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Frozen Shoulder: Evidence and a Proposed Model Guiding Rehabilitation

Frozen shoulder, or adhesive capsulitis, describes the common shoulder condition characterized by painful and limited active and passive range of motion (ROM). Frozen shoulder is reported to affect 2% to 5% of the general population,[4,13,64,88] increasing to 10% to 38% in patients with diabetes and thyroid disease.[4,5,13,64,71,88] Individuals with primary frozen shoulder are commonly between 40 and 65 years old,[79,82,83] and the incidence appears higher in females than males.[4,9,43,64,71,109] The occurrence of frozen shoulder in 1 shoulder increases the risk of contralateral shoulder involvement by 5% to 34%, and simultaneous bilateral shoulder involvement occurs as often as 14% of the time.[16,39,64,107]

To date, the etiology of frozen shoulder remains unclear; however, patients typically demonstrate a characteristic history, clinical presentation, and recovery. Codman[22] described frozen shoulder as "a condition difficult to define, difficult to treat, and difficult to explain from the point of view of pathology." Nevaiser[80] introduced the term *adhesive capsulitis* to describe the inflamed and fibrotic condition of the capsuloligamentous tissue. The term *frozen shoulder* will be used, because it encompasses both primary frozen shoulder (adhesive capsulitis) and secondary frozen shoulder related to systemic disease and extrinsic or intrinsic factors, excluding cerebral vascular accident, proximal humeral fracture, and causative rotator cuff or labral pathology. This paper will present an overview of the classification, etiology, pathology, examination, and plan of care for frozen shoulder.

CLASSIFICATION

THE ABSENCE OF STANDARDIZED nomenclature for frozen shoulder causes confusion in the literature. Lundberg[64] first described a classification system identifying primary frozen shoulder as idiopathic and secondary frozen shoulder as posttraumatic. Nash and Hazelman[77] expanded the classification system by including diseases such as diabetes mellitus, myocardial infarction, or various neurologic disorders under secondary frozen shoulder. Zuckerman[128] proposed a classification schema where primary

● SYNOPSIS: Frozen shoulder or adhesive capsulitis describes the common shoulder condition characterized by painful and limited active and passive range of motion. The etiology of frozen shoulder remains unclear; however, patients typically demonstrate a characteristic history, clinical presentation, and recovery. A classification schema is described, in which primary frozen shoulder and idiopathic adhesive capsulitis are considered identical and not associated with a systemic condition or history of injury. Secondary frozen shoulder is defined by 3 subcategories: systemic, extrinsic, and intrinsic. We also propose another classification system based on the patient's irritability level (low, moderate, and high), that we believe is helpful when making clinical decisions regarding rehabilitation intervention. Nonoperative interventions include patient education, modalities, stretching exercises, joint mobilization, and corticosteroid injections. Glenohumeral intra-articular corticosteroid injections, exercise, and joint mobilization all result in improved short- and long-term 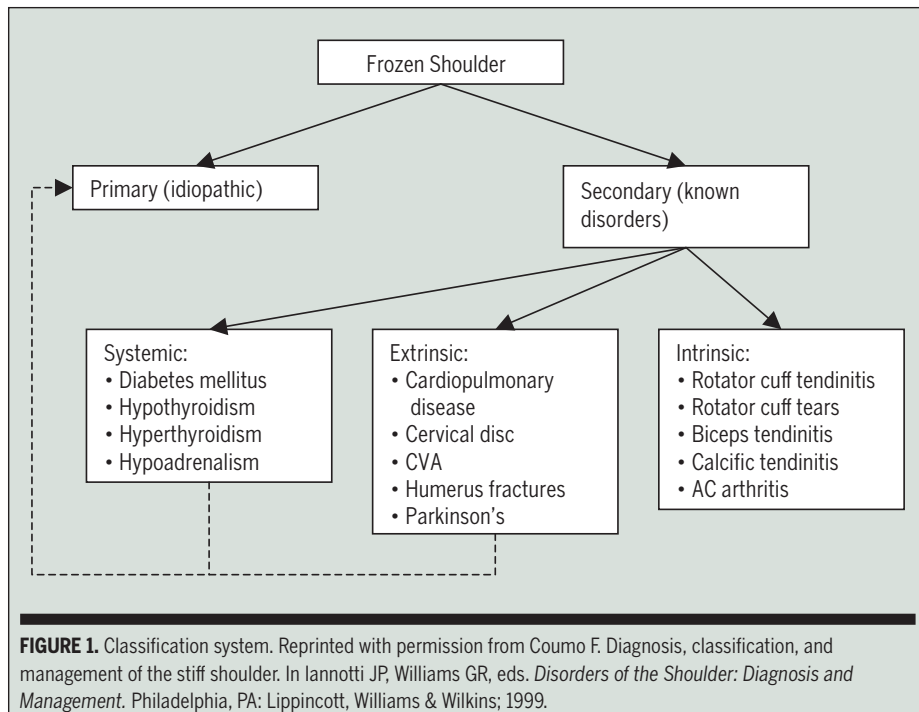outcomes. However, there is strong evidence that glenohumeral intra-articular corticosteroid injections have a significantly greater 4- to 6-week beneficial effect compared to other forms of treatment. A rehabilitation model based on evidence and intervention strategies matched with irritability levels is proposed. Exercise and manual techniques are progressed as the patient's irritability reduces. Response to treatment is based on significant pain relief, improved satisfaction, and return of functional motion. Patients who do not respond or worsen should be referred for an intra-articular corticosteroid injection. Patients who have recalcitrant symptoms and disabling pain may respond to either standard or translational manipulation under anesthesia or arthroscopic release.

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FIGURE 1. Classification system. Reprinted with permission from Coumo F. Diagnosis, classification, and management of the stiff shoulder. In Iannotti JP, Williams GR, eds. *Disorders of the Shoulder: Diagnosis and Management.* Philadelphia, PA: Lippincott, Williams & Wilkins; 1999.

TABLE 1	Irritability Classification		
High Irritability	**Moderate Irritability**	**Low Irritability**	
High pain (≥7/10)	Moderate pain (4-6/10)	Low pain (≤3/10)	
Consistent night or resting pain	Intermittent night or resting pain	No resting or night pain	
High disability on DASH, ASES, PSS	Moderate disability on DASH, ASES, PSS	Low disability on DASH, ASES, PSS	
Pain prior to end ROM	Pain at end ROM	Minimal pain at end ROM with overpressure	
AROM less than PROM, secondary to pain	AROM similar to PROM	AROM same as PROM	

Abbreviations: AAROM, active assisted range of motion; AROM, active range of motion; ASES, American Shoulder and Elbow Surgeons Score; DASH, Disabilities of the Arm, Shoulder and Hand Questionnaire; PROM, passive range of motion; PSS, Penn Shoulder Score; ROM, range of motion.

frozen shoulder and idiopathic adhesive capsulitis are considered identical and not associated with a systemic condition or history of injury.[128] Secondary frozen shoulder was defined by 3 subcategories: systemic, extrinsic, and intrinsic (**FIGURE 1**).[128] The 3 subcategories for secondary frozen shoulder identify a relationship between some disease process and shoulder symptoms. Systemic secondary frozen shoulder is more common among these patients, due to the related underlying systemic connective tissue disease processes.[13,14,88] Extrinsic secondary frozen shoulder includes patients whose pathology is not directly related to the shoulder, and intrinsic secondary frozen shoulder describes patients with a known pathology of the glenohumeral joint soft tissues or structures. Specific causes of secondary frozen shoulder may influence prognosis. For instance, individuals with secondary frozen shoulder related to insulin-dependent diabetes are more likely to have a more protracted and difficult clinical course.[85,86,88]

We also propose another classification system based on the patient's irritability level (low, moderate, and high), that we believe is helpful when making clinical decisions regarding rehabilitation intervention (**TABLE 1**). Irritability is determined based on pain, range of motion (ROM), and extent of disability. Patients with low irritability have less pain and have capsular end feels with little or no pain; therefore, active and passive motion are equal and disability lower. These patients typically report stiffness rather than pain as a chief complaint. Patients with high irritability have significant pain resulting in limited passive motion (due to muscle guarding) and greater disability. These patients typically report pain rather than stiffness as a chief complaint. While these criteria are not time based, most commonly, patients in early-stage frozen shoulder have a high level of irritability, while patients in later stages have low irritability.

ETIOLOGY AND PATHOLOGY

THE BENEFIT OF ZUCKERMAN'S CLASsification system is that it organizes the following previously described possible etiologies of frozen shoulder into subcategories: rotator cuff contracture,[69] biceps tenosynovitis,[27] subscapularis trigger points,[31,113] autoimmune response,[16,17] and autonomic reflex dysfunction.[100] Although the precise etiology remains unclear, recent evidence identifies elevated serum cytokine levels as part of the process.[19,49,101] Cytokines and other growth factors facilitate tissue repair and remodeling as part of the inflammatory process. Elevated cytokine levels appear predominately involved in the cellular mechanisms of sustained inflammation and fibrosis in primary and some secondary frozen shoulder.[19,49,75,101] Although the initial stimulus is unknown, Bunker et al[19] postulated that a minor insult could initiate an inflammatory healing response leading to excess accumulation and propagation of fibroblasts releasing type I and type III collagen. Fibroblasts differentiate into myofibroblasts, causing contraction of newly laid-down type III collagen. He proposed an imbalance between aggressive fibrosis and a loss of normal collagenous remodeling may lead to protracted stiffening of the capsule

TABLE 2	Stages of Adhesive Capsulitis*

Stage 1

Duration of symptoms: 0 to 3 months

Pain with active and passive ROM

Limitation of forward flexion, abduction, internal rotation, external rotation

Examination with the patient under anesthesia: normal or minimal loss of ROM

Arthroscopy: diffuse glenohumeral synovitis, often most pronounced in the anterosuperior capsule

Pathologic changes: hypertrophic, hypervascular synovitis, rare inflammatory cell infiltrates, normal underlying capsule

Stage 2: Freezing Stage

Duration of symptoms: 3 to 9 months

Chronic pain with active and passive ROM

Significant limitation of forward flexion, abduction, internal rotation, external rotation

Examination with the patient under anesthesia: ROM essentially identical to ROM when patient is awake

Arthroscopy: diffuse pedunculated synovitis (tight capsule with rubbery or dense feel on insertion of arthroscope)

Pathologic changes: hypertrophic, hypervascular synovitis with perivascular and subsynovial scar, fibroplasias and scar formation in the underlying capsule

Stage 3: Frozen Stage

Duration of symptoms: 9 to 15 months

Minimal pain except at end ROM

Significant limitation of ROM with rigid end feel

Examination with the patient under anesthesia: ROM identical to ROM when patient is awake

Arthroscopy: no hypervascularity seen, remnants of fibrotic synovium can be seen. The capsule feels thick in insertion of the arthroscope and there is a diminished capsular volume

Pathologic changes: "burned-out" synovitis without significant hypertrophy or hypervascularity. Underlying capsule shows dense scar formation

Stage 4: Thawing Phase

Duration of symptoms: 15 to 24 months

Minimal pain

Progressive improvement in ROM

Examination under anesthesia data not available

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and ligaments.[19] Using new histological and immunocytochemical analysis techniques, Hand et al[42] found that patients with frozen shoulder had both chronic inflammatory cells and fibroblast cells, indicating both an inflammatory process and fibrosis.

Frozen shoulder is typically considered an inflammatory process; however, this concept is being challenged. No significant inflammatory cells in the capsular tissue have been identified upon histological examination.[18,19,64,115] Numerous investigators report the visual presence of synovitis consistent with inflammation,[43,79,83,123] yet focal vascularity and synovial angiogenesis (increased papillary growth), rather than synovitis, are described by others.[18,50,124] In addition to confirmation of angiogenesis, frequent positive staining for nerve cells was found in patients with frozen shoulder.[42] However, if the synovial pathology is angiogenesis or synovitis, there is agreement that pain accompanies the change. Clinically, the idea that frozen shoulder occurs in the absence of inflammation is difficult to accept, especially because corticosteroid injections have been shown to have such a significant positive short-term effect.[3,15,20,58,104,108,117]

There is little disagreement regarding significant capsuloligamentous complex (CLC) fibrosis and contracture, which are consistently found in open or arthroscopic shoulder surgery and histologic examinations in patients with frozen shoulder.[80,115] Contracture of the rotator cuff interval (RCI) is prevalent in patients with frozen shoulder.[50,78,86,87,115,116,124] The RCI forms the triangular-shaped tissue between the anterior supraspinatus tendon edge and upper subscapularis border, and includes the superior glenohumeral ligament and the coracohumeral ligament. The interval acts as an anterior-superior hammock, restricting external rotation with the arm at the side and preventing inferior translation.[94] Imbrication of the RCI resulted in a 50% loss of external rotation with the arm at the side,[45] and RCI release in patients with frozen shoulder leads to an immediate and dramatic increase in shoulder external rotation ROM.[43,78,86,87] Others have noted significant subacromial scarring,[50,80] loss of the subscapular recess,[64,81] and inflammation of the long head of the biceps tendon and its synovial sheath[123] in patients with frozen shoulder. Clinicians attempting to regain shoulder external rotation should perform stretching and joint mobilization techniques to target the RCI as well as the anterior CLC.

Reeves[96] elaborated on the natural history of frozen shoulder and distinguished 3 sequential stages: the painful stage, the stiff stage, and the recovery stage. Hannafin and Chiaia[43] described 4 stages incorporating the arthroscopic stages described by Nevaiser,[83] the clinical examination, and the histologic findings (**TABLE 2**). Stage 1, the preadhesive stage, demonstrates mild erythematous synovitis. Patients present with mild end-range pain and are often misdiagnosed as having rotator cuff impingement. Stage 2, the acute adhesive or "freezing" stage, is characterized by a thickened red synovitis. Patients frequently have a high level of discomfort and a high level of pain near end-range of movement. Even though this phase is represented by pain, examination under anesthesia reveals connective tissue changes resulting in loss of motion. Stage

3, the fibrotic or "frozen" stage, is characterized by less synovitis but more mature adhesions. Patients note significant stiffness with less pain. These patients have motion limited by established contracture as opposed to pain based on examination under anesthesia, which reveals equal passive motion compared to when awake. Severe capsular restriction without apparent synovitis defines stage 4, the "thawing" phase. Patients in this phase present with painless stiffness and motion that typically improves by remodeling.

Arthroscopic staging clarifies the continuum of frozen shoulder and, although initially considered a 12- to 18-month self-limited process, mild symptoms may persist for years, depending on the extent of fibroplasia and subsequent resorption.[15,21,22,39,41,107] Authors report motion restrictions in 90% of patients at 6 months[15] and up to 50% of patients at greater than 3 years.[8,21,107] Mild symptoms persisted in 27% to 50% of patients at an average of 22 months to 7 years.[39,107]

EXAMINATION

History

ALTHOUGH SPECIFIC DIAGNOSTIC criteria do not exist, patients with primary frozen shoulder demonstrate a consistent history and clinical examination (**TABLE 3**).[80,83,96] Primary frozen shoulder and some secondary frozen shoulder (eg, secondary to diabetes mellitus), is characterized by an insidious onset, a progressive increase in pain, and gradual loss of motion. A minor traumatic event may coincide with the patient's first recognition of symptoms. Pain, specifically sleep disturbing night pain, frequently motivates the patient to seek medical advice. Most patients are comfortable with the arm at the side or with mid-range activities, but often describe a sudden, transient, excruciating pain with abrupt or end-range movements.

Three specific factors from the history may be useful in determining the stage or irritability level of the patient's condition. First, the ability to sleep through the night indicates less irritability. It also indicates that the painful synovitis/angiogenesis is resolving as consistent with stage 3. The second factor is whether pain or stiffness is the predominant symptom. The patient experiencing more stiffness than pain likely has less symptomatic synovitis/angiogenesis and more fibrosis. The third factor is whether the symptoms have been improving or worsening over the last 3 weeks. Improving symptoms may indicate that the patient is advancing from stage 2 into stage 3, and that the irritability level is decreasing. Recognizing the extent of tissue irritability has a direct influence on the plan of care.

Physical Examination

A full upper-quarter examination is performed to rule out cervical spine and neurological pathologies. With frozen shoulder, the examination of the shoulder typically reveals significant limitation of both active and passive elevation, usually less than 120°[18,80,95,100]; but motion limitations are stage dependent. Scapular substitution frequently accompanies active shoulder motion.[103,120] Passive motions should be assessed supine to appreciate the quality of the resistance to motion at the end of passive movement (end feel). Frequently, passive glenohumeral motions are very restricted due to pain at or before end range, and muscle guarding can often be appreciated at end range. We believe that muscle guarding can masquerade as a capsular end feel. The first author has had the opportunity to examine 6 patients prior to manipulation, both preanesthesia and postanesthesia.

All were felt to have a capsular end feel while awake, yet 5 of 6 patients had an increase in passive motion of 10° to 30° when anesthetized. Partial improvement in motion related to diminished pain, and muscle guarding has been reported after local or regional anesthetic.[109]

Cyriax[24] described a capsular pattern he believed diagnostic for adhesive capsulitis. The capsular pattern is defined as greater limitation of external rotation than abduction and less-limited internal rotation. Although the capsular pattern is often encountered, it is not consistently seen in patients with frozen shoulder when objectively measured.[103] A greater than 50% reduction in passive external rotation or less than 30° of external rotation, when measured with the arm at the side, is a common finding in individuals with frozen shoulder.[8,15,18,24,39,78,96,100,107,119,121]

Although authors of textbooks have described patients with frozen shoulder as having normal strength and painless resisted motions,[24] authors of recent studies, using handheld dynamometry, have revealed significant weakness of the shoulder internal rotators[53,59] and elevators[53,59,111] in these patients. The shoulder internal rotators were significantly weaker in patients with frozen shoulder compared to patients with rotator cuff tendinopathy; however, significant weakness of the external rotators and abductors was also found relative to the uninvolved side.[59] Special tests, such as impingement signs and Jobe's test, are not helpful in differentiating frozen shoulder from rotator cuff tendinopathy because they require painful end-range positioning.

TABLE 3	CHARACTERISTIC OF PRIMARY FROZEN SHOULDER
Patient age, 40-65 years	
Insidious or minimal, event resulting in onset	
Significant night pain	
Significant limitations of active and passive shoulder motion in more than 1 plane	
50% or greater than 30° loss of passive external rotation	
All end ranges painful	
Significant pain and/or weakness of the internal rotators	

Significant loss of passive external rotation with the arm at the side, as well as loss of active and passive motion in other planes of movement, differentiates frozen shoulder from other pathologies. However, other pathologies resulting in significant loss of external rotation with the arm at the side include proximal humeral fracture, severe osteoarthritis, acute calcific bursitis/tendinitis, and a locked posterior dislocation. Early frozen shoulder may be difficult to differentiate from rotator cuff tendinopathy because motion may be minimally restricted and strength testing may be normal. The patient with a slight loss of passive external rotation motion at the side and relatively full motion in all other directions should be cautioned to return for further evaluation if the patient experiences a rapid progression of shoulder pain and stiffness.

Diagnosing frozen shoulder is often achieved by physical examination alone, but imaging studies can further confirm the diagnosis and rule out underlying pathology. Radiography rules out pathology to the osseous structures. Arthrography has been used to determine decreased glenohumeral joint volume associated with adhesive capsulitis.[81,118,120] Although Binder et al[9] observed that over 90% of patients with frozen shoulder demonstrated an increased uptake on the diphosphonate scans (bone scan), they concluded bone scans possess little diagnostic or prognostic value for frozen shoulder.[9] Magnetic resonance imaging (MRI) helps with the differential diagnosis by identifying soft tissue abnormalities of the rotator cuff and labrum.[56] MRI has identified abnormalities of the capsule and RCI in patients with frozen shoulder.[56,70] Recently, ultrasonography has gained favor because it can help differentiating rotator cuff tendinopathy from frozen shoulder. A recent study revealed fibrovascular inflammatory soft tissue changes in the RCI in 100% of 30 patients with frozen shoulder.[57]

A comprehensive history and examination should include a patient-oriented shoulder functional outcome measure.

Multiple shoulder-specific outcome measures are available, such as the Disabilities of the Arm, Shoulder, and Hand Questionnaire (DASH),[68] Simple Shoulder Test (SST),[62] Penn Shoulder Score,[60] American Shoulder and Elbow Surgeons (ASES) score,[98] and the Constant-Murley score.[23] These forms typically include questions relative to the patient's pain and function and some include impairment data, such as ROM and strength measurements. To date, research has not identified a specific outcome tool or specified score range that is optimal for individuals with frozen shoulder.

NONOPERATIVE INTERVENTIONS

THE DEFINITIVE TREATMENT FOR frozen shoulder remains unclear even though multiple interventions have been studied including oral medications,[10,32,97] corticosteroid injections,[3,8,20,25,46,58,95,104,117] exercise,[3,15,20,25,28,39,46,55,58,72,95] joint mobilization,[15,52,84,118,119,127] distension,[37,73] acupuncture,[114] manipulation,[30,44,47,48,76,89,102] nerve blocks,[26] and surgery.[1,7,38,43,50,85-87,90,93,106,123,124] Unfortunately, varied inclusion criteria, different treatment protocols, and various outcome assessments render study comparison difficult. One of the major difficulties in assessing efficacy is success criterion. Often success is defined by return of "normal" motion rather than pain-free functional motion. It may be implausible for conservative treatment to rapidly restore full pain-free motion, considering the presence of dense fibrotic CLC tissue and the months of collagen remodeling required to regain soft tissue length. Even if an intra-articular corticosteroid injection relieves pain in someone with stage 3 frozen shoulder, the fibrotic/contractured tissue continues to limit motion. Establishing treatment effectiveness is also difficult because the majority of patients with frozen shoulder significantly improve in approximately 1 year; therefore, natural history must be considered.

Although multiple studies demonstrate improved outcomes with physical therapy, these outcomes are not always superior to other interventions.[3,15,20,46,117] Additionally, the optimal use of common physical therapy interventions (modalities, exercise, joint mobilization), frequency and timing of visits, and discharge criteria have not been established. The proposed physiologic effect and supporting literature for using modalities, exercises, and manual techniques in physical therapy will be discussed in the following sections.

Patient Education
Patient education about the natural history of frozen shoulder is probably an important treatment aspect, though no studies have specifically addressed this component. Explaining the insidious nature of frozen shoulder allays the patient's fear of more serious diseases. Discussing how the painful synovitis/angiogenesis progresses into fibroplasia and restricts motion prepares the patient for an extended recovery. Instruction in performing a consistent home exercise program (HEP) is important, because daily exercise is effective in relieving symptoms.[15,20,55]

Modalities
Little data exist to supporting the use of frequently employed modalities such as heat, ice, ultrasound, or electric stimulation. Modalities are suggested to influence pain and muscle relaxation; therefore, they might enhance the effect of exercises and manual techniques. Hot packs can be applied before or during ROM exercises. Application of moist heat in conjunction with stretching has been shown to improve muscle extensibility.[51] This may occur by a reduction of muscle viscosity and neuromuscular-mediated relaxation.[105,121] Gursel et al[40] demonstrated the lack of efficacy of ultrasound, as compared to sham ultrasound, in treating shoulder soft tissue disorders. Transcutaneous electrical nerve stimulation (TENS), together with a prolonged low-load stretch, resulted

in less pain and improved motion in patients with frozen shoulder.[99]

Stretching Exercise

The basic strategy in treating structural stiffness is to apply appropriate tissue stress.[74] It is helpful to think of the total amount of stress being applied as the "dosage," in much the same way that dosage applies to medication. The primary factors that guide this process are pain and ROM. Adjusting the dose of tissue stress results in the desired therapeutic change (increased motion without increased pain). Three factors should be considered when calculating the dose, or total amount of stress delivered, to a tissue: intensity, frequency, and duration. The total end range time (TERT)[34,66] is the total amount of time the joint is held at or near end-range position. TERT is calculated by multiplying the frequency and duration of the time spent at end range daily, and is a useful way of measuring the dose of tissue stress.[34,66] Intensity remains an important factor in tensile stress dose but is typically limited by pain. Traditional ROM exercises are considered lower forms of tensile stress, while the highest tensile stress doses are achieved by low-load prolonged stretching (LLPS), because TERT is maximized. Therefore, the goal with each patient is to determine the therapeutic level of tensile stress.

Applying the correct tensile-stress dose is based upon the patient's irritability classification (**TABLE 4**). In patients with high irritability, low-intensity and short-duration ROM exercises are performed to simply alter the joint receptors' input, reduce pain, decrease muscle guarding, and increase motion.[126] **FIGURES 2 AND 3** show commonly performed exercises for patients with high irritability. Stretches may be held from 1 to 5 seconds at the relatively pain-free range, 2 to 3 times a day. A pulley may be used, depending on the patient's ability to tolerate the exercise. These exercises primarily influence different regions of the synovial/CLC and have been used in supervised physical therapy programs and an HEP in

TABLE 4	TREATMENT STRATEGIES BASED ON IRRITABILITY LEVEL		
	High Irritability	**Moderate Irritability**	**Low Irritability**
Modalities	Heat/ice/electrical stimulation	Heat/ice/electrical stimulation	...
Activity modification	Yes	Yes	...
ROM/stretch	Short-duration (1-5 s), pain-free, passive AAROM	Short-duration (5-15 s), passive, AAROM to AROM	End range/overpressure, increased-duration, cyclic loading
Manual techniques	Low-grade mobilization	Low- to high-grade mobilization	High-grade mobilization/ sustained hold
Strengthen	...	...	Low- to high-resistance end ranges
Functional activities	...	Basic	High demand
Patient education	+	+	+
Other	Intra-articular steroid injection	...	...

Abbreviations: AAROM, active assisted range of motion; AROM, active range of motion.
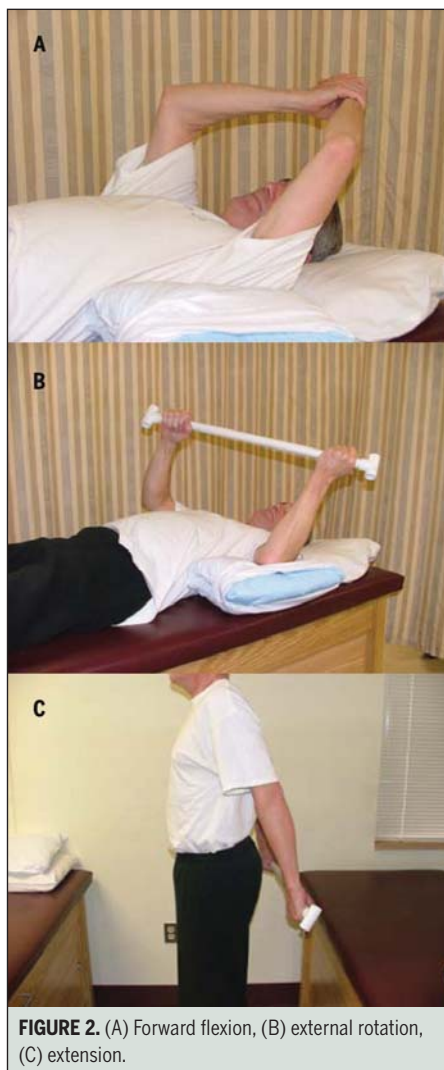

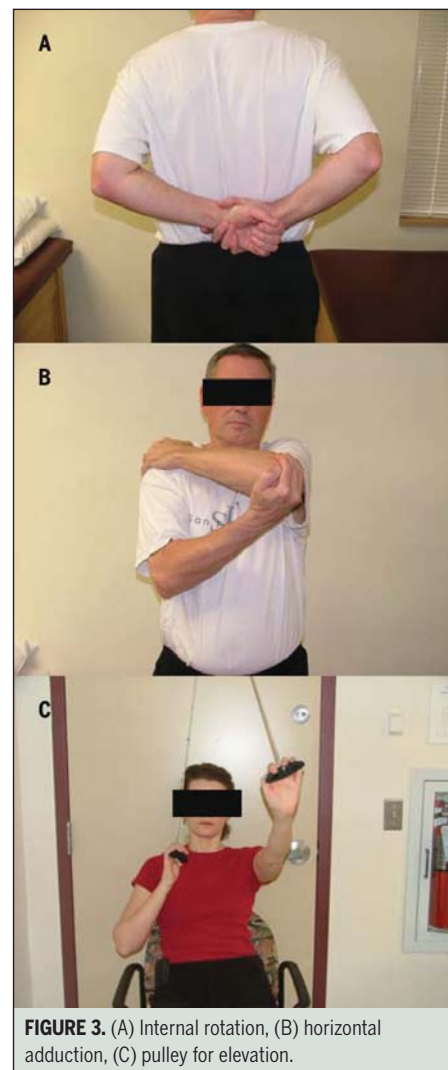**FIGURE 2.** (A) Forward flexion, (B) external rotation, (C) extension.

FIGURE 3. (A) Internal rotation, (B) horizontal adduction, (C) pulley for elevation.

patients with frozen shoulder.[20,28,39,55,104] Aggressive stretching beyond the pain threshold resulted in inferior outcomes in patients with frozen shoulder, particularly if performed in the early phase of the condition.[28] As the synovitis/angiogenesis and pain reduce, the fibrotic connective tissue wall is reached (stage 3). Tissue stress is progressed primarily by increasing stretch frequency and duration, while keeping the intensity in tolerable limits. The patient may be asked to hold the stretch for longer periods and increase the number of sessions per day. The patient is instructed to avoid excessive scapular compensation while performing exercises to minimize carryover of abnormal movement patterns as motion returns.

As the patient's irritability level becomes low, more-intense stretching and LLPS using a pulley or device (**FIGURE 4**) are performed to influence tissue remodeling. Tissue remodeling refers to a physical rearrangement of the connective tissue extracellular matrix (fibers, crosslinks, and ground substance). Collagenous tissues respond to increased tensile loading by increasing the synthesis of collagen and other extracellular components.[36,74,125] The collagen is oriented parallel to the lines of stress, and tensile strength is increased. It is important to note that biologic remodeling occurs over long periods (months), in contrast to mechanically induced change, which occurs within minutes.[2] Brand[12] describes this phenomenon as "growth," not stretch, of the contracted tissue. This growth process is consistent with the recovery process seen in primary frozen shoulder. Commercially available devices, such as the Dynasplint (Dynasplint Systems Inc, Severna Park, MD), and continuous passive motion units can provide LLPS; however, these devices require specific positioning and dedicated time during the day. Sustained end-range positioning devices are typically not tolerated in the patients with high or moderate irritability. McClure and Flowers[67] have described a simple abduction splint fabricated from thermoplastic materials that provides a LLPS.

FIGURE 4. Stick for prolonged elevation/external rotation.

Outcomes have been reported in patients with frozen shoulder treated primarily with exercise in physical therapy. Diercks and Stevens[28] prospectively followed 77 patients with idiopathic frozen shoulder for 24 months to compare the effects of "intensive physical therapy" to "supervised neglect." The intensive physical therapy group performed active exercises up to and beyond the pain threshold, passive stretching, glenohumeral joint mobilization, and an HEP. The "supervised neglect" group was instructed not to exercise in excess of their pain threshold, to do pendulum exercises and active exercises within the painless range, and to resume all activities that were tolerated. These authors found both groups made significant improvement in ROM and pain; however, 89% of the "supervised neglect" group achieved a Constant score of greater than 80, compared to only 63% of those in the intense-physical-therapy group. A conclusion of this study was that aggressive stretching beyond a pain threshold could be detrimental, especially if applied in the early phase of the condition.

As mentioned earlier, criteria for successful treatment is pain reduction and improved functional motion and patient satisfaction. Patient satisfaction may ultimately be the most important measure. Griggs et al[39] reported that 90% of 75 patients (mean follow-up, 22 months), classified with stage 2 idiopathic frozen shoulder, demonstrated good outcomes with an exercise program in a prospective functional outcome study. All patients were referred to physical therapy and performed HEP of passive stretching exercises in forward elevation, external rotation, horizontal adduction, and internal rotation. Ten percent of the patients were not satisfied with the outcome, and 7% of these patients underwent manipulation and/or arthroscopic release. Patients with the worst perceptions of their shoulder before treatment tended to have the worst outcomes.

Levine et al[61] reported that 89.5% of 98 patients with frozen shoulder responded with nonoperative management.[61] Resolution of symptoms occurred in 52.4% with physical therapy and nonsteroidal anti-inflammatory drugs (NSAIDs), while 37.1% resolved with NSAIDs, physical therapy, and 1 or more corticosteroid injections. The average time to successful treatment was 3.8 months. An impressive finding among several studies is that patients placed on a therapist-directed HEP had the same outcomes at short- (4-6 months) and long-term (12 months) follow-ups as those treated with other interventions.[15,20,55,104] Kivimaki[55] compared patients treated with an HEP to those who underwent manipulation under anesthesia and HEP. Other than a slight increase in ROM, the group performing just an HEP did not differ at any follow-up (6 weeks, and 3, 6, and 12 months) in pain or working ability.

Joint Mobilization

Many authors and clinicians advocate joint mobilization for pain reduction and improved ROM.[31,54,65,84,118,119] Unfortunately, little scientific evidence exists to demonstrate the efficacy of joint mobilization over other forms of treatment for frozen shoulder. However, patients treated with joint mobilization, with or

without concurrent interventions, had better outcomes.[15,52,84,118,119,127]

Specific joint mobilization techniques are believed to selectively stress certain parts of the joint capsule; for example, an inferior glide with the arm at the side, while in external rotation, would stress the RCI (**FIGURE 5**). While this may be true, it may be more beneficial to view the CLC through the circle concept. The circle concept refers to all regions of the CLC providing stability in all directions (ie, anterior structures providing anterior as well as posterior stability).[112] When this concept is applied to the shoulder with limited glenohumeral motion, improved extensibility of any portion of the CLC results in improved motion in all planes. This concept appears supported by the findings of Johnson et al,[52] who found significant improvement in external rotation motion in patients with frozen shoulder after performing posterior glide mobilizations sustained for 1 minute at end range of abduction and external rotation. High-grade joint mobilizations (grades III and IV) are used to promote elongation of shortened fibrotic soft tissues. High-grade mobilizations should be performed with the joint positioned at or near its physiologic end range. It should be noted that immediate ROM gains made with manual techniques (joint mobilization or end-range stretching) represent transient tissue preconditioning[12,35] and must be reinforced by an HEP. Joint mobilization techniques may be combined with hold-relax stretching methods to maximize relaxation, so that tensile load may be applied to the affected CLC. An example is performing a submaximal isometric contraction of the internal rotators, preceding an anterior glide, while at external rotation end range.

Several studies have examined the effect of joint mobilization in patients with frozen shoulder.[15,52,84,118,119] Nicholson[84] compared a group of patients who received joint mobilization and active exercise to a group receiving exercise alone. They found significantly improved motion and pain reduction in both groups,

FIGURE 5. Inferior glide with the arm at the side and in external rotation.

but the mobilization group had greater improvement only in passive abduction over the exercise group. Vermulen[118] presented a case series of 7 patients with frozen shoulder treated using only intense end-range mobilization techniques (no exercise or modalities) over a 3-month duration. They reported significant improvement in active and passive motion, pain, and joint volume. Vermullen[119] also performed a randomized prospective study comparing high-grade mobilization techniques to low-grade mobilization techniques (grades I and II). Patients were treated over 12 weeks (24 sessions) and followed for 12 months. They found significant improvement in motion and disability for both groups and the greatest amount of improvement occurred in the first 3 months. The high-grade mobilization group did better, but only a minority of comparisons reached statistical significance and the overall differences between the 2 interventions was small.[119] Bulgen et al[15] found that patients treated with joint mobilization and an HEP significantly improved in the first 4 weeks but not more than patients receiving intra-articular and subacromial corticosteroid injections. At 6 months, the mobilization group significantly improved in motion return and pain reduction, but no difference was noted compared to the other treatment groups, even the group performing just pendulum exercises. Yang et al[127] performed a multiple-treatment trial using combinations of end-range mobilization, midrange mobilization, and mobilization with motion in patients with frozen

shoulder. They found improved motion and function at 12 weeks, and concluded that end-range mobilization and mobilization with motion were more effective than midrange mobilization in increasing motion and functional mobility.

Corticosteroid Injections

Corticosteroid injections have been used to manage inflammatory processes for many years. The proposed effect of corticosteroids is to quell the inflammation, resulting in symptom reduction. Often, corticosteroid injections are administered with either a short- or long-acting local anesthetic lasting 30 minutes to 6 hours, respectively. An immediate gain in motion following a glenohumeral intra-articular corticosteroid injection is attributable to the anesthetic effect of reducing pain and thereby muscle guarding.[109] Over subsequent days, the corticosteroid's anti-inflammatory effect diminishes the painful synovitis/angiogenesis.[76]

Multiple studies have investigated the use of corticosteroids alone (either intra-articular or subacromial), in conjunction with supervised physical therapy, or with an HEP. Lee et al[58] found that all 3 exercise groups (2 receiving different site-specific corticosteroid injections), treated over a 6-week period, improved equally, though better than a fourth group treated only with analgesics. The groups performed active assisted ROM and active ROM exercises and resistive exercise. The authors noted the greatest motion improvement in the first 3 weeks of treatment. Hazelman[46] compared nonspecified physical therapy, cortisone injections, and manipulation under anesthesia and analgesics for 130 patients retrospectively. Although they found no significant recovery difference amongst the groups, 28% of the patients in the physical therapy group had symptom exacerbation. Arslan and Celiker[3] randomly allocated patients to receive either an intra-articular glenohumeral joint injection and home exercise, or physical therapy and a nonsteroidal anti-inflammatory drug. Physical therapy consisted of hot

packs, ultrasound, passive glenohumeral stretching exercises and wall climb. ROM and a pain scale were used for outcome measures. Patients in both groups improved similarly at 2 and 12 weeks. The authors concluded that corticosteroid injections and home exercise were as effective as physical therapy, but injections were much cheaper.

Several studies have also advanced the argument that intra-articular injections may be superior to therapy. Van der Windt et al[117] compared intra-articular injections to physiotherapy in a prospective randomized study on 109 patients with a stiff, painful shoulder (capsular syndrome). Physiotherapy consisted of twelve 30-minute sessions involving passive joint mobilization and exercises. Thermal modalities and electrostimulation could be used at the therapist's discretion. At 7 weeks, 77% of the patients treated with injections were considered treatment successes, compared to only 46% treated with physiotherapy, and significant differences were found in nearly all outcome measures. The main differences between groups were related to faster initial relief of symptoms with injections.

Bulgen et al[15] compared paired intra-articular and subacromial injections, joint mobilization, ice/proprioceptive neuromuscular facilitation (PNF), and no treatment (pendulum exercise), in a prospective randomized study. Pain and ROM significantly improved by the fourth week of treatment for all groups and continued until 6 months. Improvement was most obvious in the corticosteroid injection group, reaching statistical significance for motion, but not pain, during the first 4 weeks. No significant differences were seen among the groups at 6 months. The study concluded that there is little long-term advantage of one treatment over the other; however, corticosteroid injections may best improve pain and ROM in the first 4 weeks.

Carette et al[20] confirmed the benefit of intra-articular corticosteroid injections in treating frozen shoulder in a well-controlled randomized prospective study (n = 90). This study compared 4 groups, glenohumeral intra-articular corticosteroid injection with HEP, glenohumeral intra-articular corticosteroid injection with physical therapy and HEP, intra-articular saline injection with physical therapy, and intra-articular saline injection with HEP. To control for the inaccuracy of blind intra-articular injections, which can be as high as 42%,[33] fluoroscopy was used to ensure the accurate location of the injections of corticosteroid. At 6 weeks, the corticosteroid injection/physical therapy/HEP and corticosteroid injection/HEP groups demonstrated the largest change in Shoulder Pain and Disability Index (SPADI) score and were improved significantly over the noncorticosteroid groups. At 6 months, the SPADI scores were similar among the groups; however, active and passive motion were better in the corticosteroid injection/physical therapy/HEP group. This study concluded that at 6 weeks, an intra-articular injection alone, or in conjunction with physical therapy, was more effective than supervised physical therapy or an HEP; however, there was no benefit of one intervention over the others at 12 months.

Ryans et al[104] also investigated the effect of corticosteroid injections but performed both an intra-articular and subacromial injection. Their methods were similar to those of Carette et al[20] (4 groups), except they did not use fluoroscopy-guided injections, and only 8 sessions (over 4 weeks) of physical therapy were delivered instead of 12. The physical therapy program included PNF, mobilization, interferential electrical stimulation, and exercise. At 6 weeks, the injection groups significantly improved in the Shoulder Disability Questionnaire (SDQ) compared to the other groups; but patients treated in supervised physical therapy gained significantly more external rotation motion. All groups significantly improved by 16 weeks, but no difference was present between the groups. The authors recommended an intra-articular and subacromial corticosteroid injection for relieving shoulder disability and physical therapy for improving external rotation motion.[104]

Glenohumeral intra-articular corticosteroid injections, exercise, and joint mobilization all result in improved short- and long-term outcomes. However, there is strong evidence that glenohumeral intra-articular corticosteroid injections have a significantly greater 4- to 6-week beneficial effect compared to other forms of treatment.

PROPOSED MODEL GUIDING REHABILITATION

WE BELIEVE THAT REHABILITATION should be guided by the evidence in the literature, the extent of tissue irritability (as defined in **TABLE 1**), and the response to treatment. **TABLE 4** shows basic rehabilitation strategies matched with the level of irritability. While there is not strong evidence supporting the use of modalities, they may be useful in some patients with high or moderate irritability if there is a clear decrease in pain with their application. Patients with high irritability should be treated with short-duration, relatively pain-free stretching and low-grade joint mobilization to reduce symptoms and avoid exacerbation of pain and inflammation. Exercise found to be too painful or resulting in a prolonged painful response is held from the program and reintroduced when irritability reduces. Patients with low irritability should be given longer-duration stretching techniques and high-grade mobilizations performed with the joint near end range. The core exercises include pendulum exercise, passive supine forward elevation, passive external rotation with the arm in approximately 40° abduction in the plane of the scapula, and active assisted ROM in extension, horizontal adduction, and internal rotation (**FIGURES 2 AND 3**). Patients with moderate irritability may be instructed in pulley use for elevation. As the irritability level

reduces, progressive end-range stretching and mobilization may be performed. The authors encourage reassessment of motion and end-range discomfort at each session to determine the patient's response to treatment. Patients classified as having low irritability may be instructed in the same exercises and the use of pulleys, but will hold at end range for up to 30 seconds. More provocative stretching positions are used, such as stretching into external rotation with the arm in adduction (to isolate the stretch of the RCI) or with the arm in extension and adduction (**FIGURE 6**). We believe that strengthening and aggressive functional activity should be avoided when high and moderate irritability is present, and introduced gradually when individuals have low irritability; however, regaining motion should always be emphasized.

There is no clear evidence to determine which patients may need formal supervised therapy rather than simply a home program. Therefore, we recommend this decision be made based on the physician and patient preference, with input from the therapist after initial evaluation. Factors that may favor use of supervised therapy may be greater disability, more comorbidities, lower social support, lower educational level, or high fear and anxiety. Patients may initially be offered an intra-articular corticosteroid injection, and clearly those who fail to progress within approximately 3 to 6 weeks should be offered this option. A return visit to the referring physician for a corticosteroid injection should be facilitated if the patient's symptoms worsen.

There is also no clear evidence to suggest proper frequency of supervised therapy visits. We make decisions about frequency of visits based on a patient's within-session and between-session responses to treatment over the first several weeks. In general, patients with moderate or high irritability who demonstrate pain reduction and within-treatment ROM changes of greater than 10° to 15°, are seen more frequently, typically 2

FIGURE 6. Stretch to target the rotator cuff interval. The patient's hand remains fixed and the elbow is moved toward the table.

times per week. Patients with low irritability who have achieved pain reduction but minimal changes in motion are seen less frequently, typically once every week or 2, with emphasis on the home program as long as they are able to adhere to it appropriately. Success of treatment is not necessarily based on the restoration of normal motion but, rather, symptom reduction and patient satisfaction. Commonly, patients are discharged when the following occur: significant pain reduction, stagnant motion gains between sessions, improved functional motion, and improved satisfaction.

If the symptoms and motion are unresponsive to the various levels of treatment over time (3-6 months) and quality of life is compromised, a manipulation under anesthesia or surgical capsular release should be considered. If the patient is unwilling to have a manipulation or surgery, the patient is discharged but encouraged to continue with a daily stretching program.

MANIPULATION/MOBILIZATION UNDER ANESTHESIA

Manipulation Under Anesthesia

MANIPULATION UNDER ANESTHEsia remains a reasonable treatment for patients who have not responded to conservative treatment and are capable of adhering to a postmanipulation program of stretching and therapy.[30,48,83,90] The anesthesia, either general or a local brachial plexus block, completely relaxes the shoulder muscles,

ensuring that the force applied by the surgeon reaches the capsuloligamentous structures. Potential complications include glenoid, scapular, and humeral fractures, dislocations, postmanipulation pain, hemarthrosis, rotator cuff tear, labral tears, and traction injuries of the brachial plexus or a peripheral nerve.[63,76]

Manipulation under anesthesia is controlled, forced, end-range positioning of the humerus relative to the glenoid in an anesthetized patient. Surgeons try to use short lever arms to minimize potential fracture risk. Frequently, the surgeon first forcefully abducts the shoulder by stabilizing the scapula against the thorax, while elevating the humerus to release the inferior capsule. Next, the surgeon typically manipulates the shoulder into external and then internal rotation.[47] Audible and palpable release of the tissue suggests a good prognosis.[63] Arthroscopic examination following manipulation reveals significant bleeding into the joint due to tearing of the CLC.[63] The use of a glenohumeral joint intra-articular injection of corticosteroid following manipulation likely minimizes postmanipulation joint irritability.[48,90] Contraindications to manipulation of a frozen shoulder include a history of fracture or dislocations, moderate bone loss, or inability to follow through with postprocedure care. Studies assessing manipulation under anesthesia report success rates ranging from 75% to 100%, due to varied inclusion criteria, intraoperative procedures, and outcome measures.[1,30,48,90,93]

Translation Mobilization Under Anesthesia

Instead of traditional manipulation under anesthesia, Roubal et al[102] performed translational manipulation for the treatment of frozen shoulder. Translational manipulation differs from traditional manipulation in its use of translational (gliding) techniques and static end-range capsular stress, with a short-amplitude high-velocity thrust, if needed, as opposed to angular stretching forces. The translational techniques

are identical to joint mobilization techniques (anterior, posterior, and inferior gliding). The authors determined that postmanipulation average increase in flexion was 68°, abduction 77°, external rotation 49°, and internal rotation 45°. Placzek et al[92] used the same techniques and found significant improvement in outcomes at both short- (5.3 weeks) and long-term (14.4 months) follow-ups. Boyles et al[11] used translational gliding as already described on 4 patients with frozen shoulder; however, they performed additional mobilization/manipulation into directions of perceived restrictions. Translational manipulation under anesthesia appears to be a safe and efficacious alternative for the treatment of frozen shoulder.[11,92,102]

SURGERY

Open Capsular Release

FEW REPORTS OF OPEN SURGICAL RE-lease for frozen shoulder exist.[80,110] Complete or near complete return of motion has been described with open release directed toward the RCI and coracohumeral ligament.[86,87]

Arthroscopic Capsular Release

Arthroscopic surgery has replaced open capsular release as the preferred surgical treatment of primary frozen shoulder. Initially, arthroscopic surgery was used only after manipulation failed; but now it is typically performed alone or accompanies the manipulation. However, most clinicians still reserve arthroscopic surgery for patients with painful, disabling frozen shoulder unresponsive to at least 6 months of conservative treatment.[6,38,43,91,123] With arthroscopy, the surgeon can identify and address any intra-articular and subacromial pathology.[63,123] The surgeon selectively releases pathologic fibrosis in a controlled manner, versus manipulation, which ruptures capsuloligamentous structures nonspecifically.[6,43,85,91,123]

Debate continues about which structures should be arthroscopically re-leased. Several authors believe the RCI and the contracted coracohumeral ligament are the only structures requiring release.[7,86,87,124] Berghs[7] demonstrated impressive short- and long-term results (mean follow-up, 14.8 months) in 25 patients with primary frozen shoulder who had just the RCI and coracohumeral ligament released. Other authors selectively release additional portions of the CLC, such as the superior and middle glenohumeral ligament,[6,50] inferior glenohumeral ligament,[38,85,91,122] the intra-articular component of the subscapularis tendon,[29,85,91,122] and the posterior capsule.[38,122,123]

Postoperative protocols can vary from using a continuous passive motion device and exercise[50] to a an initial daily comprehensive physical therapy program.[123] In 37% of the patients, a follow-up intra-articular cortisone injection was required at approximately 4.5 weeks.[123]

SUMMARY

FROZEN SHOULDER IS A COMMONLY treated musculoskeletal problem, yet the etiology remains uncertain. Patients present with a characteristic history, physical examination, and natural course of recovery. Multiple interventions have been investigated assessing short- and long-term outcomes. Corticosteroid intra-articular injections demonstrate short-term (4-6 weeks) benefits and are favored in patients with high irritability or those who have not responded well to rehabilitation. Applying the correct tensile stress dose (intensity, frequency, and duration) while stretching is based on the patient's irritability classification. The majority of patients will respond to conservative interventions by achieving significant pain relief, return of functional movement, and patient satisfaction. CLC remodeling occurs over a prolonged period, resulting in functional motion. The patient with a recalcitrant frozen shoulder has the option of manipulation and/or capsular release, if conservative treatment fails. ◉

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