

Adhesive Capsulitis

A Treatment Approach

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Adhesive capsulitis of the shoulder is a condition of unknown etiology that results in the development of restriction of active and passive glenohumeral motion. The authors will review what currently is known about the etiology of idiopathic adhesive capsulitis, will raise unanswered questions regarding etiology and treatment, and will define a stage-based evaluation and treatment program. Treatment options including benign neglect, home-based and supervised physical therapy, intraarticular corticosteroid injections, closed manipulations, and arthroscopic capsular release will be reviewed critically and the authors' approach to the treatment of patients with idiopathic adhesive capsulitis also will be presented. Additionally, areas of future research will be defined.

Idiopathic adhesive capsulitis is a condition characterized by gradual loss of active and passive shoulder motion. Although the etiology of frozen shoulder remains elusive, the understanding of the pathophysiology recently has improved. Factors associated with adhesive capsulitis include female gender,⁴ age older than 40 years,³⁹ trauma,³⁹ diabetes,^{10,31,43,44,56}

prolonged immobilization,¹⁹ thyroid disease,^{7,71,77} stroke or myocardial infarction,^{44,45} and the presence of autoimmune diseases.^{12,66} The prevalence of frozen shoulder in the general population is slightly greater than 2%.⁴ Approximately 70% of patients with adhesive capsulitis are women, and 20% to 30% of those affected subsequently will have adhesive capsulitis develop in the opposite shoulder.

The diagnosis of adhesive capsulitis encompasses primary adhesive capsulitis, which is characterized by idiopathic, progressive, painful loss of active and passive shoulder motion; secondary adhesive capsulitis which has a similar histopathologic appearance but results from a known intrinsic or extrinsic cause; and secondary shoulder stiffness after surgical intervention. In primary adhesive capsulitis, an insidious onset of pain causes the individual to gradually limit the use of the arm. Inflammation and pain can cause reflex inhibition of the shoulder muscles, similar to inhibition of the quadriceps after injury to the knee. Disuse of the arm results in loss of shoulder mobility whereas continued use of the arm through pain can result in development of subacromial impingement. Subsequently, the individual gradually begins to lose motion and finds it increasingly difficult to perform activities of daily living that require overhead movement of the involved arm, reaching out to the side, or rotation of the humerus. Pain and muscular inhibition result in compensatory movements of the shoulder girdle to minimize pain. With

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time, there is resolution of pain and the individual is left with a stiff shoulder with severe limitation of function.

For any disease process, treatment decisions should be based on the pathophysiology of the disease and the natural history of the disease. In the case of frozen shoulder, the natural history of the disease remains controversial. Miller and Rockwood⁴⁴ reported on 50 patients during a 10-year period and found that the majority of the patients regained motion with minimal pain after home therapy, moist heat, antiinflammatory medications, and physician-directed rehabilitation. In contrast, Shaffer et al⁷⁰ reported that 50% of patients had pain or residual stiffness at 7 years followup.

The treatment of patients with adhesive capsulitis remains controversial. Many studies have been reported in the orthopaedic and rheumatology literature during the last 30 years. Treatment options documented in the literature include: benign neglect,⁴⁴ supervised physical rehabilitation,^{4,13,41,51,57,74} nonsteroidal antiinflammatory medications,^{29,64} oral corticosteroid,⁵ intraarticular injections,^{13,17,18,60,72} distension arthrography,³⁶ closed manipulation,^{2,21,26,31,40,52} open surgical release,³³ and more recently, arthroscopic capsular release.^{9,52,53,58,69} It is difficult to compare the results reported in these studies because of the lack of documentation of the stage of adhesive capsulitis being treated. It is the authors' belief that the stage of adhesive capsulitis is critical in determining the appropriate treatment and can dramatically affect the outcome of treatment. Issues surrounding the stage-appropriate treatment of patients with primary and secondary adhesive capsulitis will be discussed although the problem of the iatrogenic stiff shoulder will not be discussed.

PATHOGENESIS

To formulate a logical approach to the treatment of patients with primary and secondary adhesive capsulitis, it is necessary to understand the underlying cellular and biochemical pathophysiology of this disease. A review of

the literature reveals a multitude of strategies for treatment of patients with adhesive capsulitis, with extremely variable results provided. The lack of consistency in the published literature reflects a lack of understanding of the stages of adhesive capsulitis, which play a significant role in diagnosis and treatment. In 1945, Neviasser⁴⁷ introduced the term "adhesive capsulitis" and described pathologic changes in the synovium and subsynovium. There is disagreement as to whether the underlying pathologic process is an inflammatory condition^{22,67,76} or a fibrosing condition.¹⁴ Significant evidence exists^{12,20,66,76} in support of the hypothesis that the underlying pathologic changes in adhesive capsulitis are synovial inflammation with subsequent reactive capsular fibrosis, making adhesive capsulitis an inflammatory and a fibrosing condition, dependent on the stage of the disease. Cytokines recently have been implicated in the inflammation and fibrosis described in adhesive capsulitis. Cytokines are involved in the initiation and termination of repair processes in multiple musculoskeletal tissues and their sustained production has been shown to result in tissue fibrosis.^{1,6} Rodeo et al⁶⁷ reported an increase in transforming growth factor- β , platelet-derived growth factor, and hepatocyte growth factor staining in capsular biopsy specimens from patients with primary and secondary adhesive capsulitis and proposed that these cytokines are involved in the inflammatory and fibrotic cascades seen in adhesive capsulitis. The potential role of matrix metalloproteases in adhesive capsulitis recently has been identified. In a series of 12 patients with inoperable gastric cancer who were treated with a synthetic matrix metalloproteinase inhibitor, six had a frozen shoulder or a Dupuytren's-like condition develop.³⁰ Three cases of acute adhesive capsulitis also have been reported recently in patients treated with protease inhibitors for the human immunodeficiency virus (HIV).⁷⁸

The initial trigger in this cascade of inflammation and subsequent fibrosis is unknown. In some practices, there seems to be a seasonal variation in patients presenting with adhesive

capsulitis. Could a virus be responsible for the development of this self limited syndrome of inflammation and fibrosis? In the authors' clinical practice, it appears that adhesive capsulitis is more common in women between the ages of 40 and 60 years, which are the perimenopausal years. Do fluctuations in female hormones play a role in the development of adhesive capsulitis or is there a subtle, unrecognized autoimmune component of this syndrome? Why are patients with thyroid disease and diabetes at increased risk for the development of a frozen shoulder? It is clear that multiple factors can elicit an inflammatory condition that progresses in a continuum to a fibrosing condition; however, the common biologic trigger in initiation of this

process is unknown and warrants additional extensive study.

STAGES OF ADHESIVE CAPSULITIS

Neviaser⁴⁹ and Neviaser and Neviaser⁵⁰ described the arthroscopic stages of adhesive capsulitis and stressed the importance of an individualized treatment plan based on an understanding of the clinical stages of the disease. Hannafin et al²² described a correlation between the arthroscopic stages described by Neviaser, the clinical examination, and the histologic appearance of capsular biopsy specimens taken from patients with Stages 1, 2, and 3 adhesive capsulitis. Adhesive capsulitis can

TABLE 1. 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 the 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, fibroplasia and scar formation in the underlying capsule

Stage 3: "Frozen Stage"

- Duration of symptoms: 9 to 5 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

ROM = range of motion.

be broken down into four stages as outlined in Table 1. It is critical to remember that these stages represent a continuum of disease rather than discrete well-defined stages.

In Stage 1, patients will present with pain, which often is described as achy at rest and sharp at extremes of range of motion (ROM). Symptoms have been present for less than 3 months; however, the patient will report a progressive loss of motion. Loss of internal rotation, forward flexion, and abduction are present, and a more subtle loss of external rotation in adduction. Examination of a patient with Stage 1 adhesive capsulitis after injection of the glenohumeral joint with local anesthetic or examination of the patient under anesthesia will reveal a significantly improved, near normal ROM. In this early stage, the majority of motion loss is secondary to the painful synovitis, rather than a true capsular contraction. Arthroscopic examination reveals a hypertrophic vascular synovitis that coats the entire capsular lining. Biopsy specimens show rare inflammatory infiltrates, a hypervascular synovitis and normal underlying

capsular morphologic characteristics (Fig 1). In Stage 2, symptoms have been present for 3 to 9 months with progressive loss of ROM and persistence of the pain pattern described above. Examination of the patient after local anesthetic infiltration or scalene block reveals relief of pain, with partial improvement in ROM. The motion loss in Stage 2 adhesive capsulitis reflects a loss of capsular volume and a response to the painful synovitis. Arthroscopic examination reveals a dense, proliferative, hypervascular synovitis. A capsular biopsy specimen is notable for a hypervascular synovitis with perivascular scar formation and capsular fibroplasia with deposition of disorganized collagen fibrils and a hypercellular appearance (Fig 2). No inflammatory infiltrates have been reported in Stage 2. In Stage 3, patients will present with a history of painful stiffening of the shoulder and significant loss of ROM. Symptoms have been present for 9 to 14 months and have been observed to change with time. Patients often report a history of an extremely painful phase that has resolved, resulting in a relatively pain-free but stiff shoulder.

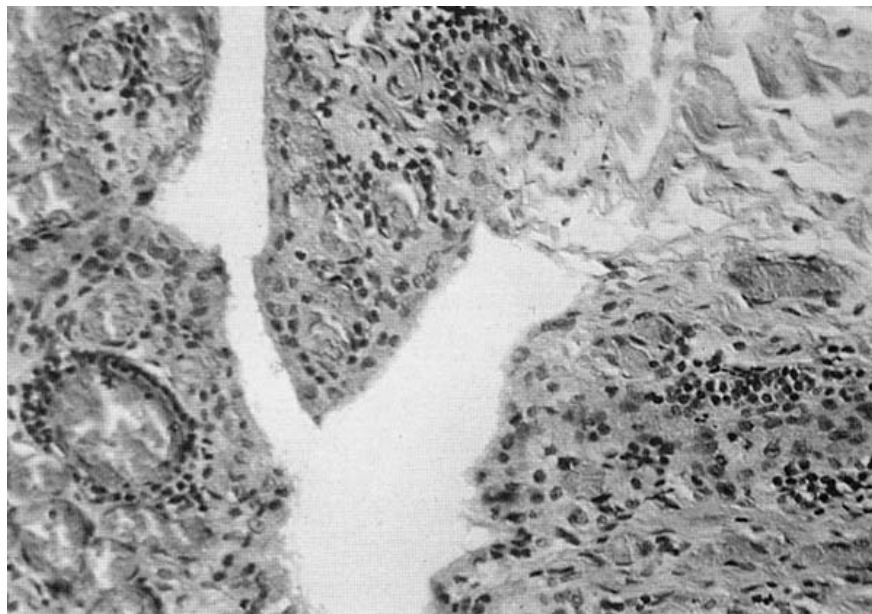


Fig 1. Histologic appearance of the synovium and underlying capsule in a biopsy specimen obtained from a patient with Stage 1 adhesive capsulitis. The hypervascular appearance of the synovium with underlying normal capsular architecture can be seen. (Stain, hematoxylin and eosin; magnification, $\times 20$).

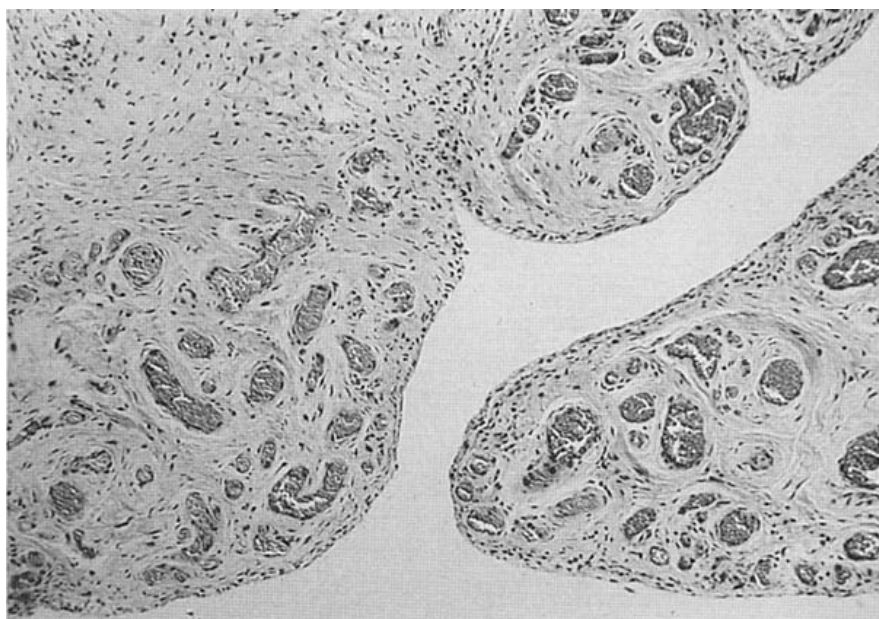


Fig 2. Histologic appearance of the synovium and underlying capsule in a biopsy specimen obtained from a patient with late Stage 2 adhesive capsulitis. The perivascular scar formation and the extensive fibrosis in the underlying capsule is shown. (Stain, hematoxylin and eosin; magnification, $\times 10$).

Range of motion examination is unchanged by injection of local anesthetic or evaluation with the patient under anesthesia reflecting the persistent loss of capsular volume and fibrosis of the glenohumeral joint capsule. Arthroscopic examination of a patient with Stage 3 adhesive capsulitis is unremarkable when compared with examination of patients with Stages 1 and 2 adhesive capsulitis. A residual filmy synovial layer is visible with patches of synovial thickening without hypervascularity. Capsular biopsy specimens reveal a dense, hypercellular collagenous tissue (Fig 3). Stage 4, the "thawing stage" for adhesive capsulitis, is characterized by the slow, steady recovery of ROM resulting from capsular remodeling in response to use of the arm and shoulder. No arthroscopic or histologic data are available for patients with Stage 4 adhesive capsulitis because these patients rarely undergo surgery. The authors think that it is essential to consider these stages when examining a patient with a stiff or painful shoulder because the stage of the disease should direct the treatment options.

DIAGNOSIS

Primary and Secondary Adhesive Capsulitis

The diagnosis of primary adhesive capsulitis is made from the history and physical examination. This is an idiopathic condition and the diagnosis is made when other causes of pain and motion loss are eliminated.^{48,49,63} The physical examination always should include an evaluation of the cervical spine and the shoulder. Patients presenting with Stages 1 and 2 adhesive capsulitis have pain on palpation of the anterior and posterior capsules and describe pain radiating to the deltoid insertion. Night pain and pain at rest are common in the early stages. Evaluation of active and passive ROM should be performed because documenting the initial ROM, especially passive motion, is critical in determining the efficacy of the treatment plan. Pure glenohumeral motion is measured while limiting scapulothoracic motion. A series of measurements are taken to define glenohumeral and combined shoulder motion. Ac-

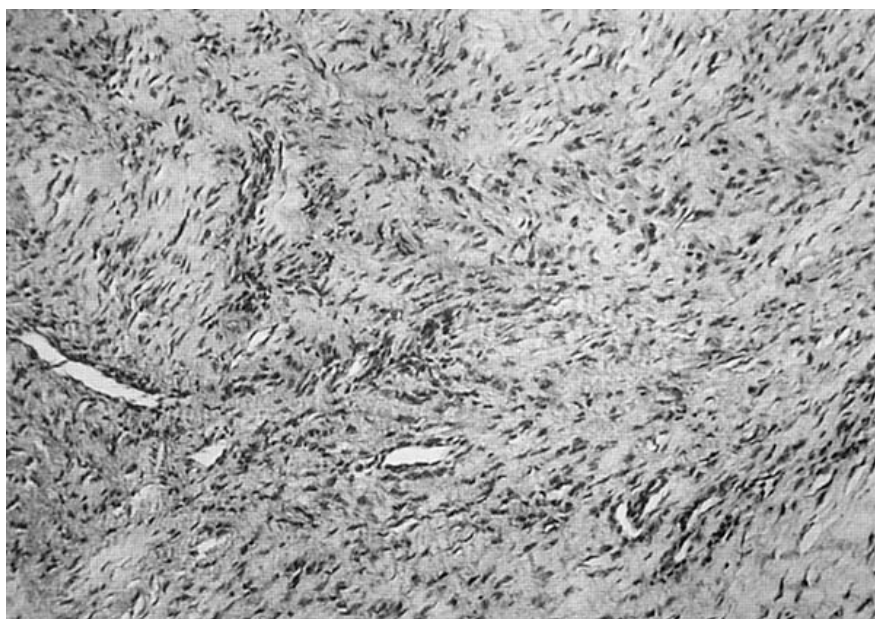


Fig 3. Histologic appearance of the capsule in a biopsy specimen obtained from a patient with Stage 3 adhesive capsulitis. The extensive capsular fibroplasia and new pericellular matrix deposition can be seen. (Stain, hematoxylin and eosin; magnification, $\times 20$).

tive and passive forward flexion, abduction, internal rotation (measured by having the patient place the thumb to the highest point possible on the spinous process) and external rotation in neutral abduction are measured and recorded with the patient standing. Passive glenohumeral motion then is measured with the patient supine and scapulothoracic motion is constrained by manual pressure on the acromion. Measurements are made in the coronal plane as opposed to the scapular plane, because the anterior capsule is more lax in the scapular plane and gives the appearance of better range ROM. With the patient in the supine position, passive internal and external rotation at 45° glenohumeral abduction and maximal glenohumeral abduction are measured and recorded.

Routine radiographic evaluation should include anteroposterior (AP) views in internal and external rotation and axillary and outlet views to rule out other causes for a stiff, painful shoulder including glenohumeral arthritis, calcific tendinitis, or longstanding rotator cuff disease. Radiographs usually are negative in patients with frozen shoulder, although there may be evi-

dence of disuse osteopenia. Leppala et al³⁷ documented significant decreases in bone mineral density associated with active phase adhesive capsulitis with recovery of normal bone density when measured 9 years after disease. Historically, arthrography, which shows a decreased joint capacity in patients with Stages 2 to 4 adhesive capsulitis, has been used for diagnostic purposes; however, it no longer is used routinely.^{46,62} If the clinical diagnosis is unclear, magnetic resonance imaging (MRI) may be useful in evaluation of the rotator cuff or labrum but it is not recommended routinely for the diagnosis of adhesive capsulitis. Magnetic resonance imaging has been used for investigational purposes in patients with adhesive capsulitis and has shown an increased blood flow to the synovium in frozen shoulder, a finding consistent with the observed histologic appearance.⁷³

STAGE 1 AND STAGE 2

Patients presenting with different stages of primary adhesive capsulitis should have individualized treatment. However, certain basic

principles apply to all stages. Patients who present with a painful limitation of motion are given oral nonsteroidal antiinflammatory medications that are supplemented with other analgesics as necessary. An intraarticular injection of steroid and local analgesic can be extremely useful in the diagnosis and treatment of adhesive capsulitis. The injection of the glenohumeral joint in patients with adhesive capsulitis is analogous to injection of the subacromial spaces in patients with impingement syndrome. Review of the stages outlined above provides the rationale for early intraarticular injection. Injection of a local anesthetic and corticosteroid in a patient with Stage 1 adhesive capsulitis can be diagnostic and thera-

peutic. After injection, passive glenohumeral ROM is reevaluated. If the patient has significant improvement in pain and normalization of motion, the diagnosis of Stage 1 adhesive capsulitis is confirmed. If the patient has a significant improvement in pain but no significant improvement in ROM, then by definition he or she has Stage 2 adhesive capsulitis. Again, it must be reinforced that these stages represent a continuum of the inflammatory and scarring processes.

There is extensive information regarding the efficacy of intraarticular corticosteroid in the treatment of patients with adhesive capsulitis.^{13,17,18,60,72} In interpreting the results of published studies, it is critical to note the stage

TABLE 2. Rehabilitation Guidelines For Adhesive Capsulitis

Stage 1:

Presentation of symptoms: empty end-feel

Goal: interrupt inflammation and pain cycle

Treatment:

education (resting position, postural correction)

activity modification

modalities are used as needed to control pain and inflammation

ROM exercises (joint mobilization, gentle physiologic movement, continuous passive motion, hydrotherapy)

closed chain scapular stabilization

home exercise program

Stage 2:

Presentation of symptoms

Limitation of ROM in capsular pattern

Pain at end of ROM

Goal: decrease pain and inflammation

minimize capsular adhesions and restriction of ROM

Treatment

Modalities are used as needed to decrease pain and inflammation and to increase tissue extensibility

ROM exercises (joint mobilization) in inferior and posterior direction)

active exercise is initiated in the plane of the scapula

training of scapular muscles

home exercise program

Stages 3 and 4:

Presentation of symptoms

capsular end-feel

resistance felt before pain is reached

Goal: increase ROM

Treatment

aggressive stretching (proprioceptive neuromuscular facilitation, soft tissue mobilization, low load prolonged stretch)

modalities to promote relaxation and tissue extensibility, reduce discomfort

strengthening continues to reestablish scapula force couples, and address any home exercise program

ROM = range of motion.

of adhesive capsulitis being treated. Bulgen et al¹³ randomized patients to treatment with steroid, physical therapy, ice, or benign neglect. The initial response to treatment was most marked in patients treated with steroid; however, no significant difference in final long-term outcome was reported when treatment groups were compared. Hazelman²⁵ summarized numerous studies on the use of intraarticular corticosteroid and reported that the success of treatment is dependent on the duration of symptoms. Patients treated within 3 months of the onset of symptoms reported a significant improvement in symptoms whereas those patients treated after 5 or more months of onset of symptoms had a more delayed recovery. The time necessary for full recovery was reported to be dependent on the duration of symptoms. Patients treated within 1 month of onset of symptoms recovered in an average of 1.5 months. Patients treated within 2 to 5 months of onset of symptoms recovered in 8.1 months; patients treated 6 to 12 months after onset of symptoms required an average of 14 months for full recovery. The authors have observed a similar pattern in treatment of patients with adhesive capsulitis at their institution. Patients treated with intraarticular corticosteroid during Stage 1 (0–3 months) had a rapid and striking recovery of a pain-free shoulder within 6 weeks. Patients treated during Stage 2 had a significant improvement in night pain and pain at rest, with the time necessary for recovery of ROM dependent on the duration of symptoms before treatment. These data and others support the hypothesis that adhesive capsulitis is an inflammatory and fibrotic condition.^{5,13,14,22,76} In the early stages of adhesive capsulitis, a hypervascular synovial hyperplasia is present that results in eventual fibrosis of the subsynovium and capsule. Early treatment with intraarticular corticosteroid may provide a chemical ablation of the synovitis, thus limiting the subsequent development of fibrosis and shortening the natural history of the disease. The self-limiting nature of adhesive capsulitis also supports the role of the synovium in initiation and regulation of the fibrotic process

in the capsule. With resolution of the synovitis and loss of the cytokine stimulus to the capsular fibroblasts, capsular remodeling and recovery of ROM take place. It remains to be defined whether the use of specific inhibitors of cytokines also will shorten this syndrome's natural history or whether the use of inhibitors of collagen synthesis will be helpful in decreasing capsular fibrosis.

The authors think that patients should be started on a supervised physical therapy program because the major objective in the treatment of individuals with adhesive capsulitis is to restore function by decreasing the inflammatory response and pain, increasing ROM, and reestablishing normal shoulder mechanics. Patient education and instruction in a home exercise program are mainstays of all rehabilitation programs. An understanding of the diagnosis of adhesive capsulitis will encourage patient compliance and decrease patient frustration. The first phase of rehabilitation correlates with Stage 1, which is characterized clinically by pain, and a reluctance to perform movements with the involved shoulder (Table 2). The patient often reports a diffuse aching pain in the shoulder that seems to settle in the deltoid. Pain is present at rest and intensifies during movement. Sleep disturbance secondary to pain is common. The humeral head is positioned anteriorly because the individual assumes a protective posture of the involved extremity with the arm splinted against the body in an adducted, protracted, and internally rotated position.²³ Range of motion assessment reveals an "empty" end-feel, where pain stops passive movement before resistance is felt by the clinician.¹⁶ No significant deficits in resisted movements are seen.

The primary goal of treatment of patients with Stage 1 adhesive capsulitis is to interrupt the cycle of inflammation and pain; therefore, activity modification is an important consideration. The individual is encouraged to use pain as a guide to limit activities of daily living because inflammation and pain can alter shoulder mechanics. The optimal resting posture with the arm positioned in comfortable abduc-

tion for improved vascularization of the cuff is demonstrated to the patient.⁶¹ Postural training is incorporated to discourage thoracic kyphosis and a forward humeral head position during forward elevation. Therapeutic modalities are used to reduce pain (high voltage galvanic stimulation, transcutaneous electrical nerve stimulation,⁶⁴ iontophoresis, cryotherapy), reduce inflammation (iontophoresis, phonophoresis, and cryotherapy), and to promote relaxation (moist heat, ultrasound).⁷⁵ Gentle joint mobilizations and physiologic movements using the opposite extremity within a pain-free ROM will assist in reducing pain by stimulating the joint mechanoreceptors and decreasing nociceptive input.⁵⁴ Hydrotherapy can be used to break the cycle of pain and muscle spasm. The buoyancy of the water provides an environment for active-assistive exercise and helps facilitate the return of normal scapulohumeral rhythm. The hydrostatic pressure on the arm during hydrotherapy may cause a "glove effect" that stimulates the proprioceptors of the skin and generates a biofeedback-like effect.⁷¹ The recommended water temperature is tepid to neutral. Basic exercises including flexion in the plane of the scapula and horizontal abduction and adduction along the surface of the water are performed. A closed chain exercise, in which the distal end of the limb is fixed, can be performed early on during the rehabilitation process, thus permitting the rotator cuff to work as a glenohumeral compressor.³⁴ This type of exercise reduces the shearing effect of the deltoid, causes cocontraction of force couples around the shoulder, and creates an axial load through the joint. Training of the scapular stabilizers should be initiated as soon as tolerated by the patient to provide a stable base for distal mobility. The home exercise program should include passive ROM exercises and pendulum exercises to allow joint distraction and increase the arc of pain-free movement.

The second phase of rehabilitation is used to treat patients with Stage 2 adhesive capsulitis. The continuum of symptoms progresses in

this stage to include pain in the upper trapezius and periscapular musculature. Painful spasm of these muscles may extend to the neck additionally altering shoulder mechanics. If the individual has responded successfully to intraarticular corticosteroid, pain will be felt only at extremes of movement. Hiking of the shoulder girdle is evident with elevation of the arm as a result of capsular contracture and inhibition of the rotator cuff musculature. Anterior translation of humeral head may result from a decrease in capsular volume associated with adhesive capsulitis.⁶⁸ Postural habitus of increased thoracic convexity will alter the resting position of the scapula causing the head of the humerus to move into internal rotation to maintain its resting position in the glenoid, and affect the balance of shoulder girdle musculature.³ The limitation of ROM is in a capsular pattern with external rotation most limited, followed by abduction, then internal rotation.¹⁶ In this stage, ROM evaluation reveals a rigid, capsular end-feel.

The goal of the second phase of rehabilitation is to decrease inflammation and pain, and to minimize capsular restriction thereby minimizing loss of motion. It is important to educate the patient regarding the improvement in ROM because the patient will continue to perceive pain at the end of the range, and may not recognize the objective improvement in function. In this phase, modalities are used to decrease pain and inflammation and to increase tissue extensibility. Range of motion exercises including passive joint mobilizations are used to restore joint glide and separation. The goal is to stretch the capsule sufficiently to allow restoration of normal glenohumeral biomechanics. Kaltenborn³² stressed the importance of promoting joint glides for increasing capsular mobility and prevention of joint compression and periarticular soft tissue injury that may occur with long lever angular mobilizations. Because ROM is improved, active exercise is performed in the plane of the scapula at an angle 30° to 45° anterior to the coronal plane. In the plane of the scapula, the inferior part of the capsule is not twisted, and an opti-

mal length-tension relationship of the rotator cuff muscles is achieved.⁵⁹ The home exercise program should emphasize frequent ROM exercises including pendulums, cane exercises for improving internal rotation and external rotation, and elevation.

Although most patients will have significant improvement by 12 to 16 weeks, some patients do not improve and may get worse. At this point, the options include continued physical therapy, surgical intervention, closed manipulation or an arthroscopy, capsular release, and manipulation. The risks and benefits of these approaches are described, including fracture, neurovascular injury, residual stiffness, instability, and infection. The authors' preference is to proceed with arthroscopic assessment before manipulation. If one accepts the hypothesis that the glenohumeral synovitis is an essential factor in the development of adhesive capsulitis, then an arthroscopic examination is critical to rule out residual synovitis or allow synovectomy before manipulation with or without capsular release.

STAGE 3 AND STAGE 4

Patients who present with Stages 3 and 4 adhesive capsulitis often report a history of long-standing pain at rest and pain at night that have resolved spontaneously.⁸ Physical examination will reveal a stiff shoulder, with striking alteration of scapulohumeral mechanics and limited use of the arm during activities of daily living. As has been observed in Stages 1 and 2, treatment should be individualized to each patient. There is no indication for the use of intraarticular corticosteroid because the inflammatory phase of the disease has passed. The decision to proceed with operative versus non-operative treatment is dependent on the degree of functional disability and the patient's response to a rehabilitative program designed to improve capsular flexibility and subsequently ROM.

Physical therapy for patients with Stage 3 adhesive capsulitis is designed to treat the significant loss of motion and abnormal scapulo-

humeral rhythm that is characteristic of this stage. Poor scapulohumeral rhythm is observed during elevation of the arm. There is dominance of the upper trapezius resulting in hiking of the shoulder girdle. This is attributed to decreased inferior glide of the glenohumeral joint, which prevents glenohumeral abduction.²⁷ A capsular pattern describes the limitation of motion. Resistance in the form of a capsular end-feel is felt before pain is reached as the glenohumeral joint is taken through passive ROM. The primary goal of treatment is to increase ROM. In this phase, aggressive stretching will be tolerated and should be the focus of treatment. Stretching can be taken beyond the limits of the available ROM. Techniques may include proprioceptive neuromuscular facilitation, soft tissue mobilization of the subscapularis and pectoralis minor,¹⁵ and joint mobilization.²⁰ Low load, prolonged stretch produces plastic elongation of tissues as opposed to high tensile resistance seen in high load, brief stretch.^{38,65} Heat may be used to promote muscle relaxation, ultrasound may be used to promote tissue extensibility in the axillary fold, and cryotherapy may be used to reduce discomfort after stretching. An active warm-up to increase soft tissue circulation is preferred over passive treatment. Strengthening of the scapula musculature continues in this phase of rehabilitation to reestablish effective force couples. As ROM improves, and if rotator cuff weakness persists, isolation of the cuff can be initiated to address strength and endurance. The home exercise program includes ROM and flexibility exercises and training of the scapular muscles.

OPERATIVE TREATMENT OF PATIENTS WITH ADHESIVE CAPSULITIS

Closed Manipulation

Closed manipulation is contraindicated in patients with significant osteopenia, recent surgical repair of soft tissues about the shoulder, or in the presence of fractures, neurologic in-

jury and instability. Closed manipulation is performed as described previously²¹ after placement of a scalene block anesthesia. The scapula is stabilized with one hand while the humerus is grasped just above the elbow with the other hand. Initially, the adducted shoulder is externally rotated and then abducted in the coronal plane. Next, the shoulder is externally rotated in abduction and then internally rotated while abduction is maintained. The shoulder then is flexed and finally brought back into adduction and internally rotated. There frequently is palpable and audible yielding of the soft tissue as motion is restored in the different planes. The authors do not advocate closed manipulation of the shoulder for patients with adhesive capsulitis, but prefer an arthroscopic inspection before any manipulative treatment is done.

ARTHROSCOPIC RELEASE

Historically, arthroscopy has been of little diagnostic and therapeutic value in patients with adhesive capsulitis of the shoulder.³⁹ However, it has been suggested that the arthroscope may be helpful for delineation of disorders, documentation of the result of closed manipulation, and treatment of concomitant intraarticular and subacromial disease.^{15,28,58,69,76} For this reason, the authors are prepared to perform arthroscopy, capsular release, and manipulation if there are no suspected extraarticular factors contributing to the motion loss. During the past 7 years, the senior author has performed arthroscopy, capsular release, and manipulation in more than 60 patients and has found that it has the advantage of allowing detection of concomitant disease, synovectomy in Stage 2, and permits a precise capsular release. Furthermore, the force of manual manipulation required to regain motion is reduced greatly by arthroscopic capsular release before manipulation.⁵²

It is essential to document glenohumeral and total ROM before initiation of the surgical procedure. The timing of arthroscopy and manipulation remains controversial. Some sur-

geons prefer to manipulate the shoulder first and follow with the arthroscopic evaluation; however, rupture of the capsule with manipulation will greatly increase the risk of fluid extravasation in the soft tissues surrounding the shoulder. The senior author prefers to perform a diagnostic arthroscopy and synovectomy before manipulation of the shoulder to minimize fluid extravasation into the soft tissues.

Although it may be difficult to insert the arthroscope into a stiff shoulder because of the capsular contracture and decreased joint volume,^{9,76} chondral damage is avoided by inserting the arthroscope over the humeral head. The capsule is more difficult to penetrate with the blunt trocar because of the capsular fibrosis and thickening and it is helpful to distend the capsule with fluid via spinal needle before insertion of the arthroscope. The smaller 3.8-mm arthroscope has been recommended⁷⁶ but the senior author routinely uses a standard arthroscope. The arthroscopic appearance of the joint is dependent on the stage of adhesive capsulitis and outlined previously. In Stage 1 adhesive capsulitis, a diffuse hypervascular synovitis is observed that may have areas of focal thickening in the anterosuperior capsule and along the proximal biceps tendon. An arthroscopic cannula is inserted just underneath the biceps tendon and this synovium is removed atraumatically with a 4.5-mm motorized shaver. It is important to perform a thorough synovectomy in patients with Stage 1 adhesive capsulitis; thus, it is necessary to view the shoulder from anterior and posterior portals. An attempt should be made to resect any areas of synovitis in the inferior pouch. This is technically feasible in patients with Stage 1 adhesive capsulitis, but often is extremely difficult in patients with Stage 2 adhesive capsulitis. A capsular biopsy specimen routinely is taken to confirm the histologic stage of the syndrome. In Stage 2 adhesive capsulitis, the synovial lining remains hypervascular but is thicker and more pedunculated in appearance. Again, a thorough synovectomy is indicated and a capsular biopsy specimen is obtained. In Stage 3 adhesive capsulitis, resid-

ual synovial thickening or scarring is seen, but the hypervascular appearance has resolved. A sheet of capsular scar, which can be debrided carefully, may obscure the rotator cuff interval region and the tendon of the subscapularis.

At this point, the arthroscopic instruments are removed and a gentle manipulation is performed. Patients with late Stage 1 or early Stage 2 adhesive capsulitis who have mild scarring and thickening of the capsule often will regain full ROM with gentle manipulation. The authors routinely perform the manipulation in the following order: forward flexion, extension, abduction, internal and external rotation. In the early stages of adhesive capsulitis, a series of small pops are heard as the anterior and inferior capsule ruptures. If the arthroscope is placed back into the shoulder, one can visualize a capsular wrent that runs from approximately 2 o'clock to 6 o'clock then passes obliquely across the inferior axillary pouch. In late Stage 2 and Stage 3 adhesive capsulitis, the capsular scarring is dense and the patient may not respond to manipulation. Before manipulation, an arthroscopic capsular release is performed with the patient under anesthesia. The capsular scar is divided using an electrocautery device and a motorized shaver. The capsular division begins superiorly anterior and inferior to the biceps tendon and continues inferiorly until the discrete upper edge of the subscapularis tendon is encountered. This constitutes a surgical release of the rotator interval region of the capsule.²⁴ Ozaki et al⁵⁵ reported that an open release of this area usually is successful in restoring external rotation in shoulders with refractory adhesive capsulitis. As the capsule is released, the humeral head moves inferiorly and laterally, creating more room in the joint for the arthroscope to be moved into the anterior and inferior regions of the joint. The capsular release then is continued inferiorly to 5 o'clock. The authors do not routinely attempt release of the inferior recess because of risk to the underlying axillary nerve. After this arthroscopic capsular release, the arthroscope is removed and a closed manipulation is performed. In

most cases, external rotation in adduction is restored with almost no manipulation force. The shoulder then can be manipulated into other planes with minimal force and with audible and palpable yielding of tissue. If the patient still lacks internal rotation in abduction, the arthroscope is reinserted into the joint via an anterior portal and a posterior capsular release is performed. The authors perform the release in the midcapsular region, and have had no cases of axillary nerve injury with this technique.

The goal of treatment after surgery is to maintain ROM achieved under anesthesia, and to decrease pain and inflammation. In the recovery room, the arm is placed in the quadrant position while the patient is still under scalene block anesthesia. A second scalene block is administered during an overnight hospital stay so the patient can tolerate exercise through the ROM.^{11,35} Continuous passive motion is applied and the patient is encouraged to use it throughout the night.⁴² On discharge from the hospital, the patient is seen as an outpatient for 5 days per week for the next 2 weeks, then 3 times per week until treatment is completed. Treatment includes aggressive ROM, continuous passive motion, modalities for pain and inflammation, and hydrotherapy. Hydrotherapy is instituted as soon as possible with the use of a water-impermeable dressing to keep the suture sites dry. Strengthening exercises gradually are incorporated into the program as outlined previously. It is the authors' opinion that treatment of patients with adhesive capsulitis of the shoulder requires an individualized approach that considers the stages and natural history of the disease. In general, a conservative approach with the use of intraarticular corticosteroid and a supervised physical therapy program is the initial method of treatment and generally is successful in patients with Stage 1 adhesive capsulitis. This conservative treatment approach often will be successful in patients with Stage 2 adhesive capsulitis; however, some patients may have a refractory motion loss. In patients who continue to have a refractory motion loss with nonoperative

treatment, arthroscopy, synovectomy, and closed manipulation may be attempted. In patients with late Stage 2 and Stage 3 adhesive capsulitis, an arthroscopic release technique often is required to successfully restore motion. This technique is demanding, and proper patient selection, anesthesia, and postoperative analgesia are critical to its success.

The unanswered questions outlined within the current study should form the basis of continued clinical research in the treatment of patients with adhesive capsulitis. The biologic trigger responsible for the development of synovitis and subsequent capsular fibroplasia must be identified. Studies must be undertaken to determine why women are at increased risk for development of adhesive capsulitis. Do fluctuations in hormone levels in the perimenopausal years somehow trigger this syndrome, or alternatively, do they determine the individual response to the biologic trigger? No data currently exist in the scientific literature regarding the role of hormone replacement therapy in the prevention or development of adhesive capsulitis. Is there an autoimmune component to adhesive capsulitis, as has been described in other fibrosing conditions such as scleroderma? It also is unknown whether women and men respond differently in the conservative or operative treatment of adhesive capsulitis, as published studies do not assess outcome with relation to gender. It is theoretically possible that treatment with specific agents designed to eliminate the synovitis, capsular fibroplasia, and scar formation may make the surgical treatment of patients with adhesive capsulitis obsolete in the future. Well-controlled prospective clinical trials will be needed to determine the efficacy of these treatment regimens for women and men, and to transform the treatment of adhesive capsulitis into a clearly defined science.

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