinatus tendon, mechanical wear, acute trauma and repetitive microtrauma all serve as potential causes or effects of primary extrinsic impingement.

This form of impingement is often seen in young athletes, especially those who engage in sports that involve overhead arm movement such as tennis, football, and baseball. It is also seen in persons whose occupations require overhead motion and in older individuals with degenerative changes of the coracoacromial arch. Classic primary extrinsic type of impingement results from entrapment of the supraspinatus tendon by the coracoacromial arch, which is caused by variations in the architecture of the coracoacromial arch, including one or more of the following: a subacromial enthesophyte, anteriorly hooked acromion, downsloping or low-lying acromion, inferior AC joint osteophytes, os acromiale, or a thickened coracoacromial ligament. Although these findings may be seen on MRI, linking them back to impingement can only be made by an examination of the patient. This part of the puzzle is a clinical abnormality not detectable on MRI.

An os acromiale is more mobile than an acromion without accessory ossification centers. Motion is increased at the deltoid tendon insertion along the lateral inferior aspect of the ossification center. Contraction of the deltoid muscle can pull down on the lateral aspect of the acromion, creating a hinge effect that narrows the subacromial outlet with resultant impingement and tears of the rotator cuff. The coracoacromial ligament also commonly attaches to the unfused segment. Hyperostosis, or a step-off, may develop along the undersurface of the os acromiale at its site of failed union, leading to impingement and eventual tear of the supraspinatus tendon (6-9). Although in the past, surgical and radiologic literature has confirmed the association between os acromiale and impingement and rotator cuff pathology (6, 8, 10), there is some controversy about this (9). Simply identifying the os acromiale does not implicate it as the source of shoulder pain or rotator cuff disease. It is important to report the presence of an os acromiale, especially in patients who are being considered for subacromial decompression for impingement (8). The os acromiale has the potential for even more mobility and there can be further weakening of the synchondrosis after this procedure, leading to more impingement (5, 8). MRI has an advantage over conventional radiographs and CT in that it can reveal underlying, frequently associated rotator cuff tendon abnormalities and abnormal signal intensity in the os acromiale. In addition to possibly causing primary extrinsic impingement of the supraspinatus, an unstable or stressed os acromiale can cause shoulder pain. On MRI, it is common to see abnormal marrow signal with T2 hyperintensity around the synchondrosis. The synchondrosis may also be abnormally elevated in signal intensity. If the pain does not diminish, small ossification centers are often excised with reattachment of the deltoid; larger or unstable ones may be fused to the rest of the acromion.

Other osseous causes of primary extrinsic impingement include a prominent greater tuberosity caused by fracture, malunion, or nonunion and Paget disease. Paget disease can produce enlargement of the acromion, resulting in narrowing of the coracoacromial outlet (11). The enlargement of the acromion and occasional abnormal signal intensity of the marrow can aid in diagnosis on MRI. Other bones may also be involved. A conventional radiograph should be consulted in such cases to identify pagetoid characteristics and confirm the diagnosis.

An impingement-injection test, first described by Neer, is a tool to assess the presence of primary extrinsic impingement (12). The examiner elevates the humerus of the patient with one hand while preventing scapular rotation with the other. Pain is produced when the greater tuberosity of the humeral head impinges against the acromion. The pain is relieved by injection of an anesthetic agent into the subacromial space. Caution should be taken when using this test alone for the diagnosis of impingement, because other causes for the pain may also be relieved by the injection.

Rotator cuff disease associated with primary extrinsic impingement may include tendinosis, partial and full thickness tears. MRI characterizes the degree of tendon pathology and displays tendon retraction and associated muscle atrophy or fatty infiltration. MRI demonstrates abnormal distention of the subacromial-subdeltoid bursa or subcoracoid bursa. MRI can also show damage to the rotator interval, located in the region devoid of tendons between the supraspinatus and subscapularis tendons. There may be synovitis, tears of the superior glenohumeral and coracohumeral ligaments and tendinosis, subluxation and/or tears of the biceps tendon in this region.

The biceps tendon often becomes impinged by the coracoacromial arch just before it enters the bicipital groove. Neer has shown that a shallow or laterally placed bicipital groove also exposes the long head of the biceps tendon to impingement by the anterior third of the acromion, resulting in inflammation or rupture of the intra-articular portion of the tendon. Fluid in the biceps tendon sheath is often seen in asymptomatic individuals because this structure communicates with the glenohumeral joint. It can be difficult to diagnose tenosynovitis of the biceps from an MRI since fluid naturally surrounds this tendon. Multiple low signal intensity bands in the tendon sheath are associated with tenosynovitis. With tendinosis, the tendon may be increased in size and may concomitantly or alternatively demonstrate internal high signal intensity on T2 weighting. This “hourglass” appearance is common in the long head of the biceps right before it enters the intertubercular groove. Sagittal images of the biceps are best for assessment of this location, but this is one area that is not well imaged by the routine planes used for shoulder MRI. In addition the magic angle phenomenon can be seen in this portion of the biceps on short TE images, simulating tendinosis. Partial thickness tears may be difficult to distinguish from tendinosis if the tendon is not thinned, split, or irregular. The full-thickness biceps tendon rupture is seen with discontinuity of the tendon and several axial MR images showing an empty intertubercular groove.

Subacromial-subdeltoid bursitis and bursal thickening and fibrosis are associated with primary extrinsic impingement. It has been shown that bursitis is often associated with shoulder pain and a perception of disability (13). Previous injection of a local anesthetic or steroid preparation may cause difficulty in interpretation of the rotator cuff tendons and bursa (14) (15) (16, 17). The area of injection is high signal intensity on MRI and can mimic a bursitis, tendinopathy, or tear. Injected fluid can remain in the subacromial subdeltoid bursa for up to three days following injection (15). Since injections are performed without imaging guidance, a misdirected injection into the rotator cuff tendons and muscles can result in high signal intensity in those structures that simulate tendon tears and muscle strains (18). This can interfere with interpretation of muscle and tendons for several months, leading to false positive diagnoses of strain and tear. Therefore, it is important to ask the patient if they have had a recent injection and to discourage clinicians from performing injections before they order an MRI.

### *Subcoracoid Impingement*

Subcoracoid impingement, also called (coracoid and coracohumeral impingement) is an uncommon form of impingement that occurs when the coracohumeral distance narrows, encroaching upon the subscapularis tendon, subcoracoid bursa, and anterior capsule (19, 20). The narrowing can be caused by different factors, including morphologic variants of the lateral tip of the coracoid, alteration in coraco-glenoid angle, microinstability that narrows the distance, coracoid fractures and coracoid surgery (21).

Subscapularis tendon tears result from this type of impingement. One may also see cystic changes in the lesser tuberosity of the humerus (20). Symptoms are produced with the humeral head in forward flexion and medial rotation reducing the distance between the coracoid and humerus. Narrowing of this distance can be caused by congenital hypertrophy, elongation of the coracoid, or acquired

conditions including coracoid or lesser tuberosity fractures, glenoid osteotomy, and coracoid process transfer during surgery (22).

It is recommended that imaging plays a supporting role in this evaluation with the confirmation of this disorder remaining a clinical and arthroscopic diagnosis. Patients present with anteromedial shoulder pain and tenderness over the coracoid process. One should look for subscapularis tendon pathology, lesser tuberosity cysts and marrow signal abnormalities, biceps tendon subluxations, as well as subcoracoid and anterior subacromial-subdeltoid bursitis (23). Measuring coracohumeral distance is not always the key to the diagnosis and at this time is not considered diagnostic. The normal coracohumeral distance has been quoted at 8.6 mm in one study (24), whereas a distance of 10.5-11.5 mm has been called clinically significant narrowing in another. The coracohumeral distance is 1.4-3mm smaller in females than in males (24). Because the coracohumeral distance is narrowed in internal rotation, it has been suggested that imaging of the glenohumeral joint be obtained in internal rotation when specifically evaluating this form of impingement (25).

There is also a correlation between narrowing of the coracohumeral interval and the presence of a full thickness supraspinatus tendon tear due to anterosuperior humeral migration (26). This secondary form of subcoracoid impingement may be seen in older individuals resulting in subscapularis tendon degeneration and tears and is a cause of continued anterior shoulder pain following supraspinatus tendon repair.

#### *Internal Impingement (Secondary Extrinsic Impingement)*

Secondary extrinsic impingement can be caused by inferior narrowing of the coracoacromial outlet from glenohumeral or scapulothoracic instability. Posterior-superior impingement is an internal form of impingement that can produce shoulder pain and can lead to partial thickness tears of the undersurface of the rotator cuff.

Internal (secondary extrinsic impingement) varies from primary extrinsic impingement in that there is no morphologic abnormality of the coracoacromial arch. This form of impingement is produced in the setting of glenohumeral microinstability, which produces narrowing of the coracoacromial outlet owing to anterior capsular laxity resulting in anterior translation during throwing (27, 28). It can also result from scapulothoracic instability, glenohumeral internal rotation deficit, and posterosuperior glenoid impingement. It is more common in younger patients and athletes who perform repetitive overhead or throwing motions that produce repetitive shear force during the late cocking and early acceleration phases. In one study, 68% of patients with anterior or multidirectional shoulder instability had impingement signs in addition to apprehension and capsular laxity (29).

#### *Anterior Superior Impingement*

Painful shoulder internal rotation and adduction, can be caused by a form of internal impingement involving abnormal anterosuperior humeral head translation on the glenoid as a result of biceps pulley lesions. The undersurface of the biceps pulley mechanism and subscapularis tendon may impinge against the anterosuperior glenoid rim resulting in biceps subluxation and subscapularis partial failure. The supraspinatus tendon may also be involved. This problem is termed *anterior superior impingement (ASI)*(30). This impingement can be seen in those who use repetitive overhead movement in an occupation (masonry) or sport (tennis, pole vaulting).

MRI can demonstrate abnormalities of the biceps pulley in the rotator interval, biceps subluxation and dislocation and tears of the subscapularis and supraspinatus tendons around the biceps pulley (31-33) (31-33). Treatment of type 1 lesions of the pulley may require suture repair of the pulley. The type 2 lesions involve debridement of the supraspinatus with a transtendon repair. Type 3 lesions

involve repair of the subscapularis tendon tear and biceps stabilization. The type 4 lesion is treated with repair of the supraspinatus and subscapularis tendon tears as well as a biceps tenodesis or tenotomy. It is of interest that there is also an increased incidence of AC joint arthritis in patients with ASI. It is debated whether this entity is associated with SLAP lesions (30, 34).

#### *Posterior Superior Glenoid Impingement*

Another form of instability is believed by some to be associated with impingement of the labrum, rotator cuff and joint capsule between the greater tuberosity and glenoid rim in certain positions, especially in the act of throwing. This is termed posteriorsuperior glenoid impingement or *PSGI*. PSGI is believed by some to be the result of impingement of the humeral head with the glenoid rim in the ABER position, commonly seen in overhead athletes, especially baseball and tennis players, javelin throwers, and swimmers. It can also be present in non-athletes (28, 35). PSGI is seen in the setting of repetitive abduction and external rotation. There is significant repetitive shear force applied to the glenohumeral joint during the late cocking phase when the shoulder is in extreme abduction and external rotation. This leads to anterior capsular failure, causing anterior glenohumeral joint laxity and increased anterior translation during throwing. Scapular dyskinesia, called the *SICK* scapula (scapular malposition, inferior medial border prominence, coracoid pain and malposition and dyskinesia of scapular movement) or hyperangulation of the humerus in relation to the scapula may further aggravate the condition (36, 37). This imbalance of the humerus sliding anteriorly in this ABER position with impaction of the greater tuberosity and glenoid margin, impinges on the undersurface of the posterior supraspinatus and anterior infraspinatus tendons and the posterior superior labrum. Findings on MRI are summarized in Table and include fraying and tears of these structures in 81-100% of these patients with the clinical diagnosis of internal impingement (28, 36, 38). Cysts and impaction deformities are also seen along the posterior aspect of the greater tuberosity (39). This condition is treated conservatively in those with minor structural damage, with surgical debridement and repair of the labrum and tendons along with capsular plication reserved for more extensive tears.

Whether PSGI is the result of an impingement process or an alteration in the biomechanics of the glenohumeral joint is a subject of debate since impingement of the supraspinatus and infraspinatus tendons on the glenoid can be a normal physiological occurrence (38, 40-43). When this results in injury to the rotator cuff, glenoid labrum and greater tuberosity is it the result of increased frequency and/or force of impingement?

#### *Glenohumeral internal rotation deficit (GIRD)*

An alternative mechanism for similar soft tissue findings to PSGI that includes articular sided supraspinatus and infraspinatus tears and superior labral tears, is that of *glenohumeral internal rotation deficit (GIRD)*, originally described by Burkhart (44-46). Thought to be a form of microinstability rather than impingement by some and not others, GIRD is seen in the dominant shoulder of throwing athletes, resulting from repetitive throwing motions in the ABER position, which leads to a posterior fibrosing capsulitis. This thickening of the posterior band of the IGHL is a precursor to the Bennett lesion, described previously and commonly seen in baseball pitchers. The proponents of this theory hypothesize that microinstability of the shoulder due to repetitive overuse during follow-through phase of throwing creates the tight posterior capsule combined with anterior capsular stretching. The center of rotation of the glenohumeral joint is shifted posterosuperiorly on the glenoid, changing the forces on the superior labrum, biceps tendon and anchor. This leads to superior labral tears with excessive twisting of the

biceps tendon, causing a “peel back” Type II SLAP lesion of the posterior superior labrum along with twisting of the supraspinatus and infraspinatus tendons producing articular sided partial tears in a similar location to what has been described with PSGI. GIRD is characterized by pain in the late cocking and early acceleration phase of throwing. The athletes have a decrease in throwing velocity, hampering athletic performance creating a “dead arm” characterized by sudden sharp pain and discomfort throwing a fastball during the late cocking or early acceleration phase created by the SLAP lesion. This posterior capsular tightening allows for further external rotation of the shoulder by increasing clearance for the greater tuberosity with limited internal rotation of the humerus.

Imaging findings of GIRD include thickening of the posterior capsule adjacent to the posterior labrum, tears of the superior labrum posterior to the biceps anchor, articular sided tears of the posterior supraspinatus and infraspinatus tendons, sclerosis and cyst formation in the posterior rim of the glenoid and posterior humeral head. Remodeling can occur in the posterior glenoid rim. One may see posterior decentering of the humeral head on routine images, but sometimes the ABER positioning can bring out subtle posterior decentering of the humeral head on the glenoid not seen with the arm at the side. The ABER position can also show the posterior peel back of the superior labrum and the undersurface tears of the tendons.

Treatment of GIRD is at first conservative with physical therapy that stretches the posteroinferior capsule as well as arthroscopic debridement and repair of the soft tissue pathology. If that fails, arthroscopic release of the posterior capsule can be performed to improve motion (45).

#### *Other Causes of Rotator Cuff Impingement*

Supraspinatus muscle hypertrophy is an intrinsic form of impingement of the supraspinatus muscle. It is common in athletes such as weight-lifters and swimmers who perform forceful overhead arm movements. This form of impingement can be seen in the presence of a normal coracoacromial arch (47). Patients tend to be most symptomatic when carrying heavy objects with the arms at the sides and when sleeping. In this condition, there is deformation of the supraspinatus musculotendinous junction beneath the AC joint and anterior acromion on oblique coronal MR images. Supraspinatus muscle hypertrophy can be treated by reduction in overhead activity.

Callus around a healed greater tuberosity fracture can also lead to impingement of the supraspinatus tendon between the remodeled prominent greater tuberosity and the lateral acromion. A prominent greater tuberosity can lead to impingement of the supraspinatus tendon by the same mechanism.

Scapulothoracic instability results from abnormal scapular motion during throwing. Stability and positioning of the scapulothoracic articulation is dependent on a delicate balance between scapulothoracic muscles (trapezius, serratus anterior, rhomboids, and latissimus dorsi). Abnormalities in the biomechanics of this articulation can result in malpositioning of the glenoid articular fossa. This may produce superior positioning of the humeral head with respect to the glenoid with narrowing of the coracoacromial outlet, leading to impingement.

#### *Shoulder instability and rotator cuff tears*

Rotator cuff tears associated with instability are especially common in the patient over 40 years of age who may have pre-existing tendinosis or partial tears. In anterior instability, the anteriorly unstable humeral head can produce narrowing of the coracoacromial outlet, leading to rotator cuff pathology. Such sequelae of instability mimics primary impingement and should be thought of, particularly in the younger patient (adolescent and young adult) who is more likely to have instability

rather than impingement. Tears of the subscapularis tendon are often associated with anterior or posterior dislocation and are important to detect, since subscapularis strengthening is one of the treatments for instability (48). With posterior humeral dislocation, extreme tension placed at the glenoid fossa on the subscapularis tendon at the time of dislocation may predispose to a tear or avulsion of the lesser tuberosity (49). Tears of the teres minor and infraspinatus tendons can be seen more frequently with posterior dislocation (50). In one study, 42% of acute posterior dislocations were associated with rotator cuff lesions (51). Rotator interval injuries may also occur with subluxation or dislocation in the form of stretching or tears. Tears of the rotator interval can be treated with surgical closure (52, 53).

Some hypothesize that microinstability can be associated with rotator interval laxity and avulsion or laxity of the SGHL or MGHL allowing for abnormal contact between the humerus and the articular surface of the anterior supraspinatus tendon and the superior glenoid labrum resulting in anterior impingement or what is termed the *superior labrum anterior cuff (SLAC Lesion)* (30). These patients present with anterior superior shoulder pain that mimics extrinsic impingement without classical impingement signs. The shoulder fatigues easily and there can be parascapular pain. Symptoms are provoked on clinical examination by shoulder extension and superior displacement of the humeral head with the arm abducted. On MRI, one will see involvement of the anterior superior labrum (type 2 SLAP lesion) and glenoid. There is a partial thickness tear of the articular side of the anterior supraspinatus tendon. The MGHL may also be involved. Treatment for SLAC lesions include imbrication of a patulous rotator interval, reattachment of the labrum, and repair of the SGHL and MGHL and tendon tear.

## REFERENCES

1. Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on magnetic resonance images of asymptomatic shoulders. *J Bone and Joint Surg* 1995; 77-A:10-15.
2. Milgrom C, Schaffler M, Gilbert S, van Holsbeeck M. Rotator-cuff changes in asymptomatic adults. The effect of age, hand dominance and gender. *J Bone Joint Surg Br* 1995; 77:296-298.
3. Miniaci A, Mascia AT, Salonen DC, Becker EJ. Magnetic resonance imaging of the shoulder in asymptomatic professional baseball pitchers. *Am J Sports Med* 2002; 30:66-73.
4. Yamaguchi K, Sher JS, Andersen WK, et al. Glenohumeral motion in patients with rotator cuff tears: a comparison of asymptomatic and symptomatic shoulders. *J Shoulder Elbow Surg* 2000; 9:6-11.
5. Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the shoulder. *J Bone and Joint Surg* 1972; 54-A:41-50.
6. Mudge MK, Wood VE, Frykman GK. Rotator cuff tears associated with os acromiale. *J Bone and Joint Surg* 1984; 66-A:427-429.
7. Edelson JG, Zuckerman J, Hershkovitz I. Os acromiale: Anatomy and surgical implications. *J Bone Joint Surg* 1993; 74-B:551-555.
8. Hutchinson MR, Veenstra MA. Arthroscopic decompression of shoulder impingement secondary to os acromiale. *Arthroscopy* 1993; 9:28-32.
9. Ouellette H, Thomas BJ, Kassarian A, et al. Re-examining the association of os acromiale with supraspinatus and infraspinatus tears. *Skeletal Radiol* 2007; 36:835-839.

10. Park JG, Lee JK, Phelps CT. Os acromiale associated with rotator cuff impingement: MR imaging of the shoulder. *Radiology* 1994; 193:255-257.
11. Boutin RD, Spitz DJ, Newman JS, Lenchik L, Steinbach LS. Complications in Paget disease at MR imaging. *Radiology* 1998; 209:641-651.
12. Neer CS. Impingement lesions. *Clin Orthop and Rel Res* 1983; 173:70.
13. Krief OP, Huguet D. Shoulder pain and disability: comparison with MR findings. *AJR Am J Roentgenol* 2006; 186:1234-1239.
14. Kieft GJ, Bloem JL, Rozing PM, Obermann WR. Rotator cuff impingement syndrome: MR imaging. *Radiology* 1988; 166:211-214.
15. Major NM. MR imaging after therapeutic injection of the subacromial bursa. *Skeletal Radiol* 1999; 28:628-631.
16. Bergman AG, Fredericson M. Shoulder MRI after impingement test injection. *Skeletal Radiol* 1998; 27:365-368.
17. Wright RW, Fritts HM, Tierney GS, Buss DD. MR imaging of the shoulder after an impingement test: how long to wait. *AJR Am J Roentgenol* 1998; 171:769-773.
18. Borick JM, Kurzweil PR. Magnetic resonance imaging appearance of the shoulder after subacromial injection with corticosteroids can mimic a rotator cuff tear. *Arthroscopy* 2008; 24:846-849.
19. Gerber C, Terrier F, Ganz R. The role of the coracoid process in the chronic impingement syndrome. *J Bone Joint Surg* 1985; 67B:703.
20. Dines DM, Warren RF, Inglis AE, Pavlov H. The coracoid impingement syndrome. *J Bone and Joint Surg* 1990; 72-B:314-316.
21. Gumina S, Postacchini F, Orsina L, Cinotti G. The morphometry of the coracoid process - its aetiologic role in subcoracoid impingement syndrome. *Int Orthop* 1999; 23:198-201.
22. Paulson MM, Watnik NF, Dines DM. Coracoid impingement syndrome, rotator interval reconstruction, and biceps tenodesis in the overhead athlete. *Orthop Clin North Am* 2001; 32:485-493, ix.
23. Stallenberg B, Destate N, Feipel V, Gevenoio PA. Involvement of the anterior portion of the subacromial-subdeltoid bursa in the painful shoulder. *AJR Am J Roentgenol* 2006; 187:894-900.
24. Gerber C, Terrier F, Zehnder R, Ganz R. The subcoracoid space. An anatomic study. *Clin Orthop Relat Res* 1987:132-138.
25. Giaroli EL, Major NM, Lemley DE, Lee J. Coracohumeral interval imaging in subcoracoid impingement syndrome on MRI. *AJR Am J Roentgenol* 2006; 186:242-246.
26. MacMahon PJ, Taylor DH, Duke D, Brennan DD, O'Brien J, Eustace SJ. Contribution of full-thickness supraspinatus tendon tears to acquired subcoracoid impingement. *Clin Radiol* 2007; 62:556-563.
27. Dillman CJ, Fleisig GS, Andrews JR. Biomechanics of pitching with emphasis upon shoulder kinematics. *J Orthop Sports Phys Ther* 1993; 18:402-408.
28. Jobe CM. Posterior superior glenoid impingement: Expanded spectrum. *Arthroscopy* 1995; 11:530-536.
29. Warner JJP, Micheli LJ, Arsianian L, Kennedy J, Kennedy R. Patterns of flexibility, laxity, and strength in normal shoulders and shoulders with instability and impingement. *Am J Sports Med* 1990; 18:366.
30. Habermeyer P, Magosch P, Pritsch M, Scheibel MT, Lichtenberg S. Anterosuperior impingement of the shoulder as a result of pulley lesions: a prospective arthroscopic study. *J Shoulder Elbow Surg* 2004; 13:5-12.

31. Morag Y, Jacobson JA, Shields G, et al. MR arthrography of rotator interval, long head of the biceps brachii, and biceps pulley of the shoulder. *Radiology* 2005; 235:21-30.
32. Bigoni BJ, Chung CB. MR imaging of the rotator cuff interval. *Magn Reson Imaging Clin N Am* 2004; 12:61-73, vi.
33. Weishaupt D, Zanetti M, Tanner A, Gerber C, Hodler J. Lesions of the reflection pulley of the long biceps tendon. MR arthrographic findings. *Invest Radiol* 1999; 34:463-469.
34. Gerber C, Sebesta A. Impingement of the deep surface of the subscapularis tendon and the reflection pulley on the anterosuperior glenoid rim: a preliminary report. *J Shoulder Elbow Surg* 2000; 9:483-490.
35. Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. *Shoulder Elbow Surg* 1992; 1:238-245.
36. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part III: The SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. *Arthroscopy* 2003; 19:641-661.
37. Laudner KG, Myers JB, Pasquale MR, Bradley JP, Lephart SM. Scapular dysfunction in throwers with pathologic internal impingement. *J Orthop Sports Phys Ther* 2006; 36:485-494.
38. Giaroli EL, Major NM, Higgins LD. MRI of internal impingement of the shoulder. *AJR Am J Roentgenol* 2005; 185:925-929.
39. Tirman PFJ, Bost F, Garvin GJ, et. al. Posterosuperior glenoid impingement of the shoulder: Findings at MR imaging and MR arthrography with arthroscopic correlation. *Radiology* 1994; 193:431-436.
40. Barber FA, Morgan CD, Burkhart SS, Jobe CM. Current Controversies. Point counterpoint. Labrum/biceps/cuff dysfunction in the throwing athlete. *Arthroscopy* 1999; 15:852-857.
41. Halbrecht JL, Tirman P, Atkin D. Internal impingement of the shoulder: comparison of findings between the throwing and nonthrowing shoulders of college baseball players. *Arthroscopy* 1999; 15:253-258.
42. McFarland EG, Hsu CY, Neira C, O'Neil O. Internal impingement of the shoulder: a clinical and arthroscopic analysis. *J Shoulder Elbow Surg* 1999; 8:458-460.
43. Gold GE, Pappas GP, Blemker SS, et al. Abduction and external rotation in shoulder impingement: an open MR study on healthy volunteers initial experience. *Radiology* 2007; 244:815-822.
44. Ticker JB, Beim GM, Warner JJ. Recognition and treatment of refractory posterior capsular contracture of the shoulder. *Arthroscopy* 2000; 16:27-34.
45. Kaplan LD, McMahon PJ, Towers J, Irrgang JJ, Rodosky MW. Internal impingement: findings on magnetic resonance imaging and arthroscopic evaluation. *Arthroscopy* 2004; 20:701-704.
46. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part I: pathoanatomy and biomechanics. *Arthroscopy* 2003; 19:404-420.
47. Crues JV, Fareed DO. Magnetic resonance imaging of shoulder impingement. *Topics in Mag Res Im* 1991; 3:39-49.
48. Blasier RB, Soslowsky LJ, Malicky DM, Palmer ML. Posterior glenohumeral subluxation: active and passive stabilization in a biomechanical model. *J Bone Joint Surg Am* 1997; 79:433-440.
49. Matsen FA, 3rd, Harryman DT, 2nd, Sidles JA. Mechanics of glenohumeral instability. *Clin Sports Med* 1991; 10:783-788.

50. Hottya GA, Tirman PFJ, Bost FW, Montgomery WH, Wolf EM, Genant HK. Tear of the posterior shoulder stabilizers after posterior dislocation: MR imaging and MR arthrographic findings with arthroscopic correlation. *AJR* 1998; 171:763-768.
51. Saupe N, White LM, Bleakney R, et al. Acute traumatic posterior shoulder dislocation: MR findings. *Radiology* 2008; 248:185-193.
52. Kim SH, Kim HK, Sun JI, Park JS, Oh I. Arthroscopic capsulolabroplasty for posteroinferior multidirectional instability of the shoulder. *Am J Sports Med* 2004; 32:594-607.
53. Kim SH, Ha KI, Yoo JC, Noh KC. Kim's lesion: an incomplete and concealed avulsion of the posteroinferior labrum in posterior or multidirectional posteroinferior instability of the shoulder. *Arthroscopy* 2004; 20:712-720.