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Physical Therapy Management of the Shoulder

ARTHUR J. NITZ

This article presents an overview of physical therapy management for patients with pathological musculoskeletal conditions of the shoulder. The importance of a thorough physical examination is stressed because proper treatment is based on this information. The primary goals of management should include the alleviation of acute inflammation and pain and the avoidance of subsequent soft tissue shortening. Medical and surgical treatments for common shoulder disorders also are discussed.

Key Words: Muscles, Physical therapy, Shoulder.

Shoulder pain and disability may arise in response to acute injuries, repeated microtrauma, or degenerative tissue alterations secondary to the normal aging process. A thorough medical history and neuromusculoskeletal examination of patients with complaints of shoulder pain should help to determine the anatomical site of the lesion (the type of tissue involved, whether it is contractile or inert) and the nature of the disability (acute or chronic). The patient's medical history and findings from a thorough examination must be interpreted correctly because the development of an appropriate management plan and the judicious choice of therapeutic procedures undertaken on the patient's behalf should be based firmly on this information.

In attempting to identify the specific tissue(s) involved in a particular lesion, clinicians must not discount the impact that disease in one structure in the shoulder complex may have on adjacent or functionally related tissues. For instance, if a primary condition such as bicipital tendinitis causes prolonged shoulder immobility, then a secondary effect, capsular fibrosis, may ensue. Recurrent injury may result if both the primary condition and the secondary effect are not recognized and treated by the clinician. Underlying postural abnormalities such as increased cervical lordosis, shortened pectoral muscle length, and increased thoracic kyphosis may precipitate additional shoulder stress and accelerate rotator cuff degeneration.

Properly planning a rehabilitation program requires further delineation of the extent of the disability. Although some confusion exists regarding the terms acute and chronic, these terms should be used to refer to the nature of the inflammatory condition and not to the length of time a pathological condition has existed. Acute inflammatory conditions are characterized by pain that is present at rest, diffuse in its distribution, and often referred from the site of the primary condition. Any passive movement restriction usually is the result of soft tissue impingement (e.g., impingement syndrome) and muscle guarding in response to pain. Tissue temperature classically increases in the acute phase, although this increase is difficult to detect in patients with pathological shoulder conditions because of the depth of the overlying muscle mass. Similarly, grossly visible edema accumulation, which accompanies many acute inflammatory conditions, often is not seen at the site of the lesion in patients with musculoskeletal injuries of the shoulder. Patients with chronic shoulder conditions, however, typically do not complain of pain at rest, although specific activities usually will elicit pain, particularly at the end of range of motion.

The hallmark sign of patients who have chronic inflammatory shoulder conditions is loss of ROM (active and passive), implying soft tissue shortening (capsular fibrosis or tendon shortening). Palpable tenderness may be noted in a discrete area although not always at the site of soft tissue shortening.

Determination of whether a patient's shoulder disorder represents an acute or chronic inflammatory condition is crucial to the specific treatment procedures used for each patient. Treatment of the patient with an acute disorder may be directed at alleviating the inflammation, reducing repeated irritation, and preventing subsequent undesirable tissue shortening. A significant corollary to treating these signs of disease rather than the symptoms is that the patient's typical chief complaint, pain, usually is reduced. The underlying causes of acute inflammation also must be established so that they may be eliminated as quickly as possible. For the patient with a chronic inflammatory disorder, the treatment plan should include a strategy for dealing with any existing soft tissue shortening that may have resulted in a loss of ROM. Additionally, muscles and ligaments that cross the region should be stressed to increase strength and to enhance appropriate tissue fiber orientation. Training that improves the patient's potential for returning to his usual level of functional activity also should be initiated as the signs of chronic inflammation diminish. The overall goal of treatment is to guide the patient through the acute or chronic inflammatory stages of a particular disorder until the preinjury level of functional activity is restored and recurrence of the pathological condition can be prevented.

In this article, I first discuss general treatment procedures of signs of pathological shoulder conditions and then outline the specific application of these techniques for several common shoulder disorders.

TREATMENT PROCEDURES

Procedures to Alleviate Inflammation

Immobilization. Rest from activity by joint immobilization is an integral part of the treatment for acute soft tissue and bony lesions of the shoulder. The purpose of such treatment is to allow the soft tissue inflammation to abate or to stabilize
fracture fragments and thereby prevent further trauma to the region. Excessive inflammation should be avoided because it may result in increased scar tissue formation (secondary to fibrin deposition) and eventual joint capsule contracture. Immobilization usually is discontinued when tissue healing is confirmed radiographically or by evidence of reduction in temperature, edema, and pain.

Although temporarily restricting joint motion is necessary so that tissue may heal, deleterious effects of these procedures are well documented. Articular cartilage has been reported to undergo biochemical changes at the light- and electron-microscopic levels during and after periods of therapeutic immobilization. The most common alterations include fraying, fibrillation, chondrocyte degeneration leading to cellular necrosis, and a reduction in staining capability of the extracellular ground material. Similar changes have been noted in aged tissue and may explain why the incidence of intra-articular lesions is somewhat more prevalent among the elderly population than younger persons. Such lesions also may account partly for the usual loss of motion over time among aged individuals. Although exercise can be initiated too quickly and undertaken too vigorously after prolonged immobilization, the potential for articular cartilage tissue changes suggests the need for early exercise.

Ligamentous tissue is known to lose tensile strength as a result of immobility and is offset only partially by prolonged rehabilitation. The precise site of ligamentous weakening secondary to immobilization has not been established definitely and may be dependent on age, but the most likely location is at the bone-ligament interface. Although the net effect of prolonged joint immobility may be capsular fibrosis, individual collagen fibers have demonstrated that they require reduced force for rupture; that is, they actually elongate more than fibers taken from exercised tissue. Pathologically weak ligamentous tissue, therefore, may result from periods of protracted joint immobilization. Age also has been shown to influence ligamentous tissue strength with a general reduction in resistance to tensile force observed in older tissue.

Immobilization also has adverse effects on muscle tissue. Muscle oxidative enzyme systems are known to decrease by 26% to 42% after prolonged immobilization. Muscle atrophy has been reported with a predilection for reduction in type I slow-twitch fiber diameter and volume. Isometric exercises performed during cast immobilization have been reported to reduce, but not eliminate, the loss of muscle strength and disuse muscle atrophy. Generalized loss of muscle mass also seems to occur with advancing age and may be a predisposing factor for the development of musculoskeletal injuries.

Heat and Cold. Cold-induced analgesia is the result of alterations in sensory fiber neurophysiological properties. Both luminous infrared radiation and moist hot packs have been shown to lower intra-articular joint temperature while simultaneously elevating the temperature in superficial soft tissues. Consequently, inflammation and ischemic pain in the deeper joint tissues are reduced with resultant analgesia after superficial local heat application. Both luminous infrared radiation and moist hot packs have been shown to lower intra-articular joint temperature while simultaneously elevating the temperature in superficial soft tissues. Consequently, inflammation and ischemic pain in the deeper joint tissues are reduced with resultant analgesia after superficial local heat application.

Transcutaneous electrical nerve stimulation. Transcutaneous electrical nerve stimulation is another modality that has been used widely for treating a variety of acute and chronic musculoskeletal conditions. Although TENS may be combined with various forms of treatment, it is distinguished from other physical agents in that, classically, TENS has been applied only for the purpose of pain relief. Recent reports, however, suggest that the absorption of calcific deposits in the shoulder muscle tendons is accelerated by low frequency TENS therapy and may be related to increased microcirculation in the region of the stimulation. Although controlled studies were identified to document those hypotheses, the most consistent and extensive pain relief appears to occur with stimulation of the acupuncture points thought to be associated with shoulder pain.

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Diathermy and ultrasound produce heat to a depth of 3 cm to 5 cm and probably are the two deep heating agents used most often in treating musculoskeletal disorders of the shoulder after acute inflammation has abated. Because diathermy can penetrate to a specific depth and can stimulate different tissues selectively, it is considered the most appropriate modality for heating muscle, but the joint capsule and intra-articular structures may benefit most from ultrasound application. Ultrasound is reported to be more effective than microwave or short wave diathermy in reducing pain because it causes a more vigorous rise in tissue temperature at the site of the lesion. Reduction in pain perception occurs because of a diminished propagation of motor and sensory nerve fiber impulses, resulting in decreased muscle fiber tension (relaxation) and less central nervous system stimulation.

Early mobilization. Codman's pendulum exercises commonly are initiated during or shortly after the periods of prolonged immobilization required for healing fractures or reducing soft tissue inflammation. Although no electromyographic data are available to substantiate their effectiveness, these exercises are intended to be a purely passive treatment of the shoulder. The effects may be reported...
to be beneficial because these exercises spread synovial fluid, stimulate tonically depressed joint mechanoreceptors, alleviate pain secondary to muscle ischemia, or prevent collagen cross-bridging. Experimental and clinical studies are needed to document the effects of such common methods of shoulder treatment.

**Procedures to Diminish Soft Tissue Shortening**

In synovial joints, the principal effect of inflammation and prolonged immobilization is a contracted joint capsule. A variety of treatment techniques are directed at alleviating this soft tissue shortening.

**Deep heating agents.** In addition to possibly resorbing calcium deposits, the application of ultrasound seems to increase connective tissue (joint capsule) extensibility by enhancing collagen fiber separation. Because, under tensile stress, connective tissue behaves like a viscoelastic material, optimal tissue extensibility can be realized by applying selective joint capsule heating simultaneously with continuous passive stretching of moderate force. After heat application, maintaining soft tissue tension (sustained passive stretch) for about 10 minutes during cooling helps to ensure that the plastic deformation achieved during the treatment will not be lost.

**Manual therapy: General concepts.** Orthopedic manual therapy has become an integral part of most treatment programs for patients with shoulder pain and is applied to increase accessory movements in an effort to improve joint mobility. Before initiating manual therapy, clinicians should determine whether restricted motion is the result of contractile or inert tissue shortening, select the mobilization grade, know the shape (convex or concave) of the joint surface to be moved, and determine whether the patient's chief dysfunction results from pain or stiffness. Because patients with complaints of shoulder pain usually do not have disorders characterized exclusively by pain or loss of motion, the therapist must be prepared to modify the treatment program to focus on both of these manifestations. In addition, the treatment should be altered as the patient's condition improves (ie, pain decreases and physiological movement increases), which demands regular patient reassessment. Additional fundamental and historical perspectives to manual therapy can be found in the reviews by Cookson and Kent and Barak et al.

For patients with pathological shoulder conditions, loss of motion is measured at the glenohumeral (GH) joint, but accessory movements may be reduced at the acromioclavicular (AC), sternoclavicular (SC), and scapulothoracic joint articulations, as well as at the GH joint itself. Therefore, a complete regimen of mobilization for patients with GH joint loss of motion should include techniques for all joints of the shoulder complex and the elbow joint. Most shoulder mobilization procedures have become fairly common and are detailed elsewhere.

**Specific joint mobilization: Rationale.** Sternoclavicular joint cranial-dorsal gliding may be required for patients with restricted scapular protraction and depression; caudal-ventral gliding might be limited at the SC joint when decreased scapular retraction and elevation are identified. Similarly, because GH joint flexion, abduction, and rotation are dependent on clavicular movement at both the sternoclavicular and acromial ends, AC joint gliding procedures (ventral and dorsal) may be indicated when restrictions in the GH joint motions are present. Mobilization procedures for the SC and AC joints usually are tolerated well by patients with acutely inflamed shoulder conditions because no irritating physiological GH joint motion is required. These techniques at the SC and AC joints should be maintained as long as their movement is restricted.

Freedom of movement at the scapulothoracic joint articulation is necessary for normal scapulohumeral rhythm. An examination of patients with shoulder musculoskeletal disorders having loss of scapulohumeral rhythm may reveal similarly restricted scapulothoracic movement requiring mobilization. The patient's scapula may be rotated on the posterior chest wall by the therapist in any restricted direction (elevation-depression, protration-retraction, upward rotation-downward rotation). The inferior angle of the scapula also may be tilted posteriorly away from the thoracic wall by the therapist in much the same manner that scapular "wining" occurs during GH joint medial (internal) rotation. Although some patients complain of discomfort during initial attempts to mobilize the scapulothoracic articulation, clinical experience indicates that many individuals with painful shoulder conditions experience a marked reduction in pain and can sleep undisturbed through the night shortly after these techniques are applied. Perhaps these results are from the effects of massage of the articular surfaces or modulation of the joint mechanoreceptor firing, although no experimental data were reported to document this possibility. Some patients with loss of scapulohumeral rhythm also may have excessive scapulothoracic movement (ie, rotator cuff tear). In such cases, scapular stabilization by manual or strapping techniques may be necessary to treat GH joint restrictions without increasing scapular hypermobility.

In addition to needing SC, AC, and scapulothoracic mobilization, patients with restricted GH joint motion also need manual therapy applied to the GH joint itself. No literature can be cited to support this contention but, in my experience, treatment techniques for GH joint restriction that permit the patient's arm to remain close to the side generally are tolerated well. The techniques seem to be quite effective in permitting pain-free GH joint motion when undertaken shortly after the inflammatory stage has abated. Examples of such procedures include inferior glide, anterior-posterior glide, and lateral (external) distraction of the GH joint.

After the pain is reduced, attempts to improve ROM by lengthening the joint capsule can become more vigorous and may begin to resemble more usual physiological motion. Dorsal glide of the humerus on the glenoid fossa is indicated in the presence of restricted flexion and medial rotation. A loss of extension and lateral rotation, however, suggests GH joint ventral gliding. With a loss of lateral rotation, the greater tuberosity of the humerus abuts against the acromion process during attempts to abduct the arm. Therefore, to avoid impinging the contiguous soft tissue during treatment, clinicians may find improving lateral rotation before moving the arm beyond 90 degrees of abduction to be beneficial. When the patient's pain diminishes and ROM is increased, additional treatment procedures that the patient can undertake at home may be emphasized, such as automobilization and passive ROM exer-
Friction massage. Another commonly used manual therapy technique is deep friction massage. Many clinicians have observed positive results in their patients after a course of treatment by friction massage; no clinical or experimental research data, however, are available to substantiate the reported benefits derived from this procedure. Proponents suggest that this form of treatment may be indicated for any chronic inflammatory shoulder condition in which abnormal modeling of fibrous tissue may be occurring. Supraspinatus tendinitis is a typical lesion for which friction massage has been advocated. Treatment objectives include preventing contracture and tissue hyperemia, believed to be caused by the release of histamine. Specific application techniques of friction massage are detailed in the work of Cyriax and Cyriax and discussed in the reviews by Chamberlain and Kessler and Hertling.

Orthopedic manual therapy has gained wide acceptance among physical therapists who treat shoulder conditions, maybe partly because of the anecdotal reports of those who use it. Unfortunately, however, my review of the literature revealed virtually no controlled studies to verify the efficacy or substantiate the claims of those who advocate these treatment procedures. Some investigators have reported finding Maitland’s mobilization techniques to be of no more benefit to the patient than steroid injection, cryotherapy, or withholding treatment. Further investigation of the efficacy of these procedures is needed.

Muscle Rehabilitation Procedures

After improved active and passive ROM have been established, exercises designed to increase muscular strength, power, and endurance may be begun. Initially, manual resistance by rhythmic stabilization and slow-reversal techniques for the axioscapular, axiohumeral, and scapulohumeral muscles may be used as the patient’s condition resolves. These exercises increase periscapular muscle strength and may lead to improved ROM. Increased strength also may result in enhanced joint protection and improved posture, thereby diminishing the possibilities of the shoulder disorder recurring. Isometric and isotonic shoulder exercises may be performed to improve muscle strength and endurance further and should be highly specific for each patient’s condition. Isokinetic equipment permits exercise at both slow and fast limb velocities and is reported to ensure a thorough recovery of functional activity. Recently, investigators have reported that high intensity electrical stimulation may improve muscle strength and endurance, but its application to shoulder disorders has not been documented in the literature.

Experimental Procedure for Tissue Healing

An experimental treatment modality, pulsed electromagnetic field (PEMF) therapy, has been used recently for patients with chronic rotator cuff tendinitis who do not respond to steroid injection or other forms of conventional conservative management. Peaked pulse electromagnetic field therapy is now being recognized as a valuable approach to the management of chronic inflammatory disorders of the shoulder. The precise mechanism of action for this modality is not known but is believed to be related to accelerated tissue healing. Many patients, however, found the treatment coils to be awkward and uncomfortable, and the lengthy duration of treatment (3–10 hours a day for 4–8 weeks) may be a disadvantage. Although the preliminary results are promising, the utility of this modality for patients with musculoskeletal disorders of the shoulder remains uncertain.

Medical Treatment

Administration of medication is an important aspect of medical treatment for most patients with pathological shoulder conditions and may include anti-inflammatory drugs and corticosteroid injection. Muscle relaxants are prescribed rarely for musculoskeletal disorders of the shoulder, and the use of narcotic analgesics should be kept to a minimum. A five-day course of phenylbutazone use (300–600 mg/d) often alleviates acute tissue inflammation, but continued use of this drug may lead to gastrointestinal tract and blood cell abnormalities. Patients with chronic inflammatory disorders may respond to nonsteroidal anti-inflammatory medications such as aspirin, although a recent report questions the efficacy of such agents. Some clinicians contend that subcutaneous or intra-articular injection of corticosteroids is the preferred treatment when oral anti-inflammatory drugs and other conservative measures do not achieve the desired patient response. Those clinicians who advocate this form of therapy indicate that success depends on the accuracy of the injection location and patient selection. The appropriateness of administering steroid injections in the conservative management of shoulder disorders has been discussed on a theoretical level and is supported by the results of recent clinical studies. Proponents of corticosteroid therapy, however, assert that multiple injections of these agents may be necessary before beneficial results are realized. Although systemic side effects from occasional corticosteroid injection are rare, local tissue alterations are common and clinically relevant. Complications such as loss of articular cartilage elasticity, depressed fibroblast protein synthesis, tissue degeneration at the cellular and ultrastructural levels, and vascular changes may be offset partially by exercise. A regimen of exercise, therefore, is believed to be an integral component of a total management program for the patient receiving steroid injections. Despite the claims of some investigators, most clinicians believe that corticosteroid administration should be limited to three or four injections a year and that indefinite series of injections are not recommended.

TREATMENT OF SELECTED COMMON SHOULDER DISORDERS

In the following sections, I will discuss briefly specific applications of these management principles for selected pathological shoulder conditions. Except for a few reports comparing various nonsurgical methods of treating adhesive capsulitis, controlled clinical studies that document or compare the efficacy of applying these treatment procedures specifically for shoulder disorders are rare. Because most studies have been unable to identify clearly etiological factors for these disease processes, a thorough discussion of potential underlying causes will not be undertaken.
Rotator Cuff Injuries

Rotator cuff tendinitis is thought by many to be the single most common cause of shoulder pain with the supraspinatus portion of the cuff most frequently involved.66,120-122 Calcific deposits in the tendon are common and, although not necessarily symptomatic, are often the cause of severe, incapacitating shoulder pain.113,115,117,123 Treatment during the acutely painful stage consists of rest (sling), TENS, and cryotherapy.66,67,120 Initial reports do suggest that low frequency TENS is more effective in eliminating calcium deposits in the rotator cuff muscles of patients with chronic tendinitis than spontaneous remission without treatment.52,124 Needling of the calcific portion of the tendon with multiple puncture sites in the acutely painful shoulder is reported to promote hyperemia and resorption of the calcium deposition,125 but no controlled study documenting the effectiveness of this method exists in the literature. Most physicians also advocate the injection of various combinations of local anesthetics in steroid suspension, although one clinician considered acutely painful tendinitis with calcium deposits to be a surgical emergency.126 As soon as pain is alleviated, gentle passive and active exercises may be initiated to prevent contracture, but mobilization should not be necessary unless loss of accessory motion has occurred. Deep heating modalities may be used, but not during the acute inflammatory stage.44 Transverse friction massage and combined stretching and strengthening of the involved rotator cuff muscles may be indicated when acute symptoms have abated, and these methods are thought by some clinicians to be instrumental in preventing soft tissue shortening in patients with rotator cuff tendinitis.8 For the estimated 10% of the patients who do not respond to nonsurgical methods of treatment, surgical excision of any calcific deposit may be necessary and usually is followed by pendulum exercises performed early and a comprehensive rehabilitation program initiated at three weeks after surgery.3,120,121

Rotator cuff degeneration may lead to a complete tear in response to minor repetitive activities.66 Many of these tears remain quiescent,127,128 but chronic cases often become symptomatic.127 If the symptomatic rotator cuff tear is partial, then a period of nonoperative management is indicated including rest, cryotherapy, and steroid injection followed by exercise.66,127,129-131 Should these measures fail to bring resolution of the symptoms, however, various surgical approaches are advocated.132-134 Most orthopedists agree that complete, massive rotator cuff tears necessitate surgical intervention as soon after the injury as possible. This repair then is followed by three to four weeks of immobilization.67,127,130,133 As a result of chronicity or prolonged immobilization, some patients with rotator cuff tears may demonstrate substantial loss of both grossly measurable movement and accessory motions. A rehabilitation program for such patients, therefore, should include the appropriate joint mobilization procedures in addition to palliative measures such as TENS and ultrasound.52,62

A postoperative muscle strengthening program is recommended strongly by many physicians but, beyond the general goal of restoring function, no specific objectives of such a regimen have been outlined in the literature.121,130,132 A change in vocation from heavy labor to less strenuous work activities is a common adjustment the patient has to make.130

Bicipital Tendinitis

Another common shoulder disorder among young or middle-aged individuals is bicipital tendinitis, which often is characterized by a painful arc, arthritis,3,120 and tenderness to palpation at the bicipital groove.3,66,120,122 The proximity of the rotator cuff tendons to the long head of the biceps brachii muscle has led Neviaser to conclude that this condition rarely occurs in isolation but, rather, concomitantly with rotator cuff tendinitis.3,122 Other investigators, however, reported that only 40% of 93 patients undergoing surgical procedures for bicipital tendinitis were found to have accompanying rotator cuff attrition or rupture.135 Avoidance of the activity that causes the condition to develop usually is combined with other nonoperative measures during the acute phase (TENS, cryotherapy, nonsteroidal anti-inflammatory agents, and steroid injection).3,66,120,122 Deep friction massage may be useful over the bicipital groove but may exacerbate acutely or chronically inflamed tissue. Although pain may limit the extremes of shoulder ROM (especially abduction and medial and lateral rotation), no true capsular contracture is believed to exist, so joint mobilization techniques may be unnecessary. Because this disorder usually occurs in response to repetitive upper extremity vocational and recreational activity,3,120,122 bicipital tendinitis often is recurrent and consequently has been treated with a variety of surgical procedures.3,122,136,137 Simon states that some investigators claim that 90% of patients treated operatively for bicipital tendinitis experience short-term relief of pain.120 No long-term follow-up studies of patients who have had operative procedures for bicipital tendinitis, however, were identified in the literature. Because muscle tendon and joint capsule tissue adaptation occurs as a result of postoperative immobilization, muscle stretching and orthopedic manual therapy procedures usually are required.

Subdeltoid Bursitis

The term bursitis often is misused66 and, as a primary condition, it is thought to be present only in patients with rheumatoid arthritis, tuberculosis, gout, and pyogenic infections.122 The intimate relationship between the subdeltoid bursa and underlying rotator cuff permits an extruding calcium salt mass (calcific tendinitis) to irritate and eventually rupture spontaneously into the bursal sac.3,6,6,120,138-140 This event produces the acute, disabling pain of bursitis that often results in sleep disturbance and voluntary spinting of the shoulder. During this stage, attempts to reduce the tissue inflammation and pain include sling immobilization of the affected extremity, ice application, and medication. Because the usual doses of narcotic medication often are ineffective in controlling acute bursitis pain, needling decompression of the engorged bursa followed by irrigation and subsequent corticosteroid injection is advocated by some clinicians.120,141 A review of the literature, however, revealed no controlled clinical investigations to substantiate the efficacy of this treatment. Fortunately, the severe pain of bursitis is short-lived (2–3 days) and may be treated with active and passive ROM exercises as soon as two days after onset.78 Kessler and Hertling state that, when movement is initiated early in the patient's treatment program, loss of motion rarely occurs.66,140 Gentlen mobilization has been advocated during the acute phase of bursitis for its purported muscle-relaxing effects and consequent improvement in muscle capillary filling.71 Use of ultrasound during the subacute phase of bursitis is thought by some clinicians to be helpful particularly in inducing local

§ 4(p302),92(p19,20),121.
hyperemia and facilitating absorption of the calcium deposit.²⁷ A controlled study, however, indicated that ultrasound had no essential effect on the resolution of the deposit.¹⁴² In addition, a recent report of a double blind investigation suggested that ultrasound produced no significant difference in pain, ROM, or function in patients with subacromial bursitis.¹⁴³ Because subdeltoid bursitis usually occurs as a result of calcific tendinitis, preventive measures may include patient instruction to avoid shoulder movements that might have led to the primary condition.³

Acromioclavicular Joint Degeneration

Degenerative changes of the AC joint are much more common than of the GH joint but often are overlooked⁶⁶ by clinicians seeking the cause of pain in patients with shoulder disorders. Meticulous palpation often will reveal tenderness at the superior and anterior aspects of the joint and painful crepitus with passive dorsal-ventral joint gliding, especially in patients over the age of 50 years.⁶⁶ Intra-articular injection of a local analgesic in steroid suspension is thought by some clinicians to be the preferred treatment for signs that result from AC joint degeneration,⁶⁶⁶⁶ although use of moist heat, ultrasound applications, and a short period of sling immobilization also have been reported.⁸⁶ Depth of heat penetration with moist heat (hot pack) application has not been shown to exceed 1 cm¹⁴⁴ and, therefore, probably has little impact on intra-articular tissues. Ultrasound and diathermy, however, may increase tissue temperatures sufficiently to cause collagen destruction and an increase in intra-articular inflammation.⁴⁴ Because limited GH joint motion may lead to increased compensatory AC joint motion and subsequent degeneration,¹⁴⁵ one case report has suggested treating AC joint arthritis with GH joint mobilization.¹⁴⁶ The literature, however, does not appear to contain any controlled studies that clearly substantiate the effectiveness of any single nonoperative management approach for this condition. Acromioclavicular joint arthroplasty (Mumford procedure) has been recommended by some clinicians when shoulder pain arising from AC joint degeneration does not abate with the previously mentioned treatments.¹⁴⁵,¹⁴⁶ Prolonged immobilization after surgery usually leads to soft tissue shortening; therefore, the postoperative treatment program should include those shoulder immobilization procedures appropriate for the diagnosed joint restrictions.

Glenohumeral Joint Dislocation

The redundant capsule, extreme mobility, and relative absence of bony stability render the GH joint particularly susceptible to dislocation.⁶⁶ Fully 95% of the cases of GH joint dislocation occur in an anterior-inferior direction.⁶⁶ This type of dislocation is most common among active men between 20 and 50 years of age.⁶⁶ If spontaneous reduction, or self-reduction, does not occur, the shoulder may be manipulated by the Hippocratic, Kocher, Stimson, or Milch methods using adequate analgesics or anesthetics.¹⁴⁸ Relocation usually is followed by a period of immobilization (GH joint adduction, medial rotation), the duration of which is dependent on the patient's age.¹⁴⁸-¹⁵⁰ A one-week period of immobilization appears to be adequate in patients over 30 years of age because the incidence of recurrent dislocation is low in this age group.¹⁵⁰ Three to four weeks of immobilization is recommended for younger individuals.¹⁵⁰ Rigid restriction of activities and strict adherence to an aggressive muscle rehabilita-

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