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Rotator Cuff Tendinopathy

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Introduction

Shoulder pain is common: 16% of the general population suffer from it [1]. Rotator cuff disease is the commonest cause of shoulder pain, forming a large proportion of the workload of the specialist shoulder surgeon (of 1500 new shoulder referrals to a UK shoulder surgeon, 310 had rotator cuff tears) [2]. The morbidity associated with rotator cuff disease in terms of pain and loss of function is variable, but can be severely debilitating. In parallel with this, there is the cost to society in terms of loss of employment, Social Security claims, and the utilization of medical resources. Controversy exists in almost every area of the subject, and this chapter gives a broad overview of rotator cuff disease.

Basic Science of the Rotator Cuff

The rotator or musculotendinous cuff is a complex of 4 muscles that arise from the scapula. As these muscle and tendons extend toward the humerus, they intersect and blend with adjacent tendons and the subjacent capsule, forming a continuous cuff around the humeral head (see Figure 12-1). This cuff has a central role in the mechanics and function of the shoulder. The rotator cuff, however, does not function alone: it is part of a complex system of muscles, ligaments, and joints that affect shoulder movement.

The mechanics of cuff action are complex, but may be thought of as having 3 main functions. They rotate the humerus with respect to the scapula; they compress the head into the glenoid fossa, providing dynamic stability, particularly in the midrange of motion; and they provide muscular balance counteracting unwanted force components produced by non-rotator cuff muscles, e.g. deltoid or latissimus dorsi. Kuhn et al. give an excellent summary of the biomechanics of glenohumeral stability and shoulder kinematics [3].

The principal muscles involved in forming the rotator cuff are the supraspinatus, infraspinatus, subscapularis, and teres minor, although the long head of the biceps should also be considered as a functional part of the cuff [4]. From the suprascapular fossa, the supraspinatus passes laterally coursing beneath the coracoacromial arch and inserts into the greater tuberosity of the humerus. The space defined by the acromion posterosuperiorly, the humeral head inferiorly, the scapular spine posteriorly, and the coracoacromial ligament anterosuperiorly is known as the supraspinatus outlet. The supraspinatus tendon must pass through this outlet to its insertion, and here it can be compressed between the unyielding humeral head and the coracoacromial arch.

Near its insertion, fibers from the supraspinatus tendon fuse posteriorly with fibers from the infraspinatus, while others extend anteriorly toward the rotator interval, the space between the anterior portion of the supraspinatus and the superior portion of the subscapularis. Medially, the coracoid projects through the rotator interval, and the tendon from the long head of the biceps passes via the interval to its glenoid attachment, ensheathed in fibers from the subscapularis and supraspinatus tendons. Fibers of the coracoacromial ligament extend from the coracoid to the rotator interval and the supraspinatus, contributing to the cuff-capsule complex. Muscle contraction can thus tension the cuff and capsule in addition to applying force to the greater tuberosity.

The infraspinatus arises from the infraspinatus fossa of the scapula and inserts into the greater tuberosity posterior to the supraspinatus. Near its insertion some fibers diverge to blend with the supraspinatus anteriorly and the teres minor inferiorly. The infraspinatus can therefore tension the entire posterosuperior cuff capsule complex.

The subscapularis is the most powerful of the cuff muscles. Originating from the subscapular fossa of the scapula, it inserts onto the lesser tuberosity and has widespread attachment to both capsule and glenohumeral ligaments.

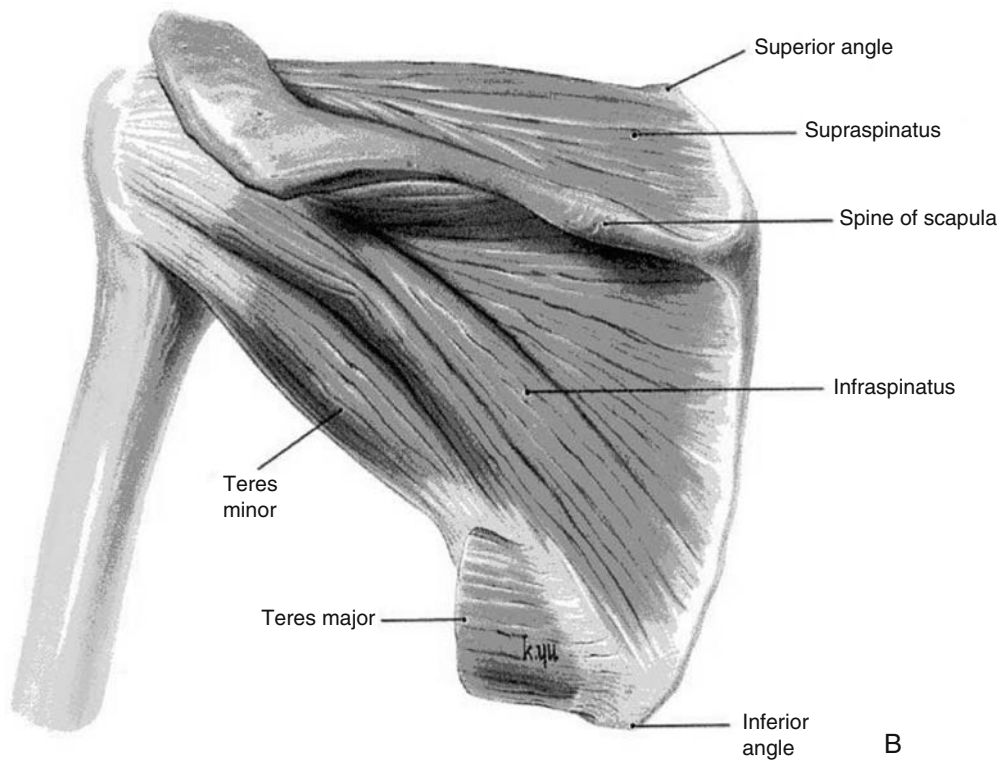
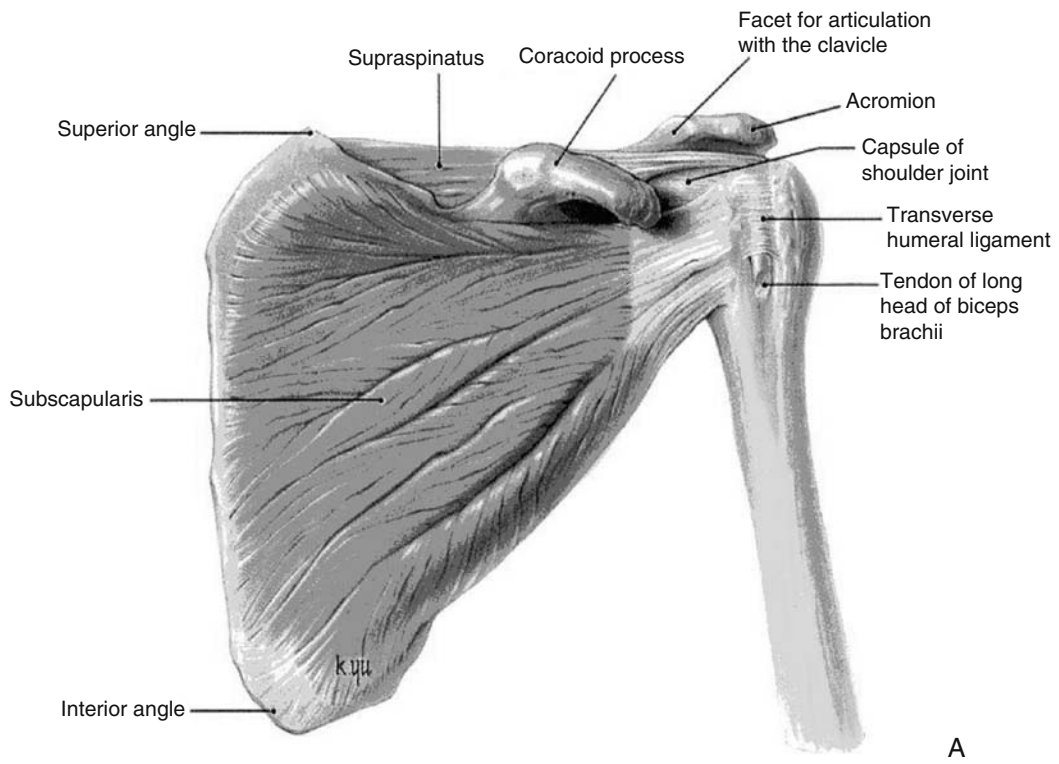


Figure 12-1. Gross anatomy of the shoulder.

Teres minor is the smallest of the cuff muscles. Originating from the lower lateral scapular border, it inserts inferiorly on the greater tuberosity.

At the insertion of the tendon fibers, a 5-layered structure of the cuff-capsule complex has been described [4]. The transverse transmission of force across the rotator cuff has been explained in terms of this structure and may be important in the initiation of cuff tears as significant shear forces occur.

The blood supply of rotator cuff structures is also important. Supraspinatus receives its blood supply primarily from the suprascapular artery, but contributions from both anterior and posterior circumflex humeral arteries and the subscapular artery have been reported. Classically, Codman [5] identified a “critical zone” at the supraspinatus tendon insertion as a region that had inadequate blood supply. More recently, a differential circulation between articular and bursal-sided tendon tissues has been reported, with the bursal side having better perfusion with respect to its articular side [2]. Despite this, laser Doppler studies have demonstrated a normal vascular supply with many anastomoses around this “critical zone” in uninjured cuff tendons, and impaired blood supply may be a secondary event in cuff tear pathogenesis.

Classification and Incidence of Rotator Cuff Tears

Rotator cuff tears are common and typically involve the supraspinatus tendon and often the posterior cuff to a variable degree. Subscapularis involvement may be present, and is easily overlooked.

Tears about the rotator interval are less common, and identification can be difficult. Tears may be vertical, horizontal or combined with widespread differences in size and degree of retraction.

There is no universally accepted classification of rotator cuff disease. However, important variables to consider when describing rotator cuff lesions include duration, depth, and size, as well as condition of the muscle and tendon. Acute tears may be associated with a traumatic event resulting in pain and dysfunction. Chronic tears may be associated with a variable degree of pain and weakness. Occasionally an acute extension of a chronic tear can follow shoulder trauma.

The depth of the tear will differentiate partial- from full-thickness tears. Partial tears can occur on the articular or bursal side of the tendon or within its substance. Classification of partial tears has been reported, but it is difficult to apply and not widely used. More concordance exists with the classification of full-thickness tears. Typically, they are described as small (less than 1 cm in diameter), medium (1 to 3 cm), large (3 to 5 cm), or massive (larger than 5 cm).

The true incidence of rotator cuff tears is unknown. By definition, such an incidence would refer to the occurrence of new rotator cuff tears. However, as individuals may remain asymptomatic, the actual incidence in a given population is impossible to measure, as only symptomatic individuals will present to the shoulder surgeon. The “true” prevalence of rotator cuff tears has been widely reported, and results vary depending on the design of the study. Cadaveric studies have shown prevalences between 5% and 30%, but these are age dependent. The Lehmanns [6] study of 235 cadavers found the prevalence of full-thickness tears to be 17%. The mean age of those with tears was 77.8 years, compared with 64.7 years in those without.

In contrast, in Tempelhof’s study of 411 living asymptomatic patients, 13% of patients under 60 years of age and 51% of patients over 80 years of age were diagnosed with cuff tears despite being asymptomatic [7], indicating the importance of age in the prevalence of such lesions.

Partial-thickness tears appear to be about twice as common as full-thickness defects. Bursal-sided lesions are more common, and studies have shown that these can cause more severe symptoms. Furthermore the progression of partial-thickness tears has been demonstrated both in terms of size and development into full-thickness tears. Much of the literature on the prevalence of rotator cuff disease has used cadaveric or symptomatic patients. The very fact that many asymptomatic individuals may have a rotator cuff lesion means that accurate data on the population as a whole are difficult to obtain, and all other data must be interpreted in view of this. Sher et al. [8] used magnetic resonance imaging (MRI) to scan 96 asymptomatic individuals with no history of shoulder complaints or evidence of pathology on clinical examination. For all age groups, the overall prevalence of rotator cuff tears was 34% (14% full-thickness, 20% partial-thickness). In the subgroup of individuals older than 60 years of age, the prevalence increased to 54% (28% full-thickness, 26% partial-thickness), thus showing the potential for normal, painless, functional shoulder activity despite observed rotator cuff abnormalities on MRI.

Etiology and Pathogenesis of Rotator Cuff Disease

The pathology of the rotator cuff includes a broad spectrum of conditions including reversible tendon inflammation, irreversible tendon degeneration, partial-thickness cuff tears, reversible calcific tendinopathy, full-thickness cuff tears, and degenerative glenohumeral arthritis (cuff tear arthropathy associated with chronic, massive cuff tears). The heterogeneity of rotator cuff disease, as well as the notion that the disease may not

represent a continuum of the same process, may explain differing viewpoints regarding its origin [2,7,8].

Postulated mechanisms of rotator cuff injury are either intrinsic or extrinsic. Studies supporting intrinsic mechanisms have included vascular and anatomical studies as well as evaluation of overuse syndromes. The critical zone described previously was thought to represent a region of poor vascularity at risk of injury and with little capacity for repair. Although generally refuted, aging, injury, or external compression may reduce perfusion, resulting in cuff injury. Degenerative changes in the cuff that may precede tearing have been confirmed histologically. These changes may in turn predispose to further injury and or tearing [6].

Normal cuff tendon is composed of collagen and small amounts of elastin, glycosaminoglycans (GAGs), proteoglycans, and water. The collagen present is predominantly Type I (>95%) and forms the main constituent of tendon fibers. Many other types exist but are found in much smaller quantities. Type III collagen is associated with the endotenon, Type IV with basement membranes, and Type VI with cellular interactions, but Types V, XII, and XIV have also been implicated in cuff tear pathogenesis. Under conditions of tendon injury and repair, collagen metabolism is significantly altered. There is an overall reduction in tendon collagen content, but gene expression of collagen Types I, VI, XIV, and particularly Type III, increases, the last thought to play an important role in stabilization of the extracellular matrix. In addition to this, Type II collagen expression is inhibited and mature crosslink formation increases.

Little remodeling is thought to occur before 50 years of age. After this age, remodeling occurs in response to tendon microruptures with the laying down of new collagen fibers that are thought to be of equal quality to pre-existing fibers. This is thought to represent the "wear, tear, repair" process of the normal rotator cuff. In chronic tendinopathy, however, remodeling is extensive in an attempt to repair tendon defects. In this case, the previously functional and carefully constructed matrix is replaced by aberrant collagen, which may result in a mechanically less stable tendon predisposing to tearing [9].

The detailed mechanism of collagen fiber remodeling is complex, but is thought to be mediated at the cellular level by matrix metalloproteinases (MMPs) such as collagenases and stromelysins [10]. These zinc-dependent enzymes are capable of degrading all components of the extracellular matrix and are produced as proenzymes by tendon tissues. MMPs in turn are under the control of tissue inhibitors of metalloproteinases (TIMPs).

Other components of the cuff tendons have been seen to be altered in rotator cuff disease. Under normal conditions, the GAG content of supraspinatus is different in comparison to that of biceps tendon and is analogous to

that found in fibrocartilage, suggesting an adaptation to mechanical stresses such as compression and shear. GAG deposition increases with both age and in disease states such as chronic tendinopathy [11]. Evidence of further adaptation has been found in diseased supraspinatus tendons by the presence of fibrocartilagenous metaplasia, in keeping with other tendons in the body that are subjected to compressive loads. This functional adaptation may have important consequences for the structural strength of the supraspinatus tendon and an influence on the ability of the tendon to repair after injury.

More recently, the localized deposition of amyloid in tears of the rotator cuff has been reported [12]. Increased sulfated GAGs and Type III collagen content have both been associated with amyloid deposition and, once deposited in tissues, amyloid is resistant to proteolytic degradation. This explains its persistence and continued accumulation leading ultimately to functional and structural failure of the affected tissue. As such, amyloid may play a role in cuff tear pathogenesis.

Calcium crystal deposition is another common finding associated with disease of the rotator cuff. The association between rotator cuff tear arthropathy and the intra-articular presence of basic calcium phosphate crystals (BCP) was identified in 1981 and termed the Milwaukee shoulder [13]. Electron microscopic analysis of synovial tissue from the glenohumeral joints of patients with cuff tear arthropathy revealed microspheroids of BCP crystals, as well as crystals within the synovial fluid. Basic calcium phosphate is a generic term used to describe crystals composed of carbonate-substituted hydroxyapatite, octacalcium phosphate, or rarely tricalcium phosphate. BCP crystal deposition is thought to occur secondarily as a result of severe joint changes associated with massive cuff tears, the crystals further accelerating joint damage by inducing synovial hyperplasia and metalloproteinase production. Calcium crystal deposition is known to occur in other periarthropathies, including calcific tendinopathy and chondrocalcinosis (where calcium pyrophosphate deposition predominates), and often a mixed picture exists in a variety of conditions, such as osteoarthritis.

Although the exact mechanism of calcium crystal deposition is poorly understood, recent studies using mice with the "ank" mutation (ank = progressive ankylosis locus) have increased our knowledge of the mechanism of calcification [14]. The ank mutation causes a generalized progressive form of arthritis accompanied by mineral deposition, osteophyte formation, and joint destruction. The ank gene codes for a multipass transmembrane protein necessary for the transport of inorganic pyrophosphate (PPi) out of cells. PPi is an important inhibitor of calcification, particularly BCP formation. Mutation of the ank gene results in a three- to fivefold decrease in extracellular PPi concentrations, thus producing a milieu conducive to crystal deposition. The

human ank protein is nearly identical to the mouse ank protein, with the human gene mapping to a region on chromosome 5p. Several human pedigrees with joint abnormalities, such as arthritis and chondrocalcinosis, have been mapped to the same locus as the human ank gene [15], although the role of the ank gene in disease of the rotator cuff remains to be seen.

Extrinsic mechanisms popularized by Neer [16] have implicated impingement against the undersurface of the acromion and coracoacromial ligament as primary factors in causing cuff tears. In support of this, it has been found that patients with Type III or hooked acromia have an increased incidence of rotator cuff tears (see Figure 12-2). Changes in the coracoacromial ligament can reduce the supraspinatus outlet area, resulting in extrinsic cuff compression. Distinctive histological changes with shortening and thickening of the coracoacromial ligament have been found in patients with cuff tears. Debate exists as to whether these are primary changes resulting in secondary cuff compression or secondary changes as a result of altered loading after a primary cuff tear. More recent evidence, including pathological changes in the acromion, has suggested that at this site the problem is predominantly intrinsic [17].

Several etiological factors have been associated with the development of rotator cuff disorders. Traumatic events such as anterior glenohumeral dislocation and fracture of the greater tuberosity can result in rotator cuff tears. However, it is difficult to determine whether trauma was the sole cause or whether a preexisting cuff lesion has extended.

Other traumatic insults occur in young athletes such as swimmers or tennis players who participate in repeated overhead activity. Such injuries generally manifest as small partial-thickness tears.

An association between an unfused acromial epiphysis or os acromiale and rotator cuff tears exists and is found in up to 8.2% of patients. Abnormal motion at the synostosis decreases the volume of the subacromial space, resulting in impingement. Congenital subacromial stenosis, a rare abnormality of the subacromial arch, may predispose certain patients to impingement. Attempts to unify intrinsic and extrinsic theories can be made to explain the natural history of rotator cuff tears.

Throughout its life, the cuff is subject to traction, compression, abrasion, inflammation, and, most importantly, age-related degeneration. Lesions of the cuff typically start at the deep surface of the anterior insertion of the supraspinatus near the long head of biceps. Tendon fibers fail when load exceeds their strength—either a few at a time or en masse—and retract after rupture. As a result, the load on remaining fibers is increased, the tendon is detached from bone thus decreasing force generation, blood supply is compromised causing local ischemia, and local tissues are exposed to lytic enzymes from the syn-

ovial fluid, which remove any hematoma. Risk from subsequent loading, and in the absence of repair, age-related degenerative processes result in extension to a full-thickness lesion and posterior propagation into the infraspinatus tendon.

With progressive dissolution of the cuff tendon, there is a loss of interposition of soft tissues between the humeral head and undersurface of the acromion, i.e., the “spacer effect” of the supraspinatus tendon is lost, resulting in superior migration of the humeral head and increased load on the biceps tendon. Further propagation of the defect crosses the bicipital groove to the subscapularis tendon, destabilizing the long head tendon.

Pain results in reflex inhibition of muscle action with less effective balance and stability. Increasing superior migration causes wear on the superior glenoid rim and labrum, and abrasion of the humeral articular cartilage on the coracoacromial arch may result in secondary degenerative joint disease known as cuff tear arthropathy. A diagrammatic representation of this “Unifying Continuum Theory” is seen in Figure 12-3.

Despite its attractive appearance, many discontinuities exist in this model, and debate continues as to its applicability. For example, the model cannot explain the variability in progression of partial- to full-thickness tears, the extension of small to large or massive tears, or why only 4% of patients with massive cuff tears develop cuff tear arthropathy. The existence of impingement without cuff tear and vice versa, as well as the heterogeneity of symptoms, simply does not fit into this model.

As a result, a more complex “discontinuous” theory has been developed in which the multifactorial nature of rotator cuff tear aetiology and pathogenesis is recognized (Figure 12-4).

History and Examination

Patient history and clinical examination are key elements in the diagnosis of rotator cuff pathology. Lyons and Tomlinson [18] concluded that preoperative clinical evaluation of a rotator cuff defect had a sensitivity of 91% and specificity of 75%. A detailed history enables the surgeon to perform a “directed” clinical examination, which, if performed correctly, should enable an accurate diagnosis to be made.

History

The overwhelming majority of patients with rotator cuff tears are over 40, the dominant extremity is most frequently affected, and up to 60% of patients can recall an exact incident to which their symptoms are attributed.

Pain, especially with overhead activity, is frequently reported with impingement and rotator cuff tears. Patients may complain of pain at night with the inability to sleep on the affected side, or describe periodic

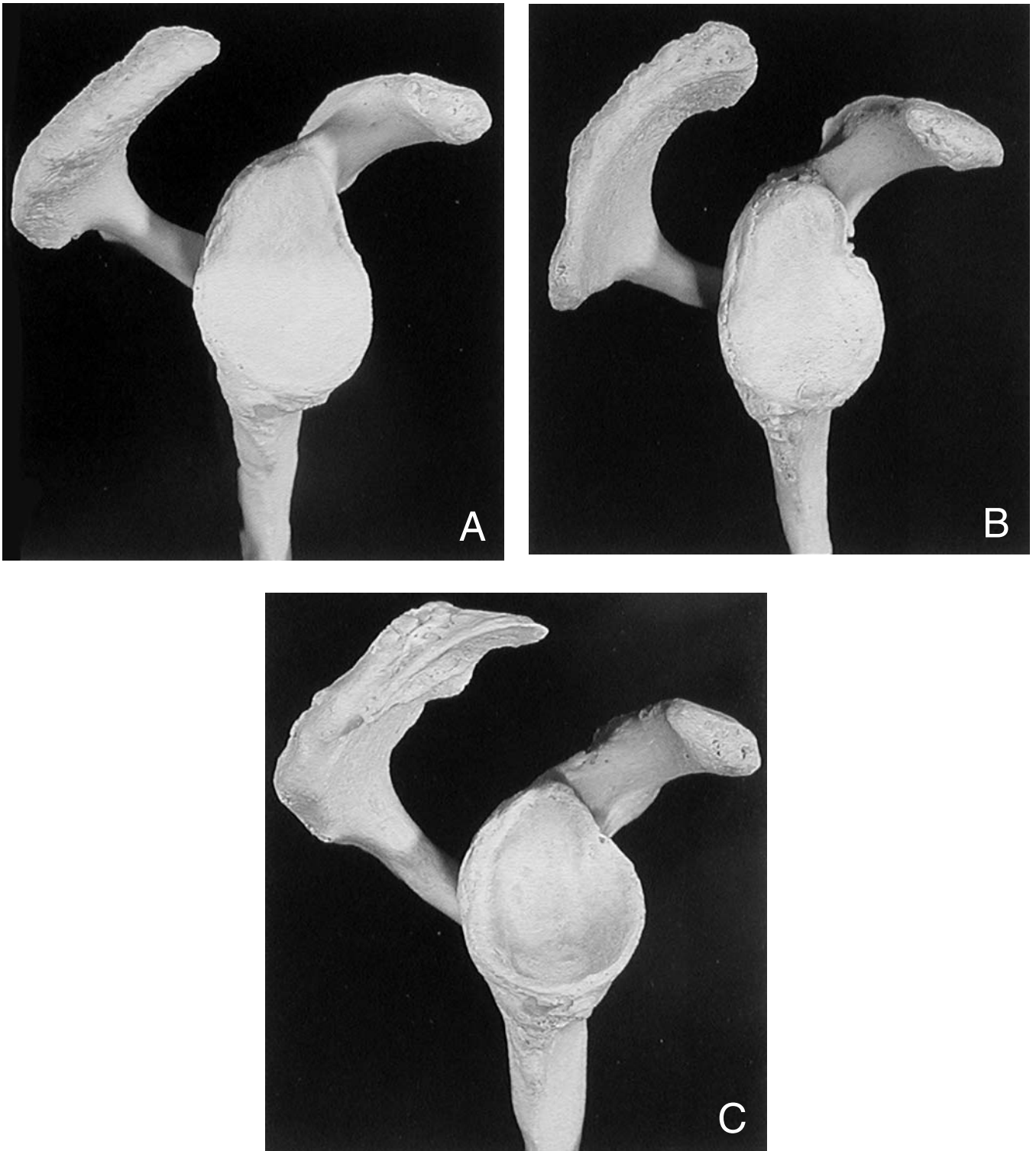


Figure 12-2. Morphology of the acromion. (A) flat, (B) curved, (C) hooked.

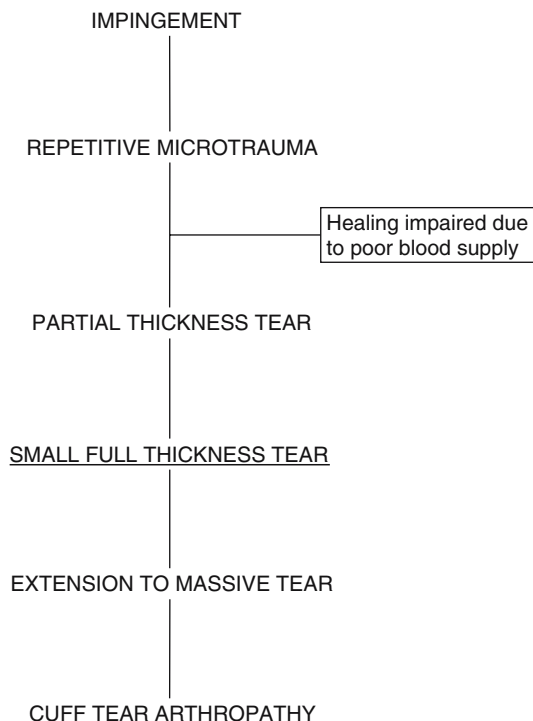


Figure 12-3. Unifying Continuum Theory of cuff tear aetiology and pathogenesis.

exacerbation of shoulder pain wrongly interpreted as “bursitis” or “tendinopathy” as small numbers of rotator cuff tendon fibers intermittently fail.

Weakness of the affected shoulder during abduction and external rotation is another frequent complaint. Full-thickness tears may produce crepitus with the patient complaining of “roughness” in their shoulder movement. Stiffness with reduced range of movement is a variable complaint. Delineating between a true reduction in range of movement and loss of movement secondary to pain can be very difficult.

Despite this, the symptomatology of rotator cuff pathology is extremely variable. Some patients with full-thickness tears have no signs or symptoms and have a normal quality of life, whereas some patients with small cuff tears have pain, marked weakness, and a significantly reduced range of movement. Such variation in symptoms, and more specifically why some patients have pain and some do not, is a question that remains to be answered.

Furthermore, when taking a history it is important to assess what impact a patient’s symptoms have on their quality of life. This is vital to establish what priorities and expectations patients have from their treatment and to enable the surgeon to accurately inform the patient whether these results are likely to be achieved.

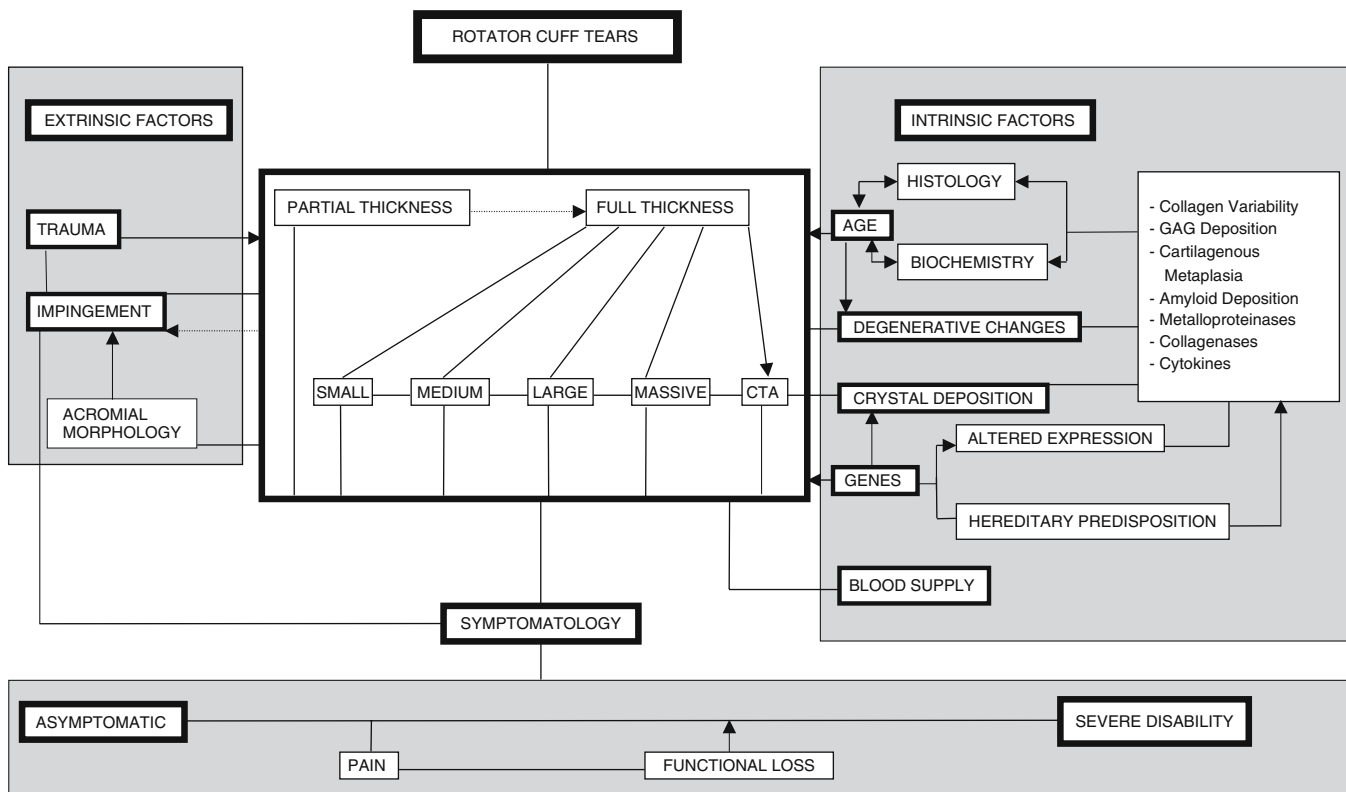


Figure 12-4. Discontinuous Multifactorial Model of cuff tear aetiology and pathogenesis.

Physical Examination

Physical examination can be divided into four parts: general inspection, palpation, range of movement/strength testing, and special tests.

i) Inspection

Inspection begins when meeting the patient. Age, body habitus, use of walking stick, and obvious systemic disease should all be noted. General inspection of the shoulder region may show muscle wasting, deformity or signs of previous surgery. More pertinent to rotator cuff pathology a prominent scapular spine may indicate supraspinatus and/or infraspinatus wasting (Figure 12-5). A ruptured long head of biceps may manifest as an obvious biceps deformity, particularly with elbow flexion.

ii) Palpation

Palpation over the greater tuberosity may elicit tenderness, as may palpation of the bicipital groove when there



Figure 12-5. Massive tear of the right rotator cuff with inability to abduct.

is associated biceps involvement. As pointed out by Codman [5], defects in the cuff can often be palpated by rotating the proximal humerus under a finger placed at the anterior corner of the acromion. Tenderness around the acromioclavicular joint should be noted, as this may represent degenerative changes in the joint as opposed to rotator cuff pathology.

iii) Range of Movement/Strength Testing

Both range of movement and strength testing can be carried out simultaneously, and it is currently recommended by the American Shoulder and Elbow Surgeons Society that 4 functionally necessary arcs of motion be recorded: forward flexion, external rotation in neutral position, external rotation at 90 degrees abduction, and internal rotation, with both passive and active ranges of movement being assessed.

iv) Special Tests

Many special clinical tests are recorded in the literature, which attempt to isolate and test specific muscles forming the rotator cuff (including the biceps tendon) and also to elicit signs of impingement. The most commonly used are mentioned below:

Tests of Rotator Cuff Integrity

1. *Supraspinatus Test.* Described by Jobe, resisted abduction by the arm extended at the elbow, flexed in the scapular plane with maximal internal rotation (thumb pointing to floor). Weakness or pain is specific for a tear of the supraspinatus tendon.

2. *Infraspinatus Test.* The external rotation lag sign originally described by Hertel [19] involves near maximal passive external rotation with the elbow flexed to 90 degrees. Holding the elbow, the wrist is released. The test is positive if the patient cannot maintain the position and a drop or lag occurs.

3. *Subscapularis Test.* Gerber's lift-off test involves internally rotating the arm with the forearm/dorsum of hand placed against the "small" of the back. Inability to lift the hand posteriorly off the back or hold the arm in a position just off the back is both sensitive and specific for a subscapularis tendon tear.

No specific test is commonly used to test teres minor. Its function is most commonly assessed by direct palpation of the muscle during external rotation of the arm.

Tests of Biceps Involvement

1. *Yergason's Test.* First described in 1931, Yergason's test describes pain localized to the bicipital groove when the examiner resists active supination with the elbow

flexed to 90 degrees and the forearm pronated. Yergason thought this pain represented wear and tear of the long head of biceps.

2. *Speed's Test*. Performed with the shoulder flexed, elbow fully extended, and hand supinated, resistance is applied by the examiner. Pain in the bicipital groove is suggestive of biceps pathology.

Tests of Impingement

1. *Painful Arc*. With the arm abducted in the coronal plane, pain is experienced typically between 60 and 120 degrees. Pain is often exacerbated by adding resistance.

2. *Neer Impingement Sign*. This involves forced passive "forward flexion" of the arm. The test is positive if the patient experiences pain with greater than 120 degrees of forward flexion. Neer's test involved a subsequent injection of 5 to 10 mL of 1% lidocaine into the subacromial space. Alleviation of pain on repeating the test confirms impingement.

3. *Hawkins' Test*. Forced internal rotation of the arm when flexed to 90 degrees.

4. *Jobe's Test*. Forward flexion to 30 degrees and abduction to 90 degrees against resistance.

In a recent prospective study, Murrell and Watson [20] compared the results of 23 commonly used shoulder tests in 400 patients with and without rotator cuff tears. Three simple tests were found to be predictive for rotator cuff tears: supraspinatus weakness, weakness in external rotation, and impingement. In patients older than 60 years with 3 positive tests, there was a 98% chance of having a rotator cuff tear. If none were present, this was reduced to 5%. Furthermore, they conclude that the predictive power of the combined clinical tests is similar to the best values for magnetic resonance and ultrasonography.

Imaging of the Rotator Cuff

Advances in medical technology have included the development of increasingly sophisticated imaging techniques, which have greatly enhanced the surgeon's ability to diagnose, stage, and treat rotator cuff disease.

It is not surprising, however, that, considering the nature of rotator cuff pathology combined with the need to image both bone and soft tissue structures, no single imaging modality has been universally accepted as being the investigation of choice. As a result, a large amount of sometimes conflicting published literature compares various techniques with findings at arthroscopy and open surgery. Combining this with additional variables such as availability, cost, and interobserver variability means that further research in this field is needed before absolute consensus is achieved.

Those techniques most commonly used will be discussed:

Plain Radiographs

In early rotator cuff disease, plain radiographs are usually normal. With more advanced disease, and particularly in patients with positive impingement signs, radiographic abnormalities are found, but to visualize such abnormalities it is necessary to obtain specific radiographic views. Views commonly used to assess rotator cuff disease include the true anteroposterior (AP), scapular outlet views, and the 30-degree caudal tilt AP.

A true AP radiograph of the shoulder may show a reduced acromiohumeral interval of less than 7 mm, which suggests the presence of a chronic tear. Subacromial calcification or sclerosis (the "sourcil" sign), sclerotic changes in the greater tuberosity, or gross changes consistent with cuff tear arthropathy may be seen (Figure 12-6).

Acromial morphology is best visualized with a scapular outlet view, whereas the 30-degree caudal tilt AP view will demonstrate an anteroinferior acromial spur or calcification of the coracoacromial ligament.

Norwood et al. [21] give a comprehensive account of 10 radiographic abnormalities associated with rotator cuff disease, and attempt to correlate the number of abnormalities found with the severity of the underlying rotator cuff pathology.

Arthrography

Under normal circumstances, no communication exists between the glenohumeral joint and the subacromial bursa. Such a communication is prevented by the presence of the rotator cuff. This anatomical relationship forms the basis of shoulder arthrography. When radiopaque contrast is injected into the glenohumeral joint in the presence of a full-thickness rotator cuff tear, contrast will leak via the tear into the subacromial bursa, which can be visualized with plain radiography. For many years arthrography has been considered the gold standard technique to evaluate full-thickness rotator cuff tears. It has a false negative rate of 0% to 8% in published data (probably due to scarring and adhesions precluding contrast leakage), but it is accurate in only 50% of cases in predicting the size of such tears [22].

Positional arthrography is also accurate in demonstrating joint side partial-thickness tears seen as contrast extravasates along the cuff tendon. Attempts at improving resolution have been made using double contrast methods (dye and air), as well as computed tomography (CT). Unfortunately, there is no one method that will allow prediction of tissue mobility, ease of repair, and long-term function. However, CT arthrograms can be useful in attempting to address these issues. The combi-

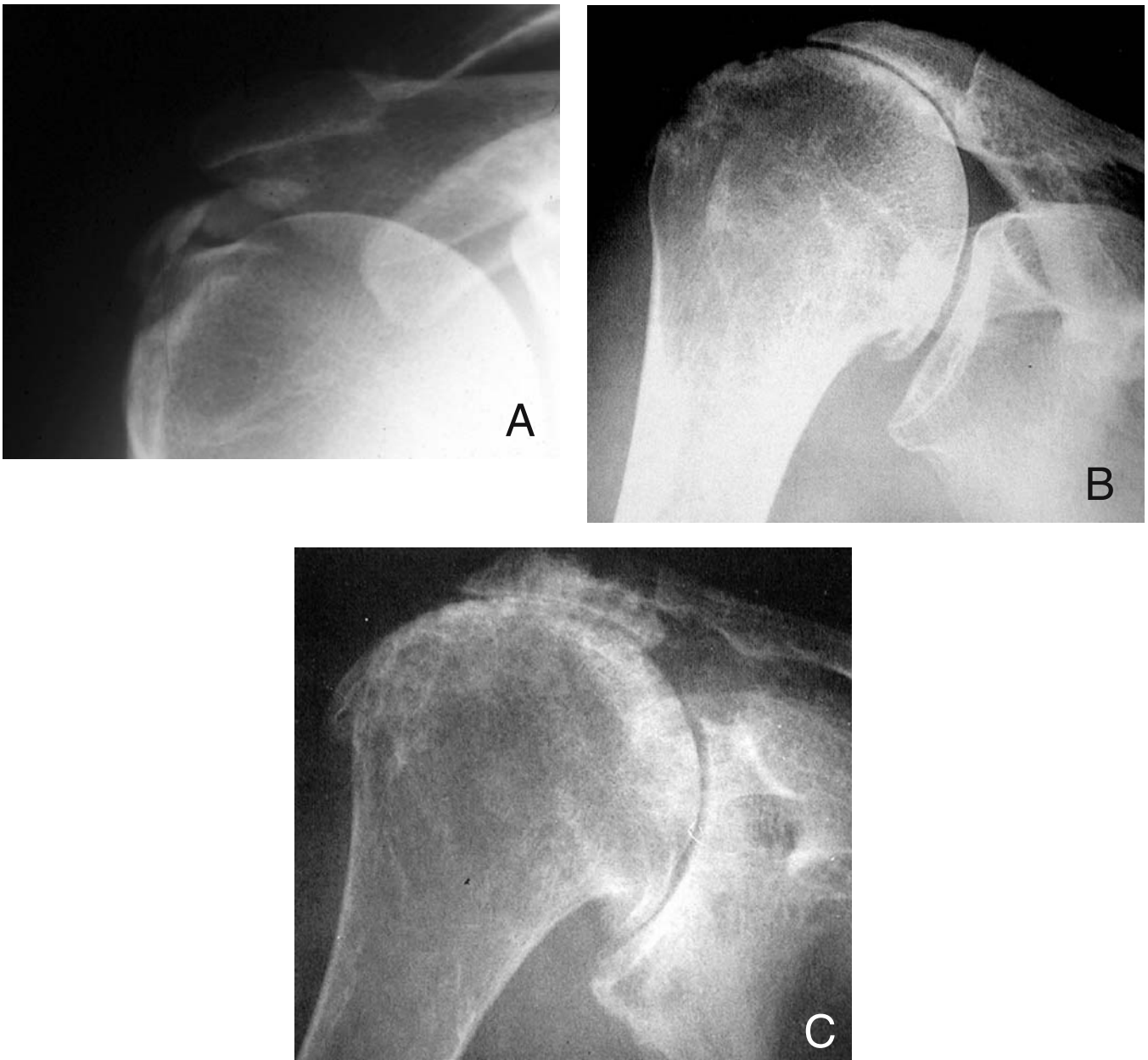


Figure 12-6. Plain radiographs showing features of rotator cuff disease. (A) Supraspinatus calcification. (B) Superior migration of humeral head. (C) Cuff tear arthropathy.

nation of arthrography and CT scanning enhances the quality of imaging of the glenoid labrum and glenohumeral capsule, as well as giving information about the degree of fatty degeneration within the torn rotator cuff. The degree of fatty degeneration of both supra- and infraspinatus is thought to be an important prognostic factor of the anatomical and functional results after rotator cuff repair. Goutallier et al. [23] proposed a 5-stage classification of such fatty degeneration based on CT scanning, and concluded that the degree of such degeneration had an influence on the final range of move-

ment (particularly in external rotation) and the final strength. Furthermore, fatty degeneration after cuff tear has a strong association with the degree of tissue retraction [24] and muscle atrophy [25], both of which predispose to difficulties in achieving tear closure at operation.

Arthrography is unable to demonstrate bursal-sided partial-thickness tears. As in all partial-thickness tears, contrast cannot communicate with the subacromial bursa and, under these circumstances, no joint side discontinuity exists in the cuff to allow contrast to extravasate along the tendon. Subacromial bursography has been used to

detect bursal side partial tears, but the procedure is both impractical and inaccurate. Complications such as infection, allergic reaction, and synovial effusions are rare, but the procedure is invasive. As a result, there has been a general tendency away from arthrography.

Ultrasonography

Shoulder ultrasonography has been used to diagnose rotator cuff pathology since the early 1980s. It is an inexpensive, noninvasive modality that does not use ionizing radiation, and is widely available. Furthermore, it allows dynamic evaluation of the rotator cuff with results in real time and the important benefit of practical bilateral examinations. Its main drawback is that it is highly user dependent.

Ultrasonography can reliably detect full-thickness cuff tears (Figure 12-7). Teefey et al. [26] compared ultrasonography with arthroscopic findings in 100 shoulders. Ultrasonography correctly diagnosed all 65 full-thickness cuff tears (sensitivity 100%, specificity 85%) confirmed at arthroscopy and was accurate in predicting tear size in 86% of cases. Sensitivities of 96% and above for the detection of full-thickness tears are widely published.

Ultrasonography is much less reliable at detecting partial-thickness cuff tears. The distinction between a partial tear, tendinopathy, and cuff degradation can be very difficult. The presence of fluid in the glenohumeral joint and subacromial bursa correlates highly with the presence of a rotator cuff tear and is helpful in diagnosing partial tears. In the aforementioned study, 67% of partial-thickness tears were identified correctly by ultrasonography, but sensitivities of above 90% have been published.

Given its user-friendliness, cost, and performance in comparison with other imaging modalities, ultrasonogra-

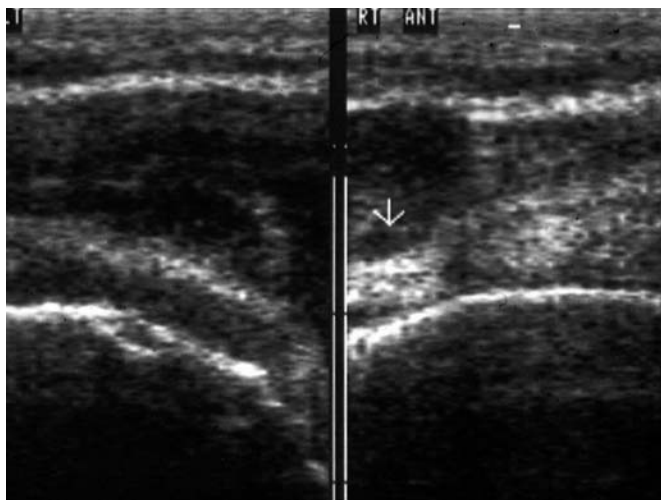


Figure 12-7. Ultrasound scan. (Left) Normal supraspinatus tendon. (Right) Full-thickness supraspinatus tear.

phy is now the “screening” modality of choice in the identification of rotator cuff tears.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) noninvasively produces high-resolution images of the bones and soft tissues of the shoulder, but, unlike arthrography, does not use ionizing radiation.

Its significant advantage is the diagnosis of midsubstance and bursal side rotator cuff tears, which allows detection of cuff pathology earlier in the disease process. Such tears can be difficult to diagnose with other imaging modalities. Bony pathology associated with rotator cuff disease may also be identified using MRI. Acromial morphology and subacromial spurs are easily demonstrated (Figure 12-8).

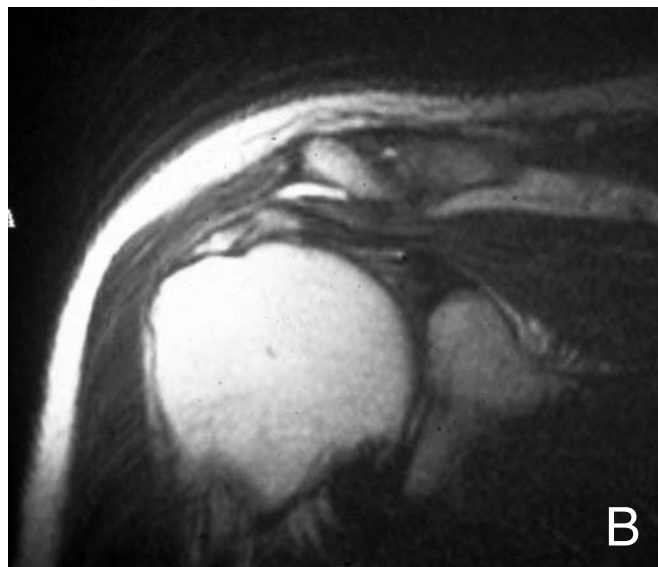
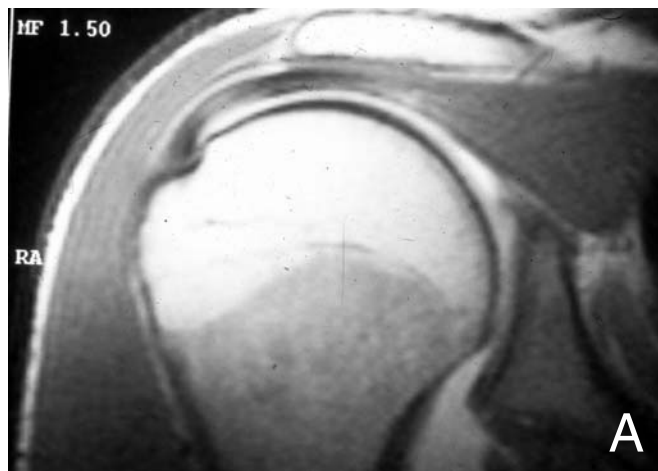


Figure 12-8. Magnetic Resonance Image. (A) Normal supraspinatus tendon. (B) Full-thickness supraspinatus tear.

As with ultrasonography, MRI is excellent at identifying full-thickness cuff tears. In Iannotti's series [27] comparing MRI with arthroscopic/open surgical findings, MRI had a sensitivity of 100% and specificity of 95% for full-thickness tears.

In addition, MRI offers information regarding cuff tear size, specific tendon involvement, and the degree of retraction. Tendon edges can be identified and comments made on the reparability of the tear. MRI can assist the surgeon in formulating a surgical strategy. However, MRI scans alone should not form the basis for surgery indications, as significant numbers of individuals with MRI-proven rotator cuff tears are in fact asymptomatic [8].

Arthroscopy

Diagnostic shoulder arthroscopy is indicated for the evaluation of the glenohumeral joint prior to arthroscopic subacromial decompression to rule out intra-articular or rotator cuff pathology. Concomitant pathological entities such as impingement, labral tears, and partial tears of the cuff on the articular side often exist.

Arthroscopy remains the definitive investigation in establishing the difficult diagnosis in the shoulder, all major pathological processes of the glenohumeral joint and subacromial space being amenable to arthroscopic diagnosis. It remains the benchmark by which other imaging modalities are compared (Figure 12-9, see color insert).

Despite this, arthroscopy should not replace, but instead enhance a detailed history, thorough clinical examination, and suitable radiographic tests, providing the indications and limitations of each are understood.

Management of Rotator Cuff Disease

Understanding what is the correct treatment for all forms of rotator cuff disease is both difficult and controversial. Patients with rotator cuff disease are a heterogeneous group, both in terms of their symptoms and underlying pathology. Furthermore, the methods and scoring systems used for functional assessment, symptoms, and quality of life evaluation as well as chosen outcome measures seem almost equally heterogeneous. Because of this, it is difficult to make comparisons and as a result draw sound conclusions from much of the vast array of published literature.

For the purpose of this chapter, an outline of the approach to the treatment of rotator cuff disease is offered, deliberately avoiding much of the minutiae and details of operative techniques that can be found in reference texts [28].

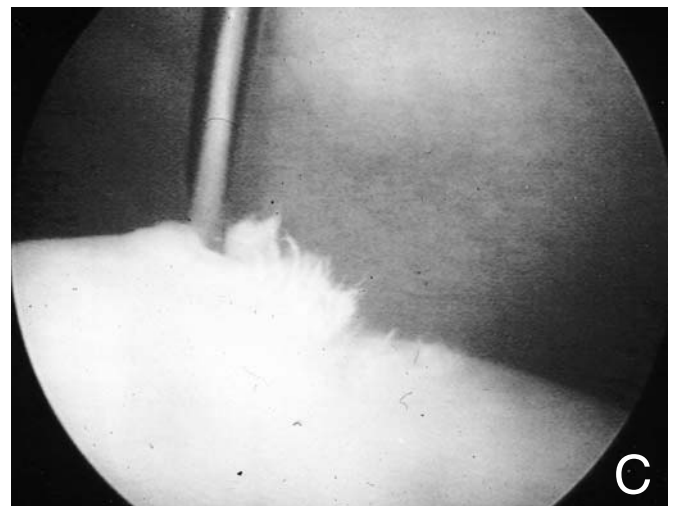
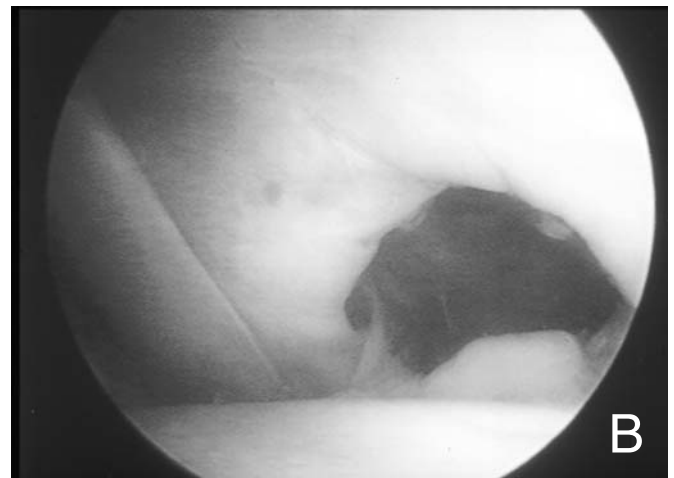


Figure 12-9. Arthroscopic appearances. (A) Bursal side full-thickness tear. (B) Joint side full-thickness tear. (C) Bursal side partial-thickness tear. (See color insert.)

Nonsurgical Management of Rotator Cuff Disease

The majority of shoulder surgeons will today recommend an initial trial of nonsurgical treatment for most patients with rotator cuff tears and would expect to continue this for at least 6 months before considering surgical intervention. Despite this, the length of nonsurgical treatment will vary depending on the degree of cuff involvement and with the patient's response to treatment. Continued pain despite an adequate rehabilitation protocol warrants consideration for surgical intervention.

The success of nonsurgical treatment has been reported from less than 50% to greater than 90%, and probably represents a lack of definitive indications for such treatment as well as variations in the protocols used. As a result, each patient needs to be individually assessed with regard to age, occupation, size of cuff tear, loss of function, mechanism of injury and, most importantly, pain.

Various nonoperative rotator cuff treatment programs have been described both for the general population and for athletes, and some surgeons devise individual treatment programs. Studies have consistently shown that better outcomes are achieved with well-structured, goal-directed rehabilitation programs tailored to each patient's specific needs. Within these programs a variety of treatment modalities can be used. These include:

1. *Physiotherapy*

Most physiotherapy regimes have common goals, which include the relief of pain, maintenance of good range of movement in the shoulder, and progressive strengthening of the rotator cuff, ultimately aiming for unrestricted movement. Typically patients are taught exercises which they need to perform several times per day for maximal benefit. Compliance is paramount, and regular patient review by the physiotherapist is important.

2. *Nonsteroidal Anti-Inflammatory Medication*

Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used despite very little evidence as to their efficacy in the management of tendinopathies. Particular concern should be given to their side effect profile, specifically with respect to nephrotoxicity and gastrointestinal bleeding in older patients.

3. *Corticosteroid Injection*

Few studies conclusively show that subacromial injection of corticosteroids is of benefit in the treatment of rotator cuff disease. Blair et al. [29] showed a significant improvement in pain, range of motion, and relief of impingement signs in patients injected with 1% lidocaine and 80mg triamcinolone compared with those receiving lidocaine

alone. However, other studies have found no evidence of the efficacy of such treatments. Despite this, the design of studies investigating the efficacy of corticosteroid injections is highly variable. The systematic review of randomized clinical trials of corticosteroid injections by Van der Heijden et al. [30] highlights this. The poor methodology of the majority of studies is emphasized, while the more robust and better-designed trials provide little conclusive evidence as to corticosteroid injection efficacy. In parallel, but on a much broader scale, the systematic review of randomized control trials of interventions for shoulder pain by Green et al. [31] was equally critical. This included all randomized controlled trials of NSAIDs, subacromial corticosteroid injection, oral steroids, physiotherapy, manipulation under anesthetic, hydrodilatation, and surgery for shoulder pain. The only positive finding was that subacromial corticosteroid injection was better than placebo in improving the range of abduction, with little evidence to confirm or refute the efficacy of other interventions.

Adverse effects from subacromial corticosteroid injections are well known, with infection and tendon atrophy with rupture being reported. Tissue quality after repeated injection can be so poor as to render surgical repair impossible. In light of this, one can appreciate the potential hazard of making a diagnosis of "bursitis" or "tendinopathy" and treating the situation with repeated steroid injections until the realization of a major cuff tendon degeneration becomes apparent. Speed [32] gives an excellent review on the use of corticosteroid injection and recommends a maximum of 3 injections at one site with a minimum interval of 6 weeks between each.

4. *Ultrasound and Phonophoresis*

Ultrasound has been widely used for more than 30 years and, as with NSAIDs, little evidence exists to confirm its benefit in the treatment of rotator cuff disease. Ultrasound has a thermal effect on tissue causing local hyperemia, which is thought to be beneficial to the soft tissue healing process.

Phonophoresis is the use of ultrasound to enhance the delivery of topically applied drugs, e.g. nonsteroidals and steroid creams. Such topically applied drugs avoid the risk of systemic complications and eliminate first-pass hepatic metabolism. Published data offer good results, though most studies were poorly designed, incorporating many different musculoskeletal pathologies.

In the study by Morrison et al. [33] of 616 patients with positive impingement signs (mean follow-up 27 months), 67% had a satisfactory result with nonoperative treatment, while 28% with no improvement went on to have arthroscopic subacromial decompressions. Similarly, Itoi and Tabaton [34], in a 3- to 4-year follow-up of 114

patients with full-thickness cuff tears, report satisfactory results in 82% of patients treated conservatively.

Surgical Treatment of Rotator Cuff Disease

Shoulder surgery is technically demanding. Those surgeons with sufficient surgical expertise who can accurately select patients and perform the most appropriate operations for those patients consistently achieve the best outcomes. Patient selection can be difficult.

Patients with positive impingement signs, a failure of nonsurgical treatment, continued pain, and evidence of supraspinatus outlet narrowing are candidates for surgical intervention. Such patients may also have evidence of cuff degradation or partial-thickness tears. The indications for surgery on patients with full-thickness tears depend on the size of the defect and the mechanism of injury. Early surgical intervention is often warranted with acute tears, particularly in the younger patient who sustains a traumatic tear associated with marked functional impairment and weakness [35]. In chronic cuff tears with an insidious onset of symptoms, surgical treatment is only considered after a full nonsurgical rehabilitation program.

Full patient assessment is important. Other factors that may influence earlier surgical intervention include high pre-morbid activity, high expectations for future activity, and younger physiological age. Whenever a worker's compensation claim is an issue, both nonsurgical and surgical outcomes are less satisfactory. For this reason, compensation claims have been used as exclusion criteria in some studies.

Shoulder Impingement Syndrome

Approximately one-third of patients with shoulder impingement will continue to have pain and show positive impingement signs despite nonoperative treatment. The majority of these patients will undergo acromioplasty.

Open decompression of the subacromial space was first described by Neer [16] in 1972. Ellman [36] published the first large series of 50 arthroscopic decompressions in 1987.

Arthroscopy is now generally favored. Proponents have argued that this procedure requires less surgical dissection and produces less scarring or postoperative morbidity. Additionally, cosmesis is good, and patient acceptance is high. Arthroscopy offers the ability to inspect the glenohumeral joint and subacromial space, as well as identifying any partial or full-thickness rotator cuff tear that may coexist.

Despite this, the goals for both open and arthroscopic techniques are identical. Subacromial decompression should recontour the acromion such that its undersurface is smooth and flat, impingement relieved, and pain alle-

viated. Recovery takes 2 to 4 months, with 83% to 94% of results proving satisfactory. As this procedure is carried out to relieve pain, such results are entirely dependent on the patient's subjective interpretation of their pain levels at review.

Less commonly, acromioclavicular resection is employed. Acromioclavicular arthritis or joint osteophytes can result in impingement and mechanical irritation of the cuff tendons. In this technique, 1.0 to 1.5 cm of the distal clavicle are excised, using either open or arthroscopic techniques, leaving a flat and smooth bone surface while preserving the superior and posterior capsular ligaments for stability.

Partial-Thickness Tears

Partial-thickness tears of the rotator cuff are common, with the majority of such tears occurring on the articular surface of the supraspinatus insertion site. Partial tears are not a single condition, but represent the common outcome of a variety of insults to the rotator cuff. Degenerative changes due to aging, anatomical impingement, and trauma may all be etiological factors. Because of this, several approaches to surgical treatment exist in the event of failed nonsurgical treatment.

There is no surgical treatment that reliably restores the tendon to its normal condition. Historically, arthroscopic debridement of the cuff defect and subacromial decompression have been used, either singularly or combined, with good results. Open repair by excision of the partial tear defect and repair of the subsequent full-thickness tear has also been used. The use of either technique is determined by the size of the defect, acromial morphology, age, and activity level of the patient.

For patients with significant supraspinatus outlet narrowing and partial-thickness tear of the supraspinatus alone with a defect involving <50% of cuff thickness, subacromial decompression alone, with or without arthroscopic debridement of the cuff tear, provides good or excellent results in the majority of cases. In younger patients or those with high functional demands and a tear of >50% of cuff thickness, open repair may be necessary, with or without subacromial decompression, depending on acromial morphology. In the series of 39 patients [37] with partial-thickness tears treated with debridement, decompression, and tendon repair, 90% regarded their general condition as improved at review (mean 55 months).

Full-Thickness Tears

Many patients with full-thickness cuff tears have excellent outcomes with nonsurgical treatment. In those who do not improve, open surgical repair is commonly undertaken. While the diagnosis of a full-thickness tear is not difficult, it is important to realize that in a small group

the cuff defect is irreparable due to the poor quality of the underlying tissues.

The greatest challenge in this type of surgery is dealing effectively with the torn retracted cuff tendons. The fresher the tear, the easier it is to repair and the less traction is required to bring the tendons back to their near normal anatomical position—re-rupture being one of the major complications of such surgery. Chronic tears are characterized by significant retraction and fibrosis, often necessitating partial resection of the thickened and proliferative subacromial bursa to delineate cuff tendon anatomy.

Usually irreparability can only be determined on inspection of the tissues, but information bearing on the reparability of the cuff defect can sometimes be obtained from the history and examination. Acute tears in younger, healthy patients are more likely to be repairable. Long-standing tears associated with major weakness in older patients carry a poorer prognosis. The prognosis for a durable repair is even worse if the history reveals local or systemic steroid usage, smoking, or previous surgery.

Several series have reported the results of surgery for full-thickness rotator cuff tears. Most describe clinical results that, on average, support satisfactory (good or excellent) result in 85% to 90% of patients [38]. Preoperative cuff tear size strongly correlates with other prognostic factors, including tendon tissue quality and the difficulty of tendon mobilization. Women with associated rupture of the long head of biceps often did worse.

It is not within the scope of this chapter to describe detailed surgical procedures; the reader is referred to reference textbooks.

Massive Tears

With massive tears, the complexity of the problem is magnified. Cofield suggested that tears greater than 5 cm should be termed massive, whereas Patte requires a tear diameter of 5 cm with acromial migration and glenohumeral arthrosis to consider a tear massive. Lack of uniformity in classification schemes makes it difficult to perform comparative research, and it is a source of controversy in diagnosis and management of such injuries. Disagreement also exists as to whether surgical results on patients with massive tears are affected by tear size. It appears that surgery for patients with massive tears is better at eliminating pain than improving function.

A multitude of surgical techniques exist for the surgical management of massive cuff tears. In patients with an irreparable cuff tear, debridement and decompression is thought to be the treatment of choice by many surgeons. Those patients whose main complaint is pain with an intact anterior deltoid and long head of biceps do best. Both short- and medium-term results have been satisfactory, however these initial results have been seen in some

studies to deteriorate with time. Any decompression should leave the coracoacromial arch intact. This structure is thought to prevent superior migration of the humeral head, especially in the presence of massive cuff tears, which may otherwise predispose to cuff tear arthropathy.

Many techniques for local rotator cuff repairs or muscle transfers have been described, for example using subscapularis or teres minor. Depending on the size of the tear and mobility of the muscle, these techniques may or may not be suitable to close massive rotator cuff defects. If unsuccessful, many other techniques have been described, and include distant muscle transfers, for example using trapezius or latissimus dorsi, and autogenous free fascia lata grafts.

Despite this, reconstruction of the rotator cuff may not eliminate end-stage rotator cuff arthropathy and pain. Treatment in these circumstances, as with the majority of rotator cuff surgery, is aimed mainly at the resolution of the patient's pain. Function is almost always improved with relief of pain, but functional goals are variable. Possible methods of treatment include conservative treatment, glenohumeral arthrodesis, resection arthroplasty, hemiarthroplasty, and total shoulder arthroplasty. Almost all published series of surgical management show very high complication rates.

Complications of Rotator Cuff Surgery

Patient selection is crucial for satisfactory outcomes in rotator cuff surgery, and good results can be expected if careful selection criteria are employed by a suitably skilled surgeon. With poor patient selection, complication rates increase. Such complications may be related to misdiagnosis, errors of technique, or unforeseeable postoperative factors such as poor rehabilitation and wound healing problems.

Complications Relating to Misdiagnosis

With a detailed history and examination, the diagnosis of impingement or a symptomatic rotator cuff tear can usually be made with a high degree of confidence. Conversely, the role of taking a history and performing a detailed clinical examination is to enable the surgeon to confirm or exclude other differential diagnoses that may be responsible for a patient's symptoms.

Other diagnoses that must be excluded include those related to referred pain such as cervical radiculitis, thoracic outlet syndrome, and suprascapular nerve entrapment. Intra-articular pathology, such as glenohumeral instability, arthritis, labral tears, or adhesive capsulitis may be present, as may extra-articular conditions including acromioclavicular joint arthritis and unrecognized rotator cuff tears. Occasionally, issues relating to sec-

ondary gain may be pertinent either in the form of compensation claims or in patients with psychiatric disorders. Furthermore, impingement and rotator cuff tears may occur with a secondary, coexistent pathology, which may complicate the diagnostic process.

Complications Related to Decompression

The deltoid, in concert with the rotator cuff, is responsible for generating synchronized and powerful glenohumeral motion. Deltoid detachment is a serious complication that results in significant disability, often in excess of the presence of an isolated cuff tear. Detachment typically occurs in the first 6 weeks postoperatively and is diagnosed clinically by observing a defect at the deltoid origin and a bulge in the deltoid muscle distal to its normal origin. Magnetic resonance imaging may confirm the diagnosis. Risk factors for deltoid detachment are complete or lateral acromionectomy, infection/hematoma, trauma, and early aggressive resistive physiotherapy. This complication has recently been reported with arthroscopic techniques.

Both conservative and surgical treatment (with reattachment or rotational deltoplasty) have shown poor functional results, and prevention of this disabling complication is stressed.

Neer has been credited with describing the anatomical importance of the anterior edge and undersurface of the anterior third of the acromion, coracoacromial ligament, and, in some cases, acromioclavicular joint in impingement. Inadequate decompression is one of the most common causes of poor results after performing acromioplasty. In cases where partial lateral acromionectomy has been performed, a significant number of patients continue to have symptoms because a portion of the "impingement anatomy" persists. Also, inadequate decompression has been associated with poor judgment regarding the amount of bone to be resected with respect to the anterior acromioplasty.

Acromial fracture is an infrequent complication of both open and arthroscopic acromioplasty, and has been associated with deltoid avulsion. Fracture is associated with overaggressive decompression (especially in osteoporotic bone) and, although rare, problems with healing are common (even with surgical intervention), resulting in pain and severe limitation of movement. Prevention by attention to careful surgical technique is therefore important to prevent this infrequent but significant complication.

Occasionally, symptoms of impingement recur as a result of heterotopic ossification at the site of previous acromionectomy. Rates of 3.2% are being reported. Risk factors include hypertrophic pulmonary osteoarthropathy, active spondylitic arthropathy, and chronic pulmonary disease.

Complications Related to Rotator Cuff Repair

Numerous techniques have been described for rotator cuff repair, particularly for large and massive tears, but no technique had been immune from the problem of recurrent tears. Recurrent tears have been attributed to size of tear at time of repair, inadequate tendon mobilization at operation, poor fixation techniques, trauma, and spontaneous rupture. Decompression of the subacromial space has been recommended as a concomitant procedure with rotator cuff repair. In addition to pain relief, the risk of recurrent tear is also reduced.

Neurological injury is a rare complication of rotator cuff surgery. Most commonly the axillary nerve is involved, but subscapular nerve injury is also reported. Most cases of axillary nerve injury are a result of overzealous deltoid retraction during deltoid splitting approaches, and are most frequently associated with an aberrant nerve course. Diagnosis is with electromyography.

Complications Related to Rehabilitation

Prolonged postoperative immobility, poor compliance, and deltoid detachment may result in a frozen shoulder. Treatment may be nonoperative with further physiotherapy, although manipulation under anesthetic or adhesion release may be needed. Less frequently encountered complications include reflex sympathetic dystrophy and rupture of long head of biceps.

Complications Relating to Wound Healing

Deep infections are rare, but necessitate aggressive management with drainage, debridement, and lavage, as well as culture-specific antibiotics. Such infections result in a significant negative impact on the final outcome of surgery. Less frequent complications include draining sinuses, suture granulomas, and keloid scars.

Treatment Failure

1. Failed Acromioplasty

In these circumstances, patients are dissatisfied with the results from previous arthroscopic or open acromioplasty, and usually continue to have marked pain. Such results occur in all series of acromioplasty, with incidences of failure ranging from 3% to 11%.

Treatment failure may be due to the presence of coexisting pathology. Diagnoses other than continuing impingement, including acromioclavicular joint problems, cervical spondylosis, thoracic outlet syndrome, and rotator cuff tears have been found in up to 45% of cases of failed acromioplasty. As previously mentioned, patients with workers' compensation claims often have poor outcomes even after revision surgery.

Other causes of failed acromioplasty include failure to achieve subacromial smoothness, failure of deltoid reattachment, excessive acromial resection, and postoperative complications such as dense scarring and poor rehabilitation.

Patients with unsatisfactory primary surgery need careful evaluation from a clinical, social, and vocational perspective. Often a nonsurgical treatment protocol may be commenced (even if this has previously been unsuccessful), and further imaging may be necessary.

Reoperation is considered in those well-motivated patients with residual subacromial "roughness." Patients with refractory shoulder stiffness may also be offered reoperation, as scarring between the acromion and rotator cuff cannot be managed nonoperatively. The revision procedure is usually identical to the primary acromioplasty.

Failed Rotator Cuff Surgery

Poor outcomes for rotator cuff surgery may occur for many reasons. Failure to ascertain patient expectations, infection, deltoid denervation or detachment, failure of cuff repair, and failure of rehabilitation are but a few causes.

Effective management of treatment failure depends on establishing the correct diagnosis. Infection needs culture-specific antibiotics, irrigation, and drainage if pus is present. Failure of deltoid reattachment needs prompt surgery before retraction becomes fixed. Chronically painful and functionally limiting scarring often responds to stretching exercises. Shoulder manipulation is not advisable due to the risk of cuff damage, but open lysis and removal of adhesions may be beneficial.

Persistent weakness needs evaluation for neurological injury or cuff failure, with denervation injuries being diagnosed with selective electromyography. Cuff failure is suggested by weakness of external rotation or abduction and superior instability of the humeral head. Dynamic ultrasonography is useful in such situations. Repeat cuff explorations with debridement or repair may be considered, but the risks of finding poor-quality tissues should be explained to the patient. Superior instability can result from loss of the coracoacromial arch without reestablishing stability with a durable cuff repair causing significant morbidity.

Results for revision cuff surgery are inferior to those for primary repair. DeOrio and Cofield found that at an average of 4 years after repair, 76% of patients had sustained diminution of pain but 63% still had moderate or severe pain [39].

In cases where a shoulder had been devastated by infection, denervation, or intractable cuff failure, consideration is given to arthrodesis. The best candidates for this procedure are those patients with severe weakness,

good bone quality, and a good understanding of the limitations and complications of the procedure.

Conclusion

Many aspects of rotator cuff disease are controversial, and further research is necessary in areas such as imaging, pathophysiology, and natural history to further our understanding of the disease and make improvements in diagnosis and treatment.

Many questions remain to be answered. What causes pain in rotator cuff disease? Why are some patients asymptomatic? What role does genetics have in rotator cuff disease, and could prevention be possible? More theoretical concepts such as preoperative assessment systems and outcome measures need to be standardized to facilitate comparative research and provide statistical power to evidence-based management. A uniform method of classification for rotator cuff tears would also be welcomed.

Patients with rotator cuff disease are a heterogeneous group, and because of this an individualized approach based on a detailed history and clinical examinations is vital. Despite this, rotator cuff disease represents a complex clinical challenge, and therefore its management should very much remain in the hands of the specialist shoulder surgeon.

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