

Shoulder Stiffness: Current Concepts and Concerns



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Abstract: Shoulder stiffness can be caused by various etiologies such as immobilization, trauma, or surgical interventions. The Upper Extremity Committee of ISAKOS defined the term “frozen shoulder” as idiopathic stiff shoulder, that is, without a known cause. Secondary stiff shoulder is a term that should be used to describe shoulder stiffness with a known cause. The pathophysiology of frozen shoulder is capsular fibrosis and inflammation with chondrogenesis, but the cause is still unknown. Conservative treatment is the primary choice. Pain control by oral medication, intra-articular injections with or without joint distension, and physical therapy are commonly used. In cases with refractory stiffness, manipulation under anesthesia or arthroscopic capsular release may be indicated. Because of various potential risks of complications with manipulations, arthroscopic capsular release is preferred. After the capsular release, stepwise rehabilitation is mandatory to achieve satisfactory outcome. **Level of Evidence:** Level V, evidence-based review.

The members of the Upper Extremity Committee of ISAKOS met in Amsterdam in May 2014 to create a consensus statement on the definition, classification, and treatment of the stiff shoulder. A detailed analysis was published in the monograph titled “Shoulder

Stiffness: Current Concepts and Concerns.”¹ This manuscript provides a summary of this meeting.

Classification and Epidemiology

The term “frozen shoulder” was coined by Codman to describe “many conditions which cause spasm of the short rotators or adhesions about the joint or bursae.”² Previous authors have divided joint stiffness into intrinsic and extrinsic causes.^{3,4} Because of its unique position, the capsule is the structure most at risk of developing a contracture. Primary capsular pathology has a specific cause, treatment, and prognosis; thus, it is considered as a separate category.^{5,6}

Our definitions are as follows:

Stiff Shoulder

This global term should be used to describe a patient who presents with a restricted range of motion. The etiology can be due to primary or secondary causes.

1. Frozen shoulder or primary idiopathic stiff shoulder. This term should be used exclusively to describe the primary idiopathic stiff shoulder. It develops without any trauma or specific shoulder disease period. If a patient has a condition that may be linked to a stiff shoulder, but not known to specifically cause the stiffness, it will still be considered idiopathic. Examples include predisposing factors such as diabetes, thyroid conditions, Dupuytren contracture, smoking, etc.

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Table 1. Key Points of Classification and Epidemiology

Classification	Cause	Examples
Frozen shoulder (primary stiff shoulder)	Unknown Predisposing conditions	Diabetes mellitus, Dupuytren contracture, thyroid disorders, myocardial infarction, Parkinson disease
Secondary stiff shoulder	Intra-articular Capsular Extra-articular Neurologic	Chondral lesion, labral tear, loose bodies After capsular injury, surgery, joint immobilization Muscle tightness, heterotopic ossification, skin scarring of burns Injuries to the cervical spine or brachial plexus

2. Secondary stiff shoulder. This term should be used to describe shoulder stiffness with a known cause, such as a stiff shoulder after trauma or surgery.

Adhesive Capsulitis

This term is not recommended, as it does not reflect the pathologic processes present.

Normal movement of a joint requires all components of the motor and lever system to be functional. Abnormal joint motion is the result of pathologic changes to normal anatomic structures. The pathologic process that creates a decreased range of motion of the shoulder is the sum of the premorbid state, the initial insult, the healing response, and the secondary changes that occur with time. The ISAKOS Upper Extremity Committee developed a classification that divides the causes of secondary stiff shoulder into 4 groups: intra-articular, capsular, extra-articular, and neurologic causes.

1. Intra-articular causes such as chondral lesions, labral tears, synovitis, or loose bodies can all be managed with articular surgery, such as arthroscopic debridement or repair.
2. Capsular causes include contracture after capsular injury or immobilization. Capsular release or resection can be performed as an open or arthroscopic procedure. If this is an isolated capsular pathology, then the prognosis is good.
3. Extra-articular causes include muscle tightness, heterotopic ossification, or skin contracture from burns. A localized lesion can be managed with an extra-articular resection or release of the offending structures. The prognosis is usually good.
4. Neurologic causes need treatment directed to the primary neurologic disorder.

The incidence of frozen shoulder is 2% to 5% of the general population.⁷ It affects 3.38 women and 2.36 men per 1,000 person-years.⁸ In a study in the Brazilian population on 88 stiff shoulder patients, of whom 52.3% were female, with a mean age at onset of 50.5 years, there was a family history of the disease in 9.5% of the patients.⁹ Males with frozen shoulder are at greater risk for longer recovery and greater disability.¹⁰⁻¹² Frozen shoulder can present

bilaterally, especially in diabetics,^{13,14} but it rarely affects the same shoulder twice.¹⁵

The cause of frozen shoulder is not clearly understood. Comorbidities are present in 85% of the patients with a frozen shoulder, and 37.5% had more than 3 comorbidities.⁹ Multivariate analysis identified thyrotoxicosis (especially hypothyroidism), diabetes, nephrolithiasis, and cancer being statistically significant. Other possible factors related to stiff shoulder include Parkinson disease, Dupuytren contracture, immobility, smoking, and neck and cardiac surgery. Frozen shoulder has a strong association with diabetes mellitus (Table 1).

Etiology

Predisposing Factors of Shoulder Stiffness

Epidemiologic data has established the relationship between diabetes mellitus and frozen shoulder, but a definitive pathophysiological association has not yet been reported. Presumed cause might be related to micro- and macrovascular disease. Patients with established diabetes have a greater likelihood of developing frozen shoulder than the normal population, and a diagnosis of frozen shoulder may be the first warning sign of a diabetic condition. Significant associations of frozen shoulder with the age of the patient and duration of diabetes have been reported.¹⁶⁻²¹ Other recognized predisposing factors for shoulder stiffness are immobilization, thyroid disorders, cardiac diseases (e.g., myocardial infarction), pulmonary disorders (e.g., tuberculosis, emphysema), neoplastic disorders, neurologic conditions (e.g., Parkinson disease, cerebral hemorrhage), medications (e.g., treatment with protease inhibitors for HIV), smoking, and Dupuytren contracture.^{18,22-32} Psychological factors may also play a role.³³ Intercellular adhesion molecule-1 (ICAM-1) plays a crucial role in synovial inflammation. An increased level of ICAM-1 in serum, capsular tissue, and fluid of the glenohumeral joint, in both patients with diabetes or frozen shoulder, has been reported. However, symptoms of frozen shoulder seem not to be correlated to ICAM-1 levels in the serum.^{34,35}

Secondary stiff shoulder can occur after shoulder surgery, but also after breast cancer surgery or cardiovascular procedures (e.g., cardiac catheterization in the axilla, coronary artery bypass grafting).³⁶⁻⁴⁰

Etiology of Frozen Shoulder

Frozen shoulder or primary shoulder stiffness occurs when no findings on history, examination, or imaging explain the onset of disease. Thus, the cause of frozen shoulder is still unknown. In other words, a stiff shoulder of unknown cause is called frozen shoulder. Cytokines might be involved in the inflammatory and fibrotic process, as an increased deposition of transforming growth factor, tumor necrosis factor- α , and platelet-derived growth factor in the capsule and in the synovium has been described.^{17-19,28,41-43} In particular, capsular fibrosis may result from the persistent stimulus of the matrix-bound transforming growth factor- β . Also notable is the absence of matrix metalloproteinase 14 in patients with frozen shoulder. Matrix metalloproteinase 14 is required to activate the proteolytic enzyme gelatinase A.⁴⁴

Multiple authors have identified an association between Dupuytren contracture and frozen shoulder, with rates of association up to 52%. Histologic changes of the glenohumeral joint capsule in patients affected by frozen shoulder have been compared with those seen in Dupuytren contracture of the hand. In both groups, no inflammation or synovial involvement have been reported. Inflammation might occur in an early stage of the disease, whereas fibrosis, as a result of collagen and matrix synthesis, occurs in a later stage.⁴⁴ Type I and III collagens were found in specimens of the capsular structures in patients with frozen shoulder, whereas vimentin, a cytocontractile protein present in Dupuytren disease, was found only in the anterior capsular structures. This may support the theory that contracture in patients with frozen shoulder selectively involves only the anterior capsule, and the fibroplasia the entire capsule.⁴⁵ Frozen shoulder has also been associated with hypoparathyroidism. It is still discussed if frozen shoulder is a manifestation of hypoparathyroidism or if primary hypoparathyroidism and frozen shoulder have a common genetic or immunologic basis.⁴⁵

Histology of Frozen Shoulder

During surgery, one can see inflamed synovia with neoangiogenesis (related to inflammation), and thickened and stiffened joint capsule (related to fibrosis),⁴⁶ but the pathogenesis of frozen shoulder is still unclear. It has been thought to be a combination of synovial inflammation and capsular fibrosis,^{47,48} similar to Dupuytren disease.⁴⁹ The cell density is significantly higher and the capsular tissue was significantly stiffer in frozen shoulders than in shoulders with rotator cuff

tears. Transformation from fibroblasts to myofibroblasts is a key to understanding the pathology of frozen shoulder. Cytokines and growth factors related to fibrosis and inflammation increased in the joint capsule from frozen shoulder. In addition to fibrosis and inflammation, which used to be considered the main pathology of frozen shoulders, chondrogenesis is likely to have a critical role in the pathogenesis of frozen shoulders.⁵⁰ There is no specific blood test for frozen shoulder.

Posttraumatic Shoulder Stiffness

Posttraumatic changes might cause limitations of the physiologic glenohumeral and scapulothoracic motion, resulting in stiff shoulder. No general consensus exists on the cause of posttraumatic shoulder stiffness. In contrast to the idiopathic shoulder stiffness or frozen shoulder, patients affected by posttraumatic shoulder stiffness are usually able to remember a specific trauma to the shoulder joint, often combined with a certain period of immobilization. Identifying structural damage of the joint is important for treatment planning, although determining the real causative factor is not always simple, as patients might correlate the onset of symptoms to some minor trauma. Additionally, in a specific group of patients, posttraumatic shoulder stiffness does not correlate with any specific structural change that can be diagnosed by today's image-based or clinical methods. Causes for onset of posttraumatic shoulder stiffness are dynamic and very complex. They can grossly be structured into 3 major groups: extra-articular adhesions impairing the motion interface and its gliding structures or neurophysiological deficits (e.g., hematomas, large burn injuries, injuries to the cervical spine, plexus); direct injuries to the intra-articular structures (e.g., fractures, loose bodies, tendon ruptures); intra-articular scarring of the capsule or secondary fibrosis of the capsular structures after a defined trauma.^{51,52}

Postoperative Shoulder Stiffness After Rotator Cuff Repair

One of the most important and frequent complications of surgical repair of the rotator cuff is the postoperative shoulder stiffness, with an incidence of 4.9% to 32.7% of all repairs.⁵³⁻⁵⁵ Etiology and the most efficacious method(s) by which to predict and prevent occurrence of postoperative shoulder stiffness after rotator cuff repair are still discussed. Postoperative shoulder stiffness may be related to tear morphology, postoperative immobilization, glenohumeral adhesion, capsular contracture, or underlying predisposing patient comorbidities such as diabetes. Postoperative shoulder stiffness is most likely caused by the surgical violation of tissue planes, resulting in contractures of the soft tissue surrounding the articulations, pathologic

connections between motion interfaces, and/or shortened muscle-tendon unit excursion. Shoulder stiffness after rotator cuff surgery is typically global, but posterior capsular stiffness is often accentuated. Patients may also complain of pain and/or weakness. Three different basic forms of shoulder stiffness after rotator cuff repair have been defined: intra-articular capsular contracture; contractures and/or adhesions of gliding tendons such as the rotator cuff or biceps; and adhesions within the extra-articular glenohumeral motion interface. Shoulder stiffness after rotator cuff repair can be categorized broadly into 1 of 4 subgroups: stiffness without re-tear; stiffness with re-tear; stiffness with untreated osteoarthritis; and stiffness with deltoid or neurovascular injury with or without re-tear.⁵⁶

These patients can be difficult to assess and manage. A detailed history is required to determine if there has been preoperative stiffness or postoperative infection. On examination, the wound should be assessed for swelling and erythema. The deltoid and rotator cuff should be assessed for wasting and strength. The passive and active range of motion should be measured. Radiographs are performed to assess the glenohumeral joint, humeral superior migration, and anchor placement. Unfortunately, ultrasonograph and magnetic resonance image (MRI) are difficult to interpret in this group of patients. From all of this information, determine the cause of stiffness.

If activity-limiting stiffness persists after 9 months postoperatively, arthroscopic release of adhesions with capsular release and subsequent manipulation under anesthesia should be considered. Common intraoperative findings in this group of patients are extensive subacromial and subdeltoid bursal adhesions. These adhesions can be impressively thick and are particularly prevalent at the sites of previous soft-tissue trauma where tissue planes have been violated by arthroscopic instrumentation or cannulas.

Postoperative positioning in an abduction brace can help keep the inferior glenohumeral joint capsule stretched out and, if the repair is felt to be strong enough to withstand the stress, passive stretching exercises can be initiated immediately after surgery. Nevertheless, the importance of implementing early postoperative motion is still discussed.^{57,58} Too-rapid advancing motion protocols could also lead to an inflammatory response and increased risk of re-tear.⁵⁹⁻⁶⁴

Different contributing factors have been proposed to the development of shoulder stiffness after rotator cuff repair: tears less than 3 cm in diameter; partial articular-sided tears; workers' compensation; age less than 50 years; calcific tendinosis; concomitant labral repair; diabetes; single tendon cuff repair; limited preoperative active forward elevation, active external rotation, and passive internal rotation; and open repair.^{55,58,65}

Postoperative Shoulder Stiffness After Surgical Repair of Shoulder Instability

Both open and arthroscopic instability repair procedures can result in loss of external rotation range of motion.⁶⁶⁻⁷⁶ Loss of range of motion has also been shown to be greater after revision surgery than primary repair.^{77,78} Open repair has a higher incidence of this complication, most probably because of longer periods of specific immobilization and compromise to subscapularis muscle and capsular structure. A subscapularis-splitting approach may help to minimize range of motion loss. Procedures that expose the deltopectoral interval or tighten the anterior glenohumeral capsule have also shown higher rates of contracture.^{72,79}

Histologic evaluations of stiff shoulders after surgical repair of shoulder instability have reported that the most common areas for the development of fibrosis include the coracohumeral ligament, between the rotator cuff and the overlying acromion, the rotator interval, between the conjoined tendon and the subscapularis, and the musculature and bursae in the scapulothoracic region.⁸⁰ A significantly smaller capsular volume in the area of the axillary recess has also been described.

The rotator interval opens with internal rotation and closes with external rotation. Loss of external rotation, as a result of overtightening, might therefore occur if the arm is positioned in internal rotation during imbrication.⁸¹

Postoperative Shoulder Stiffness After Arthroplasty

Significant stiffness is a relatively rare complication in patients undergoing unconstrained shoulder arthroplasty. When considering stiffness, it is important to investigate whether it was already present preoperatively and whether motion was gained postoperatively and then stiffness occurred or if motion was never regained. In a recent report, the French group of Walch and Boileau found significant stiffness to be relatively rare in unconstrained shoulder arthroplasty patients.⁸² According to them, only 0.9% of total shoulder arthroplasty patients had a problem with stiffness. The most common cases of stiffness are associated with either posttraumatic arthroplasty or in the presence of subtle infection, most commonly *Propionibacterium acnes*.⁸³

Other Causes of Shoulder Stiffness

Shoulder stiffness can be secondary to different causes. **Neurologic** disorders affecting either or both upper and lower motor neurons can result in shoulder stiffness. Upper motor neuron damage may result from progressively degenerative (multiple sclerosis, amyotrophic lateral sclerosis), traumatic (brain or spinal cord

Table 2. Key Points of Etiology

Predisposing conditions of frozen shoulder	Diabetes mellitus, Dupuytren contracture, thyroid disorders, cardiac diseases, pulmonary disorders, neoplasms
Etiology of frozen shoulder	Cytokines might be involved in the inflammatory and fibrotic process.
Histology of frozen shoulder	Fibrogenesis, angiogenesis, chondrogenesis
Posttraumatic stiffness	Extra-articular adhesions (e.g., hematoma) Capsular fibrosis
Postoperative stiffness after rotator cuff repair	Intra-articular injuries or scarring (e.g., fracture) Capsular contracture Adhesion of gliding tendons (cuff tendon, biceps)
Postoperative stiffness after instability surgery	Adhesion of extra-articular motion interface Loss of external rotation: typical finding Fibrosis of the coracohumeral ligament, rotator interval capsule, and between the conjoint tendon and subscapularis Rotator interval closure with the arm in internal rotation may limit external rotation
Postoperative stiffness after arthroplasty	Relatively rare after unconstrained shoulder arthroplasty

injury), or cardiovascular disorders (cerebral palsy, cerebrovascular accident).

Lower motor neuron damage commonly results from external trauma (crush, stretch, division), but also compressive (paralabral cyst, suprascapular notch stenosis) or inflammatory mechanisms (brachial neuritis, Parsonage-Turner syndrome) may be responsible.

A stiff shoulder can also be caused by extra-articular conditions, such as heterotopic ossification. Heterotopic ossification is the deposition of bone in extraskeletal tissue, with the presence of cellular, protein, and mineral components of bone.⁸⁴ Usually, it develops around the hip or the elbow. Heterotopic ossification rarely involves the nearby articular surfaces; thus, joint stiffness occurs in the setting of a normal articular surface. Fascial restrictions and muscular tightness can also be the extra-articular cause of shoulder stiffness (Table 2).⁴⁸

Anatomy and Biomechanics

Muscular Imbalance and Altered Kinematics

Frozen shoulder patients may exhibit significant alterations in shoulder kinematics, including increased elevation and upward scapular rotation. This indicates compensation due to lack of capsular extensibility as well as a change in central nervous system motor patterning with adaptive postural deviations such as anterior shoulder compensation or increased thoracic kyphosis.⁸⁵⁻⁸⁷ Upper trapezius muscles are more activated than lower trapezius, creating an imbalance of the scapular stabilizers. Patients with frozen shoulder have higher EMG ratios of the upper trapezius to lower trapezius during arm elevation compared with asymptomatic subjects, indicating a muscular imbalance.^{88,89} The subscapularis may have an independent role in frozen shoulder that is separate from the known capsular contractures. It has been proposed that a greater loss of external rotation at 45 degrees of

abduction versus 90 degrees of abduction indicates more specific involvement of the subscapularis.⁹⁰

Capsular-Ligamentous Complex

The glenohumeral joint capsule, coracohumeral ligament, and glenohumeral ligaments (superior, middle and inferior) make up the capsular ligamentous complex. These structures connect the humerus and glenoid and also attach to the glenoid rim via the labrum.⁹¹

The rotator interval is a confluence of tissue in a triangular shape located between the anterior supraspinatus leading edge and the upper border of the subscapularis tendon. The rotator interval includes the coracohumeral ligament, the superior glenohumeral ligament, and the anterior-superior capsule. Contracture of these structures, the most commonly affected in frozen shoulder, results clinically in loss of flexion and external rotation in adduction.^{81,92-95}

The anterior inferior quadrant of the glenohumeral joint contains the anterior inferior capsule and the middle glenohumeral ligament. The anterior capsule is frequently adhered to the middle glenohumeral ligament and the subscapularis in patients with frozen shoulder resulting in further limitations of external rotation at the side.^{7,92,96}

Research based on arthroscopic findings suggests that there is contracture of the inferior capsular tissues.⁴⁶ This results in significant losses of abduction, forward flexion, and both external and internal rotations owing to involvement of the anterior and posterior bands of the inferior glenohumeral ligament as well as the remainder of the capsular tissue.⁹²

Clinically, internal rotation is affected later during the development of frozen shoulder. When the posterior capsule is involved, this results in loss of internal rotation both in adduction and in abduction.^{92,97,98} There has been recent evidence to suggest the existence of a posterior superior glenohumeral ligament.⁹⁹ The relevance of this ligament to frozen shoulder is unknown.

Table 3. Key Points of Anatomy and Biomechanics

Alteration in scapular kinematics	Lack of capsular extensibility Change in motor firing pattern of periscapular muscles with adaptive postural deviations
Pathomechanics of capsular ligamentous complex	Anterosuperior capsule and upper subscapularis muscle: lead to loss of external rotation in adduction Inferior capsule: leads to significant losses of abduction, forward flexion, and both external and internal rotations Posterosuperior capsule: rarely involved but causes loss of internal rotation both in adduction and in abduction

The expected effect of contracture of this ligament would be a loss of internal rotation in adduction (Table 3).

Examinations

Clinical History

The diagnosis of frozen shoulder is based on the history of an insidious onset.^{7,51,100,101} An important differential aspect is that in frozen shoulder, there is no anatomic abnormality, no imaging abnormality, and no systemic abnormality or disease that can directly explain these typical signs and symptoms.⁵¹ Three phases have been described.¹⁰² The first phase or the freezing phase is distinguished mainly by pain and gradually increasing stiffness and lasts 2 to 9 months. Pain starts often in the night and is sometimes so severe that it prevents the patient from sleeping on the affected side. Gradually, pain increases and is present all the time. During the freezing phase, when pain is the principal problem and stiffness is not yet evident, differentiation between other causes of shoulder pain on purely clinical grounds is difficult. In the second phase or the frozen phase lasting from 4 to 12 months, pain is less severe and there is minor discomfort in the shoulder, but stiffness is substantial. In the third phase or the thawing phase, function is gradually restored and pain is resolved. This can take a further 5 to 26 months. Some patients may regain full use of their shoulder within 12 to 18 months, whereas others may have persistent symptoms for several more months.

Clinical Examination

On examination, essential measurements of the range of motion are forward flexion, external rotation with the arm at the side, and internal rotation in the standing position. The typical finding in frozen shoulder is a global reduction of the range of motion, by definition in 2 or more planes, and equal in passive and active examination.^{47,96,103} In addition, measuring the external rotation with the patient in the supine position can be helpful through stabilization of the scapula.¹⁰⁴ Loss of external rotation is typically an initial symptom. Important feature is that the reduction in the range of

motion is fixed, and not influenced by pain. This means that even in a fully anesthetized patient, the range of motion will be similarly reduced. Strength is unaffected when tested within the pain-free range. During the evolution from the freezing phase to the frozen phase, pain diminishes, but stiffness persists. The Upper Extremity Committee of ISAKOS advises as a guideline: if the range of motion is less than 100 degrees in forward flexion, less than 10 degrees in external rotation, and less than L5 level in internal rotation, we define this as global limitation of the range of motion, which is typically seen in the frozen phase of frozen shoulder.

Imaging

In the frozen phase with global motion loss, a typical MRI finding is a thickening and shortening of the inferior capsule with concomitant volume reduction of the axillary pouch. During the freezing phase, on the other hand, no abnormalities can be seen on imaging modalities. Most shoulders with full-thickness rotator cuff tears demonstrate passive range-of-motion deficit, but this deficit is usually slight or moderate and does not show severe and global motion loss. In patients with severe and global motion loss, 91% showed an intact rotator cuff on MRI or ultrasonography, only 9% showed a partial-thickness cuff tear, and none of them showed a full-thickness tear (Table 4).¹⁰⁵

Conservative Treatment

Oral Medication and Intra-articular Injection

Although NSAIDs are one of the most common interventions in treating frozen shoulder, only 1 comparative study was found in which treatment with oral analgesics was inferior to all other conservative interventions.¹⁰⁶ There is evidence that treatment of frozen shoulder with only NSAIDs has no effect on the natural course of frozen shoulder.¹⁰⁷ Moreover, some reports describe that a short course of oral steroids for frozen shoulder may be of significant short-term benefit than placebo or no treatment, but the effect is not maintained at 6 weeks. It is suggested that further improvement can be obtained with a more prolonged course of therapy and/or more gradual withdrawal of

Table 4. Key Points of Examinations

Clinical history	History of an insidious onset without any anatomic or imaging abnormalities
Clinical examination	Global reduction of the range of motion and equal in passive and active ranges of motion (flexion < 100°, external rotation < 10° degrees, internal rotation < L5)
Imaging	Thickening and shortening of the inferior capsule on magnetic resonance imaging. No other abnormalities.

treatment.¹⁰⁸ Intra-articular corticosteroid injection is a common intervention in treating frozen shoulder. There is a suggestion that intra-articular steroid injection may be beneficial in the short term for frozen shoulder. Multiple injections are beneficial until 16 weeks from the date of the first injection. There is no evidence supporting more than 6 injections. There is no difference in the outcome between lower and higher doses of corticosteroids.^{107,109} Intra-articular corticosteroid injections compared with subacromial injections result in greater improvements in terms of pain and passive range of motion in the short term but did not result in better outcomes in the long term.¹¹⁰ In the setting of diabetes mellitus, steroid injections may be contraindicated.^{111,112}

Physical Therapy

Physical therapy is one of the most used interventions in frozen shoulder. Nevertheless, there is a persistent discussion about the effect, the intensity, and the timing. Some publications consider that there is no effect of physical therapy by itself on the outcome of the disease. Most studies with good end results include physical therapy as an adjunctive intervention together with mobilization, joint distension, steroid injection, or capsular release. A large number of studies have been performed on the different techniques of physical therapy, manual therapy, mobilization techniques, and stretching techniques. Small differences in the outcomes are reported, but in most cases either the inclusion criteria are not stringently defined or the results are operator-dependent. There is no evidence supporting passive capsular and muscle stretching, soft tissue manipulation, and joint mobilization techniques.¹¹³ A program of home exercises is as effective as practice-based therapy.¹¹⁴ In general and according to the existing evidence, physical therapy program to treat this disease should be executed in the pain-free zone during the frozen phase. Gently handled therapy is more effective than intensive therapy. Progressive, painless scapulothoracic exercises should be added to achieve decrease in pain, increase in the range of motion, and recovery of the scapulohumeral rhythm. During the thawing phase, exercise intensity advances and the range of motion increases so as to achieve maximum activities-of-daily-living functionality.¹⁰⁴

Joint Distension

Capsular distension is often performed, but there is no proven superiority of joint distension over other therapeutic options.¹¹⁵ There is no difference in effects between frozen shoulder (primary stiff shoulder) and secondary stiff shoulder.¹¹⁶ Generally, improvement in function is maintained. There is no difference in the outcome shown between adding steroids and different volumes. More than 1 repeated distension after 2 weeks has no added effect.¹¹⁷ In a study comparing joint distension with manipulation under anesthesia, similar improvements were noted after 6 months (Table 5).¹¹⁸

Surgical Treatment

Manipulation Under Anesthesia

Manipulation under anesthesia is commonly used when other conservative treatment does not work in patients with frozen shoulder (Video 1, available at www.arthroscopyjournal.org). This is an effective treatment option for refractory frozen shoulder.^{119,120} Randomized clinical trials comparing manipulation under anesthesia and other treatment options showed no significant difference in the outcome.^{118,121,122} It is as effective as but less costly than arthroscopic capsular release.¹²¹ Manipulation can be performed under general anesthesia,⁴⁷ but it can also be performed under interscalene block.^{123,124} Serious complications are fractures or neurovascular lesions and should be avoided with the use of a short lever arm with slow and gentle motion. High-quality evidence needs to be established.¹²¹ Although manipulation under anesthesia improves shoulder elevation and abduction, rotation remains limited because of surgeons' concerns about forceful rotation maneuvers.^{125,126}

Arthroscopic Capsular Release

Even though the mainstay of treatment for frozen shoulder is conservative, in refractory cases, arthroscopic capsular release can be performed (Video 2, available at www.arthroscopyjournal.org). Arthroscopic capsular release includes a few steps (block anesthesia, capsular releases, ligament splitting, resection of adhesions, and shoulder manipulation) followed by a postprocedural oral cortisone plan and a supervised rehabilitation program.^{51,127}

Table 5. Key Points of Conservative Treatment

NSAIDs	Have no proven effect on the natural course of the disease.
Oral steroids	Have a significant short-term benefit and probably more lasting effects with a prolonged therapy course and gradual withdrawal of treatment.
Steroid injections	Yield substantial improvement. More than 6 injections or higher dosages have no additive effect.
Joint distension	Provides sustainable improvements in motion.
Physical therapy	Is an adjunct to other conservative or surgical treatment modalities. Painless and gently handled exercises increase range of motion and prevent further adhesions.

NSAIDs, nonsteroidal anti-inflammatory drugs.

This surgical procedure provides precise and controlled release of the capsule and ligaments, reducing the risk of traumatic complications observed after forceful manipulation. Release of the capsule and the involved structures with a radiofrequency device delays healing, which prevents adhesion formation. The procedure can be performed with the patient fully awake under inter-scalene block, which boosts patients' confidence and adherence to the physical therapy protocol.

Additional oral postoperative steroid therapy decreases pain and allows for a better pain control during the early rehabilitation program.

This technique plays a role in patients with frozen shoulder and recalcitrant symptoms. Studies have shown that arthroscopic capsular release provides complete and long-lasting improvement in shoulder pain and function, faster than any other treatment modality. In addition, long-term (mean 7 years, range 4-13 years) outcomes after arthroscopic capsular release are encouraging.^{126,128}

There is considerable debate regarding the optimal amount of surgical release during arthroscopic capsular release. Along with the coracohumeral ligament and rotator interval release, some authors advocate releasing other structures, including the subscapularis tendon, the inferior and posterior capsule, and the global capsule,^{93,95} to improve shoulder elevation and internal and external rotations. Pearsall et al. reported that preoperative assessment of motion loss should guide the degree of capsular release.¹²⁹

There is consensus to postpone surgical treatment of coexistent shoulder injuries such as rotator cuff repair or biceps tenodesis until recovery of shoulder motion and relief of symptoms. In diabetic patients, the less encouraging surgical results than in nondiabetic counterparts are possibly related to suboptimal glycemic control and the presence of microvascular derangement.

The best timing to proceed with arthroscopic capsular release is also a matter of controversy. Traditionally, surgeons used to wait for failure of conservative measures for 6 to 12 months. There is no evidence whether

an early surgical intervention would shorten the recovery time of this disease.

A recent systematic review compared outcomes after manipulation under anesthesia or arthroscopic capsular release.¹³⁰ The differences between the 2 groups were minimal. As the quality of evidence is low, it is difficult to provide any strong clinical recommendation based on the existing published literature. Randomized controlled trials are needed to support the use of either treatment modality, but at the present time, in patients with refractory frozen shoulder, arthroscopic capsular release emerges as a suitable option.

The procedure is performed with the scope at the posterior portal, and the anterolateral portal is established using an outside-in technique just anterior to the biceps tendon. Rotator interval release with a radiofrequency wand entails complete dissection of the coracohumeral ligament, while freeing anterior structures allows for complete recovery of external rotation. The radiofrequency device is used through the posterior portal to release the posterior capsule and the posterior band of the inferior glenohumeral ligament. Occasionally, gentle shoulder manipulation is needed to obtain full forward flexion and abduction, which in turn, extends the reach of the cutting device to the inferior capsule. The surgeon must be aware of the anatomic location of the axillary nerve.¹³¹ Any release of the inferior capsule should be performed close to the labrum to avoid the risk of nerve injury. Lastly, adhesions located at the subacromial space are released.

Postoperative pain control is critical for obtaining early range of motion and improving rehabilitation compliance. Many options are available that can decrease dependence on opioids, including oral or intra-articular steroid injections, interscalene nerve block or catheters, and more recently, liposomal bupivacaine injectable suspensions. As a positive incentive to the patient, it is critical to share the immediate postoperative range of motion with the patient.

The reported benefits of a brief hospital admission after capsular release are early physical therapy, improved postoperative pain control, and early initiation and compliant use of continuous passive

Table 6. Key Points of Surgical Treatment

Manipulation under anaesthesia	Can be as effective as but is less costly than arthroscopic capsular release. Complications such as a fracture should be avoided with use of a short lever arm with slow and gentle motion.
Arthroscopic capsular release	With the advantage of a precise release of the involved structures (coracohumeral ligament and capsule) leads to a faster and long-lasting recovery in recalcitrant cases.
Postoperative management	A multimodal approach with pain control (blocks or catheters) and early mobilization are critical to achieve satisfactory results.

motion.^{96,132,133} However, there is no evidence to clearly support routine clinical application of continuous passive motion. A systematic review confirmed that continuous passive motion may provide better pain reduction in the early phase of rehabilitation¹³⁴ but cited no difference in long-term range of motion or function.¹³⁵

Most authors recommend early discontinuation of sling immobilization. Some have even advocated brief immobilization in 90 degrees of abduction and external rotation.^{96,128,132} Unfortunately, data are lacking to recommend any particular regimen of immobilization.

The protocol specifically designed to address the patient undergoing arthroscopic capsular release consists of 4 phases of rehabilitation: early motion, active motion, strengthening, and advanced strengthening. It is important to note that phases will overlap to varying degrees. Treatment should always be modified according to each individual patient's needs. Ideally, treatment should begin within 24 hours to minimize scar tissue formation. Goals for the early phase of rehabilitation are to control pain and inflammation, prevent scar tissue and adhesions, and increase shoulder range of motion. It is important to tailor postoperative management to a patient's individual needs and accommodate individual goals (Table 6).

Limitations

The ISAKOS Upper Extremity Committee undertook this expert meeting to collect the highest available evidence on cause, diagnosis, and management of the stiff shoulder from a practical surgeons' point of view. The level of evidence in most studies, however, is low, and is often supported by a single or a few noncontrolled cohort studies or expert opinions. In most studies, a clear definition of "stiff" or "frozen" shoulder is not mentioned, which made it difficult to compare the results. As only descriptive but no causal histologic or biochemical factors are known and as there is a paucity of evidence on frozen shoulder, further research is needed to obtain a better understanding of this disease.

In this article, we defined different types of stiff shoulders by clinical appearances. Reasonable scientific evidence to support interventions could only be found for medication and conservative treatment. We were

not able to define clear guidelines on the indications for and choices of more aggressive interventions like manipulation under anesthesia, joint distension, and open or arthroscopic capsular release. These results should be interpreted with care.

Summary

There is very little evidence in the field of shoulder stiffness, and buildup of high-quality evidence is strongly required. The ISAKOS Upper Extremity Committee has made a start by developing a classification system based on clinical and imaging findings and summarizing the existing evidence on treatment modalities in shoulder stiffness. The goal of this review is to support the orthopaedic surgeons in differentiating between the 2 main types of shoulder stiffness, that is, frozen shoulder and secondary stiff shoulder, and to provide them with the evidence to choose the best patient-tailored intervention. In frozen shoulder or primary stiff shoulder, conservative therapy, including injection therapy and corticosteroid medication, should be the first choice. Arthroscopic capsular release can be offered to patients with refractory symptoms. In secondary stiff shoulder, defining and treating the underlying cause of restricted mobility is essential.

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