Frozen Shoulder: Diagnosis and Management

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Abstract

"Frozen shoulder" comprises a group of conditions caused by different processes. Effective treatment depends on recognition of the underlying pathologic disorder in each individual case. Idiopathic adhesive capsulitis usually responds to nonoperative therapy or closed manipulation, but shoulder stiffness due to trauma or surgery may necessitate either an arthroscopic or an open-release procedure. Both of these technically demanding techniques are effective in restoring motion in cases of frozen shoulder refractory to nonoperative treatment.

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Although many researchers have considered the etiology of frozen shoulder,[1-4] few have presented an organized treatment approach based on the underlying diagnosis as well as on variations in natural history related to the specific diagnosis. The purpose of this article is to present an organized overview of the various causes of motion loss in the shoulder and the treatment options available for each.

Epidemiology and Natural History

"Frozen shoulder" is a general term denoting all causes of motion loss in the shoulder. The condition is one in which there is both active and passive limitation of motion due to soft-tissue contracture that results in a mechanical block. This soft-tissue contracture can occur in combination with other conditions, such as rotator cuff tear and degenerative arthritis. In the latter, joint incongruity may also limit motion. In all cases, the soft-tissue contractures must be treated concurrently with the other underlying or associated disorders. This article will address all causes of motion loss that involve soft-tissue contracture and scarring about the region of the glenohumeral joint.

When discussing the idiopathic form of motion loss in the shoulder, the term "primary adhesive capsulitis" is preferable to "frozen shoulder," as it more precisely describes the pathologic changes in the joint capsule. The pathogenesis of this idiopathic condition remains a subject of debate. Possible causes include immunologic, inflammatory, biochemical, and endocrine alterations.[1,3] Systemic disorders, such as diabetes mellitus, cardiovascular disease, and neurologic conditions, can also be contributing causes. In particular, patients with diabetes are at greater risk of adhesive capsulitis than the general population; the condition is often bilateral and resistant to all forms of treatment.[1,5]

Regardless of the biologic cause, adhesive capsulitis is characterized by thickening and contracture of the joint capsule,[6] which results in decreased intra-articular volume and capsular compliance so that glenohumeral motion is limited in all planes. The natural history of primary adhesive capsulitis is well described and has been termed benign because it tends to resolve over the course of 1 to 3 years.[3,7] Despite this favorable natural history, some patients are unwilling to endure the painful limitation of motion while they wait for resolution of the condition. Most patients will have some residual loss of motion even after many years, although the literature suggests that most do not have serious functional limitations or pain.[7]

Secondary, or acquired, shoulder stiffness develops when there is a known intrinsic, extrinsic, or systemic cause.[8-11] Examples include postsurgical and posttraumatic stiffness, which can occur with or without an associated fracture. Both types of shoulder stiffness usually occur in association with prolonged immobilization. While the management of primary adhe-

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sive capsulitis is usually conservative with physical therapy, most surgeons believe that acquired stiffness of the shoulder after surgical procedures for instability merits more aggressive treatment because of the potential unfavorable consequences.[1,8-14] For example, if an excessively tight anterior soft-tissue repair for instability causes an internal rotation contracture, it may result in chronic posterior subluxation of the humeral head, leading to incongruity of the joint and rapid deterioration of articular cartilage.[8,9] In the case of motion loss after immobilization of a traumatized shoulder, a supervised therapy program might be successful in restoring motion.

Normal Anatomy and Pathology

The inherently loose articulation of the normal shoulder joint is a necessary anatomic feature that permits the large range of multiplanar motion required for normal shoulder function. In this nonconstrained articulation, the larger humeral head has a nearly perfect congruity with the smaller osseous glenoid. The surface area of the articulation is enlarged by the fibrous glenoid labrum attached around its periphery. The stability of this joint is largely maintained by rotator cuff muscle action, which creates compression of the convex humeral head into the matched concave articular glenoid fossa. The glenohumeral ligaments and capsule, which are normally lax or under minimal tension during most shoulder rotations, function mainly at the extreme positions of rotation and translation to statically constrain the joint against excessive movement of the humeral head on the glenoid.[15]

During motion of the normal shoulder, the tightening and loosening of the glenohumeral ligaments and capsule encircling the humeral head are accompanied by lengthening and shortening of the rotator cuff and deltoid tendons and muscles.[16] Loss of motion in the shoulder can be the result of any condition that directly affects these structures or their ability to glide relative to one another during shoulder movements. A coexisting joint incongruity, such as arthritis or an osseous block due to a malunited or displaced greater tuberosity fracture, may be present. Both arthritis and fractures are also commonly associated with capsular contracture and scarring between soft-tissue planes.

The etiology of shoulder stiffness includes contracture or shortening of the capsule and glenohumeral ligaments, contracture or shortening of the extra-articular tissues, and scarring between the tissue planes of the shoulder (Fig. 1). The exact cause can usually be predicted with some degree of accuracy. For example, in primary adhesive capsulitis, the soft tissue principally affected is the joint capsule, while the rotator cuff and soft-tissue planes remain normal (Fig. 1, A). In cases of postsurgical stiffness, the specific disorder is related to the nature of the antecedent surgical procedure. When surgical treatment was for instability, capsular contracture and/or extra-articular constraint may be the cause of the motion loss. For example, when a capsular shift or a Bankart procedure for recurrent dislocation results in excessive loss of external rotation, the main cause of motion loss is capsular scarring rather than extra-articular constraint. In most instances, arthroscopic treatment can improve motion, although subscapularis shortening can sometimes be

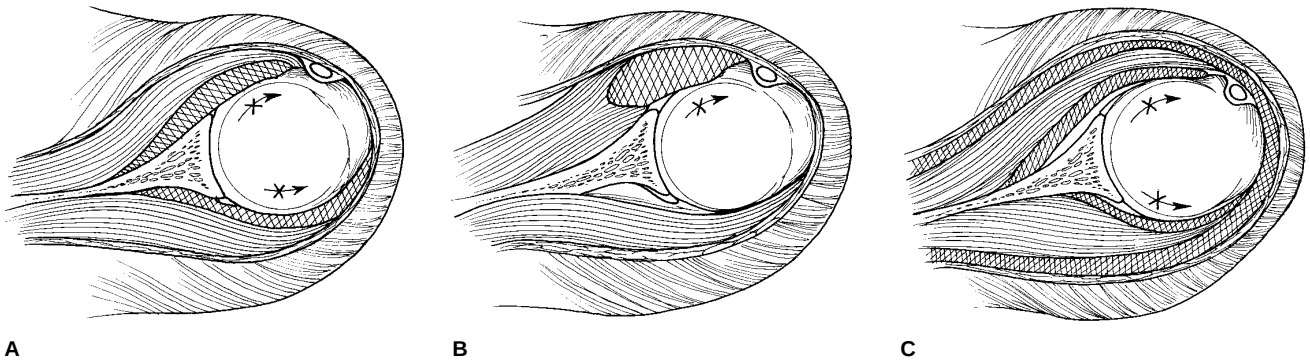

Fig. 1 Etiology of shoulder stiffness. **A,** Contracture of the capsule (cross-hatched areas) results in loss of all rotation (arrows). **B,** Extra-articular entrapment of the subscapularis (cross-hatched area) results in loss of external rotation (arrow). **C,** Scarring between tissue planes (cross-hatched areas) results in loss of all rotation (arrows).

a factor. In contrast, if stiffness after a Bristow procedure is due in part to entrapment of the subscapularis, an open procedure is necessary for definitive treatment (Fig. 1, B).[9,17]

If the shoulder has been immobilized for a prolonged period of time after a traumatic event or surgical procedure, associated scarring between tissue planes may concurrently restrict motion. For example, the motion loss that follows a fracture and prolonged immobilization is usually characterized by marked scarring in the tissue planes between the deltoid and the humerus, as well as in the deeper layers of the rotator cuff and capsule (Fig. 1, C).

Clinical Evaluation

Assessment of Motion

Shoulder motion must be assessed and documented carefully and consistently to obtain an accurate and ongoing measure of the efficacy of a given treatment. Both active and passive motion losses must be recorded and compared, since concomitant conditions, such as a rotator cuff tear, can result in loss of active motion in a shoulder that is also stiff due to adhesions. In patients with glenohumeral stiffness, there may often be the appearance of relatively good motion due to increased scapulothoracic motion or trunk lean. The examiner must be careful to identify and control these compensatory motions in order to measure only pure glenohumeral motion. Active shoulder flexion is measured anterior to the scapular plane, with the patient seated, and is referenced to the patient's thorax, not to a line vertical to the floor. This avoids measurement of any associated trunk tilt or increased scapulothoracic contribution to overall motion. Active pain-free motion is recorded. Active external and internal rotation are measured with the shoulder in adduction as well.

In some situations, pain inhibition may result in poor active motion. For example, in the case of a subacromial disorder, an injection of 1% lidocaine into this region will reveal the true active motion possible when pain is eliminated. Recording the increase in motion after injection helps differentiate motion loss due to pain from that due to a soft-tissue contracture. I perform this test on patients with painful flexion through an arc of at least 90 degrees. An individual with limited motion due to painful flexion from rotator cuff disease will have relief of pain with improved motion; an individual with a soft-tissue contracture will continue to have limited motion.

Passive motion should be evaluated with the patient supine, which restricts excessive scapulothoracic movement, therefore providing a more accurate assessment of pure glenohumeral rotation. Passive flexion, external rotation in adduction (arm at the side), external and internal rotation in abduction (arm to 90 degrees abduction), and cross-chest adduction must be measured.

Patterns of Motion Loss

Primary adhesive capsulitis is usually associated with global motion loss, whereas postsurgical or posttraumatic shoulder stiffness may present with global loss of motion in all planes or with a more discrete limitation of motion affecting some planes while relatively sparing others. Recognition of these different patterns of motion loss is important in planning surgical treatment when shoulder stiffness is refractory to nonoperative care. Motion loss often correlates with the location of a capsular contracture. For example, limitation of external rotation of the adducted shoulder is associated with contracture in the anterosuperior capsular region and the rotator interval; release of this area will usually improve the arc of motion.[12-14,18] Limitation of external rotation when the shoulder is abducted is usually associated with scarring in the anteroinferior region of the capsule. Limitation of internal rotation in adduction and abduction is associated with scarring of the posterior capsule, which is also reflected in loss of horizontal or cross-chest adduction.[15]

Even though these observations are useful guidelines for surgical treatment, it should be remembered that any capsular contracture will limit motion in more than one plane. Extra-articular contractures, such as subscapularis entrapment and scarring between tissue planes, may also contribute to global motion loss.

Secondary Findings

Both postsurgical and posttraumatic shoulder stiffness are characterized by the presence of some intrinsic shoulder disorder, such as rotator cuff disease, postsurgical scarring, or trauma to the soft tissues with or without fracture. The key finding in this setting is passive motion loss. Therefore, the physical examination should carefully document motion loss in all planes. Prolonged immobilization of the shoulder may also be a factor.

Some patients with postsurgical or posttraumatic shoulder stiffness, as well as those with primary adhesive capsulitis, will have pain patterns that suggest impingement or rotator cuff disease or another concomitant condition, even though there is no objective evidence. The

plain radiographs (specifically, the supraspinatus outlet view) may show a flat acromion (Bigliani type I),[19] but the magnetic resonance imaging study may be normal.

The biomechanical consequences of soft-tissue contractures may be "nonoutlet"-type impingement, in which the capsular contracture causes excessive translation of the humeral head on the glenoid during attempted shoulder rotation. Both anterior and posterior capsular contractures have been shown experimentally to cause increased superior translation of the humeral head during attempted flexion.[15,18] This abnormal superior movement of the humeral head has the effect of compressing the rotator cuff and subacromial bursa between the coracoacromial arch and the humeral head, thus causing the impingement-type symptoms. Some patients have partial pain relief with a subacromial injection of lidocaine. Experimental work by Harryman et al[18] has shown that capsular release eliminates abnormal translation and restores normal ball-and-socket kinematics (concentric rotation) to the glenohumeral joint.

Loss of glenohumeral motion will not only profoundly restrict overall upper extremity function but also alter the normal kinematic relationship of the glenohumeral and scapulothoracic joints. A compensatory increase in scapulothoracic motion can create additional symptoms, described by the patient as discomfort medial to the scapula.

Radiologic Studies

Plain Radiography

In most cases, but particularly in instances of primary adhesive capsulitis, radiographic studies do not help to clarify the causation of stiffness of the shoulder, but they do confirm the presence of a normal glenohumeral joint by identifying fractures, arthritis, or metallic implants that may be contributing to motion loss. Disuse osteoporosis may occasionally be evident, especially in patients who have the clinical features of reflex sympathetic dystrophy.

Arthrography

Many authors have asserted that arthrographic confirmation of decreased joint capacity, defined as the inability of the joint to accept more than 5 to 10 mL of contrast medium, is essential to a definitive diagnosis of adhesive capsulitis.[3,5,20] However, it has been shown that there is no direct correlation between arthrographic findings and motion loss.[20] Therefore, I do not use this test unless I want to rule out the possibility of a concomitant full-thickness rotator cuff tear.

Magnetic Resonance Imaging and Computed Tomography

Magnetic resonance imaging can be of use in selected cases in which there is a question of an associated disorder, such as a rotator cuff tear. Contrast medium–enhanced computed tomography can also provide information about articular injury and placement of hardware about the joint that might be impinging on the articular surface.

Physical Therapy

For most patients, a supervised physical therapy program will be successful in treating primary adhesive capsulitis,[1,2,4,7] but there has never been a careful study of the cost-effectiveness of this approach. Some orthopaedic surgeons do not believe that supervised therapy is important for these patients, and instead prefer a home program. In cases of idiopathic adhesive capsulitis, I combine a home program with supervised physical therapy three times a week for an initial 6-week trial. If the patient is making progress, the combined program is continued for an additional 6 weeks, followed by a home program.

In contrast to primary adhesive capsulitis, postsurgical or posttraumatic shoulder stiffness is often more resistant to a conservative approach.[1,8-11] The likely natural history can be predicted from an understanding of the pathologic features and the potential for future articular injury, such as that associated with a fixed subluxation of the joint. Although the literature clearly shows that limitation of external rotation to less than neutral can be associated with the development of arthritis, the minimal acceptable external rotation loss remains unclarified.[8,9,11] It is my impression that after instability surgery, limitation of external rotation to less than 60% of that of the contralateral shoulder should be treated aggressively to avoid development of arthritis from eccentric articular contact. A supervised physical therapy program is tried first, but my experience has been that even an aggressive stretching program by a knowledgeable shoulder therapist is often ineffective when there is a history of surgery or trauma. The length of time for which the supervised physical therapy program is continued depends on the cause of the motion loss and the patient's response to this treatment. If after 12 to 16 weeks the patient is getting progressively worse or there has been no improvement, an operative intervention is recommended. The risks and benefits of the operative approach have been

carefully described and may include fracture, neurovascular injury, residual stiffness, instability, and infection.

Selection of Operative Treatment

It is important to emphasize that treatment of primary adhesive capsulitis should not be considered while the patient is experiencing severe pain in addition to motion loss because this may represent the inflammatory phase of the disease. Neviaser and Neviaser[3] have pointed out that any surgical treatment in this stage will likely exacerbate the patient's motion loss by increasing capsular injury. It is important to wait until pain is present only at the end of the range of motion, indicating that the active inflammatory process has resolved.

When an operative approach is contemplated, it is also important to reemphasize that the diagnosis can be used to predict the likelihood of extra-articular scarring as well as capsular contracture. The surgical treatment must be tailored to address all of these factors. In all cases of refractory primary adhesive capsulitis, closed manipulation should be the initial approach. This approach is also used in relatively acute cases of motion loss after surgery or trauma when therapy has failed to restore motion. However, when there is a suspected or known extra-articular contracture (e.g., after a Bristow or Putti-Platt procedure), an open approach is usually required, and closed manipulation should not be attempted.

Closed Manipulation

Closed manipulation is performed with the patient under general anesthesia with drug-induced complete muscle relaxation. However, if the patient has marked osteopenia or underwent an antecedent surgical repair within the previous 3 months, this approach is contraindicated because of the risk of fracture, disruption of the soft-tissue repair, nerve injuries, and postmanipulation instability.[1,3-5]

The anesthetic technique is an extremely important aspect of the overall treatment. There must be complete muscle paralysis during the procedure. In my experience, while general anesthesia is an adequate method, patients often have subsequent pain that interferes with their therapy in the immediate postoperative period. It is therefore recommended that an interscalene block with a long-acting agent (bupivacaine) be used. The block is administered either as a single percutaneous injection or by placement of an indwelling interscalene catheter.[21-23] If a simple block is performed, it is repeated in the morning on the first and second postoperative days. Use of 0.5% bupivacaine will provide about 12 hours of analgesia, which will markedly reduce the patient's requirement for narcotics while increasing tolerance to physical therapy. If an interscalene catheter is used, a continuous slow drip of bupivacaine is administered.

The method of closed manipulation has been described by Neviaser and Neviaser[3] and Harryman.[1] The scapula is stabilized by one hand while the humerus is grasped with the other hand just above the elbow. The forearm should not be used as a lever in this manipulation. An attempt is first made to recover external rotation with the arm at the side. As firm pressure is gradually exerted, palpable and audible yielding of soft tissue occurs and motion improves. Although there may be some concern about fracture with this initial rotatory force, I attempt this motion first because it is easier to recover flexion and abduction if the greater tuberosity can be rotated out from under the acromion. If external rotation cannot be recovered, flexion is attempted, followed by abduction and then external and internal rotation. Finally, the arm is brought back into adduction and internally rotated. Motion is usually restored in all planes.

Arthroscopic Release

Although most patients with primary adhesive capsulitis respond to physical therapy, some will require closed manipulation to achieve and maintain sufficient improvement in motion. A small percentage of those patients will continue to have motion loss that is refractory even to manipulation of the shoulder under anesthesia. I have found the technique of arthroscopic capsular release very helpful in this situation. Similarly, in cases of postsurgical or posttraumatic shoulder stiffness in which closed manipulation fails, arthroscopic release can also be attempted, provided there is no extra-articular component to the motion loss.[9-14]

This approach continues to be controversial, however. Some surgeons suggest that arthroscopy is of little diagnostic and therapeutic value in treating patients with adhesive capsulitis of the shoulder.[3] Others suggest that the arthroscope may be helpful in delineating pathologic changes, documenting the results of closed manipulation, and treating concomitant intra-articular and subacromial disorders.[23,24] Over the past 4 years, I have used arthroscopic release in

selected cases in which motion loss appeared to be principally due to capsular contracture that was unresponsive despite closed manipulation. This technique has the advantage of allowing the detection of coexisting disorders, as well as permitting a controlled and precise capsular release. It also allows concurrent treatment of concomitant subacromial disease, as documented by preoperative temporary relief from local anesthetic injection and the intraoperative finding of an inflamed subacromial bursa. Furthermore, in cases of both idiopathic and postsurgical motion loss, I have found that the force of manual manipulation required to regain motion is greatly reduced by arthroscopically releasing the capsule before manipulating the shoulder. The arthroscopic technique also has the advantage over an open release of avoiding the morbidity associated with an extensive open surgical dissection. If motion loss remains unchanged intraoperatively after attempted arthroscopic release and manipulation, conversion to an open release is possible.

Anesthetic technique is an important component of the surgical plan for arthroscopic capsular release. The method of obtaining complete muscle paralysis with an interscalene block, as already described for closed manipulation, is recommended for both anterior and posterior release procedures.

Anterior Release

Anterior capsular release (Fig. 2) is performed with the patient seated in the "beach chair" position.[25] With use of this position, arm traction is unnecessary, although some surgeons prefer the added joint distraction produced when traction is applied with the patient in a lateral decubitus position. It is always difficult to insert the arthroscope into a stiff shoulder because of the capsular contracture and decreased joint volume, and articular injury from forceful insertion of the arthroscope is a concern. Chondral damage can be avoided by gently inserting the arthroscope over the humeral head. Although some surgeons[24] recommend use of a smaller arthroscope (3.9-mm diameter), such as that used for small-joint arthroscopy in the wrist, I have used the standard 30-degree arthroscope (5.5-mm diameter) without difficulty.

The biceps tendon is the first anatomic landmark that should be identified. It marks the upper edge of the "rotator interval region," which is formed by the anterior edge of the supraspinatus tendon and the cranial border of the subscapularis tendon.[18] This region is usually composed of a thick band of scar tissue, which obscures the

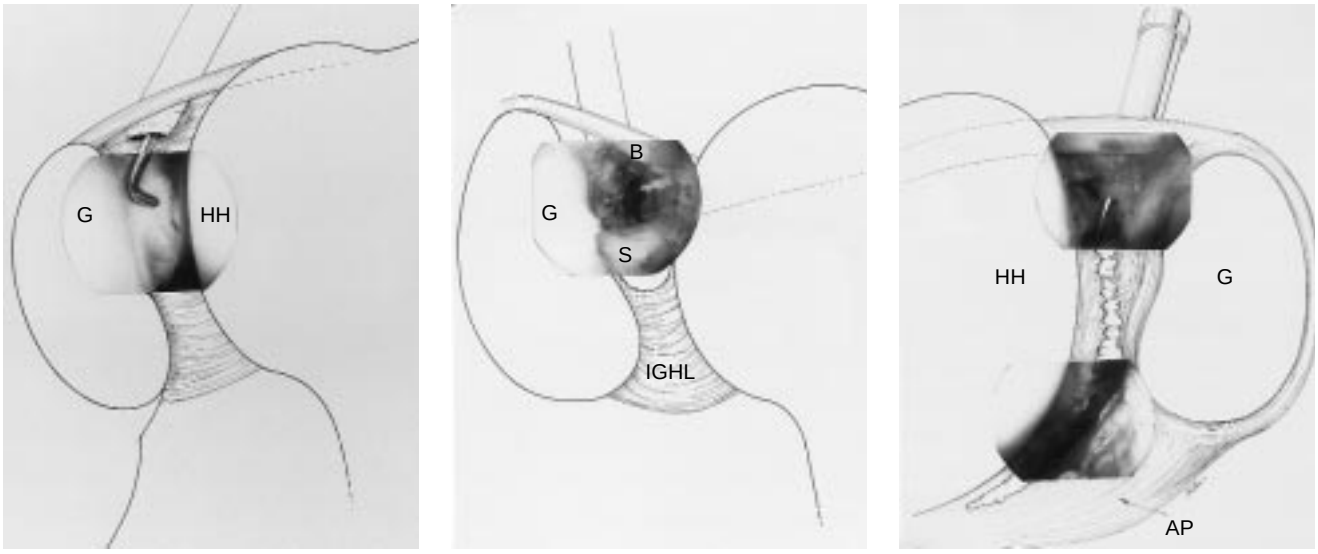

Fig. 2 Anterior arthroscopic capsular release is performed through an anterosuperior portal (G = glenoid; HH = humeral head). **A,** Left shoulder as viewed through a posterior portal. An arthroscopic probe is placed onto the thick wall of scarred capsule in the anterosuperior (rotator interval) region of the capsule. **B,** An arthroscopic electrocautery device has divided the anterosuperior region of the capsule, and the subscapularis tendon (S) and the remaining thickened inferior glenohumeral ligament (IGHL) are visible. B = biceps tendon. **C,** If necessary, the anteroinferior capsule is released down to the bottom of the glenoid, but not through the axillary pouch (AP).

normally visible upper edge of the subscapularis tendon. A varying amount of synovitis may also be present.

An arthroscopic cannula is inserted just beneath the biceps tendon, and the capsular scar tissue is divided with the use of an electrocautery device and a motorized shaver (Fig. 2, A). The capsular division begins superiorly from just anterior and inferior to the biceps tendon and continues inferiorly until the discrete upper edge of the subscapularis tendon is encountered (Fig. 2, B). This is a surgical release of the rotator-interval region of the capsule.[18] Both Ozaki et al[14] and Neer et al[12] have shown that such a release performed through an open approach is successful in restoring external rotation in shoulders with refractory adhesive capsulitis.

After release of this region of the capsule, the humeral head moves inferiorly and laterally, allowing more space in the joint for the arthroscope to be moved both anteriorly and inferiorly. The arthroscope is removed, and a manipulation is performed to restore motion in all planes. External rotation in adduction is usually restored with almost no manipulation force. The shoulder can then be manipulated into other planes with minimal force, usually accompanied by an audible and palpable yielding of tissue and improved motion. If there continues to be minimal or no improvement of motion in the remaining planes, the arthroscope is reinserted into the joint, and the remainder of the anteroinferior capsule is released. The capsular release is performed in the midcapsular region, extending down to the five-o'clock position for the right glenoid and down to the seven-o'clock position for the left glenoid (Fig. 2, C).

Some surgeons have expressed concern about risk to the axillary nerve with a capsular release in this region,[23] but I have not encountered any neurologic complications. The subscapularis is interposed between the anteroinferior capsule and the axillary nerve when the arm is adducted. It should be reemphasized that the axillary pouch should not be released with this technique. Release of the anteroinferior capsule will usually restore external rotation in abduction, but some patients will still lack internal rotation in abduction after release of the entire anterior capsule; if that is the case, the posterior capsule must be released as well.

When the capsular release is begun in the rotator interval region, it is extremely important to identify a discrete subscapularis tendon. If this structure cannot be identified, the procedure must be converted to an open release. Failure to curtail the arthroscopic release in this situation can result in division of the subscapularis tendon as well as the anterior capsule. When such a conversion has been necessary, distortion and scarring of the subscapularis and extra-articular scarring were identified, all of which were successfully managed with open release.

Posterior Release

In those few instances in which there is continued loss of internal rotation and flexion after an anterior release, arthroscopic release of the posterior capsule is also necessary to accomplish a global capsular release (Fig. 3). This technique is indicated when the loss of internal rotation in abduction exceeds 40 degrees compared with the contralateral side.

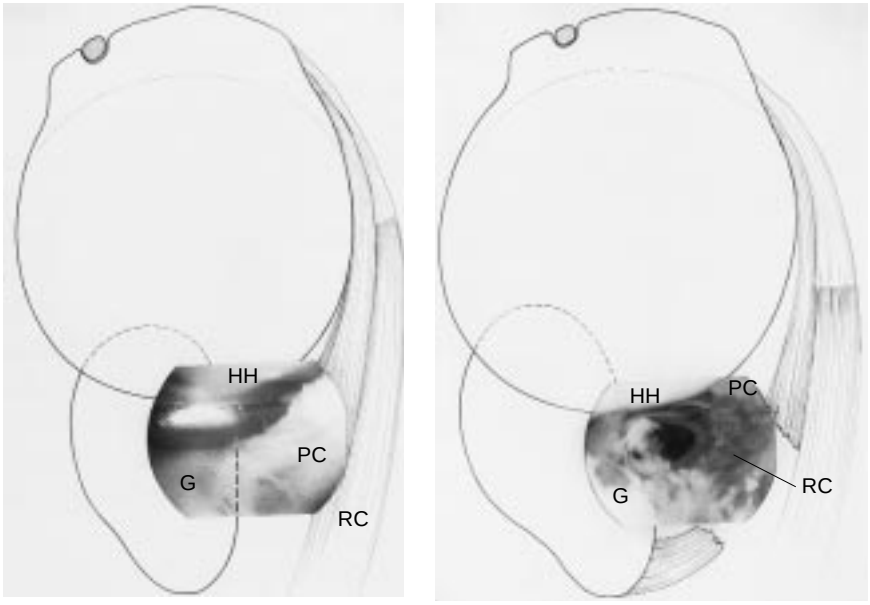

Fig. 3 Technique of arthroscopic posterior capsular release, depicted in a right shoulder viewed through an anterosuperior portal (G = glenoid; HH = humeral head; PC = posterior capsule; RC = rotator cuff). **A,** An arthroscopic shaver has been placed through the thickened posterior capsule. **B,** After release of the posterior capsule, the rotator cuff muscle can be seen.

A small subset of patients may have an isolated posterior capsular contracture characterized by motion loss primarily limited to internal rotation, cross-chest (horizontal) adduction, and flexion with relative preservation of external rotation. These patients often have impingement-type pain and in some cases have undergone acromioplasty or other surgery without relief. Their capsular contracture may result in a form of nonoutlet-type impingement by causing increased obligate anterosuperior translation during shoulder flexion and internal rotation. This condition is treated with a posterior capsular release to restore lost motion and normal kinematics.

I typically perform posterior release with the patient in the beach-chair position, although lateral decubitus positioning may be used alternatively. The arthroscope is placed through a cannula in an anterosuperior portal, while the arthroscopic sheath in the posterior portal is removed over a switching stick and is replaced with an operative cannula. An electrocautery device and a motorized shaver are then used to release the posterior capsule from just posterior to the biceps tendon origin down to the posteroinferior rim of the glenoid. The posterior capsule is always observed to be markedly thickened and without the redundancy seen in a normal shoulder, in which it is no more than 1 mm thick and is redundant when the arm is adducted and in neutral rotation (Fig. 3, A).

The arthroscopic release of the posterior capsule must be just at the glenoid rim. Because the muscle of the infraspinatus is superficial at this point, this is taken as the endpoint for capsular division (Fig. 3, B). If the capsular division is performed more laterally, there is a risk of dividing the infraspinatus tendon, because it conjoins with the capsule lateral to the joint line; this would potentially weaken external rotation by creating a tear in the infraspinatus tendon.

Open Release

In cases in which arthroscopic release is contraindicated or fails to restore motion, an open release can be performed.[8-13] Since arthroscopy is performed with the patient in a seated position, immediate conversion to an open approach is feasible.

As with closed manipulation and arthroscopic release, use of an interscalene block is the preferred anesthetic technique. A deltopectoral incision is used. Extensive scarring through all layers of the dissection is often identified. The deltopectoral interval is gently dissected, and the adhesions between the deltoid and the humerus are sharply released (Fig. 4, A). This must be done with care, as the axillary nerve may be at risk. The axillary nerve can often be palpated on the deep surface of the deltoid muscle approximately 3 to 5 cm below the lateral border of the acromion. The dissection is easier if the shoulder is abducted (Fig. 4, B), which allows the deltoid to become lax and more easily retracted. Internal rotation of the arm while gently retracting the deltoid muscle will allow anterior-to-posterior release of subdeltoid

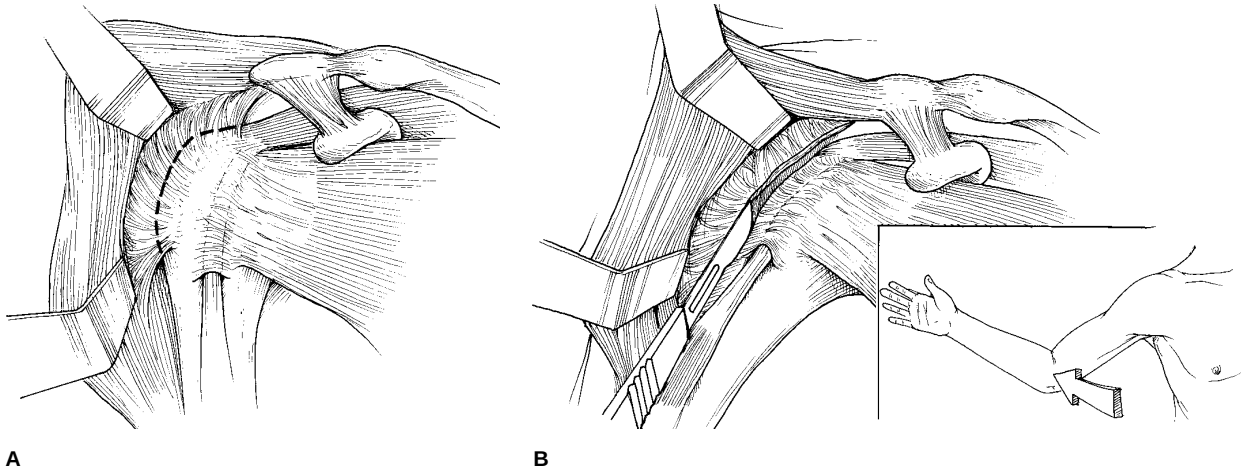

Fig. 4 Release of subdeltoid scar. **A,** Scar obliterates the plane between the deltoid and the proximal humerus and rotator cuff. **B,** Abduction of the shoulder allows the deltoid to relax and makes release of scar tissue easier.

adhesions until the deltoid can move freely over the proximal humerus when the arm is rotated.

The dissection then proceeds medially into the subacromial space. After the coracoacromial ligament is excised, the subacromial space may be found to be filled with dense scar adhesions between the rotator cuff and the acromion, which should be sharply released. Care must be taken with deltoid retraction, as overzealous retraction can either tear the muscle or avulse it from its origin or insertion. The conjoined tendon is then separated from the scarred area joining it to the underlying subscapularis and retracted medially. This can usually be accomplished with a combination of blunt and sharp dissection. The surgeon should be mindful of the musculocutaneous nerve; it is essential to keep the dissection lateral to the base of the coracoid process to prevent injury to neurovascular structures.

The superior border of the subscapularis tendon is identified, and the rotator interval is released, extending from the humerus to the coracoid.[12-14] As the dissection proceeds from superficial to deep between tissue layers, the shoulder should be gently manipulated to regain motion. If there is still marked limitation of external rotation due to scarring in the interval between the subscapularis and the capsule, the subscapularis is split between its fibers, and an elevator is used to release adhesions between its tendon and the underlying capsule. If there is still limitation of external rotation, a coronal Z-plasty lengthening of the subscapularis and capsule is performed (Fig. 5). This is done by dividing the scarred capsule and subscapularis tendon in the coronal plane so that the superficial half of the tendon remains attached to the muscle; the remaining deep half is divided at the glenoid and remains attached to the lesser tuberosity. The orientation of this coronal dissection can be guided by determining the thickness of the scarred tendon and capsular tissue once the rotator interval region is opened. The surgeon can usually both see and palpate the thickness of the anterior tissues through this interval.

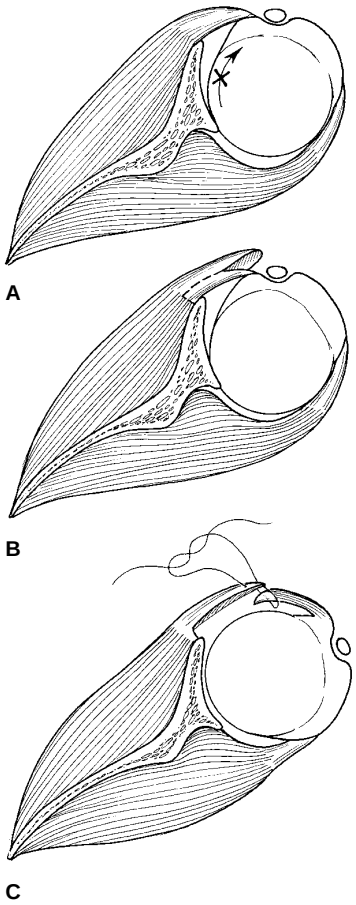

Fig. 5 Coronal Z-plasty lengthening of subscapularis and capsule. **A,** Entrapment of the subscapularis causes loss of external rotation (arrow). **B,** The subscapularis and capsule are divided in the coronal plane, beginning laterally at their insertion and developing a superficial layer. **C,** The capsule (deep layer) is then divided at the glenoid, and the arm is externally rotated for completion of the Z-plasty lengthening procedure.

When the anterior capsule and subscapularis have been dissected, the subscapularis is usually found to be entrapped in scar tissue. To achieve full mobility, it may be necessary to visualize and dissect the axillary nerve (Fig. 6, A and B). A vessel loupe is placed around the axillary nerve, and the subscapularis is released globally on its superior, inferior, deep, and superficial surfaces.

If abduction and internal rotation are still limited, the inferior and posterior capsule can be released through the joint. To do this, I place a humeral head retractor to displace the humeral head posteriorly and also put a blunt retractor beneath the inferior capsule to protect the axillary nerve (Fig. 6, C). The capsule is then released from inferior to posterior and superior under direct vision. The retractors are removed, and the shoulder is placed through the range of motion to evaluate motion gains. The arm is positioned in the maximum external rotation that will allow secure closure of the Z-plasty of the anterior capsule and subscapularis tendon with the use of large, nonabsorbable, braided suture. The patient usually gains at least 40 degrees of external rotation; however, this depends on the quality of the tendon and capsular tissue. Range of motion is assessed again to determine where there is tension on the soft-tissue repair and thus define a "safe zone" for early passive range-of-motion activity.

Postoperative Treatment

On the morning of the first postoperative day, either a repeat interscalene block is instituted or the interscalene catheter infusion is continued. Therapy is performed twice a day, in the morning and the

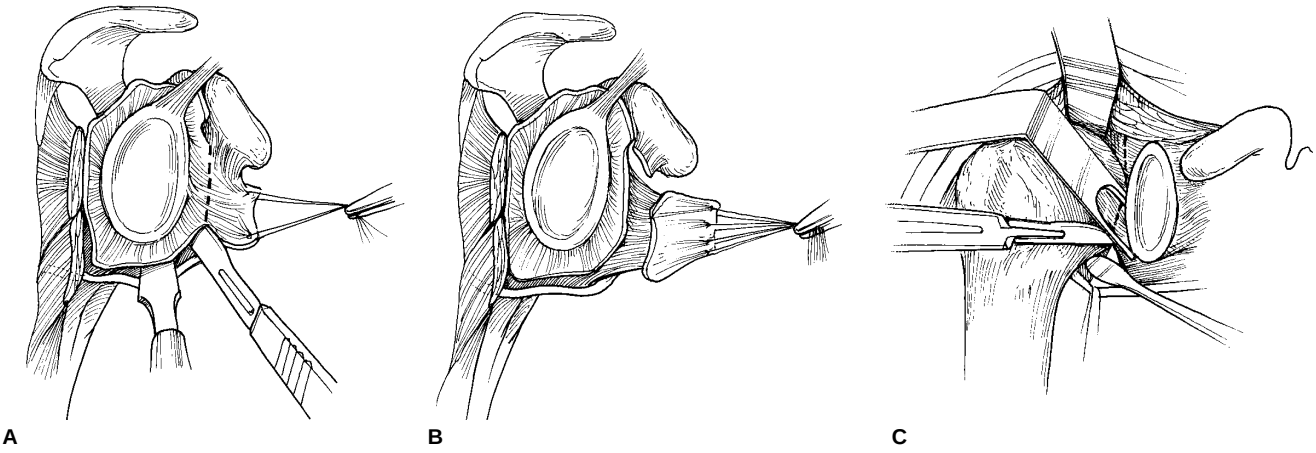

Fig. 6 Subscapularis mobilization in a left shoulder (humeral head omitted from drawing for better visualization). **A,** Sutures are placed into the contracted and shortened subscapularis tendon. An elevator protects the axillary nerve as the inferior and anterior capsules are divided. The coracohumeral ligament (dotted line) is also divided. **B,** After the capsular release and coracohumeral ligament division, the subscapularis is mobilized. **C,** An inferior and posterior capsular release can be performed if the axillary nerve is carefully mobilized and protected (shown elevated with a vessel loupe around it and an elevator beneath it).

afternoon. The patient is discharged after the second therapy session on the second postoperative day. Narcotic analgesia is used as necessary to supplement the interscalene analgesia. Therapy consists of an aggressive stretching program in all planes, and the patient is instructed in self-assisted stretching exercises as well. As the interscalene block usually results in only partial muscle paralysis, the patient can perform some stretching independently.

When a soft-tissue repair has been performed, as with a Z-plasty lengthening in the front of the shoulder, motion should be only passive for 4 weeks. The positions at which resistance is felt and the repair is observed to be under tension should be noted at the time of surgery and specified for the treating therapist as the limits of passive motion. After 4 weeks, an aggressive active-motion program with therapist-assisted stretching is begun.

The patient who has undergone an arthroscopic capsular release is discharged on the afternoon of the second postoperative day. The patient is encouraged to use the surgically treated arm for activities of daily living, and a sling is not worn. The patient continues a home program with self-assisted stretching and a pulley device, as well as supervised physical therapy on an outpatient basis five times per week for the first 2 weeks and then three times per week for the next 2 weeks.

After 4 weeks, the patient's progress is assessed, and the need for additional therapy is individualized. I do not recommend use of a continuous passive motion machine, as it has been my experience that this device is not reliable for maintaining motion gains. Whether the patient was treated arthroscopically or with open release, the strengthening phase of the postoperative therapy program is delayed until a nearly full pain-free arc of motion has been achieved. This usually takes about 3 months.

Summary

Proper treatment of motion loss in the shoulder depends on an initial recognition of the causative disorder and its natural history. Although a nonoperative approach of supervised or unsupervised therapy is usually successful in treating adhesive capsulitis, it may fail in patients whose stiffness is due to surgery or trauma. Closed manipulation restores motion in most cases of idiopathic adhesive capsulitis, but is often ineffective in the treatment of postsurgical motion loss. An arthroscopic release technique allows precise and controlled release of capsular contractures in cases of both idiopathic adhesive capsulitis and postsurgical motion loss. When there is an extra-articular component to the soft-tissue contracture, an open approach will improve motion. Postoperative treatment must emphasize pain control and maintenance of motion gains achieved at the time of manipulation or surgery.

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