Rotator cuff disease is a common cause of shoulder disability, particularly in patients beyond the fourth decade of life. Anterior acromioplasty, combined with rotator cuff repair when indicated, generally provides predictable pain relief and improved function. However, when pain continues in spite of surgery for rotator cuff disease, patient management becomes more complicated and less predictable. It is important to recognize that persistent rotator cuff disease is only one of the many potential causes for such pain (Table 1). Possible extrinsic causes include cervical radiculopathy; suprascalular, long-thoracic, or spinal-accessory neuropathy; and adjacent or metastatic neoplastic disease. Potentially causative intrinsic shoulder disorders may be intra-articular, such as osteoarthritis, adhesive capsulitis, recurrent anterior subluxation, and labral or bicipital tendon abnormalities; and extra-articular, such as subacromial impingement. Successful management requires an accurate diagnosis, maximal rehabilitation, judicious use of surgical intervention, and a well-motivated patient. The results of revision surgery in patients with persistent subacromial impingement, with or without an intact cuff, are inferior to reported results after primary acromioplasty or rotator cuff repair.

Extrinsic Shoulder Disorders

It is important to recognize that persistent pain after rotator cuff surgery may be the result of pathologic processes extrinsic to the shoulder. Extrinsic causes of persistent shoulder pain include cervical radiculopathy, suprascapular neuropathy, abnormalities of scapular rotation (due to long-thoracic or spinal-accessory neuropathy), and adjacent or metastatic neoplasms. Causes of persistent pain that are intrinsic to the shoulder include both intra-articular conditions (e.g., glenohumeral osteoarthritis, adhesive capsulitis, recurrent anterior subluxation, and labral and bicipital tendon abnormalities) and extra-articular conditions (e.g., persistent subacromial impingement, persistent or recurrent rotator cuff defects, acromioclavicular arthropathy, and deltoid muscle deficiency). Successful management requires an accurate diagnosis, maximal rehabilitation, judicious use of surgical intervention, and a well-motivated patient. The results of revision surgery in patients with persistent subacromial impingement, with or without an intact cuff, are inferior to reported results after primary acromioplasty or rotator cuff repair.

Evaluation

In most cases, an initial diagnostic impression can be formulated on the basis of the history, physical examination, and routine radiography. Additional studies that may be useful include arthrography, ultrasonography, magnetic resonance (MR) imaging, electromyography, and scintigraphy. Selective injections into the subacromial space and the acromioclavicular joint can help localize the pain or quantitate how much pain is attributable to each area when both are involved. Diagnostic arthroscopy may be useful, especially when extrinsic disorders have been excluded, the previously performed acromioplasty has been judged adequate by radiographic criteria, and the rotator cuff is intact.
 When an extrinsic cause for the persistent pain has been identified, treatment should be directed accordingly.

 Of the extrinsic causes of persistent shoulder pain, cervical radiculopathy involving the fifth or sixth cervical root is perhaps the most common. The symptoms of neck pain accompanied by radiation into the upper extremity, numbness, or paresthesias suggest this diagnosis. Routine radiography may reveal cervical spondylosis or neural foraminal encroachment. If indicated, MR imaging of the cervical spine and electromyography may confirm the diagnosis.

 Long-thoracic and spinal-accessory neuropathies result in scapular winging and poor scapular rotation during overhead elevation. Secondary impingement symptoms may develop as scapular rotation lags behind glenohumeral elevation. Although true scapular winging is an uncommon cause of persistent pain after rotator cuff surgery, many patients will exhibit varying degrees of scapulohumeral dysfunction. Scapulothoracic and scapulohumeral rhythm should be observed in all patients with persistent symptoms after acromioplasty or cuff repair. In patients with severe scapular dysfunction associated with winging, electromyography may confirm the neurologic lesion.

 Suprascapular neuropathy may also result in impingement-like symptoms because of the posterior cuff weakness that results from chronic nerve compression. Patients present with severe atrophy of either the supraspinatus and infraspinatus or the infraspinatus alone. This is associated with weakness of external rotation with the arm at the side. Electromyography is helpful in confirming the diagnosis and localizing the site of compression to the infraspinatus alone or to both the supraspinatus and the infraspinatus. Magnetic resonance imaging may reveal a ganglion cyst compressing the suprascapular nerve (Fig. 1).

 Neoplastic processes are a very rare but devastating cause of persistent shoulder pain after rotator cuff surgery. The apical lung fields should always be inspected on shoulder radiographs, because apical lung tumors (i.e., Pancoast tumors) cause referred shoulder pain through extension to the brachial plexus or cervical roots. If a lung mass is suspected, appropriate chest radiographs and medical consultation are indicated. Persistent pain may also be caused by direct involvement of the shoulder by a neoplastic process. Magnetic resonance imaging may be used to further characterize masses or unusual prominences discovered on physical examination (Fig. 2).
Intrinsic Shoulder Disorders

Causes of persistent pain that are intrinsic to the shoulder include both intra-articular conditions (e.g., glenohumeral osteoarthritis, adhesive capsulitis, recurrent anterior subluxation, and labral and bicipital tendon abnormalities) and extra-articular conditions (e.g., persistent subacromial impingement, persistent or recurrent rotator cuff defects, acromioclavicular arthropathy, and deltoid muscle deficiency).

Intra-articular Causes of Persistent Pain

Unrecognized glenohumeral disorders may be responsible for persistent postsurgical shoulder pain. Intra-articular causation should be suspected when postoperative radiographs reveal adequate decompression of the supraspinatus outlet, and the acromioclavicular joint is asymptomatic.

Articular Cartilage Abnormalities

Glenohumeral osteoarticular disease may be a cause of persistent pain in at least two circumstances: (1) unrecognized or underappreciated preoperative osteoarthritis and (2) cuff tear arthropathy, or Milwaukee shoulder syndrome. Primary glenohumeral osteoarthritis is characterized by subchondral sclerosis and cyst formation, glenohumeral jointspace narrowing and osteophyte formation, asymmetric posterior glenoid wear, and an intact or repairable rotator cuff. The management of primary osteoarthritis does not differ substantially whether or not there has been prior impingement or rotator cuff surgery.

Cuff tear arthropathy is characterized by destruction of the glenohumeral articular surfaces, accompanied by chronic, massive rotator cuff insufficiency and proximal humeral migration, that persists or recurs in spite of one or more previous attempts at cuff repair. Persistent pain may be improved by humeral hemiarthroplasty. Functional improvement is less predictable than pain relief, especially if the coracoacromial ligament was sacrificed during previous cuff repair.

Traumatic articular cartilage defects of the humerus and glenoid may cause persistent shoulder pain in the absence of generalized articular degeneration. A history of a single traumatic event is often elicited. Examination may reveal painful glenohumeral crepitus during glenohumeral rotation. Radiographs and MR images are often normal. In this circumstance, diagnostic arthroscopy may be necessary to confirm a humeral or glenoid articular defect.

Adhesive Capsulitis

The hallmark of capsular contracture or adhesive capsulitis is a symmetric decrease in both active and passive range of motion, which can be localized or can involve all planes of motion. Localized posterior capsular contracture is common with subacromial impingement syndrome and is characterized not only by limited elevation but also by decreased cross-body adduction and internal rotation, both of which are more pronounced with the arm at 90 degrees of elevation in or anterior to the scapular plane. The presence of localized posterior capsular contracture postoperatively is a sign of an incompletely rehabilitated shoulder and can be a factor contributing to continued pain and disability. Generalized capsular contracture is less common with primary rotator cuff disease or subacromial impingement syndrome than localized posterior contracture. It is characterized by loss of motion in all planes (especially passive external rotation with the arm at the side) and is an important source of persistent pain and disability after surgery for rotator cuff disease.

The initial management of adhesive capsulitis consists of physiotherapy for joint mobilization and capsular stretching. If motion cannot be restored through the use of nonoperative joint-mobilization techniques, then closed manipulation or surgical capsular release is indicated. Postoperative frozen
shoulder is often unresponsive to closed manipulation. Traditionally, surgical capsular release was performed through an anterior deltopectoral approach in combination with subscapularis lengthening. Arthroscopic capsular release has recently been reported as an alternative, but this procedure requires advanced arthroscopic surgical skills and may be contraindicated in the presence of extra-articular adhesions.

**Recurrent Anterior Subluxation**

In patients less than 40 years of age, particularly those who engage in sports involving overhead motion, there is an overlap between rotator cuff overuse and recurrent anterior subluxation. Young patients with persistent shoulder pain after acromioplasty may be experiencing secondary impingement symptoms as a result of subtle anterior subluxation. They may report a forceful abduction–external rotation injury, a distal traction injury, or “dead arm” symptoms while throwing.

Examination may reveal increased passive external rotation with the arm at 90 degrees of elevation in the scapular plane, underlying multidirectional laxity or generalized ligamentous laxity, or a positive relocation test. Radiographic evaluation should include specialized views such as the apical oblique or Garth view, the West Point view, and the Stryker notch view. These may demonstrate small Hill-Sachs defects and calcification or fracture of the glenoid rim consistent with recurrent posttraumatic anterior subluxation (Fig. 4).

Treatment includes activity modification and strengthening exercises for the rotator cuff, deltoid, and scapular stabilizers. If this treatment fails, surgical stabilization may be considered.

**Labral or Bicipital Tendon Abnormalities**

The tendon of the long head of the biceps traverses the bicipital groove, enters the glenohumeral joint slightly anterior to the supraspinatus insertion, becomes confluent with the superior labrum, and attaches to the supraglenoid tubercle. Because of its course, the biceps tendon may become involved in the subacromial impinge-
ment process. In addition, attri-
tional changes to the tendon within
the groove, primary biceps tendini-
tis, and anterior-to-posterior
lesions of the superior labrum
("SLAP" lesions) may result in per-
sistent symptoms after surgery for
impingement syndrome.

The physical findings are non-
specific but may include painful
resisted forearm supination with
the elbow at 90 degrees of flexion.
Diagnostic arthroscopy allows
visualization of the superior la-
brum and the biceps tendon. The
extra-articular portion of the ten-
don within the bicipital groove can
be visualized by advancing the ten-
don into the joint with the assis-
tance of a probe or other instru-
ment placed through an anterior
portal (Fig. 5). Treatment options
include labral repair, labral de-
bridement, and biceps tenodesis.

Extra-articular Causes of
Persistent Pain

Persistent Subacromial
Impingement

Insufficient supraspinatus outlet
decompression may result from
residual anterior acromial spurring,
regrowth of bone or sub-
acromial calcification, inferior
projecting acromioclavicular osteo-
phytes, and persistence or
regrowth of the coracoacromial lig-
ament. Persistent impingement
syndrome related to residual
supraspinatus outlet narrowing is a
common cause of continued shoul-
der pain after surgery for rotator
cuff disease and has been reported
in 18% to 79% of patients with
failed acromioplasty.

Physical examination reveals a
positive impingement sign and the
impingement reinforcement sign
(i.e., Hawkins, or abduction inter-
nal rotation ["ABIR"], sign). Substantial reduction in the pain

Fig. 5 Arthoscopic image of severe par-
tial tearing of the long head of the biceps in
a patient with continued pain after open
acromioplasty and cuff repair followed by
open distal clavicle excision.

associated with these maneuvers
after subacromial injection of lidocaine (i.e., a positive impingement test) helps to confirm the presence of continued subacromial impinge-
ment. Radiography should in-
clude a supraspinatus outlet view
and a 30-degree caudal tilt view
to evaluate for continued anterior
acromial spurring and a Zanca view (standing anteroposterior
view with 15- to 30-degree cephalic
tilt) to visualize any inferiorly pro-
jecting acromioclavicular osteo-
phytes (Fig. 6).

The results of revision acromio-
plasty are less reliable than the re-

results of primary acromioplasty.
Flugstad et al reported the cases
of 13 patients who underwent revi-
sion acromioplasty with an intact
cuff. Six patients described their
shoulders as “much better”; the
other 7, as “better.” Hawkins et
al reported the cases of 51
patients in whom acromioplasty
had failed. Twelve of these pa-
tients underwent repeat acromio-
plasty, one with a rotator cuff
repair. All 12 patients were receiv-
ing workmen’s compensation.

Only 1 achieved a satisfactory
result. Ogilvie-Harris et al evalu-
ated 67 shoulders in 65 patients
more than 2 years after an initial
acromioplasty for impingement
syndrome without a cuff tear.
Eighteen of the 65 patients under-
went revision rotator cuff surgery
(6 rotator cuff repairs and 12 revi-
sion acromioplasties). There was a
good result in 9 of the 12 patients
(75%). Rockwood and Williams re-
ported 67% good or excellent
results in 27 patients who under-
went revision acromioplasty with
an intact or repairable cuff.

Because of the inconsistent re-

sults of revision acromioplasty,
successful management of patients
with persistent subacromial outlet
narrowing requires careful patient
selection. Nonoperative manage-
ment should be maximized in all
cases. Repeat surgery is reserved
for patients with radiographic evi-
dence of continued impingement
who obtain pain relief with sub-
acromial lidocaine. In spite of
these stringent selection criteria,
the results of revision acromio-
plasty will likely not approach those of
primary acromioplasty.

Persistent or Recurrent Rotator
Cuff Defect

Evaluation

The presence of a full-thickness
rotator cuff defect can be compat-
ible with asymptomatic shoulder
function. Furthermore, some
authors have reported high per-
centages of patients with good or
excellent results after acromioplasty
and cuff repair in spite of arthro-
graphically and ultrasonographi-
cally proven persistent or recurrent
rotator cuff defects. Therefore,
when evaluating patients with
continued pain and a persistent or
recurrent rotator cuff defect after
rotator cuff repair, it is important
to eliminate other causes of persis-
tent pain before focusing on the residual rotator cuff defect.

Physical findings are variable and depend on the size of the recurrent rotator cuff defect. Small defects, which primarily affect the supraspinatus tendon, are characterized by an intact anterior (i.e., subscapularis) and posterior (i.e., infraspinatus and teres minor) rotator cuff force couple. The impingement and impingement-reinforcement signs may be positive and accompanied by subacromial crepitus. However, range of overhead elevation, shoulder strength, and function are relatively normal.

Large defects extend anteriorly and/or posteriorly into the subscapularis and infraspinatus–teres minor, respectively. Posterior extension results in weakness of external rotation with the arm at the side and the humerus in neutral rotation. If the posterior rotator cuff insufficiency is severe enough, the patient will be unable to raise the arm overhead, in spite of full passive motion.

The signs of anterior (i.e., subscapularis) rotator cuff insufficiency can be more subtle than the signs of posterior rotator cuff insufficiency. Increased passive external rotation with the arm at the side is suggestive of subscapularis involvement. Subscapularis insufficiency is verified by a positive “lift off” test. This test is performed by passively resting the back of the patient’s hand against the ipsilateral buttock and then asking the patient to actively lift the hand off the back and away from the body without simultaneously extending the shoulder or the elbow (Fig. 7). This requires maximal internal rotation with the subscapularis.

Abnormalities of tendon signal intensity in the absence of alterations in signal morphology may and correlated carefully with the overall clinical impression. In particular, MR imaging of the rotator cuff is not as sensitive or specific as in the shoulder that has not been treated surgically.

Ultrasonography, arthrography, and MR imaging have all been used to evaluate rotator cuff pathology. When there has been prior surgery, the presence of subacromial scarring, subacromial bursal thickening, and postsurgical tendon irregularities may complicate the interpretation of the images obtained with these modalities. Therefore, imaging studies must be interpreted with caution
have no clinical relevance and should be interpreted with caution (Fig. 8, A). However, the presence of a well-defined gap in the tendon with synovial fluid traversing the entire thickness of the tendon into the subacromial space is definitive evidence of a persistent or recurrent defect (Fig. 8, B-D). When a full-thickness defect is present, MR imaging can accurately quantitate the size of the defect in both the anteroposterior and medial-lateral planes and can estimate atrophy in each of the four rotator cuff muscles.

**Treatment**

In many patients, a persistent cuff defect is accompanied by continued supraspinatus outlet narrowing. DeOrio and Cofield reported the data on 27 patients (27 shoulders) who underwent a second attempt at repair of a rotator cuff tear. Seven patients had physical findings consistent with continued subacromial impingement, and only 12 of the 27 shoulders had undergone an anterior acromioplasty at the time of the initial repair. Neviaser and Neviaser reported on 46 cases of revision cuff repair, in all of which repeat acromioplasty was necessary, presumably because of persistent supraspinatus outlet narrowing. Bigliani et al documented a 90% incidence of inadequate prior acromioplasty in their 31 patients who underwent a repeat repair.

The reported results of revision rotator cuff repair are inconsistent and, in general, inferior to the results of primary cuff repair. In the study by DeOrio and Cofield, 7 of the 27 patients (26%) who underwent revision rotator cuff repair required a third operative procedure before study completion and were not, therefore, included in the final results. None of the remaining 20 patients had excellent results, and only 42% had good results. Bigliani et al reported satisfactory results in 52% of 31 patients who underwent repeat rotator cuff repair. Neviaser and Neviaser reported on 46 revision rotator cuff repairs and critically evaluated return of range of motion in their outcome analysis. Twenty-two patients gained motion (mean, 45 degrees), 22 had no change, and 2 lost motion.

Given the relatively disappointing results of revision acromioplasty and rotator cuff repair, the merits of nonoperative management should not be overlooked. An important component is activity modification, which should involve employment, daily-living, and recreational activities. Physical therapy, including capsular stretching and strengthening exercises for the remaining portions of the rotator cuff, the deltoid, and the scapular rotators, should be maximized. Revision rotator cuff repair should be considered if nonoperative man-
Painful Shoulder After Rotator Cuff Surgery

mgement has failed and the patient is willing to accept the reality of inconsistent results.

The goal of all revision rotator cuff procedures is to achieve a surgical repair that ultimately heals to bone at the operative site and remains intact over the long term. Patients who achieve this goal are most likely to experience the best results with regard to pain, strength, and function. With smaller, more mobile cuff tears, this goal is often attainable. Revision acromioplasty and/or removal of inferior acromioclavicular osteophytes is performed in conjunction with rotator cuff repair when residual supraspinatus outlet narrowing from anterior acromial or inferior acromioclavicular spurring exists.

The rotator cuff tears most likely to rupture after repair are the large tears with two- or three-tendon involvement, particularly in older patients. In addition, large initial tears are most likely to be difficult to repair, primarily because of poor tissue quality. Therefore, revision of failed repairs of large rotator cuff tears is technically difficult and would be expected to be less likely to result in a permanently healed tendon.

The most important aspects of surgical technique in these difficult cases are tendon identification and mobilization. The subacromial bursa may be abnormally thickened and must not be mistaken for the torn rotator cuff tendon edge. Once the retracted tendon edge has been identified, it is systematically mobilized laterally. First, the superficial surface of the retracted tendon is freed from any overlying adhesions to the bursa, the spine of the scapula, and the deep surface of the posterior deltoid and trap-

ized if present. Finally, if necessary, any tenodesis effect of the underlying capsule is addressed by stretching the posterior capsule with an intra-articular “metal finger” or by releasing the capsule sharply slightly distal to the labrum. The mobilized tendon is then repaired to bone on the greater tuberosity or at the anatomic neck, slightly medial to the anatomic insertion site.

The subscapularis tendon should routinely be inspected for partial or complete avulsion, especially in patients with a positive preoperative lift-off test. This can be accomplished through a standard superior incision by flexing the humerus to bring the subscapularis into the wound. Alternatively, if preoperative evaluation indicates an isolated subscapularis injury, an anterior deltopectoral approach can be utilized. In either case, the subscapularis tendon is mobilized laterally and repaired to bone. Sufficient mobilization to allow repair may require release of the underlying anterior capsule.

Continued shoulder pain associated with a failed previous cuff repair in an irreparable persistent rotator cuff defect is a potentially difficult problem, which may not have a good solution. The interaction between the deltoid, the rotator cuff, and the coracoacromial arch (anterior acromion, distal clavicle, and coracoacromial ligament) during elevation of the arm is complex and not completely understood. In the presence of an intact and normally functioning rotator cuff mechanism, the potential proximal humeral migration generated by deltoid contraction is resisted by the rotator cuff; the humerus remains relatively centered on the glenoid fossa, and normal overhead elevation is accomplished. Under these circumstances, the relative role of the coracoacromial arch as a humeral-head containment mechanism is minor.

In some cases involving irreparable rotator cuff tears, enough anterior and posterior rotator cuff function remains to effectively resist proximal humeral migration during deltoid contraction. The humeral head again remains relatively centered, and overhead elevation is normal or near normal in range but may be weak. The rotator cuff function lost to the irreparable cuff defect is “compensated” for by the remaining balanced anterior and posterior rotator cuff force couple. The degree to which the coracoacromial arch functions as a humeral-head containment mechanism is variable and is probably dependent on the amount of anterior and posterior rotator cuff remaining.

If the persistent rotator cuff defect is too large, the associated loss of rotator cuff function cannot be compensated for. In this relatively “uncompensated” shoulder, the remaining anterior and posterior rotator cuff mechanism is unable to effectively resist the proximal humeral migration associated with deltoid contraction. Consequently, the coracoacromial arch becomes more important as a humeral-head containment mechanism. Incompetence of the coracoacromial arch due to prior acromioplasty and coracoacromial ligament resection combined with a poorly compensated or uncompensated rotator cuff defect may result in severe compromise of overhead shoulder function.

Surgical treatment of a patient with persistent pain and an irreparable rotator cuff defect is potentially difficult and is dependent on the supposed cause of the continued pain as well as the size of the defect. In the presence of continued supraspinatus outlet narrowing, as documented on supraspinas-
tus outlet and 30-degree caudal-tilt radiographs, persistent pain is likely to be the result of continued subacromial impingement. If pain is relieved with subacromial lidocaine and the irreparable rotator cuff defect is compensated for, as evidenced by intact overhead function and relative preservation of the acromiohumeral interval (i.e., an acromiohumeral interval of 7 mm or greater), repeat subacromial decompression without repair should provide acceptable pain relief while preserving overhead function.

Rockwood et al. have reported satisfactory results with subacromial decompression and partial cuff debridement in patients with subacromial impingement syndrome associated with chronic irreparable rotator cuff defects. The results were less satisfactory in patients who had undergone prior rotator cuff surgery. However, many of these patients also had iatrogenic deltoid insufficiency. Although more complicated surgical options for management of the irreparable cuff defect have been reported, none has been demonstrated to be superior to debridement alone when the defect is well compensated.

Debridement alone for patients with persistent pain associated with an uncompensated irreparable rotator cuff defect is unlikely to either alleviate pain or improve function. If the patient is unable to actively raise the arm overhead preoperatively, even when pain is relieved with subacromial lidocaine, it is unlikely the ability to raise the arm overhead postoperatively will be regained unless some of the lost anterior or, more commonly, posterior rotator cuff function can be reestablished. In fact, repeat subacromial decompression and partial rotator cuff debridement may further compromise shoulder function by removing the humeral-head containment provided by any remaining portions of the acromion and coracoacromial ligament.

The painful shoulder with an uncompensated irreparable rotator cuff defect and an incompetent coracoacromial arch is currently a problem without a solution. Many techniques have been described to reconstruct massive irreparable rotator cuff defects. However, few of them have the potential to restore lost rotator cuff function, as opposed to merely filling the defect. Reconstruction of the superior defect with autograft fascia lata, allograft fascia lata or rotator cuff, or prosthetic material may provide a tenodesis effect, but is not likely to restore function to severely atrophic rotator cuff musculature. Superior transposition of the teres minor and/or the subscapularis has the potential advantage of improving head depression but has the potential disadvantage of destabilizing the anterior-posterior force couple. It provides a functional musculotendinous unit without sacrificing any remaining anterior or posterior rotator cuff function. In addition, the resultant line of action provides potential head depression. The indications for unipolar latissimus dorsi transfer continue to be defined. The reported results have been variable and seem to be best when the subscapularis is not also deficient.

The role of coracoacromial arch reconstruction in this setting has yet to be established. Wiley described the use of a coracoacromial interpositional iliac-crest autograft in five patients with persistent symptoms associated with irreparable rotator cuff defects and deficient coracoacromial arches after a failed acromioplasty and rotator cuff repair. The results were disappointing, and useful overhead function could not be restored. At least three of these patients had anterior deltoid deficiency, which may have contributed to the poor postoperative elevation. The importance of a functional coracoacromial arch in patients with an uncompensated irreparable rotator cuff defect seems clear. However, additional work is required to define surgical techniques and indications for coracoacromial arch reconstruction or repair.

**Acromioclavicular Joint Arthropathy**

Acromioclavicular arthropathy is a relatively common cause of persistent pain after acromioplasty with or without cuff repair. Resectional arthroplasty or distal clavicle excision is indicated if the following criteria are met: (1) the acromioclavicular joint is tender to palpation and painful during cross-body adduction, (2) there is radiographic evidence of arthritis, and (3) temporary pain relief follows a local intra-articular injection of lidocaine.

The optimal amount of bone to be resected from the distal clavicle remains somewhat controversial. Displacement of the clavicle along its longitudinal axis, toward the acromion, is primarily controlled by the trapezoid portion of the coracoclavicular ligament. With large displacements, the acromioclavicular ligaments primarily resist anteroposterior displacement of the clavicle, and the coracoclavicular ligament (especially the conoid portion) resists superoinferior displacement. Results of distal clavicle excision may be negatively affected by excessive translation of the distal clavicle in both the anteroposterior and superoin-
ferior planes. Therefore, the amount of bone resected should be sufficient to prevent axial compression or contact between the residual clavicle and the acromion, but not so much as to compromise the capsular and coracoclavicular ligaments.

Resection can be performed arthroscopically or by traditional open techniques. Our current practice in most cases is to arthroscopically remove 1.0 cm of distal clavicle, which results in a final gap distance of 1.2 to 1.5 cm.

Deltoid Insufficiency

Denervation or postoperative detachment of the deltoid after acromioplasty and cuff repair is a devastating complication, which is best managed by prevention (Fig. 9). The axillary nerve exits the quadrilateral space and divides into a posterior branch, which innervates the teres minor and the posterior portion of the deltoid, and an anterior branch, which innervates the middle and anterior deltoid. As the anterior branch courses from posterior to anterior, it lies approximately 4 to 5 cm distal to the lateral edge of the acromion. In this position, the nerve is vulnerable to injury if the surgical incision splits the deltoid beyond the 4- to 5-cm “safe zone.”

If this occurs, all portions of the deltoid anterior to the deltoid incision can be denervated, which results in substantial functional impairment. Therefore, extreme caution should be used when splitting the deltoid in line with its fibers, so that the length of the split does not exceed 4 to 5 cm.

Postoperative deltoid detachment can be minimized by using a deltoid-preserving approach during acromioplasty and cuff repair. \(^{50}\)

Once the interval between the anterior and middle deltoid fibers has been identified, the deltoid split is extended proximally into the deltotrapezius aponeurosis, at the anterior edge of the acromion. The incision in the deltotrapezius aponeurosis should be carefully placed so that it leaves a strong tendinous edge on the anterior deltoid to allow secure reattachment. Deltoid reattachment is accomplished by intratendinous repair of the deltotrapezius aponeurosis, which can be supplemented by transosseous sutures through the acromion.

If detachment of the deltoid is recognized early in the postoperative period, repair is much easier and more likely to yield a satisfactory result than if the postoperative detachment is discovered late, when the tendon has retracted and the muscle has atrophied. Therefore, the deltoid repair should be routinely inspected at each postoperative visit. The findings associated with deltoid dehiscence can be subtle. If the patient is requested to gently contract the deltoid while the arm is supported by the examiner, the integrity of the deltoid origin can be verified. Early postoperative failure of the deltoid repair is often associated with large hematoma formation, which should always raise the index of suspicion for possible deltoid disruption. When deltoid detachment is suspected, operative repair is warranted. If the initial repair was not transosseous, attempting reattachment to bone should be considered. Because the tissue quality is often suboptimal, an abduction brace or pillow may be used for protection.

The surgical management of chronic postoperative deltoid detachment or denervation includes primary repair, local muscle transposition, and distant muscle transfer. \(^{51,52}\) When the defect is small to moderate in size, primary repair is attempted. Complete closure of larger defects may require anterior transposition of a portion of the middle deltoid. Loss of the entire anterior deltoid due to denervation is a very difficult problem. If the deltoid deficiency is accompanied by a massive, potentially irreparable rotator cuff defect and coracoacromial arch incompetence, arthrodesis may be the most prudent option. If rotator cuff integrity has been maintained, however, bipolar transfer of the latissimus dorsi may be indicated. \(^{52}\)

Patients who have undergone radical or complete acromiectomy to represent a specific subgroup of patients with postoperative deltoid insufficiency that is even more difficult to treat than the group as a whole. \(^{53}\) Satisfactory results with radical acromiectomy have been reported. \(^{54}\) However, when deltoid dehiscence occurs after radical or complete acromiectomy, absence of the acromion makes reattachment of the deltoid technically difficult, if not impossible. In addition, radical acromiectomy, by definition, results in coracoacromial arch insufficiency. Postoperative deltoid detachment after radical acromiectomy combined
with a persistent uncompensated rotator cuff defect results in severe functional disability, which is probably not salvageable without arthrodesis. For these reasons, radical acromionectomy is unpopular.

**Summary**

Shoulder pain that persists after rotator cuff surgery may be the result of many causes, both intrinsic and extrinsic to the shoulder. Appropriate evaluation may identify a subset of patients with intrinsic shoulder disorders amenable to surgical correction. When continued pain is the result of persistent subacromial impingement or a persistent rotator cuff defect, the results of revision surgery are inferior to the reported results of primary arthroplasty and cuff repair. The goals of revision rotator cuff repair are a decompressed supraspinatus outlet and a permanently healed tendon. If the rotator cuff defect is irreparable but compensated, satisfactory results can be obtained with repeat subacromial decompression and partial rotator cuff debridement. The combination of an irreparable uncompensated rotator cuff defect and coracoacromial arch incompetence is currently an unsolved problem.

**References**


