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Review

Imaging shoulder impingement

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Abstract. Appropriate imaging and clinical examinations may lead to early diagnosis and treatment of the shoulder impingement syndrome, thus preventing progression to a complete tear of the rotator cuff. In this article, we discuss the anatomic and pathophysiologic bases of the syndrome, and the rationale for certain imaging tests to evaluate it. Special radiographic projections to show the supraspinatus outlet and inferior surface of the anterior third of the acromion, combined with magnetic resonance images, usually provide the most useful information regarding the causes of impingement.

Key words: Shoulder impingement syndrome – Painful arc syndrome – Subacromial spur – Acromioclavicular spur – Supraspinatus outlet view – Os acromiale

The shoulder impingement syndrome (SIS) is a common, progressively painful and disabling disorder resulting primarily from compression of the subacromial bursa or supraspinatus tendon between the greater tuberosity of the humerus and the coracoacromial arch. A combination of clinical findings and appropriate imaging evaluations may lead to an early diagnosis, permitting conservative, nonsurgical treatment or arthroscopic decompression before the disorder progresses to a complete tear of the rotator cuff. Even at a later stage of disability, imaging studies may delineate a specific cause of impingement, facilitating surgical intervention for pain relief.

The SIS, also known as the painful arc syndrome, is characterized by intense pain in the shoulder when the arm is either abducted and externally rotated or elevated forward and internally rotated. The pain is caused by repeated entrapment and compression of the supraspinatus tendon between the proximal end of the humerus inferiorly, particularly its greater tuberosity, and one or more components of the coracoacromial arch superiorly. The coracoacromial arch is composed of five basic structures: the distal clavicle, acromioclavicular joint, anterior third of the acromion, coracoacromial ligament, and anterior third of the coracoid process (Fig. 1). Repetitive trauma leads to progressive edema and hemorrhage of the rotator cuff and hypertrophy of the synovium and subsynovial fat of the subacromial bursa. In a vicious cycle, the resultant loss of space predisposes the soft tissues to further injury, with increased pain and disability. The SIS most commonly affects young athletes who engage in repetitive overhead throwing, and older individuals with degenerative changes of the coracoacromial arch.

As long ago as 1931, Meyer [20] proposed that attrition of the rotator cuff by the undersurface of the acromion was the main cause of cuff rupture. Codman [3], in 1934 described a "critical zone" of hypovascularity

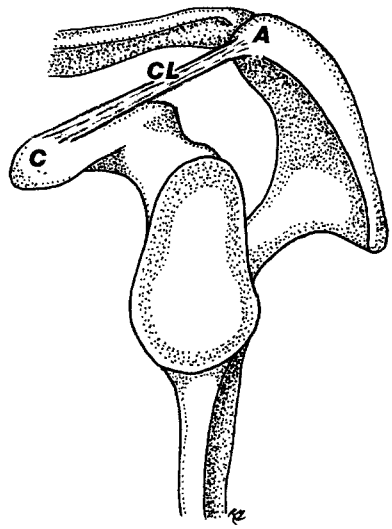

Fig. 1. Coracoacromial arch as it appears in a supraspinatus outlet view. The arch consists of the distal clavicle, acromioclavicular joint, anterior third of the acromion (*A*), coracoacromial ligament (*CL*), and anterior third of the coracoid process (*C*)

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in the supraspinatus tendon in the region of its attachment at the greater tuberosity of the humerus. In 1949, the "supraspinatus syndrome" (the term anteceding "SIS") and the effect of acromionectomy upon its clinical course were characterized [1]. Neer [25], in 1972, recounted his success with anterior acromioplasty in treating the syndrome, which he attributed primarily to abnormalities of the anterior third of the acromion. Investigating dissected scapulae, Neer found that some specimens contained abnormalities of the acromion, including proliferative spurs along the anterior lip and undersurface of the anterior third. He hypothesized that the abnormalities were caused by impingement, including excessive traction by the coracoacromial ligament subsequent to its repeated impaction by the humeral head. Neer's clinical observations confirmed that the critical site for degenerative tendinitis and tendon rupture was the supraspinatus tendon, extending at times to include the tendons of the infraspinatus and long head of the biceps. Neer described an "impingement-injection" clinical test in which the examiner elevates the humerus of the patient with one hand while preventing scapular rotation with the other [26]; pain is produced when the greater tuberosity impinges against the acromion. Although this maneuver alone cannot be used to distinguish SIS from other painful disorders of the shoulder, a diagnosis of SIS may be made should the resultant pain be relieved by injection into the subacromial space of a short-acting anesthetic agent.

Prior to Neer's investigations, the treatment of SIS consisted of complete or lateral acromionectomy, procedures which not only weakened the deltoid muscle, whose strength is essential when the rotator cuff is deficient, but also removed a portion of the acromion that was normal. Neer subsequently stressed the importance of excising only that part of the acromion that manifested the spurs and roughened surface upon which the supraspinatus tendon was rubbing. He advocated excision of no more than the anterior third of the acromion along with the attached coracoacromial ligament. If other sites of pathology were discovered at operation, such as a hypertrophied acromioclavicular joint, spurs of the greater tuberosity, or adhesions involving the long head of the biceps, Neer proposed that they also should be removed. Should the supraspinatus tendon become impinged between the greater tuberosity and a degenerated acromioclavicular joint which has undergone osteophyte formation and capsular hypertrophy, treatment might combine anterior acromioplasty and partial or complete excision of the acromioclavicular joint [34]. Rockwood and Lyons [30] have increased their rate of favorable operative results by using a modification of the Neer acromioplasty, in which more of the anterior aspect of the acromion is resected than had been proposed by Neer [26].

According to Neer [26], 95% of tears of the rotator cuff result from impingement. Furthermore, most of these tears occur in the hypovascular "critical zone" described by Codman. Neer [25] described three progressive pathologic stages of impingement lesions and a treatment plan for each: stage I – edema and hemorrhage of the rotator cuff, typically seen in individuals under 25 years of age, and often reversible with conservative treatment; stage II – fibrosis of the subacromial bursa and inflammation of the supraspinatus tendon, typically found in individuals between 25 and 40 years of age, and treatable with bursectomy or division of the coracoacromial ligament; and, with further wear, stage III – a tear of the rotator cuff or tendon of the long head of the biceps, and osteophytes and other alterations of the anterior acromion and greater tuberosity, typically seen in individuals over 40 years of age, and treatable with anterior acromioplasty combined with repair of the rotator cuff. Neer [25] also showed that a laterally placed or shallow bicipital groove exposed the tendon of the long head of the biceps to impingement by the anterior third of the acromion, resulting in inflammation or rupture of the intra-articular portion of the tendon, and demonstrated that a timely anterior acromioplasty may lead to clinical improvement. Should conservative treatment fail, current opinion favors subacromial or acromioclavicular therapeutic decompression. While the therapeutic approach to impingement lesions has evolved since Neer's earliest work, his concept of the progressive nature of impingement continues to guide clinical practice.

Impingement thus results from depression of one or more components of the coracoacromial arch or roughening of its undersurface. Abnormalities of the acromion that may lead to the SIS include an acromion that is excessively low-lying in relation to the distal end of the clavicle (Fig. 2), an acromion whose anterior third is excessively downward-sloping (Fig. 3) [5] or downward "hooking" [6, 9], osteophytic spurs along the inferior surface of the anterior third of the acromion (Fig. 4) [25], an os acromiale (Fig. 5) [24], and hypertrophic changes of the acromioclavicular joint (Fig. 6) [34]. Rarely, a downward sloping coracoid process causes impingement of the subscapularis tendon or indirectly results in supraspinatus impingement by depressing the coracoacromial ligament [4, 11]. Another rare cause of impingement is post-traumatic calcification or ossification of the coracoacromial ligament (Fig. 7) [23].

An os acromiale (Fig. 5) results from a failure of fusion of one or more of the three ossification centers of the acromion: the metaacromion (the most proximal center), the mesoacromion, and the preacromion (the most distal center). The most common site of nonunion is the junction of the mesoacromion and metaacromion [24]. Instability may cause repeated trauma to the rotator cuff. The abnormality, bilateral in over 60% of individuals, is best seen on radiographs in the axillary projection, where it may be mistaken for a fracture.

In summary, SIS primarily reflects supraspinatus impingement related to abnormalities of the coracoacromial arch which cause excessive narrowing of the supraspinatus outlet. A less common type of impingement, "nonoutlet" impingement, has other causes: a prominent greater tuberosity, such as may result from fracture malunion; loss of the normal suspensory mechanism of the shoulder due to injury of the acromioclavicular joint; septic or calcific bursitis; and glenohumeral instability. Identification of one of the causes of impingement enables therapy to be appropriately directed.

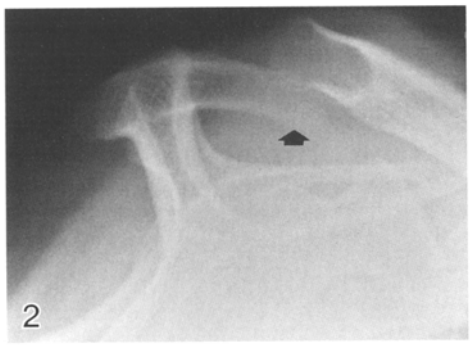
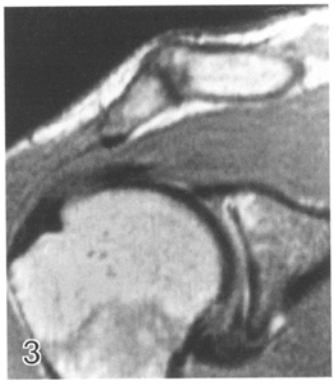
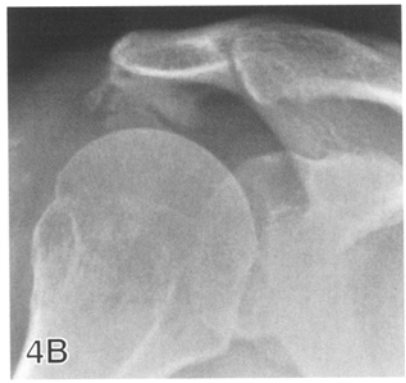


Fig. 2. Acromion that is excessively low-lying (*arrow*) in relation to the distal end of the clavicle, causing impingement, as seen in a supraspinatus outlet view of the shoulder of a 55-year-old man. Although there may be asymptomatic minor variations, the lower margins of both bones usually are at the same level

Fig. 3. Excessively downward-sloping anterior third of the acromion, causing impingement in a 28-year-old man, as shown in coronal magnetic resonance (MR) image (SE 1800/15). The anterolateral aspect of the acromion is projected inferiorly, producing a sharp projection which compresses the subacromial bursa and supraspinatus tendon

Fig. 4A, B. Osteophytic spurs along the anterior third of the acromion, causing impingement. **A** Anthropologic specimen. (From [5], courtesy of J.G. Edelson, C. Taitz, and the Editor of the *Journal of Bone and Joint Surgery*). **B** Anteroposterior radiograph, made with the X-ray beam angled 30° caudally, shows spurs and an unfused preacromion





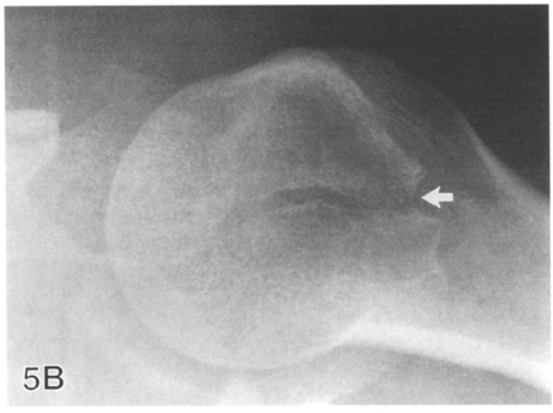
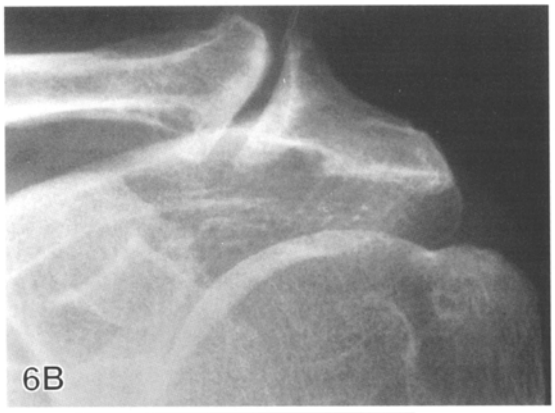


Fig. 5A, B. Unstable os acromiale as a cause of impingement in a 48-year-old man. **A** Anthropologic specimen showing os acromiale as seen from above. Degenerative changes are present on both the proximal and distal sides of the ununited apophysis. (From [5], courtesy of J.G. Edelson, C. Taitz, and the Editor of the *Journal of Bone and Joint Surgery*). **B** Axial radiograph showing failure of fusion (*arrow*) of the meta-acromion and mesoacromion. The latter is the most common type of os acromiale. The irregular margins at the site of nonunion mirror the degenerative findings in **A**



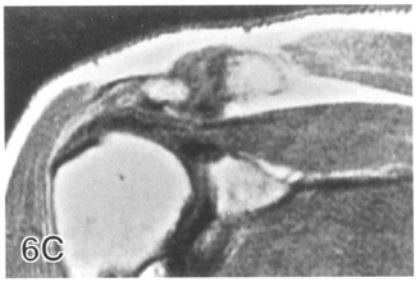
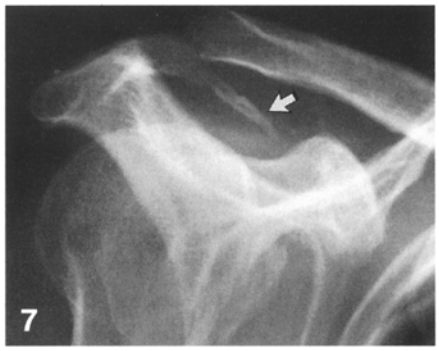


Fig. 6A–C. Hypertrophic changes of the acromioclavicular joint as a cause of impingement. **A** Anthropologic specimen as seen from below, showing extensive degenerative changes of the acromial facet of the acromioclavicular joint (*long arrow*). Hypertrophic spurs along the anterior surface of the acromion are also present (*short arrow*). (From [5], courtesy of J.G. Edelson, C. Taitz, and the Editor of the *Journal of Bone and Joint Surgery*). **B** Anteroposterior radiograph showing hypertrophic spurs projecting inferiorly from the acromioclavicular joint of a 72-year-old man. **C** Coronal MR image (SE 480/30) of the shoulder of a 49-year-old man with symptoms of impingement. Extensive degenerative capsular hypertrophy of the acromioclavicular joint, represented by the intermediate-intensity signal extending from the joint, compresses the subacromial bursa and supraspinatus tendon



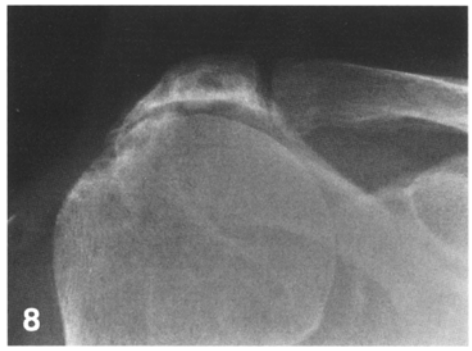
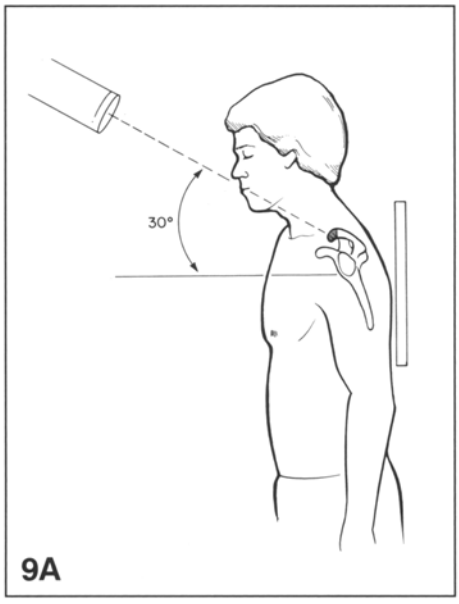
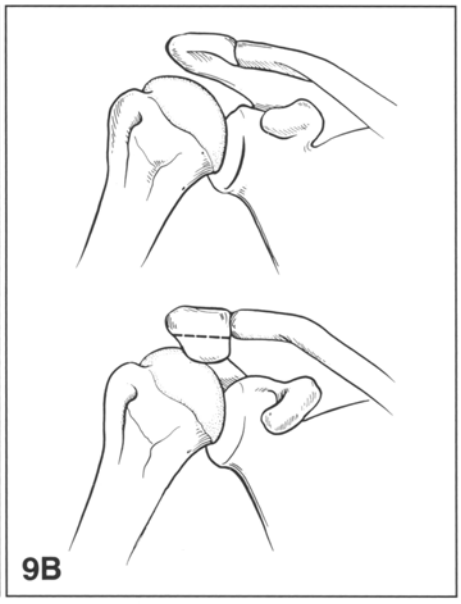


Fig. 7. Post-traumatic ossification of the coracoacromial ligament (*arrow*), causing impingement in a 54-year-old man, as seen in a supraspinatus outlet view

Fig. 8. Anteroposterior radiograph of the shoulder showing loss of the interspace between the humeral head and acromion, with resultant concavity of the inferior surface of the anterior acromion, the effects of long-standing impingement in an 81-year-old man





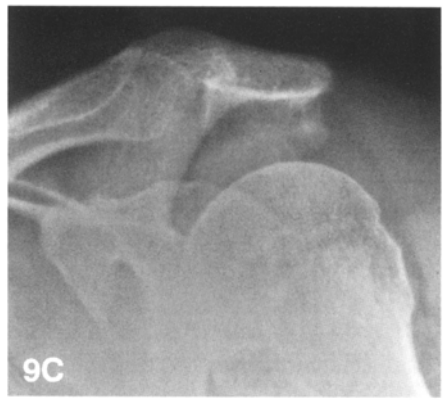
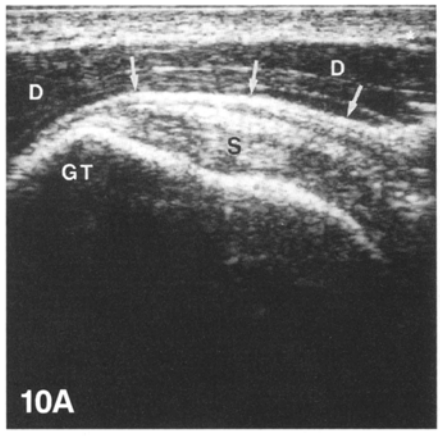


Fig. 9A–C. Anteroposterior view of the shoulder made with 30° caudal angulation of the X-ray beam shows the anterior part of the acromion. **A** Positions of the standing patient and the X-ray tube. **B** Diagram of the image as projected in a standard anteroposterior radiograph (*top*) and in an anteroposterior radiograph made with 30° of caudal angulation of the X-ray beam (*bottom*). (**A** and **B** from [17], courtesy of R.F. Kilcoyne et al. and the American Roentgen Ray Society). **C** Anteroposterior radiograph with 30° caudal angulation of the X-ray beam shows hypertrophic changes of the anterior third of the acromion, causing impingement in a 44-year-old woman



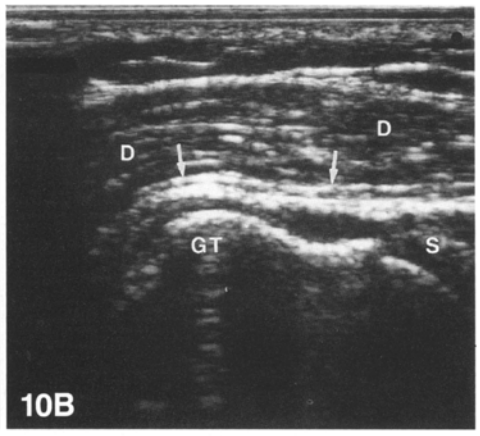
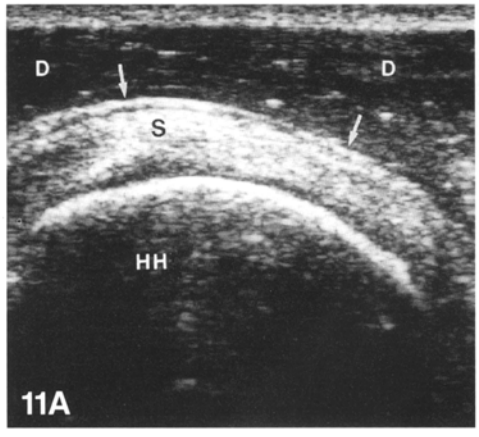


Fig. 10A, B. Longitudinal sonograms of supraspinatus tendons (*S*). *GT*, Greater tuberosity; *D*, deltoid muscle; *arrows*, subacromial-subdeltoid bursal complex. **A** Normal supraspinatus tendon is a beak-shaped structure attached to the greater tuberosity. **B** Complete tear of the supraspinatus tendon. Note the absence of the tendon at the site of its attachment to the greater tuberosity. Instead, the tendon is retracted, leading to concavity of the superior margin of the bursal complex. (Courtesy of L.A. Mack)



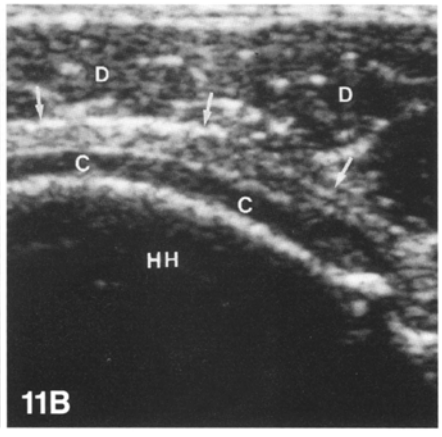


Fig. 11A, B. Transverse sonograms of supraspinatus tendons (*S*) of shoulders different from Fig. 10A, B. *HH*, Humeral head; *D*, deltoid muscle; *C*, articular cartilage of humeral head; *arrows*, subacromial-subdeltoid bursal complex. **A** Normal supraspinatus tendon. The tendon appears as a band of medium-level echoes superior to the humeral head and inferior to the deltoid muscle and subacromial-subdeltoid bursal complex. **B** Tear of the supraspinatus tendon, more extensive than in Fig. 10B. The tendon is completely absent. The thickened subacromial-subdeltoid bursal complex abuts the hypoechoic articular cartilage. (Courtesy of L.A. Mack)

Plain radiographic evaluation

Conventional anteroposterior radiographs of the shoulder may show abnormalities that have resulted from long-standing SIS, but do not necessarily reveal the lesion that caused them. As examples, the greater tuberosity of the humerus may show bone proliferation and cyst-like rarefactions, or it may be flattened and sclerotic; the humeral head may undergo superior migration, with the interspace between the head and acromion (the acromiohumeral distance) narrower than 6 mm [29]; ultimately, the unrelenting pressure by the unopposed humeral head may cause pressure atrophy and resultant concavity of the inferior surface of the anterior acromion (Fig. 8).

Although conventional anterioposterior radiographs of the shoulder may reveal osteophytes of the inferior surface of the acromion and along the inferior margin of the acromioclavicular joint [12], the anterior third of the acromion is partially obscured. Fluoroscopically controlled spot radiographs of a standing patient who leans forward, toward the image intensifier, have been used to view the undersurface of the anterior acromion, and may show impinging osteophytes [28]. The anterior acromion is also shown to advantage on an anteroposterior radiograph made with the central ray angled 30° in a caudal direction (Figs. 4B, 9). Another view that shows the anterior third of the acromion is the supraspinatus-outlet view. In this modification of the scapular Y (transscapular) view, the patient is positioned 30°–40° posterior-obliquely or 40°–60° anterior-obliquely, and the central ray is angled 10°–15° in a caudal direction (Figs. 2, 7) [17]. A combination of the anteroposterior view, with 30° caudal angulation of the central ray, and outlet view provide near-orthogonal projections of the anterior third of the acromion and the acromioclavicular joint. In one series of 100 patients with subacromial spurs found on anteroposterior radiographs made with the standing patient rotated until the scapula was parallel to the film, and 22°–25° of caudal angulation of the X-ray beam, subsequent shoulder arthrograms showed an incidence of rotator cuff tear that strongly and directly correlated with the size of the spur [14]. An os acromiale is best depicted on the axillary view (Fig. 5B).

Ultrasonographic evaluation

The diagnostic accuracy of ultrasonography is largely operator-dependent. Although ultrasonography can detect increased fluid in the subacromial bursa, and refinements in equipment and technique have increased its capability of providing information beyond the presence or absence of a complete tear of the rotator cuff, the examination has many pitfalls [22]. Some investigators have found it insensitive to small or incomplete tears, while others have had good results in the detection of incomplete tears and excellent results in the detection of full-thickness tears [35].

The examination is usually performed with the patient seated. Imaging begins with the patient's arm adducted and in neutral position, continues as the arm is internally rotated, and terminates after the arm is elevated laterally and, finally, anteriorly.

The normal subacromial-subdeltoid bursa is thinner than 2 mm at ultrasonography and is usually poorly seen in its nondistended state [2]. In a series of 37 patients with clinical evidence of impingement and surgical evidence of inflamed and thickened subacromial bursae, there were 30 in whom ultrasonography revealed gradual distention of the subacromial bursa with lateral pooling of fluid when the arm was elevated [8]. The remaining 7 patients had false-negative ultrasonographic findings. In 21 of the 37 patients, the stage of impingement was so early that a tear of the rotator cuff could not be detected at ultrasonography or surgery.

Cuff inflammation or fibrosis may appear ultrasonographically as a diffuse or focal abnormality in echogenicity [19]. A cuff tear may be manifest as a nonvisualized cuff, a focal defect or discontinuity in the cuff, or a focal abnormality in echogenicity (Figs. 10, 11) [21]. According to Mack et al. [19], rotator cuff ultrasonography is most useful in patients over 50 years of age who might be expected to have complete tears; however, in younger patients with persistent symptoms, a negative sonogram should be followed by further imaging examinations.

Glenohumeral arthrographic and subacromial bursographic evaluation

Magnetic resonance imaging has largely supplanted glenohumeral arthrography and subacromial bursography for evaluating SIS. During glenohumeral arthrography, the subacromial bursa opacifies only in the presence of a full-thickness tear of the rotator cuff. Arthrography may also disclose partial tears of the articular surface of the cuff, bicipital lesions, and adhesive capsulitis. In the presence of an intact rotator cuff, the relationship between the greater tuberosity and the coracoacromial arch can be demonstrated by subacromial bursography [18, 33]. The result of the bursogram is considered to be positive if the contrast material pools in the lateral aspect of the bursa when the arm is elevated, reflecting compression, or when the volume of the bursa is diminished to less than 5 ml. Although ligamentous and capsular stretching due to repetitive overuse of the glenohumeral joint may cause symptoms and signs impossible to distinguish from those related to impingement, they are usually associated with a normal bursogram. False-positive results may arise from failure to completely opacify the bursa or from adhesive bursitis in the absence of impingement. False-negative results may occur if the range of motion of the arm is restricted.

Computed tomographic and CT-arthrographic evaluation

The glenohumeral joint is the most mobile joint in the body, largely because only a small portion of the humeral head is in contact with the glenoid at any given time. The trade-off for this exceptional mobility is a loss of inherent stability. Glenohumeral instability may lead to

secondary impingement, i.e., a contraction of the supraspinatus outlet due to abnormal superior migration of the humeral head. The associated abnormalities of the glenoid labrum and joint capsule are usually well depicted on computed tomographic (CT-)arthrographic or magnetic resonance (MR) images [27]. The treatment of this type of secondary impingement varies from exercise to strengthen the rotator cuff [9], to subacromial decompression, to surgical correction of the underlying instability.

Goldthwait, in 1909 [10], demonstrated that abnormalities in the shape, size, or slope of the coracoid process could cause the subcoracoid bursa to be compressed between the coracoid process and the humeral head. It has since been shown that these anatomical variations may be idiopathic, traumatic, or iatrogenic [11], and that coracoid impingement may lead to pain and clicking, especially during forward flexion, medial rotation and adduction. Coracohumeral decompression may be accomplished by excising the distal 1.5 cm of the coracoid process and reattaching the conjoined tendon (coracobrachialis and short head of the biceps) to the remainder of the coracoid process [4]. Computed tomography has been claimed to be useful to compare the coracohumeral space of the symptomatic shoulder to that of the asymptomatic one [4, 11].

Magnetic resonance imaging evaluation

MR imaging is useful to evaluate the soft tissue abnormalities associated with SIS, and also provides information regarding the offending structures above the rotator cuff and subacromial bursa [13, 16, 31, 32]. Subacromial bursitis is usually evident as a thickening of the normally thin band of bursal fat. Interestingly, excessive fluid is not commonly seen in association with bursitis due to impingement syndrome. The presence of fluid in the subacromial-subdeltoid bursal complex should alert one to search for an alternate explanation, such as a rotator cuff tear or bursitis due to inflammatory arthritis or infection. This is especially true if the fluid collection is large.

The MR findings of supraspinatus tendinitis may be nonspecific. The inflamed tendon may show focal or diffuse intermediate signal intensity in T1-weighted images which may decrease or slightly increase in intensity with T2-weighted imaging. This same appearance may be associated with tendon degeneration, and also has been detected in asymptomatic subjects, in some of whom foci of increased signal intensity in the distal part of the supraspinatus tendon may be attributable to the "magic angle" phenomenon [7]. A tendon tear is diagnosed by its focal or diffuse increased signal intensity in T1-weighted or proton density images, accompanied by a well-defined high-intensity signal signifying a focal fluid collection at the site of the tear in T2-weighted images. In the case of a partial-thickness tear, the fluid collection may extend to the bursal or articular surface of the joint (Fig. 12). It is particularly important to identify the exact site of a partial-thickness tear on the MR images, because arthroscopy that is limited to the glenohumeral joint will not show a partial-thickness tear which communicates only with the subacromial bursa.

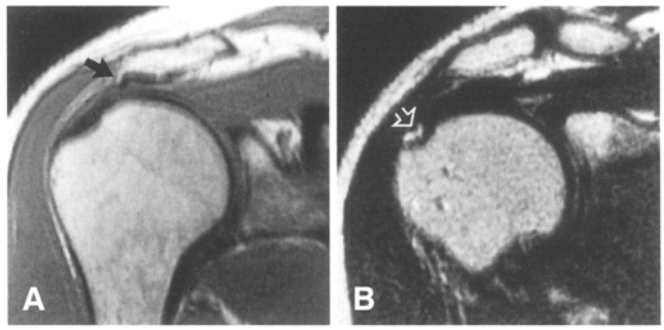


Fig. 12A, B. Partial tear of the supraspinatus tendon, caused by an impinging spur of the anterior acromion in a 43-year-old man. **A** Coronal MR image (SE 1800/15) shows a prominent spur of the lateral aspect of the anterior acromion (*arrow*). **B** Coronal MR image (SE 1800/70) reveals a focus of high-intensity signal, representing fluid in the inferior aspect of the tendon (*arrow*). The signal does not extend to the subacromial bursa

Identification on the MR scan of the cause of impingement assists the surgeon in planning appropriate intervention. Specific abnormalities include an anterior acromion which is abnormally large or lies too low with respect to the distal clavicle (Fig. 2), a downward-"hooking" [6] or downward-sloping anterior acromion (Fig. 3), and degenerative spurs or capsular hypertrophy of the acromioclavicular joint (Fig. 6C). A small- to moderate-sized spur of the anterior acromion may appear as a focus of signal void that is difficult to distinguish from a hypertrophied coracoacromial ligament. However, large spurs often contain marrow and are therefore seen as extensions of the high-intensity signal of the marrow of the parent bone. A word of caution: when the deltoid tendon is not imaged in continuity, coronal images may disclose a triangular pseudospur formed by a slip of deltoid tendon projecting from the acromion in an inferolateral direction [15].

Summary

The SIS results primarily from compression of the subacromial bursa or supraspinatus tendon between the greater tuberosity of the humerus and an abnormal coracoacromial arch. Clinical findings combined with appropriate imaging examinations may lead to early diagnosis and successful treatment prior to rupture of the rotator cuff. Plain radiographic examinations of the shoulder, consisting of an anteroposterior view with the central ray angled 30° in a caudal direction and a scapular Y view with the central ray angled 10°–15° in a caudal direction, optimally show the anterior third of the acromion and the acromioclavicular joint, the most common sites of impingement-causing lesions. Magnetic resonance imaging is also a key source of information aiding in the evaluation of the bony and soft-tissue structures of the coracoacromial arch, and the status of the su-

praspinatus tendon. Ultrasonography is useful to detect complete tears of the rotator cuff. Computed tomography, especially when combined with arthrography, is useful to evaluate the glenoid labrum and joint capsule in cases of secondary impingement. Glenohumeral arthrography and subacromial bursography have largely been supplanted by MR imaging.

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