Rotator interval lesions and their relation to coracoid impingement syndrome

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In the last 10 years we have found impingement of the coracoid process on the rotator interval in 12 patients (14 shoulders). Seven of these patients were women and 5 were men; the average patient age was 48.5 years. One patient had a calcified coracohumeral ligament, another patient had an anterior tear of a repaired deltoid flap, and a third had an aberrant pectoralis minor tendon inserted into the rotator interval. Eleven patients had a weak rotator interval, and in 4 cases the rotator interval had a small tear. We closed the rotator interval in all 12 patients. We also performed a coracoidoplasty in 5 of the patients. The condition of all shoulders improved clinically after operation; the average follow-up was 4.2 years. Three patients (4 shoulders) still had moderate pain, and 7 patients (9 shoulders) lacked strength. Internal rotation was the only shoulder movement that remained limited. Although impingement seemed obvious during surgery, experimental studies have reported contradictory findings. J Shoulder Elbow Surg 1999;8:130-5.

Mechanical impingement on the rotator cuff by the overlying acromial arch was postulated as early as 1909 by Goldthwait.[12] Goldthwait also considered the possibility of an impingement on the rotator cuff by the anteromedial portion of the arch, that is, by the coracoid process. Coracoid impingement was also described by Meyer[21] in 1937 and Bennett[2] in 1941. However, Neer's 1972 account[23] of the impingement on the rotator cuff by the anteroinferior portion of the acromion process stands as the classic in the field, overshadowing earlier and later[7,10,11] accounts alike.

The coracoacromial arch includes the acromion, the coracoacromial ligament, and the tip of the coracoid process. Although Bigliani et al[3] focused on the acromion's morphologic characteristics, Renoux et al[28] showed that in most cases it is variation in the height and length of the coracoid process that is responsible for altering the size and shape of the space between the coracoacromial arch and the rotator cuff. Goldthwait had already noted the variability of the shape and length of the coracoid process and stressed the proximity of the coracoid process to the lesser tuberosity, especially in internal rotation.[12]

Anatomic variations in the coracoacromial ligament are frequent. The lateral part of the ligament often joins the coracobrachialis tendon, thus creating a sharp fibrous falx in the anteroinferior part of the coracoacromial arch (Figure 1). This part of the arch faces the rotator interval. Some authors consider the rotator interval a weak point,[15,30] but others regard it as an area of solidity or stability.[9,13] The rotator interval is certainly an important transition area, because structures must pass under or above the coracoid process, especially during adduction and internal rotation, when the cuff passes under the narrowest part of the coracoacromial arch (Figure 2).

We report 12 surgical patients (14 shoulders) who had an apparent impingement on the rotator cuff by the anteroinferior part of the coracoacromial arch.

MATERIALS AND METHODS

From 1984 to 1994 we operated on almost 500 patients with shoulder impingement syndrome. In reviewing the surgical records, we found that 12 patients (14 shoulders) had an impingement of the rotator interval by the coracoid process or the fibrous falx at the time of surgery (Table I). Seven of the 12 patients were women; the other 5 were men. The average age was 48.5 years (range 17 to 60 years). In 11 patients the right shoulder was affected, and in 11 patients the dominant side was affected.

We excluded all patients with a subscapularis rupture because we believe that the signs of impingement by the coracoid process that they sometimes display are caused by the rupture; moreover, the physiopathologic features and treatment of subscapularis ruptures are different.[24] We also excluded cases of secondary impingement caused by superomedial migration of the humeral head in large rotator cuff tears (Figure 3).

We were led to suspect coracoid impingement syndrome in 5 of the shoulders before operation because of patient descriptions of very anterior pain around the coracoid process, with the pain increasing during forward elevation and internal rotation. It was during surgery that

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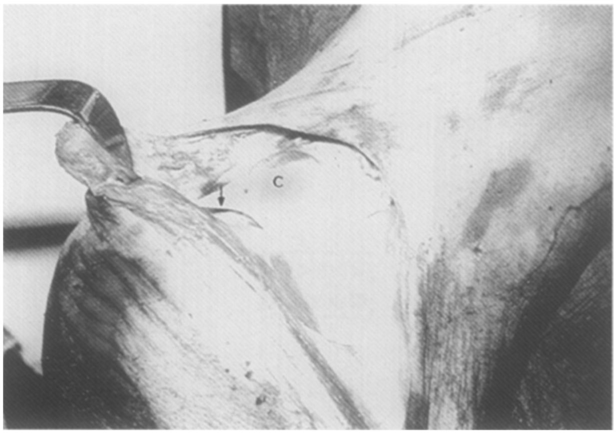

Figure 1 Fibrous falx is formed by coalescence of lateral part of coracoacromial ligament and coracobiceps tendon. *Arrow,* fibrous falx; *C,* coracoid process.

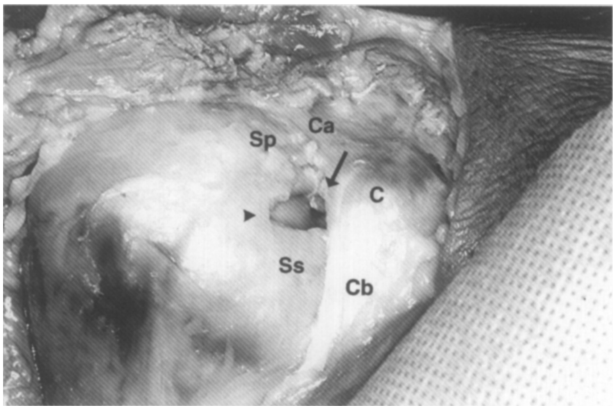

Figure 2 Anatomic view of rotator interval after removal of articular capsule. *Sp,* Supraspinatus; *Ca,* coracoacromial ligament; *arrow,* rotator interval; *C,* coracoid process; *Cb,* coracobrachialis tendon; *Ss,* supraspinatus; *arrowhead,* fibrous falx.

the other 9 cases of coracoid impingement syndrome were discovered. Preoperative imaging techniques were no help for diagnosis, even retrospectively; all patients had plain radiographs. Imaging techniques before surgery also included arthro-computed tomography (arthro-CT) with external and internal rotation, ultrasonography, and a combination. Ultrasonographic results led us to suspect that 2 patients had a rotator cuff tear in the anterior part of the supraspinatus, but both had negative arthro-CT scans. A third patient had an enlarged subscapularis bursa with thinning of the subscapularis, and a fourth had calcifications on the anterior edge of a deltoid flap repair. In the latter case there was no diminution of the coracohumeral space on the CT scan, even with internal rotation.

Six patients had previously undergone shoulder operations. Three had been operated on for calcific tendinitis and 2 for subacromial impingement. The mean preoperative shoulder pain score for the 14 shoulders was 5.5 out of 15 on the Constant[5] scale, and in 9 shoulders the pain was located in the coracoid process. Active shoulder motion was slightly limited; the mean shoulder motion score was 27.5 of 40 on the Constant scale. Except for the patient with calcification of the coracohumeral ligament, whose arm was stiff in both abduction and internal rotation after removal of a calcification, all patients had complete passive motion. One patient had marked limitation of active forward flexion, but only when the arm was in internal rotation (Table II).

With the exception of patient 4, all patients were operated on by two of us (CD, AA). The deltopectoral approach was used for 6 shoulders and the superolateral approach for 8 shoulders. To treat the patient with the very thickly calficied coracohumeral ligament, we divided the ligament but left the rotator interval open. One patient initially had to undergo a deltoid flap repair for a rotator cuff tear. The flap had split in two, and the anterior edge of the flap was being impinged by the coracoid process. After the deltoid flap was repaired, the patient underwent coracoidoplasty. Another patient had an abnormal pectoralis minor tendon that ended in the rotator interval and that abutted the coracoid process during internal rotation. The other 11 patients had rotator intervals that were "too wide

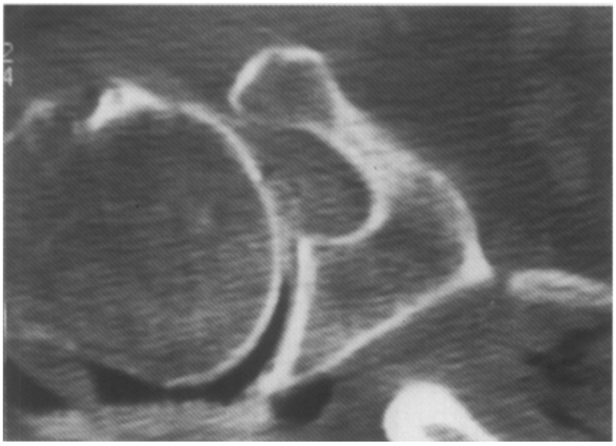

Figure 3 CT scan of impingement on humeral head by coracoid. Impingement was caused by anterosuperior migration of humeral head that was caused, in turn, by massive rotator cuff tear. Distance between bones was 2 mm on CT scan.

and weak," that is, abnormally thin and large. In these patients the rotator interval abutted against the coracoacromial falx or the coracoid process when the arm was elevated and internally rotated. There was a small tear (<1 cm^2) of the rotator interval in 4 of the shoulders; but the biceps tendon, which was exposed as a consequence, remained centered and stable. In no case was there a lesion of the subscapularis or of the superior glenohumeral ligament. In 2 patients the rotator interval was filled by an aberrant pectoralis minor tendon that we did not believe responsible for the impingement.

We closed the rotator interval in all 14 cases. We performed coracoidoplasty in 5 cases to remove the lateral and distal parts of the coracoid. We mainly removed the lateral part of the coracoid until disappearance of the abutment. The coracobrachialis tendon was left intact so that it remained continuous with the periosteum on the medial side of the coracoid. The coracoacromial ligament was divided in all patients, but the coracohumeral ligament, when present, was left intact.

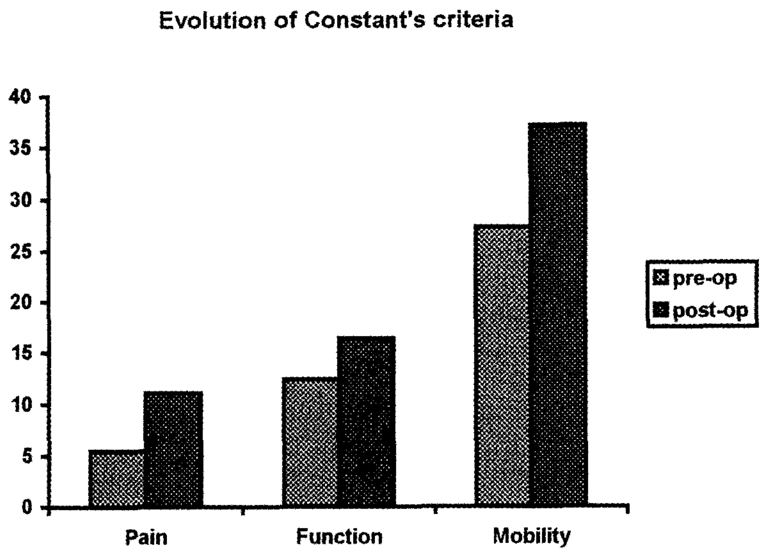

Figure 4 Preoperative and postoperative Constant scores. In most patients strength was not measured before operation.

RESULTS

After an average follow-up of 4.2 years (range 7 months to 11 years; SD ± 5.2 months), all shoulders had improved. The average Constant score, with strength not factored (strength was not recorded preoperatively), increased from 45.2 to 64.6 points on a 75-point scale. Two patients with only 7 months of follow-up were included in these calculations because they both had excellent results. Shoulder mobility was usually complete except for internal rotation. Three patients (4 shoulders) still had a fair amount of pain (score 5 of 15). Seven patients (9 shoulders) reported shoulder weakness, but this was documented only in 3 cases; moreover, there was no possible comparison with preoperative values.

DISCUSSION

Although we were aware of the possibility of coracoid impingement from the start, we discovered most of the impingement lesions during surgery. Our diagnoses of coracoid impingement syndrome were essentially clinical, as reported by others.[7,20,26,27] Pain in an unusual location should alert the physician to this syndrome. In this syndrome pain is very anterior, far beneath the acromion and coracoacromial ligament, and localized on or close to the coracoid process.[7,10,12,18,26,31] Pain may radiate to the arm or even the forearm.[10,12] It increases with shoulder movements combining adduction, flexion, and internal rotation that close the coracohumeral space.[2,7,10-12,18,20,26] Gerber et al[10] reported that for their patients with coracoid impingement, abduction and internal rotation were always painful and that the pain was maximal between 80° and 130°. Dines et al[6] reported that in their series, local anesthetic injections relieved patient pain. Kessel and Watson[17] reported a series of patients with pain in the anterior part of the shoulder, but they believed that the pain, which was maximal at the rotator interval, resulted from impingement by the coracohumeral ligament.[17]

We believe that the tests devised by Yocum[34] and Hawkins and Kennedy[14] are the most likely to yield positive results in cases of coracoid impingement. However, as Patte[26] has already pointed out, none of these tests is specific and the diagnosis is usually made during surgical exploration.

Like most other authors, we found that imaging techniques were useless in diagnosing coracoid impingement syndrome. The exception is Masala et al,[20] who found CT scans and MRI useful for diagnosis. But Masala et al[20] did not clearly define sensitivity and specificity in their series of 15 patients, all of whom were diagnosed with coracoid impingement. Twelve of the 15 patients had narrowing of the coracohumeral distance, and 6 of the 12 had cysts consistent with impingement. However, 2 of their patients had supraspinatus tears that should not be included with coracoid impingement, and 8 had a subscapularis tendon lesion present on MRI. In addition, the patients of Masala et al[20] were mainly athletes (tennis players, swimmers, etc), unlike our patients. Even retrospectively, we have been unable to find any specific pattern that might have alerted us to the presence of a subcoracoid impingement, whatever the imaging technique used. Gerber et al[10,11] performed CT scans and found that the minimal distance between the coracoid process and the lesser tuberosity was 8.6 mm with the arm straight at the side and 6.7 mm with the arm in flexion and internal rotation. Bonutti et al[4] used MRI measurements and found that a distance between the coracoid process and the lesser tuberosity of less than 11 mm is

Table I Patient characteristics

Patient	Sex	Age at surgery*	Affected side/ dominant side	Previous surgery
1	M	36	R/R	Calcification removal 6 months earlier
2	M	50	R/R	Calcification removal 2 years earlier
3	F	49	R/R	
4	F	17	R/R	
5	F	43	L/R	
5	F	49	R/R	
6	M	48	R/R	
7	F	50	L/R	
8	M	31	R/R	Arthroscopic decompression 16 months earlier
9	M	50	R/R	Surgical decompression 2 years earlier
10	F	43	R/R	
11	F	60	R/R	Deltoid-flap repair of rotator cuff tear 1 year earlier
12	F	57	L/R	Arthroscopic calcification removal 2 years earlier
12	F	59	R/R	
Totals	9 F, 5 M	45.8 (17-60)†	R = 11, L = 3, Dom = 11	

*Age in years.
†Mean age in years with range in parentheses.
R, Right side; L, left side; Dom, dominant side.

pathologic. We have little experience in MRI measurements, and we did not find CT scans to be of much use in this series. In the series of 87 cases reported by Gerber et al,[10] only 4 patients were operated on, 37 had coracoid impingement suspected on CT scan, and 46 had a clinical diagnosis.

We think that cases of coracoid impingement syndrome should be divided into 2 classes. The first class comprises cases of mechanical impingement, including calcification or cyst of the subscapularis, sequelae of the humeral head, and fractures of the coracoid process.[7,10,18,20] Coracoid processes that are too long may also cause impingement, but the normal values for the length are not known; Dines et al[7] suggest measuring its length on CT views, with a line perpendicular to the glenoid axis used as a reference point. Contrary to authors who initially addressed this issue,[2,12,21] we believe that cases caused by an excessively lengthy coracoid process are rare and that no coracoid process can induce a cam effect. Aberrant tendons of the pectoralis minor constitute another mechanical cause of coracoid impingement. According to Le Double,[18a] humeral insertion of the pectoralis minor may be present in 15% of patients with coracoid impingement syndrome. However, such abnormalities are rarely symptomatic.[30,31] Technical mistakes during surgery may also be responsible for mechanical impingement. We encountered 2 such cases after performing Gosset's procedure (costal rib graft for treatment of anterior instability), but the patients involved were not included in this report because no information on their postoperative condition was available. Gerber et al[10] reported coracoid impingement after Scott's glenoid osteotomy.[7] Roberts et al[29] pointed out that a posterior offset may be necessary in the design of humeral head prostheses to avoid potential impingement on the coracoid process. CT scans during internal rotation may sometimes be useful for the diagnosis of bony impingement. However, these are rare lesions; we agree with Gerber et al[10] that an osseous impingement can only occur late or be iatrogenic. Dines et al[6] reported 8 cases of osseous impingement in patients who were younger than ours and mostly men. An anatomic abnormality or a surgical complication was the cause of the impingement in most of these cases.[6] This may explain why 7 of the 8 patients in a different study by Dines et al[7] reported an audible snap.

The second class is more controversial and consists of cases in which impingement occurs on the rotator interval near the soft tissue around the coracoid process, mainly the fibrous falx. Goldthwait emphasized that lesions of the subcoracoid bursa may occur during daily movements, especially during internal rotation.[12] Patte reported that communication between the articular capsule and the subcoracoid bursa occurred frequently in his series.[26] At surgery, we found 11 patients with weak and wide rotator intervals. Gerber et al[10] reported 1 case of coracoid impingement caused by anterosuperior migration of the humeral head, but they did not state whether there was a lesion of the rotator interval. Walch[32] reported 5 cases of rotator interval lesions with no instability of the biceps tendon; he could not find a satisfactory physiopathologic explanation for the cases. Ikeda[15] and Nobuhara and Ikeda[25] reported 106 cases of rotator interval lesions. However, their patients had different symptoms than ours. Their younger patients had shoulder instability, and their older patients had shoulder stiffness.

Their patients had increased pain during abduction and external rotation, and some had subluxations.[15,25]

Table II Mean Constant scores of patients' shoulders*

Constant score	Pain (15)	Function (10)	Level of function (10)	Forward flexion (10)	Lateral elevation (10)	External rotation (10)	Internal rotation (10)	Sum (75)
Before operation	5.5	3.62	8.75	7.55	7.25	7.0	5.5	45.17
Afer operation	11.07	7.13	9.28	9.85	9.33	9.23	8.71	64.60

*Constant scores were calculated without taking strength into account because the preoperative strength score was not recorded for most patients.

Unlike the patients of Ikeda,[16] our patients did not have inferior instability or abnormal scapulohumeral rhythm on examination. Ikeda[15] also reported that his patients had intraarticular lesions, which we did not encounter. Only 1 of our patients had rotator interval lesions that conformed to Ikeda's description, and since the publication of his study[15] this rare pathologic condition has been described only in case reports.[1,33] Rowe and Zarins[30] reported a weak rotator interval in 54% of the patients that they operated on for anterior shoulder instability and suggested that a weak rotator interval may explain the presence of inferior lesions in the capsule. None of our patients had a positive apprehension sign, and most of their symptoms appeared when the arm was moderately elevated, usually during internal rotation. Le Huec et al[19] reported 10 cases of traumatic rupture of the rotator interval after forced internal rotation injury had exposed the biceps tendon without destabilizing it. Most of the patients in Le Huec's series had anterior pain, a positive Hawkins' sign,[14] a positive result on the palm-up test, and negative results on the Neer test or the Jobe's relocation test. Arthrography revealed leakage in front of the subscapularis in these patients. Le Heuc et al[19] operated on 8 of the patients; closure of the rotator interval led to good results.

There are 2 objections to the possible existence of coracoid impingement. First, coracohumeral ligament resection, that is, the iatrogenic creation of a large rotator interval, was recently proposed[22] and adopted for treatment of frozen shoulder; no secondary coracoid impingement has since been reported for cases so treated. Second, division of the rotator interval has been shown in experiments to increase the mobility and translation of the humeral head on the glenoid in an inferior and posterior direction.[13] Weakness of the rotator interval should increase posterior and inferior translation, whereas its closure should limit flexion and external rotation.[13] However, in our series internal rotation was the most restricted of the motions measured after operation, which seems logical given that closure of the rotator interval limits potential gliding of the subscapularis and supraspinatus over the coracoid. Although the experimental results of Harryman et al[13] argue against the theory that a weak rotator interval may induce coracoid impingement, the anatomic dissections reported by Gagey et al[9] show that the rotator interval is a solid structure. However, Gagey et al do not state whether this structure controls anterosuperior subluxation.

Surgical treatment of rotator interval lesions remains controversial. Coracoidoplasty seems necessary in cases of suspected impingement. We chose to resect the coracoid process no further than the extent necessary to relieve impingement; we did not follow the recommendation of Dines et al[7] that the tip of the coracoid process be removed. Of the 8 patients treated by Dines et al,[7] 6 were cured and 2 had improved shoulders. Gerber et al[10] reported good results in 3 of 4 surgically treated patients. However, as in our series, all authors,[6,7,12,15,16,18,19,26-28] except for Gerber et al[10] in 1 case, removed the coracoacromial ligament during surgery in order to be able to treat any potential subacromial impingement.

CONCLUSION

Whether coracoid impingement exists as a distinct syndrome and, if so, how variations of it should be classified, has yet to be fully established. We believe that lesions of the biceps and the subscapularis should not be attributed to coracoid impingement syndrome. In addition to 3 cases of mechanical impingement, we encountered 11 cases in which a weak rotator interval seemed to induce impingement by the soft tissues around the coracoid process. Closure of the rotator interval, with or without coracoidoplasty, led to improvements in all 14 cases. Such a rate of success seems to confirm our hypothesis. However, we cannot offer a satisfactory physiopathologic explanation for coracoid impingement, which in any event is rare. The most common impingement syndrome in the shoulder, subacromial impingement, also has an unclear physiopathologic mechanism.[8]

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