XIV. Shoulder Stiffness

A. Adhesive Capsulitis. Also known as *frozen shoulder*, adhesive capsulitis has no known etiology and is characterized clinically by pain and global loss of active and passive GH motion. The typical patient is a middle-aged woman with nondominant extremity involvement. The differential diagnosis includes posterior dislocation, rotator cuff tears, severe impingement syndrome, cervical arthritis, Pancoast tumor, and neuromuscular disease. Factors associated with the development of adhesive capsulitis include trauma following chest or breast surgery, diabetes, prolonged immobilization, thyroid disease, and other medical conditions (e.g., pulmonary disease, myocardial infarction, cerebrovascular accident). Similar to calcific tendonitis, the disease may have a genetic focus or autoimmune component.

Histologically, adhesive capsulitis is an intra-articular process associated with chronic inflammation, fibrosis, and perivascular inflammation of the subsynovial capsular layer suggesting an overreactive reparative inflammatory process. Macroscopically, it is characterized by contracture of the joint capsule leading to decreased capsular compliance and intra-articular volume limiting motion in all planes.

1. Stages. There are three stages of varying duration.

Stage 1. Painful phase (2–9 months): insidious or abrupt onset of diffuse shoulder pain.

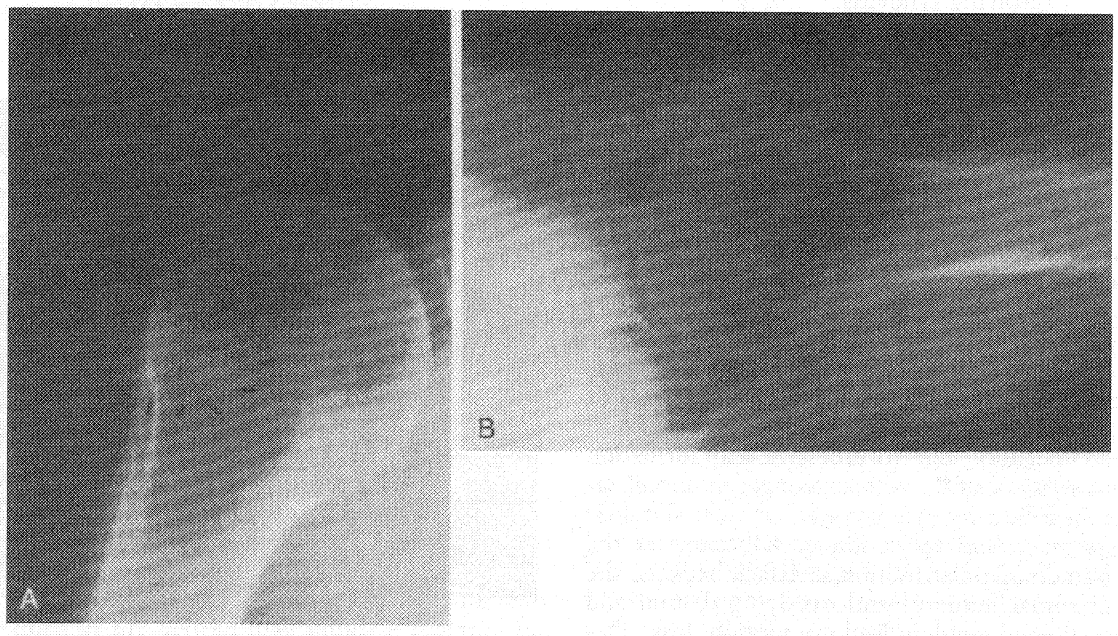

Figure 4–114. AP *(A)*, and axillary *(B)*, lateral radiographs demonstrating calcific tendonitis near the subscapularis insertion on the lesser tuberosity. (From Re P, Karzel RP: Management of rotator cuff calcifications. Orthop Clin North Am 24:128, 1993.)

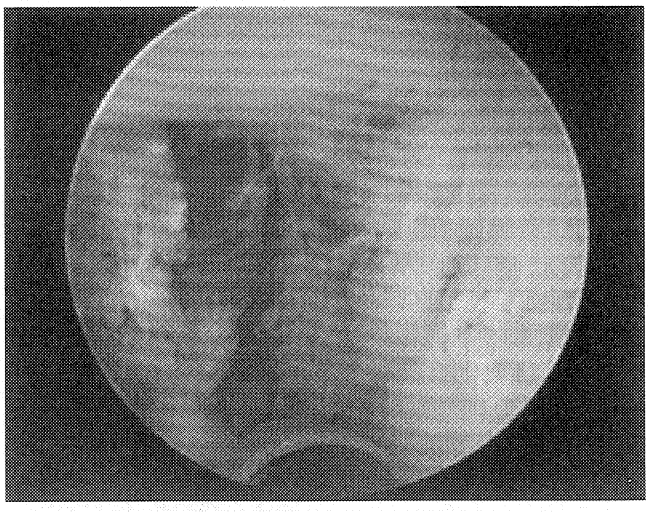

Figure 4–115. Adhesive capsulitis. Arthroscopic view from posterior of rotator interval region following capsulotomy in region of hyperemia and capsular thickening leading to increased external rotation of the adducted arm.

Stage 2. Stiffening phase (4–12 months): gradual decrease in GH motion and compromised activities of daily living.
Stage 3. Thawing phase (months to years): gradual motion return.

2. Arthroscopic Findings (Fig. 4–115). As described by Neviaser, the arthroscopic findings are as follows:

Stage 1. Patchy fibrinous synovitis without capsular contracture.
Stage 2. Capsular contraction, fibrinous adhesions, and synovitis.
Stage 3. Increased capsular contraction with resolving synovitis.
Stage 4. Severe capsular contraction.

3. Imaging. Plain radiographs are typically unremarkable, but are required to rule out unrecognized dislocations, fractures, severe osteoarthritis, locking osteophytes, or calcific tendonitis. Similarly, MRI is helpful to rule out associated disorders such as a rotator cuff tear. Shoulder arthrography may demonstrate a significant reduction in capsular volume and loss of the normal axillary recess (Fig. 4–116).

B. Post-Traumatic Shoulder Stiffness. Asymmetric loss of GH motion may occur following soft tissue trauma, fracture, or as a postoperative complication due to excessive scar formation or in association with prolonged immobilization following arthroscopic or open shoulder surgery. Scar formation or adhesions at the humeroscapular motion interface between the proximal humerus and overlying deltoid and conjoined tendon lead to motion loss. Depending on the etiology, the subdeltoid or the subcoracoid interface is obliterated with scar tissue. Motion loss is typically asymmetric and related to the areas affected by trauma or surgery. Contractures may also involve the rotator cuff and capsule.

C. Treatment
1. Nonsurgical. A supervised physical therapy program for 6 weeks combined with a home program for an additional 6 weeks will successfully treat the vast majority of patients with idiopathic adhesive capsulitis. This may be combined with NSAIDs and steroid injections, although their therapeutic benefit is not completely known. Post-traumatic shoulder stiffness or stiffness lasting greater than 24 months is unlikely to respond to nonsurgical treatment. If no improvement after 12 to 16 weeks of nonsurgical treatment is achieved, then surgical intervention is recommended.
2. Surgical. Surgical indications are evolving and many surgeons perform arthroscopic release and manipulation under anesthesia simultaneously. While patients with long-standing motion loss are less likely to respond to nonsurgical treatment, all patients should engage in physiotherapy before considering surgical intervention. The principal indication for surgery is an absolute failure to progress after a minimum of 3 months of physiotherapy.
   a. Manipulation under Anesthesia (MUA): MUA may be performed in isolation in patients with <6 months of symptoms who have otherwise failed 3 months of physiotherapy. However, patients who have >6 months of motion loss are less likely to respond favorably and may benefit from the addition of arthroscopic release (see below). MUA is contraindicated

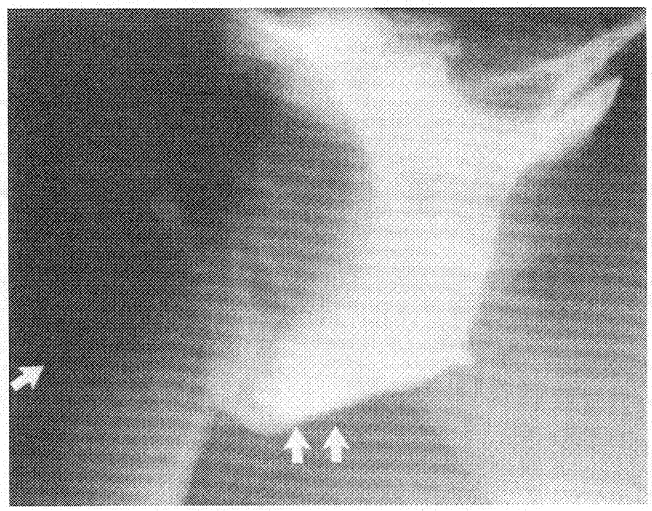

Figure 4–116. Arthrogram of a frozen shoulder demonstrating loss of axillary fold *(double arrows)*. (From Rockwood CA, Matsen FA III [eds]: The Shoulder. Philadelphia: WB Saunders, p 819.)

as an isolated procedure in osteopenic patients, longstanding diabetes, and in most cases of post-traumatic stiffness. Risks include fracture, dislocation, recurrent stiffness, and subscapularis rupture.

b. Arthroscopic Capsular Release and Lysis of Adhesions. This is a useful method to achieve significant motion in patients who have contraindications to MUA or who may benefit from release prior to MUA (i.e., post-traumatic stiffness). It allows the detection of coexisting pathology and a controlled capsular release. The procedure is facilitated with a slightly proximal posterior portal entering the superior aspect of the GH joint to minimize the chance of damage to the articular cartilage. The biceps and subscapularis tendons are typically encased in scar and require careful exposure through the use of soft tissue ablation devices and hand instruments. The rotator interval is released first, which usually allows significant increases in external rotation. The anteroinferior capsule is released next while avoiding the dependent fold of the axillary recess. If internal rotation is limited, a posterior capsular release is performed through the posterior portal while viewing anteriorly. Residual motion loss and disruption of the inferior capsule is achieved through gentle MUA. If arthroscopic release is unsuccessful in releasing the motion interface, then a limited open approach is used.

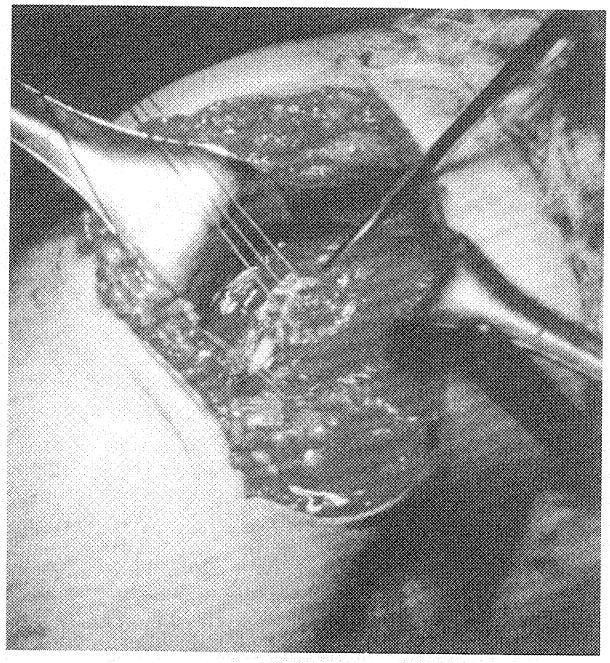

Figure 4–117. Repair of pectoralis major avulsion with sutures to be placed through a bone slot and drill hole configuration located at the native insertion site of the tendon just lateral to the long head of the biceps tendon.