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### **Evaluation and Treatment of the Painful Shoulder after Rotator Cuff-Related Surgery**

R. John Naranja Jr., M.D. and Joseph P. Iannotti, M.D., Ph.D

University of Pennsylvania, Department of Orthopaedics/Shoulder & Elbow Service, Presbyterian Medical Center, 1 Cupp Pavillion, 3900 Market St., Philadelphia, PA 19104.

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**Abstract:** The management of both rotator cuff tears and symptoms related to rotator cuff disease has historically demonstrated good results with surgical treatment when conservative measures fail. In cases of persistent pain and/or disability after rotator cuff surgery, reasons for failure include misdiagnosis, post-operative complications, errors in surgical execution, and problems related to post-operative rehabilitation. The purpose of this review is to outline these major categories for failure after rotator cuff-related surgery and to define methods for their evaluation and treatment.

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#### **Introduction**

Our understanding of rotator cuff pathology has been fostered by identifying the intrinsic vulnerability of the rotator cuff to degenerative injury secondary to its blood supply [67], as well as the extrinsic anatomic considerations which have impinged on the cuff [52,53]. The spectrum of clinical manifestations range from an asymptomatic cuff tear to advancing stages of impingement. Current consensus regarding initial treatment of cuff impingement lesions revolves around a regimen of rest, anti-inflammatory medications, and physical therapy. If these conservative measures fail, surgical treatment may be indicated. The ultimate outcome is determined by several factors, but generally, favorable results can be expected. The etiology of a poor result requires careful evaluation, however. The purpose of this review is to outline the major categories for failure after rotator cuff-related surgery and to define the methods for their evaluation and treatment.

## **Misdiagnosis**

The diagnosis of impingement and/or the symptomatic rotator cuff tear requires a complete history, physical examination, and appropriate confirmatory tests that include radiographs. Impingement is classically diagnosed by pain at the anterior aspect of the shoulder which is aggravated by forced forward elevation of the humerus against the acromion (positive impingement sign), and relief of pain after injection of local anesthetic into the subacromial space (positive impingement test). Other diagnoses which must be excluded may be classified as those related to referred pain (cervical radiculitis, thoracic outlet syndrome, suprascapular nerve entrapment), those involving both intra-articular (glenohumeral instability, labral tears, glenohumeral osteoarthritis) and extra-articular pathology (acromioclavicular joint arthritis, adhesive capsulitis, unrecognized or untreated rotator cuff tear), and secondary gain issues (worker's compensation) [60].

Referred pain related to cervical disc disease is a common source of misdiagnosis [36,37,52,60,75]. Cervical pathology can occur concomitantly with impingement syndrome or present alone as a cause of chronic shoulder pain. The patient's response to treatment will depend on the proportion of findings related to cervical pathology as opposed to those attributed to impingement at the coracoacromial arch. Evaluation must focus on location and distribution of symptoms as they relate to imaging and electrodiagnostic studies. Prognosis is more difficult to interpret for those who undergo surgery for impingement in the context of coexisting cervical disc disease.

The course of the suprascapular nerve through the confining anatomy of the suprascapular notch makes it susceptible to compression and resulting symptoms which may mimic the findings associated with a rotator cuff tear. The diagnosis must be considered in the young patient with no history of trauma and loss of power associated with vague pain at the posterior aspect of the shoulder. Confirmation with electromyographic examination is useful. Treatment for these patients is directed at the etiology of nerve compression.

Thoracic outlet syndrome is an additional cause of referred pain to the shoulder [36,37,60]. This phenomenon is thought to be related to compression of the nerves and vessels to the upper limb as they exit the interval between the scalene muscles, travel over the first rib, and course down into the axilla. The history typically includes pain and paresthesias extending from the neck and shoulder to the medial aspect of the forearm and hand in an ulnar distribution. Exacerbation of symptoms with over-head activity clouds the distinction between impingement and thoracic outlet syndrome. Perhaps the most important physical sign in thoracic outlet syndrome is the ability to reproduce the patient's symptoms by abducting and laterally rotating the arm at the shoulder while palpating the wrist pulses [44]. Loss of pulse is helpful, but not pathognomonic. Rather, reproduction of symptoms confirms the diagnosis.

The most common cause of misdiagnosis caused by intra-articular pathology comes from glenohumeral instability [19,28,36,37,60]. Instability tends to occur in young athletic individuals with some element of joint laxity who

later develop a secondary impingement syndrome. The distinction may be difficult to identify and many series report cases of instability initially diagnosed and treated as impingement syndrome. Clues to the diagnosis include signs of apprehension with provocative positioning and the presence of joint laxity, which may require determination under general anesthesia. Treatment should be directed at the underlying instability rather than the impingement to optimize outcome, and even then, this population represents a significant challenge in attempting to return to pre-injury competitive levels [38,76,77].

Glenohumeral arthritis and labral tears are also identified as sources of intra-articular misdiagnosis. These are often encountered during the diagnostic arthroscopy portion of the surgical treatment of impingement syndrome, and the diagnosis may be complicated by coexisting pathology related to the rotator cuff/impingement [36,37,60].

Extra-articular pathology as a source of misdiagnosis often includes unrecognized acromioclavicular joint arthritis [1,31,36,37,40,58,60]. This is a very common cause for recurrent impingement and re-operation in patients who have failed initial decompression surgery. The dilemma stems from the poor correlation between radiographic findings of acromioclavicular joint degeneration and clinical symptoms. Direct palpation, provocative testing (cross body adduction), and lidocaine injection tests into the acromioclavicular joint help confirm the diagnosis. Distal clavicle resection has been demonstrated to positively influence outcome after failed initial decompression.

Adhesive capsulitis or primary frozen shoulder may manifest as shoulder pain, but with an additional component of restricted range of motion. Absolute numbers regarding the limitation of motion are variable, but most agree that there is a significant restriction of glenohumeral motion with both active and passive attempts at range of motion. In contrast, impingement syndrome has a relatively full range of motion with pain localized anteriorly during forward flexion.

Decompression alone in the context of an unrecognized full thickness cuff tear has been demonstrated to cause continued shoulder pain requiring re-operation for either repair or debridement of the cuff. As more surgeons use arthroscopy for decompression with visualization of both the articular and bursal sides of the cuff, the number of misdiagnoses should decrease.

Finally, there are patients who have undergone surgical management for impingement syndrome with results that may be clouded by factors related to secondary gain or patient personality. Several reports have documented less reliable results in patients with worker's compensation issues still pending [25,35--37].

## **Post-Operative Complications**

### **Deltoid detachment**

The early operative treatment of impingement syndrome consisted of acromionectomy [2,32--34] and lateral acromionectomy [48]. Favorable results were possible, but the potential for complications was realized once

the anatomy of impingement syndrome was better conceptualized. Additionally, the complete removal or lateral resection of the acromion was soon found to increase the risk for disrupting the proximal deltoid attachment. In 1981, Neer [54] treated 30 consecutive patients who had previously undergone a radical acromionectomy. All had poor results which included persistent pain, marked weakness of the shoulder, and the inability to raise the arm above the horizontal. Neer concluded that radical acromionectomy weakened the deltoid both by removing its lever arm and by encouraging retraction of the deltoid origin. The implications of disrupting the deltoid attachment may be appreciated by understanding that the deltoid muscle, in concert with the rotator cuff, is responsible for generating synchronized and powerful glenohumeral motion. Loss of deltoid muscle integrity results in significant disability which far outweighs the presence of an isolated rotator cuff tear.

Thus, the risk factors shown to correlate highly with deltoid detachment include a history of complete or lateral acromionectomy. In these procedures, a major portion of the fulcrum for the deltoid has been removed. Other conditions noted with this complication include a history of infection/hematoma, post-operative trauma, and/or early aggressive post-operative rehabilitation [29,74]. Detachment typically occurs in the first six weeks after surgery. In general, any situation that involves detaching a portion of the deltoid for exposure increases the risk for subsequent detachment. This complication with modern arthroscopic techniques of decompression, in which the deltoid attachment to the acromion is theoretically preserved, has not been demonstrated but could result if meticulous technique is not followed.

The diagnosis of deltoid detachment depends on identifying the retracted enlargement of the detached deltoid distal to an indentation where the deltoid immediately originates. This is accentuated with active elevation of the arm. Less reliable signs include decreased abduction strength, often disabling enough to prevent raising the arm above the horizontal, and/or decreased motion secondary to adherence of the retracted portion of the deltoid to the underlying rotator cuff and humerus. Conservative treatment of this complication typically demonstrates poor function [29]. Other treatment options including deltoid reattachment, deltoid rotationplasty, or salvage with glenohumeral arthrodesis have also displayed disappointing results [71]. These relatively poor treatment results stress the importance of preventing this disabling complication.

### **Heterotopic ossification**

Heterotopic ossification as a complication of rotator cuff surgery was first reported as early as 1949 [2]. Its occurrence at the site of previous acromionectomy caused recurrent impingement symptoms that required re-excision. Subsequent reports of heterotopic ossification regarding the development of recurrent symptoms have varied [4,5,34,42,62,78,79]. Lazarus et al. [42] attributed the incidence of these symptoms to bone dust remaining after arthroscopic acromioplasty but Berg and Ciullo [4] associated them with underlying medical disorders. In one large series of patients who developed heterotopic ossification after distal clavicle excision or subacromial decompression, the incidence of heterotopic ossification was 3.2% and was disproportionately seen in patients with a history of chronic

pulmonary diseases. No correlation between the method of bone resection and incidence of heterotopic ossification was found. The results of surgery after the formation of heterotopic ossification are related to the size of acromioplasty, site of acromioplasty, and the presence of risk factors. Risk factors include a profile of hypertrophic pulmonary osteoarthropathy, active spondylitic arthropathy, and a history of chronic pulmonary disease. It should be understood that bone present on postoperative radiographs does not always represent heterotopic ossification, but may signify inadequate initial bone resection. The difference between these two causes of residual bone are best resolved by obtaining post-operative radiographs within the first four weeks after surgery.

### **Superior glenohumeral instability**

The role of the rotator cuff in preventing superior migration of the proximal humerus with shoulder abduction and forward flexion is diminished with large full thickness tears. Recent biomechanical studies have stressed the importance of the coracoacromial arch, more specifically the coracoacromial ligament (CAL), as a secondary restraint in these cases [43,51]. These authors suggested avoidance of CAL release and acromioplasty in these cuff-deficient patients and in those with high functional demands who cannot tolerate changes in coupled motions (e.g., athletes who throw). Several reports of superior dislocation/subluxation after acromionectomy with an associated large rotator cuff tear have been described [3,50]. As a result, careful consideration must be given to re-attaching the coracoacromial ligament in these patients, and further removal of the coracoacromial arch complex is deleterious.

### **Clavicular instability**

In 1988, the results of the Mumford procedure in 23 athletes with a history of grade I or grade II dislocation were analyzed. Ten athletes in the series demonstrated increased horizontal clavicular motion [14].

Additionally, in 1993, the complication of a dropped shoulder with the clavicle protruding into the trapezius secondary to distal clavicle resection was reported. The authors identified damage to the superior acromioclavicular capsular ligament as the inciting cause of this instability and recommended resecting only one to one-and-one-half centimeters of the distal clavicle with a burr in an attempt to preserve the superior acromioclavicular capsular ligament [11].

In 1996, Blazar et al [8] reviewed 17 patients who had a distal clavicle resection and correlated anteroposterior instability based on stress radiographs with postoperative pain and functional outcome. They found that increased translation of the distal clavicle after distal clavicle resection was associated with increased post-operative shoulder pain and poor surgical outcome.

Although this phenomenon does exist, it is rare among the general population. Attempts to restore stability in this plane once this condition exists have produced poor outcomes, and one should try to preserve the

capsular and soft tissue structures that provide restraint in this direction.

### **Recurrent tear**

Tears of the rotator cuff have been classified according to their size, and numerous techniques have been described for rotator cuff repair, particularly for those of massive size. No technique, however, has been immune from a recurrent tear. Reasons for recurrence have been attributed to: the quality of the cuff and size of tear at the time of repair, inadequate intra-operative mobilization of the cuff, failure to remove extrinsic impingement processes, post-traumatic falls, inadequate post-operative protection, and spontaneous rupture [7,16,27,57,63,64,66].

Recurrent tears from inadequate mobilization and poor exposure of the torn edges of the rotator cuff has been well documented. For example, in 1990, one author reported that at re-operation for failed rotator cuff surgery, thickened hypertrophied bursal tissue was found sutured and closed over a rotator cuff defect. Bursal tissue must be resected if one cannot gain sufficient exposure to adequately mobilize the underlying cuff [63]. In another series, 25 of 32 patients who had undergone post-operative arthrography demonstrated a recurrent or persistent tear. Reasons identified included inadequate exposure and/or mobilization of the cuff as determined by review of the operative notes [10].

Gerber et al [27] has also shown the importance of suture type, configuration, and bone quality with regard to strength of repair. The authors evaluated the mechanical properties of several current techniques of tendon-to-bone sutures used in rotator cuff repair and found #2 non-absorbable braided polyester and absorbable polygalactin and polyglycolic acid sutures best for combined ultimate tensile strength and stiffness. A modified Mason-Allen suture technique was superior with regard to tendon grasping.

Decompression of the subacromial space has been recommended as a concomitant procedure with rotator cuff repair. In addition to pain relief, this also minimizes the chance for a recurrent tear. In 1984, 27 patients were evaluated after initial failed rotator cuff repair; inadequate decompression of the coracoacromial arch was a major factor for recurrent tears [16].

Post-operatively, patients require cautious rehabilitation, as falls and inadequate immobilization have been cited as reasons for recurrent tear [13,39]. For example, in one series, four cases of traumatic disruption caused by a fall were described [59]. In another case of a traumatic recurrent tear, non-compliance with post-operative immobilization resulted in a recurrent tear of a free-biceps graft reconstruction. The attempt at a second repair with free-biceps graft was unsuccessful [26].

Clearly, meticulous surgical technique and cautious post-operative rehabilitation will minimize the chance for recurrent tears. However, the necessity to completely surgically close the gap occurring at the site of tear has recently been questioned. Initial recommendations for direct repair advocated obtaining a watertight closure but this concept was disputed in 1986 when the use of arthrography after operative repair of a torn rotator cuff in 20 patients, at an average of 30 months post-operation, was reported [10]. In 18 of 20 patients, contrast medium leaked into the subacromial

bursa indicating a defect in the cuff. Results, however, did not correlate with this finding because 17 patients no longer complained of pain and 15 had achieved full range of shoulder motion. The authors concluded that a watertight closure is not essential for a good functional result.

The usefulness of radiographic techniques in diagnosing these recurrences has been studied extensively. In 1986, the sensitivity, specificity, and accuracy of post-operative rotator cuff findings at second look surgery was compared with arthrography and ultrasonography. Arthrography was only 66% sensitive, 50% specific, and 62.5% accurate; ultrasonography was 85% sensitive, 100% specific, and 90% accurate [24]. The utility of ultrasonography was further confirmed in an independent study which correctly diagnosed recurrent cuff tears in 26 of 26 shoulders, and confirmed an intact cuff in 10 of 11 cases [45].

Similar studies have been performed with magnetic resonance imaging (MRI) [61]. This study correctly identified six of seven tears found at the time of second surgery for a sensitivity of 86%. MRI was also able to exclude the presence of a tear in 22 of 24 patients in which no full-thickness tear was found at surgery (a specificity of 92%).

Although none of these modalities are perfect in diagnosing recurrent rotator cuff tears, MRI and ultrasound both seem to display more promise in confirming this condition in the post-operative patient. They should be used in conjunction with a physical examination when recurrence is suspected.

## **Errors in Surgical Execution**

### **Inadequate decompression**

Neer [52] has been credited with articulating the anatomy for impingement to include the anterior edge and undersurface of the anterior third of the acromion, the coracoacromial ligament, and in some cases, the acromioclavicular joint. Inadequate decompression is one of the more common reasons for failure after initial surgical intervention for impingement [52]. Analysis of those cases with inadequate decompression reveal that it often results when the decompression technique neglects the anatomy of the impingement lesion. Consequently, it is not surprising to find that in those cases in which lateral acromionectomy procedures were performed, a high rate of continued impingement occurred because a portion of the impinging anatomy is left behind.

Other cases of inadequate decompression have been related to poor judgment regarding the amount of bone resected with respect to the anterior acromioplasty. In an experimental and computer simulation of anterior acromioplasty, the elimination of impingement was specific to an acromioplasty represented by flattening of the acromion from a location extending from the anterior third to the mid-line. Anterior acromioplasty alone (flattening of the anterior ridge) resulted in residual impingement, and a flattening of the entire acromion was excessive [6].

In summary, decompression of the subacromial space requires a thorough understanding of anatomic structures that cause impingement, combined with an ability to judge the adequacy of the decompression. Pre-operative

evaluation should include a clinical examination to determine whether acromioclavicular symptoms are contributing to the impingement syndrome, as well as appropriate radiographic projections. Appropriate radiographic projections for the assessment of acromial morphology has been shown to have good inter-observer reliability and correlation with intra-operative measurements of acromial spur size in a recent study [41]. If an inadequate decompression results, treatment options include conservative therapy with repeat injection and cuff strengthening, or repeat surgical decompression, the results of which have been relatively good.

### **Acromial fracture**

Acromial fracture is a complication seldom related to subacromial decompression. It has been associated with over-aggressive decompression of the acromion, as well as over-enthusiastic retraction of the acromion during exposure of the rotator cuff. Evaluation is best defined by plain radiographs. The incidence has been identified to be less than 1% in several studies and seems to be related to osteoporotic bone and overzealous bone resection [47]. Additionally, the prognosis for these patients has been poor because of the lack of optimal operative fixation in those who have sustained this fracture and experienced delayed healing response [47,78,79].

### **Neurologic injury**

Most open-anterior surgical approaches for rotator cuff surgery are performed through a limited deltoid muscle split. The relationship to the axillary nerve may be understood by recalling its anatomy; it arises from the fifth and sixth cervical roots and forms the posterior cord of the brachial plexus. At the inferior border of the subscapularis, it travels posteriorly under the inferior capsule and joins the posterior humeral circumflex artery to exit the quadrangular space, and at this point, divides into anterior and posterior trunks. The posterior trunk gives off branches to the teres minor and posterior deltoid and terminates at the superior lateral cutaneous nerve of the arm. The anterior trunk passes anteriorly around the humerus approximately five centimeters distal to the lateral border of the acromion. Tremendous variation in the course and position of the axillary nerve in anatomical studies suggests that this safe zone is only a guideline, and careless, over-exuberant retraction must be avoided [9]. In 1992, a case of deltoid denervation after acromioplasty and rotator cuff repair was reported with a deltoid split of four centimeters [29]. Subsequently, in 1994, two cases of axillary nerve palsy after rotator cuff repair for massive tears were described [59], but these recovered within three months. The axillary nerve is also at risk in cases of subscapularis repair and care must be taken in the surgical approach to protect this nerve [26].

### **Pain Related to Post-Operative Rehabilitation**

#### **Post-operative stiffness**

The development of a frozen shoulder after rotator cuff surgery may be associated with prolonged immobilization post-operatively, poor patient compliance, deltoid detachment, and pigmented villonodular synovitis [3,17,24,33,45,55,60,66,69]. Clinical examination reveals decreased passive range of motion, and treatment options include physical therapy, manipulation under anesthesia, and/or open or arthroscopic release of adhesions [24,54,58,72]. Care must be taken during manipulations under anesthesia to avoid excess force and subsequent iatrogenic humeral fractures.

### **Reflex sympathetic dystrophy**

Reflex sympathetic dystrophy is a condition characterized by pain, hyperesthesia, vasomotor and sudomotor disturbances, and increased muscular tone, followed by weakness, atrophy, and trophic changes involving the skin, its appendages, muscles, bones and joints. The etiology is thought to be a result of noxious stimuli (such as surgery) stimulating an aberrant sympathetic response. Its occurrence after rotator cuff surgery is approximately 0--2% [35]. Treatment includes pharmacologic therapy, nerve blocks, and if this is unsuccessful, surgical or chemical sympathectomy. Consultation with a pain management service is helpful in addressing this complication.

### **Complications related to wound healing**

This group of complications includes hematomas, draining sinuses, suture granulomas, superficial infections, and keloids or uncosmetic scars [12,18,20,21,49,52,54--56,65,66,68,73,78]. The risk factors for these complications are generally unpredictable, and early recognition and removal of offending tissues will typically result in resolution. The issue of deep infection is more complex [23,30,54,69,74]. This situation represents a significant negative impact on the final outcome of surgery, and aggressive debridement and culture-derived parenteral antibiotics are the principles of treatment.

### **Summary**

Although conservative and operative treatment for rotator cuff pathology demonstrates predictably good results, one must systematically approach the patient who fails this standard treatment protocol. Under these circumstances, one must critically evaluate both the original diagnosis and objectively critique both the operative technique used and the rehabilitative program followed by the patient. Once the specific etiology of the post-operative problem is determined, one can then decide on the best treatment modality to optimize final clinical result of the patient.

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