REVIEW

Rotator Cuff Disorders

Recognition and Management Among Patients With Shoulder Pain

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Introduction

Shoulder pain is the third most common musculoskeletal symptom encountered in medical practice after back and neck pain (1), accounting for almost 3 million patient visits each year in the United States (2). A wide range of potential pathoanatomic entities can give rise to shoulder pain, from simple sprains to massive rotator cuff tears. The majority of these conditions are amenable to conservative treatment. Rotator cuff dysfunction is a particularly important entity because it occurs frequently and may necessitate surgical treatment. This report will provide a critical overview of current diagnostic and treatment techniques for rotator cuff disease.

Epidemiology

The point prevalence of shoulder pain has been estimated to be 7–25% and the incidence as 10 per 1,000 per year, peaking at 25 per 1,000 per year among individuals ages 42–46 years (3,4). The overall number of individuals with rotator cuff dysfunction is expected to grow coincident with an aging population that is increasingly active and less willing to accept functional limitations. A large proportion of patients with rotator cuff tears remain asymptomatic. Magnetic resonance imaging (MRI) scans of participants without shoulder pain

revealed partial- and full-thickness rotator cuff tears in 4% of individuals <40 years old and in more than 50% of individuals >60 years old (5). Furthermore, autopsy studies have demonstrated a 6% prevalence of full-thickness rotator cuff tears in subjects <60 years old and 30% prevalence in those >60 years old (6), although it was unknown how many of these subjects had shoulder pain.

Anatomy and pathophysiology

Anatomy (Figure 1). The shoulder has the greatest range of motion (ROM) of any joint in the human body. The size mismatch between the smaller glenoid and larger humeral head creates a risk of instability. Stability is provided both statically by the capsule and labrum, and dynamically by the rotator cuff musculature. Dysfunction of any of these structures can lead to pain, weakness, and instability.

The rotator cuff is a tendinous confluence of 4 muscles that initiate shoulder motion and maintain the normal relationship between the articular surfaces. The supraspinatus muscle provides abduction, the infraspinatus and teres minor muscles provide external rotation, and the subscapularis muscle provides internal rotation. In addition, the muscles of the rotator cuff balance the forces of other shoulder muscles, most importantly the deltoid muscle. Contraction of the deltoid muscle in the absence of supraspinatus function leads to superior translocation of the humeral head, making wide abduction difficult.
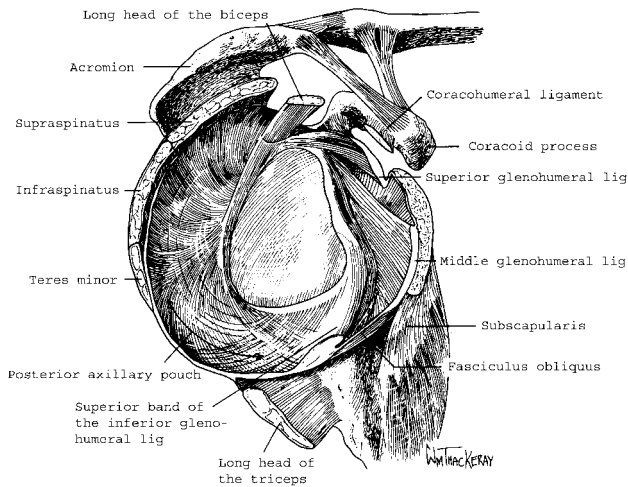
Rotator cuff dysfunction. Since Codman's report on rotator cuff tears in 1934 (7), a continuum ranging from impingement syndrome to partial- and full-thickness rotator cuff tears has been described as the basis of rotator cuff dysfunction. Impingement syndrome is a chronic process that manifests as shoulder pain and is, at least initially, reversible with rest or other conser-

Figure 1. Anatomy of the shoulder. Lateral view of the glenoid fossa (with humeral head removed). lig = ligament. Reproduced, with permission, from Turkel SJ, Panio MW, Marshall JL, Girgis FG. Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. J Bone Joint Surg Am 1981;63:1208.

vative measures. Left untreated, the pain can progress to permanent changes and eventual tearing of the rotator cuff, resulting in painful weakness. Impingement syndrome is classified into external, internal, and secondary impingement.

External impingement. External, or outlet, impingement, the most common form, is caused by compression of the rotator cuff tendons as they pass underneath the coracoacromial arch (8). Narrowing of the humeroacromial motion interface, which lies between this arch and the humeral head, causes compression of the intervening rotator cuff tendons. Inflammation of the subacromial bursa can ensue, leading to pain and further compression due to secondary swelling. Narrowing of the humeroacromial interface can occur for a variety of reasons, such as acromioclavicular (AC) joint osteophytes, acromial bone spurs, or malunions after proximal humeral fractures, especially if there has been displacement of the greater tuberosity (9). Neer has described several stages of external impingement, and he estimated it as the cause of ~95% of rotator cuff tears in his practice (8). Stage I affects younger patients, is fully reversible, and has hemorrhage and edema as anatomic correlates. Stage II is a disease affecting patients of middle age, is only partially reversible, and presents as tendon degeneration and fibrosis, also called tendinosis. Stage III occurs in elderly patients and is characterized by further tendon degeneration and rupture.

Internal impingement. Internal impingement has been described more recently and occurs primarily in

athletes who participate in overhead and throwing sports activities (10). Its anatomic correlate consists of undersurface fraying of the infraspinatus tendon, wherein it contacts the posterior glenoid as the arm is placed in maximum abduction and external rotation, such as the late cocking phase of throwing. Although this contact may often be present physiologically, the repetitive injury and eccentric loading associated with throwing can lead to labral and rotator cuff tears.

Secondary impingement. Secondary, or nonoutlet, impingement is a dynamic process caused by mild glenohumeral instability. Subtle subluxation of the humeral head brought on by activity can severely narrow the humeroacromial interface and thus lead to impingement symptoms. Posterior capsular contractures, such as occur with frozen shoulder, can cause obligate anterosuperior humeral head translation with forward flexion of the humerus. This also can narrow the acromiohumeral interval and result in secondary impingement

Intrinsic tendon degeneration. In contrast to Neer's theory, Ogata and Uhthoff (11) attributed most changes in the shoulder joints to intrinsic degeneration of the rotator cuff tendons. This degeneration was thought to arise from relative hypoperfusion of a watershed area close to the insertion on the greater tuberosity, in conjunction with repetitive microtrauma. Currently, most experts believe that both intrinsic tendon degeneration and impingement are contributing factors in the etiology of rotator cuff dysfunction (12).

Rotator cuff tears. The majority of rotator cuff tears occur in tendons with preexisting degeneration (12), which can progress to partial- and full-thickness tears, most commonly in the supraspinatus tendon. Full-thickness tears also may be precipitated by acute events; however, trauma with acute onset of weakness has been estimated to account for only 8% of patients undergoing rotator cuff repair (13).

Partial tears generally involve <50% of the tendon thickness and do not lead to retraction of the muscle (12). Depending on the location within the rotator cuff tendon, partial-thickness tears can be classified as intrasubstance, bursal-sided, or articular-sided (undersurface), the latter constituting ~90% of partial tears (14). Weakness is uncommon in partial-thickness tears but can arise from pain, which is often greater than that in complete tears.

In contrast, full-thickness tears represent complete discontinuity of rotator cuff fibers, resulting in communication between the articular and bursal spaces. The extent of the lesion on imaging studies is described in both the anteroposterior (AP) and mediolateral di-

Table 1. Differential diagnoses according to affected anatomic systems

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Articular
  Glenohumeral arthritis
    Osteoarthritis
    Inflammatory arthritis
  Instability
  Labral tears
Periarticular
  Calcific tendinitis
  Adhesive capsulitis
  Biceps tendinitis
  Fibromyalgia
  Scapulothoracic bursitis
  Acromioclavicular joint arthritis
  Acromioclavicular joint sprains
Osseous
  Proximal humerus or clavicle fracture
  Bone cysts, infections, or tumors
Referred
  Cervical radiculitis and arthritis
  Myofascial neck pain
  Cardiac, splenic, hepatic, and diaphragmatic processes
Neurogenic
  Parsonage-Tuner syndrome (brachial neuritis)
  Carpal tunnel syndrome

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rections. One centimeter is generally considered a small lesion, 1–3 cm is considered medium, 3–5 cm is considered large, and >5 cm is considered massive. Tears that involve ≥2 tendons can also be classified as massive, and these require more complex reconstruction (15). In larger tears, chronically retracted muscles undergo fatty degeneration over time that may be irreversible, and thus may make results of direct repair unsatisfactory (16).

Natural history. The natural history of rotator cuff dysfunction is not well understood. Yamaguchi et al demonstrated that in 50% of individuals with asymptomatic tears pain developed within 5 years, even though only 30% demonstrated increases in tear size (17). Studies investigating partial tears of the rotator cuff have demonstrated enlargement or progression to full-thickness tears in 80% of patients over a period of 2 years, and these were managed with nonoperative therapy (18). Once a tear occurs, there seems to be little or no evidence of spontaneous healing. A histopathologic study showed no signs of healing in pathologic specimens from partial-thickness tears (12). Furthermore, although shoulder symptoms may be short-lived, persistence or recurrence of symptoms in 40–50% of individuals within 1 year after the initial presentation has been reported (4,19,20).

Differential diagnoses. Differential diagnoses to consider in the evaluation of the painful shoulder can be grouped based on the affected anatomic structures (Table 1) or chronicity of symptoms. The differential diagnoses for the acute onset of shoulder pain include traumatic events such as shoulder dislocation, AC joint sprains, and clavicle and proximal humerus fractures. Nontraumatic etiologies of acute shoulder pain include calcific tendinitis, biceps tendinitis, and, much less frequently, gout, septic arthritis, or septic bursitis. Shoulder pain can also be caused by the sudden exacerbation of chronic processes such as glenohumeral and AC osteoarthrosis or inflammatory arthritis. Shoulder symptoms that occur chronically are suggestive of frozen shoulder (adhesive capsulitis), polymyalgia, or, rarely, osteomyelitis or neoplastic disorders. Referred pain from disorders of the cervical spine is a very common source of shoulder pain, and a careful clinical examination is essential to distinguish between local and referred processes. Rarely, visceral radiation, as occurs with splenic, hepatic, diaphragmatic, and cardiac processes, may be discerned as shoulder pain.

Evaluation and diagnosis

Individuals with rotator cuff disorders can be divided into 2 groups according to their presenting symptoms: 1) those with impingement-type symptoms, frequently manifested as pain at night and at rest as well as a painful arc of motion, which can often be successfully treated by conservative measures; 2) those with symptoms of a torn rotator cuff tendon, manifested as painful weakness and atrophy, which frequently do not respond fully to conservative measures alone and for which surgical intervention should be considered.

History. Rotator cuff pain is frequently described as a dull ache of insidious onset, extending over the lateral arm and shoulder. Overhead activities exacerbate the pain, and the pain frequently increases at night and may awaken the individual from sleep. Weakness with the inability to abduct and elevate the arm is seen in more advanced cases; patients frequently describe difficulties combing hair, holding a hair dryer, and removing the wallet from their back pocket. Immediate onset of weakness, especially in association with trauma, may indicate an acute tear.

Clinical examination. *Examination of cervical spine.* The cervical spine is a frequent source of referred pain. Therefore, it should always be included in a clinical examination.

Inspection and palpation. <u>Inspection.</u> The shoulder girdle musculature often shows evidence of muscle atrophy. The supra- and infraspinatus muscles typically demonstrate atrophy in advanced rotator cuff tears.
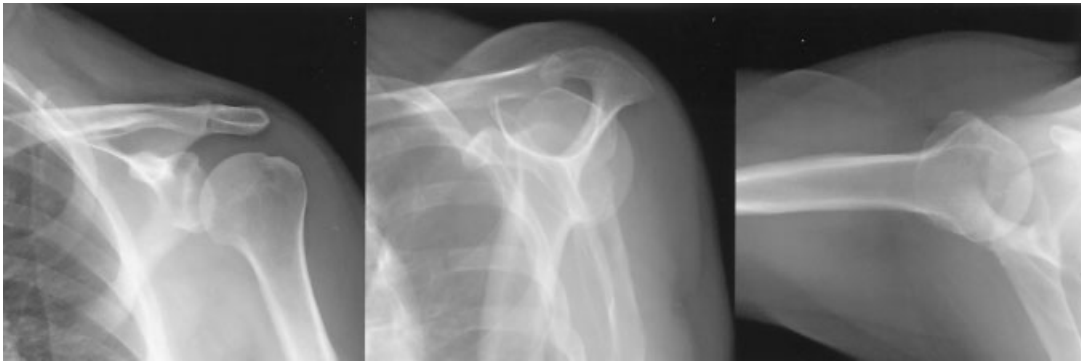
Figure 2. Shoulder radiographs for imaging studies of rotator cuff dysfunction. **Left,** Anteroposterior view. **Middle,** Outlet view. **Right,** Axillary view.

Swelling over the AC joint can be a sign of traumatic or degenerative changes.

Palpation. The arthritic AC joint is a frequent source of shoulder pain and will manifest as point tenderness. Bicipital tendinitis can be detected with palpation over the anterior shoulder, with the arm in slight internal rotation. The greater tuberosity can be tender to palpation, due to the bursitis often observed in conjunction with rotator cuff disease and calcific tendinitis, and can be palpated by extending the humerus.

Motion testing. ROM testing should first be performed actively by the patient, and then be performed passively by the examiner, with the shoulder in forward elevation, abduction, external rotation, and internal rotation. The contralateral shoulder can serve as a baseline referent if it is uninvolved. Comparison of active and passive ROM provides insight into the diagnosis. For example, greater passive ROM than active ROM, with a painful arc between 60° and 120° of abduction, is common in rotator cuff dysfunction, whereas globally decreased active and passive ROM is typically noted in adhesive capsulitis and osteoarthrosis.

Impingement signs. Provocative tests of impingement attempt to recreate shoulder pain by compressing the rotator cuff between the humeral head and other bony structures, such as the acromion or coracoid process. The 3 most commonly used provocative maneuvers are Neer's impingement sign, Hawkins' impingement sign, and Neer's impingement test. It should be noted, however, that the first 2 signs can be relatively nonspecific and may yield positive results in the setting of other pathologic entities such as AC joint arthritis or biceps tendinitis. Tests of the biceps tendon and AC joint can be used to assess the pathologic condition of these structures. Speed's test and Yergason's test elicit pain in the bicipital groove when an inflamed biceps tendon is stretched. Both tests, however, have questionable value, due to their relatively low sensitivity and specificity (21). The cross-body adduction test reproduces pain in the AC joint when the arm is adducted horizontally in front of the body.

Functional tests. Functional tests are performed for each of the 3 muscle groups of the shoulder: the subscapularis, the infraspinatus and teres minor, and the supraspinatus. Tests of the biceps are conducted as well, to determine if positive impingement test results are a result of biceps tendinitis. When evaluating strength, it is important to consider whether perceived weakness is secondary to loss of muscle or due to voluntary or involuntary inhibition secondary to pain. Frequently, subacromial anesthetic and/or corticosteroid injections are helpful to distinguish between the 2 causes. In addition to evaluating gross strength, the examiner can obtain important information by the re-creation of pain with specific functional tests.

Gross muscle strength is tested first. More specific tests of muscle function are then conducted: the subscapularis is tested with resisted internal rotation, the supraspinatus is tested with resisted abduction in the plane of the scapula, often referred to as Jobe's testing, and the infraspinatus and teres minor are tested with resisted external rotation. Side-to-side comparison is helpful. Lag signs, which are pathognomonic of rotator cuff tears, have been described by several authors: the lift-off and modified lift-off are tests for the subscapularis, the drop arm sign is used for the supraspinatus, the external rotation lag is used for the infraspinatus, and Hornblower's sign is used for teres minor dysfunction (22).

Imaging studies. *Radiographs (Figure 2).* In the AP radiographic view, joint space narrowing and osteophyte formation may indicate arthritis of the glenohumeral or AC joints. Calcium deposits from calcific

tendinitis usually occur just proximal to the rotator cuff insertion. Elevation of the humeral head on AP radiographs, especially when the subacromial space is decreased to <5–7 mm, has been associated with large rotator cuff tears (23). The axillary view is essential to exclude the possibility of a dislocation. This view also shows the joint space and helps identify the rare, but occasionally symptomatic, os acromiale, which is a persistent and nonunited ossification center at the end of the acromion (24). Finally, the supraspinatus outlet view allows visualization of the bony structures of the scapulohumeral motion interface and shows acromial spurs or calcification of the coracoacromial ligament that might compress the underlying rotator cuff.

Arthrography. Arthrography has been largely replaced by other imaging techniques, such as MRI and ultrasound. Although relatively inexpensive, arthrography is invasive and less accurate than MRI, especially for the diagnosis of partial-thickness tears, but remains of value in patients with contraindications to MRI.

Ultrasound. Ultrasound is noninvasive, readily available, and inexpensive. Recent studies utilizing arthroscopy or MRI for validation of ultrasound have demonstrated sensitivities of 58–100% and specificities of 78–100% for full-thickness tears (25,26). It is less accurate in the detection of partial-thickness tears, with sensitivities ranging from 25% to 94% (26–28).

MRI and MR arthrography (Figure 3). MRI has sensitivities close to 100% for full-thickness tears, and has all but replaced arthrography for the diagnosis of rotator cuff disease (29). Moreover, the additional quantitative and qualitative information gleaned from this cross-sectional study aids in the surgical planning and prognosis. The combination of MRI and gadolinium-enhanced arthrography further improves sensitivity, especially for the detection of partial tears, to more than 90% (30), and in the detection of labral disease, the sensitivity is improved to more than 80% (31). Important concerns regarding MRI include the associated cost and high frequency of false-positive results. Up to 30% of asymptomatic volunteer subjects have findings of rotator cuff anomalies, and up to 50% show labral anomalies (32).

Management options and outcome

The ultimate goal of any therapeutic intervention for shoulder pain is the restoration of pain-free function. Specific patient factors, such as age, preinjury functional level, demand, and general health, guide the physician in the selection of attainable goals and choice of therapy.
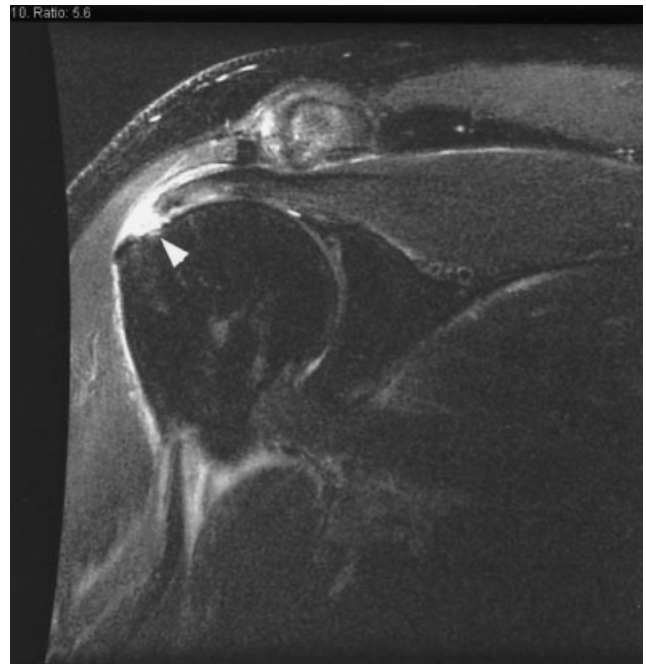
Figure 3. Oblique coronal T2 magnetic resonance image depicting a full-thickness rotator cuff tear (**arrowhead**).

For the purposes of this review, we searched Cochrane's Database of Systematic Reviews and Medline to identify relevant articles (see Tables 2–4), utilizing the keywords rotator cuff tear, rotator cuff tendinitis, and shoulder pain, with special consideration of publication types designated as randomized controlled trials and reviews.

Nonoperative treatment. Nonoperative treatment for shoulder pain due to rotator cuff impingement and tears generally includes appropriate physical therapy, antiinflammatory medication, corticosteroid injections, and other approaches. Pain relief and restoration of function have been observed in 62–74% of patients with symptomatic, radiologically proven rotator cuff tears. Predictors of good outcomes are greater preoperative muscle strength, such as the ability to lift the arm above the level of the shoulder, and duration of symptoms <6–12 months (33,34).

Medical management. Options for the medical management of rotator cuff disease include systemic and local approaches. Nonsteroidal antiinflammatory drugs (NSAIDs) decrease symptoms of cuff irritation and inflammation, and should be prescribed around-the-clock to maximize the antiinflammatory effects. This can be augmented by subacromial injections of local anesthetic agents and corticosteroids. Injections should not be instilled directly into the tendon substance, because

Table 2. Overview of outcomes of medical interventions*

First author, year (ref.)	Condition treated	Treatment arms	Study design	Outcome measure	Results
Blair, 1996 (67)	Impingement syndrome	Subacromial steroid injection, subacromial local anesthetic injection	RCT (n = 40), FU at 33 weeks	Pain, ROM	Significant pain reduction for treatment group, from 2.4 to 1.2 (0–4 scale). Control group showed reduction from 2.3 to 2.0. At FU, 15 of 19 treatment patients were without impingement sign vs. 4 of 21 in control group.
Petri, 1987 (68)	Shoulder pain	Subacromial steroid injection, oral naproxen, naproxen + injection, placebo	RCT (n = 100)	Pain, ROM, function	Both treatments significantly superior to placebo. Responders: 4% and 8% with placebo, 12% and 20% with naproxen, 8% and 28% with steroid, 20% and 28% for the combination, at 2 weeks and 4 weeks, respectively.
van der Windt, 1998 (69)	Painful, stiff shoulder	Intraarticular steroid injection, PT	RCT (n = 109), FU at 12 months	Pain, ROM, function	Statistically significant difference with 77% (injection) vs. 46% (PT) of patients improved at 7 weeks. Trend toward smaller difference between groups with increasing FU time.
Winters, 1997 (70)	Shoulder pain	PT, manipulation, steroid injection (intraarticular, subacromial, and AC joint)	RCT (n = 114), FU at 11 weeks	Pain, ROM	At 5 weeks, 75% (injection), 40% (manipulation), and 20% (PT) of patients rated themselves as "cured."
Hay, 2003 (71)	Shoulder pain	PT, subacromial steroid injection	RCT (n = 207), FU at 6 months	Pain, ROM, function	Improvements (defined as minimum of 50% drop in disability scores) in 60% of patients in PT group and in 53% in injection group. No statistically significant difference between treatment arms at 6 weeks and 3 months.
Adebajo, 1990 (72)	Rotator cuff tendinitis	Diclofenac + steroid injection, placebo + steroid injection, placebo + placebo injection	RCT (n = 60), FU at 4 weeks	Pain, ROM, function	Average improvements in VAS pain score (% responders) of 1.35 (0%) with placebo, 3.6 (30%) with diclofenac, and 4.95 (70%) with triamcinolone injections. Both treatment groups significantly improved, but no difference between the 2 groups.
White, 1986 (73)	Rotator cuff tendinitis	Indomethacin, subacromial steroid injection	RCT (n = 40), FU at 6 weeks	Pain, ROM	No statistically significant difference between groups. Improvement of 60% (injection) vs. 66% (NSAID).

* RCT = randomized controlled trial; ROM = range of motion; FU = followup; PT = physical therapy; AC = acromioclavicular; VAS = visual analog scale; NSAID = nonsteroidal antiinflammatory drug.

this and multiple injections within a short period of time increase the risk of associated tendon rupture (35). Several studies (Table 2) have investigated the efficacy of oral NSAIDs and injectable steroids. Overall, results demonstrate significant improvements with either form of treatment, although somewhat faster and greater pain relief is achieved with injections.

Several studies have investigated the accuracy of injections, with results ranging in accuracy from 29% to 87% (36–38), underscoring the technique-dependence of injections. These studies also demonstrated a significant difference in outcome, with far better results for accurate injections than for missed injections. Given the invasiveness of injections, many practitioners prefer an initial trial of oral NSAIDs and physical therapy, with injections reserved for patients with persistent pain or severe pain at the time of the initial presentation.

Physical therapy. Physical therapy and rehabilitation for rotator cuff signs and symptoms are conducted in 3 phases (34). Phase 1 consists of activity modification in addition to pain control with NSAIDs and injections. In phase 2, gentle ROM exercises are initiated to prevent adhesions (39). Only after restoration of full ROM should physical therapy transition to phase 3, which consists of a strengthening program for the rotator cuff and scapular stabilizers. Very little data comparing physical therapy with no treatment are available (Table 3). One retrospective study investigating the

Table 3. Overview of outcomes of physical management techniques*

First author, year (ref.)	Condition treated	Treatment arms	Study design	Outcome measure	Results
Ginn, 1997 (74)	Shoulder pain of mechanical origin	PT for 4 weeks, no treatment	RCT (n = 66), FU at 1 month	Pain, ROM, function	At 4 weeks, 11% of patients in the treatment group scored worse for ROM and disability each, while in the control group 32% of patients had decreased ROM and 50% had worse disability scores.
Goldberg, 2001 (41)	Full-thickness rotator cuff tears	PT home exercise program	Prospective (n = 46), FU at 12 months	Pain, function	After 1 year, 59% demonstrated improvement, 30% worsened, and 11% showed no change.
Bang, 2000 (75)	Impingement syndrome	PT alone, PT with manual PT	RCT (n = 52), FU at 2 months	Pain, strength, function	Both groups had significant improvements. Pain reduction significantly better in PT with manual therapy group, with a decrease in pain scores from 575.8 to 174.4, while PT alone reduced pain from a pretreatment mean of 557.1 to a posttreatment mean of 360.6 (VAS 0–1,000).
Morrison, 1997 (40)	Impingement syndrome	PT + NSAIDs	Retrospective (n = 616), FU at 27 months	Pain, ROM, function	Retrospective study. 67% of patients had satisfactory result. 28% without improvement proceeded with SAD, 5% without improvement declined surgery. 18% of patients with initially satisfactory outcome had recurrence and were treated nonoperatively.
Downing, 1986 (76)	Supraspinatus tendinitis, bursitis, adhesive capsulitis	PT + NSAIDs with US, PT + NSAIDs with placebo US	RCT (n = 20), FU at 4 weeks	Pain, ROM, function	Both groups improved from a moderate-to-severe pain rating to mild-to-moderate rating without statistically significant differences between the groups. Trial limited by small sample size.
Vecchio, 1993 (77)	Rotator cuff tendinitis	Low-level laser therapy, placebo laser therapy	RCT (n = 35), FU at 8 weeks	Pain, ROM, function	Improvement in VAS pain scores of 2.2 (4 weeks) and 3.9 (8 weeks) from 6 at baseline for laser treatment, 1.4 and 2.2, respectively, for control. Improvement in VAS functional scores of 2.9 (4 weeks) and 3.6 (8 weeks) with laser, 2 and 2.9, respectively for control. All patients improved over time. No statistically significant differences between groups.
England, 1989 (78)	Rotator cuff tendinitis	Low-level laser therapy, placebo laser therapy, NSAIDs	RCT (n = 30), FU at 2 weeks	Pain, ROM, function	Active laser therapy significantly better than either placebo laser or NSAIDs. NSAIDs better than placebo laser therapy.
Schmitt, 2001 (79)	Rotator cuff tendinitis	Extracorporeal shock wave therapy, placebo treatment	RCT (n = 40), FU at 12 weeks	Pain, function	Improvement in pain from 5.4 (0–10 VAS) to 3.2 in the control group, from 5.4 to 2.3 in treatment group. No statistically significant difference between groups.
Speed, 2002 (80)	Rotator cuff tendinitis	Extracorporeal shock wave therapy, placebo treatment	RCT (n = 74), FU at 6 months	Pain, ROM disability index (SPADI)	Mean change in SPADI of 16.1 in the treatment group and 24.3 in the placebo group at 3 months. At 6 months the mean changes were 28.4 and 30.4, respectively. No significant differences between groups.

* SAD = subacromial decompression; US = ultrasound; SPADI = Shoulder Pain and Disability Index (see Table 2 for other definitions).

concomitant use of physical therapy and NSAIDs obtained satisfactory results in 67% of patients with impingement symptoms (40). Another study demonstrated improvement of symptoms in 59% of patients treated conservatively for full-thickness tears, whereas the symptoms worsened in 30% of patients (41).

Several studies (Table 3) have investigated the use of adjuvant therapies such as ultrasound, electrotherapy, or laser therapy, but these studies were unable to demonstrate a significant improvement over placebo. Extracorporeal shock wave therapy has been used successfully for the treatment of calcific rotator cuff tendi-

Table 4. Overview of outcomes of surgical interventions*

First author, year (ref.)	Condition treated	Treatment arms	Study design	Outcome measure	Results
Brox, 1999 (43)	Stage II impingement	Supervised PT for 3–6 months, arthroscopic SAD, placebo laser therapy	RCT (n = 125), FU at 2.5 years	Functional score	Successful outcome, defined as Neer score >80, in 68% (SAD), 61% (PT), and 16% (placebo) of patients. 22% and 50% of patients in PT and placebo groups, respectively, underwent SAD within 30 months.
Rahme, 1998 (81)	Impingement syndrome	Open SAD, supervised PT	RCT (n = 39), FU at 1 year	Pain score	Success, defined as 50% reduction in VAS pain score, in 57% of surgical patients vs. 33% of PT group at 6 months. Success after 1 year in 76% of surgical patients, while 13 of 18 initially conservatively treated patients had gone on to surgical intervention.
Miller, 2002 (47)	Partial-thickness rotator cuff tears	Tear <50% = débridement, tear >50% = repair	Retrospective (n = 39)	Functional score	Unsatisfactory results in 26% of débridement group vs. 12.5% of repair group.
Cofield, 2001 (53)	Full-thickness rotator cuff tears	Repair	Prospective (n = 105), FU at mean 13 years	Functional score	Successful outcome in 80%. Recurrent tear in 5 of 105 shoulders.

* See Tables 2 and 3 for definitions.

nitis; however, several studies investigating its use in noncalcific tendinitis were unable to demonstrate its efficacy (Table 3).

Operative treatment (Table 4). Most shoulder pain secondary to rotator cuff disease responds well to nonoperative, conservative measures. The dilemma for the practitioner is when to forego conservative treatment in favor of surgical intervention, especially in light of reports demonstrating more favorable outcomes with early surgical repair (42). Surgical decision-making should take into consideration the functional demands and comorbidities of the individual patient. The focus in younger patients should be on restoring anatomy and maximizing strength and function, whereas in older and lower-demand patients the goal is to minimize surgical risk and achieve pain relief, albeit with the realization that there will be more limited gains in strength and function. In general, absolute indications for surgical repair are the onset of acute, posttraumatic weakness in physiologically younger, active individuals without pre-existing rotator cuff dysfunction. Relative indications for surgery are pain or weakness that has been refractory to an appropriate course of nonoperative management, which is usually considered a period of 3–6 months. Although there is an abundance of evidence supporting various types of procedures, there are, unfortunately, few prospective randomized trials that compare surgical with nonsurgical interventions (43).

Impingement syndrome. Impingement-type symptoms of pain brought on by overhead activities, with preserved strength and ROM, can be treated by arthroscopic or mini-open subacromial decompression that involves removal of the thickened bursa, thus alleviating the compression that is the cause of chronic irritation and inflammation. In more advanced cases with associated partial- or full-thickness rotator cuff tears, a tendon débridement or repair is performed during the same procedure. This provides substantial pain relief and functional improvement in 75–86% of patients (44,45). Predictors of good outcome include shorter duration of symptoms and a positive result on the preoperative impingement test (45).

Partial-thickness rotator cuff tears. The choice of treatment for partial-thickness rotator cuff tears remains controversial. For many years, experts recommended repair of tears extending across more than 50% of the tendon substance, and simple débridement of lesser tears (46). However, recent studies comparing the outcomes of débridement versus repair of partial-thickness tears demonstrated significantly better results with repair (47,48). Furthermore, the long-term results of débridement alone seem to deteriorate with longer followup (49). This explains the current trend for repair of substantial partial-thickness tears unresponsive to medical treatment (47).

Full-thickness rotator cuff tears. The type of treatment for full-thickness tears depends on the extent of the tear, the tear pattern, and the appearance of the musculature on MRI. Although, traditionally, rotator cuff tears were repaired with open surgery, most tears can now be repaired arthroscopically (50), which decreases morbidity for the patients. Some larger or more complex tears still require open procedures. Between 77% and 98% of patients are satisfied with their outcome after rotator cuff repair, with excellent pain relief and functional improvement in more than 80% of patients (51–53).

Massive rotator cuff tears. Tears larger than 5 cm or tears affecting 2 tendons (usually the supra- and infraspinatus) are considered massive tears. These tears are often associated with retraction and fatty degeneration of the torn muscle. When there is significant atrophy of the muscle and fatty replacement, the tears are considered, by some authors, to be irreparable. When such tears are repaired, there may be some pain relief, but often ROM and strength are not fully restored (functionally irreparable tear). In addition, the poor surgical results, with rerupture rates of more than 50% (15), have led some experts to recommend only simple débridement for the treatment of massive cuff tears, which leads to satisfactory outcomes in 83% of cases (54). New techniques, however, allow for the reconstruction of massive rotator cuff tears that were previously believed to be irreparable. These techniques utilize advanced mobilization of retracted tendons, as well as the transfer of adjacent muscle-tendon units such as the teres major, pectoralis major, or latissimus dorsi (15,55,56). Most outcome studies of advanced techniques are limited by small sample size, but many have shown promising results that indicate significant improvements in pain and function in these previously untreatable conditions (55,57).

Revision rotator cuff repair. Primary rotator cuff repair is highly successful for the relief of pain and restoration of function. Persistent or recurrent pain and weakness are largely attributable to failure to heal or to tear recurrence. The incidence of revision for recurrent rupture closely correlates with initial tear size. It has been estimated that 5–6% of primary repairs of small-to-large tears are complicated by recurrent rupture (53,58). Interestingly, although long-term results in patients with recurrent ruptures are worse than those in patients with intact repairs, there is still a significant improvement when compared with the patients' preoperative function and pain (59). Not unexpectedly, outcomes after revision rotator cuff repair are worse than those after primary repair, with persistent weakness in more than 70% of cases. In spite of this, pain relief was achieved in the majority of patients (60,61).

Complications. Postoperative stiffness, defined by some authors as decreased ROM to <80% of that in the contralateral shoulder (62), occurs in 4% of cases (63). Perioperative antibiotic prophylaxis has decreased the rate of surgical wound infections to 1%. Deltoid muscle dysfunction due to intraoperative avulsions of the muscle insertion on the acromion or postoperative disruption of a repair occurs in 0.5%, and nerve damage occurs in 1% of cases (58).

Postoperative course. The postoperative course after rotator cuff repair depends on the location and extent of the tear and the strength of the repair. Partial-thickness tears are immobilized for a period of 1–2 weeks postoperatively, followed by a physical therapy regimen with quick progression from passive ROM to active-assisted ROM and then to active ROM exercises. After restoration of relatively pain-free ROM, gentle strengthening exercises can be started. Overall, patients can expect further improvement in pain and function over a course of 6 months. Larger tears have a less predictable clinical course. In most repairs, the patient will be restricted to passive ROM for 6 weeks to allow for tendon healing while preventing stiffness. After 6 weeks, therapy will progress to active and active-assisted ROM with strengthening at 10–12 weeks postoperatively. Although the exact timing of healing is unknown and is influenced by a variety of biologic and mechanical factors, animal studies have demonstrated sufficient repair strength at ~3 months (64). Complete recovery usually is achieved by 6–8 months postoperatively.

Summary

Rotator cuff disease is a frequent cause of shoulder pain and encompasses a spectrum of pathologic changes, ranging from tendinosis to subacromial impingement to partial- and full-thickness tears. Most rotator cuff injuries can be adequately diagnosed on the basis of a careful history review and physical examination, and respond well to conservative measures. The subset of individuals who experience acute onset of weakness, especially in the setting of trauma in younger patients, requires early diagnostic investigation to exclude the possibility of a significant rotator cuff tear. These patients should be referred to a shoulder specialist early on for potential surgical intervention.

Overall, studies have found satisfactory results of

nonoperative treatment in more than 50% of patients with full-thickness tears and in more than 70% of patients with impingement syndrome (40). Failures in the treatment of full-thickness tears were generally due to persistent weakness even in the face of substantial improvements in pain and motion (65,66). Prognostic factors for poor outcome are a tear size >3 cm, and duration of symptoms for longer than 6–12 months. Among those patients undergoing surgical repair, ~85% can expect substantial pain relief and at least partial restoration of strength (65).

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