Arthroscopic Capsular Release for Stiff Shoulders: Effect of Etiology on Outcomes

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Purpose: The etiology, pathogenesis, time course, and response to treatment of stiff shoulder pathology is still under investigation and debate. This prospective study evaluated arthroscopic capsular release to treat stiff shoulder pathology that was resistant to conservative management. The etiology of the shoulder stiffness was categorized and analyzed for effect on outcomes. **Type of Study:** Operative technique and prospective evaluation. **Methods:** In 68 stiff shoulders (41 in women, 27 in men) that underwent arthroscopic capsular release, 5 distinct etiologies were identified: postsurgical in 20, idiopathic in 17, post-traumatic in 15, diabetic in 8, and impingement syndrome (prior primary impingement developing stiffness) in 8. Average age was 50 years (range, 29 to 72), and follow-up averaged 3 years (range, 2 to 8). Prior to this procedure, duration of symptoms averaged 7.3 months (range, 3 to 48), and formal physical therapy averaged 3.7 months (range, 1 to 12). Preoperative average American Shoulder and Elbow Surgeons Score (ASES) was 35.5 (range, 10 to 77), median Simple Shoulder Test (SST) was 3 (0 to 10), and median Visual Analog Score (VAS) for pain was 6 (0 to 10). Average active forward elevation (FE) was 92°, external rotation (ER) at side was 12°, and median internal rotation (IR) was to the buttock. All patients underwent arthroscopic capsular release with a standard aftercare protocol. **Results:** The study population showed significant improvement ($P < .0001$) for all outcome scores and active motion parameters. Average and median outcome parameters for the population, with improvement in parenthesis were: ASES 93 (+57.5), SST 10 (+7), VAS 0 (−6), FE 165° (+73°), ER at side 56° (+44°), and IR to T −12 (+7 spinal segments). The time in formal physical therapy averaged 2.3 months (2 to 20 weeks) and time to attain final, pain-free range of motion averaged 2.8 months (1 to 6). Outcomes for, and between, each etiology were analyzed. There was no difference in time to final motion between the etiologic groups. **Conclusions:** Stiff shoulder pathology can result from a variety of differing etiologic factors. Arthroscopic capsular release was equally effective across the 5 identified etiologic groups, and provided significant pain relief, restoration of motion, and function within an average of 3 months. **Key Words:** Shoulder—Adhesive capsulitis—Arthroscopy—Capsular release—Etiology.

The diagnosis of frozen shoulder, or adhesive capsulitis, is one of exclusion. The disorder is characterized by a limitation of both active and passive range of motion of the glenohumeral joint that is not primarily due to an underlying condition such as arthritis, rotator cuff tear, cervical radiculopathy, or peripheral neuropathy. The etiology, diagnostic criteria, classification terminology, treatment methods, and natural history of this condition are still under debate and investigation.[1-7] It is believed that the pathophysiology first involves inflammation of the synovium, then subsynovial fibrosis, leading to capsular fibrosis, thickening, and ultimately contracture of the glenohumeral capsule.[4,8]

Even as our knowledge of the potential pathologic components has increased, the etiology, pathogenesis, time course, and response to treatment of stiff shoulder pathology are still under investigation and debate. Treatment methods have encompassed a wide variety

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of techniques. Reports on patient home therapy,[5,9] formal physiotherapy,[5,6,9,10] and intra-articular steroid injections and brisement,[11-13] have each shown success for adhesive capsulitis. When patients do not respond to conservative management, other treatment options are available. Operative intervention in the form of manipulation under anesthesia has restored motion and decreased pain,[7,14-17] but it has been associated with complications such as fracture, tendon rupture, and neurologic injury.[6,13,17]There are reports that manipulation has not been effective and patients remained symptomatic.[7,18-22] There are reports that patients cannot undergo effective manipulation under anesthesia and require conversion to an arthroscopic release.[21,23] The more invasive open release through a deltopectoral approach has been used to restore glenohumeral motion.[6,16,24,25]

Arthroscopy has been used in a variety of ways with regard to frozen shoulder. It has been used solely for diagnosis, for brisement,[12,13] to observe post-manipulation effect and then to treat associated pathology,[26-28] and to grossly and histologically document the pathology.[4,8,29] Recently the arthroscope has been used to perform the capsular release itself. The reports on arthroscopic capsular release and arthroscopic capsulotomy have shown promising results comparable to other treatment options.[21-23,29-34]

We encountered a series of patients who exhibited severe limitation of glenohumeral joint motion, shoulder dysfunction, pain, and sleep disturbance. They had been symptomatic for an average of 7 months and in treatment for a known diagnosis of "frozen shoulder" for an average of 3 months without resolution of their pain and with no progress in restoring motion and function. They were quite frustrated with their pain and lack of progress.

The purpose of this prospective study was to evaluate an arthroscopic capsular release technique for the treatment of symptomatic stiff shoulder pathology that was recalcitrant to conservative treatment. The efficacy of the technique was analyzed by outcome scores; range of motion gains; and the effect of treatment on the time course of the disease process. The etiology of the stiff shoulder pathology was categorized. The etiologic categories were analyzed for their effect on outcomes and the time course of recovery.

METHODS

This prospective evaluation involved a consecutive series of arthroscopic capsular release procedures for stiff shoulder pathology performed with consistent technique by one surgeon. There were 68 shoulders with an average age of 50 years (range, 29 to 72), in 41 women and 27 men. The dominant shoulder was involved in 47% of the cases. The average follow-up was 3 years (range, 2 to 8 years). The average duration of symptoms was 7.3 months (range, 3 to 48 months), and patients had been in formal physical therapy an average of 3.7 months (range, 1 to 12 months) with no resolution of their symptoms.

All patients presented with limitation of shoulder motion (both passive and active), pain, functional limitations, and sleep disturbance. The diagnosis of frozen shoulder was given due to the clinical presentation. Patients with an anatomic, degenerative, radiologic, or neurologic component to the stiffness were excluded. Therefore, any patient with a history or clinical evidence of shoulder arthrosis, cervical radiculopathy, peripheral neuropathy, calcific tendinitis, malunited fractures, previous shoulder arthroplasty, or previous instability surgery was excluded from this study. We also excluded from this series patients who had undergone any procedures in which the goal had been to tighten the capsule, such as capsulorrhaphy. Subacromial space surgeries, such as acromioplasty or rotator cuff repairs, were not excluded.

We defined a recalcitrant frozen shoulder as causing shoulder complaints for at least 5 months, with a known diagnosis of shoulder stiffness with clinical symptoms of at least 3 months' duration. Most importantly, patients had no response to or worsening symptoms from a surgeon-directed therapy program, which was directed toward that known stiffness diagnosis. All patients had prior shoulder complaints and had undergone treatment for those symptoms, but the correct diagnosis of shoulder stiffness had not been made and previous therapy efforts had not been directed toward regaining motion. Therefore, in addition to any previous therapy efforts, patients in this series must have been in the surgeon-directed therapy program for at least 6 weeks. They must also have continued to exhibit night pain, limitation of active and passive motion, and shoulder dysfunction that affected their occupation, recreation, or sleep before considering arthroscopic capsular release.[30]

Our conservative therapy program consisted of a gentle home stretching program in conjunction with formal physical therapy that concentrated on stretching, range of motion, and pain control. No strengthening or forceful motion exercises were allowed. Patients typically did home stretching 2 or 3 times per day, and underwent therapy 2 times per week. Nonsteroidal anti-inflammatory medication and narcotic

analgesia were used as needed. Seventeen patients received glenohumeral corticosteroid injections, and 17 patients underwent subacromial space corticosteroid injections with only transient improvement before coming to arthroscopic capsular release.

We made an effort to categorize the shoulders by etiology of the stiffness. Five distinct etiologic categories were identified: idiopathic, diabetic, post-traumatic, postsurgical, and prior subacromial impingement syndrome. This etiologic classification allowed us to compare the outcome scores, range of motion, and time course of recovery between etiologies, and to the study population as a whole. Of the 68 shoulders, 20 were classified as having a postsurgical etiology, 17 were classified as idiopathic, 15 as post-traumatic, 8 as diabetic, and 8 were classified as being from antecedent subacromial impingement syndrome. These eight prior impingement cases were unique in that the patients were initially evaluated and treated and followed up by the author. They did not manifest any limitation of motion initially and were treated for impingement pathology. They developed significant stiffness over a period of 6 months to 2 years after the initial evaluation.

Patients who had undergone surgery on the affected shoulder were questioned about symptoms and possible stiffness before that surgery. If the procedure was believed to be the inciting event to the now-recalcitrant stiffness, the shoulders were categorized as postsurgical (Table 1). As Table 1 shows, these surgeries primarily involved subacromial space and rotator cuff procedures. If after patient history or physical examination, a trauma was determined to be the inciting event to progression of shoulder stiffness, the shoulders were categorized as post-traumatic. The types of trauma are shown in Table 2.

All patients were prospectively evaluated using history, physical examination, plain radiographs, and patient outcome scores for the American Shoulder and Elbow Surgeons (ASES) score, Simple Shoulder Test (SST), and Visual Analog Score (VAS) for pain. Active and passive range of motion was determined by goniometer in relationship to the long axis of the thorax. Motion was recorded for forward elevation (FE) in the scapular plane, external rotation (ER) of the side, and internal rotation (IR) to a spinal segment.

Table 1. *Postsurgical Etiology*

Etiology	No.
Arthroscopic Acromioplasty	8
Open RC Repair	7
Mini Open RC Repair	3
Open Coracoidplasty*	1
ORIF Surgical Neck Fracture with Blade Plate	1

Abbreviations: RC, rotator cuff; ORIF, open reduction internal fixation.

*Performed for subcoracoid impingement.

Table 2. *Posttraumatic Etiology*

Etiology	No.
Nondisplaced Greater Tuberosity Fracture	5
Fall	4
Force to Arm While Lifting	3
Car Accident	2
Dislocation	1

Statistical Methods

The data in this analysis fell into 2 categories. Ranges of motion and scores over a broad range of possible values were categorized as continuous variables and were tested by either the 1- or 2-sample *t* test. These tests evaluate equality or lack of equality between means and are based on the Student *t* test distribution. Ordered measures of limited possible range characterized by evenly spaced jumps in possible values, such as the SST, VAS, and active IR, were considered discrete ordinal categorical variables and were tested by the 1- or 2-sample nonparametric Wilcoxon statistic. These Wilcoxon statistics test for equality or lack of equality between median values and have no underlying distributional assumptions.

Preoperative and postoperative comparisons were made using the 1-sample or paired *t* test or, where appropriate, the paired Wilcoxon signed rank test. Tests between etiologic groups were performed with the 2-sample *t* test, or where appropriate, the 2-sample Wilcoxon rank sum test.

Operative Technique

All patients underwent arthroscopic capsular release performed by the author. A long-acting scalene regional anesthetic in combination with general anesthesia was used. All procedures were performed in the beach chair position. Passive range of motion was assessed under anesthesia, but no manipulation was performed. The glenohumeral joint was insufflated with saline with an 18-gauge spinal needle from the posterior approach. The humeral head was tightly opposed to the glenoid, and the capsule was thicker and less compliant than in other shoulder conditions.

The blunt trocar and sheath were carefully introduced angling toward the biceps origin to prevent joint surface damage. Intra-articular placement was confirmed by fluid backflow through the sheath.

Visualization of the contracted synovitic arthroscopic triangle between the long head of biceps, the upper surface of the subscapularis, and the glenoid rim provided orientation. The pump was then connected and started at 200 mL/min of flow and 20 mm Hg pressure. A spinal needle was placed anteriorly just lateral to the coracoid tip, into the arthroscopic triangle and was seen with the arthroscope. A smooth 7-mm cannula was then placed.

A gelatinous, proliferative synovial material at the root of the biceps, over the rotator cuff interval, and typically down the anterior capsule into the axillary pouch was encountered in 55 of 68 (81%) cases (Fig 1). This material was debrided with a motorized shaver. It was interesting to note that this material did not bleed as it was debrided. It was scraped free of the capsule without any friability or nuisance bleeding. As much synovial hyperplasia as could easily be debrided was performed at this time.

The capsular release began with the rotator cuff interval. At this point, the cannula was removed anteriorly, and a 3.0-mm 90° Arthrowand (Arthrocare, Sunnyvale, CA) was introduced down the track of the cannula and into the joint. The 90° tip allowed the surgeon to rotate the instrument and cut into the thickened capsule and back toward the entry point of the instrument. This was especially helpful in the rotator cuff interval because the coracohumeral ligament and interval tissue was thick and shortened (Fig 2). The interval was released along the base of the arthroscopic triangle medially, from the biceps down to the upper subscapularis, paralleling the glenoid rim. Then the cautery was used to release the tissue parallel to the thickened upper border of the subscapularis. The release of the interval and anterosuperior capsule allowed increased mobility of the joint to proceed with the capsular release inferiorly.

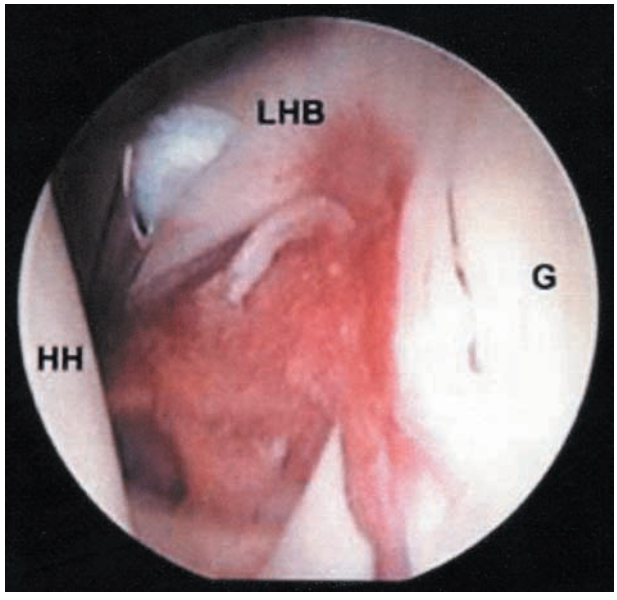

FIGURE 1. Initial arthroscopic view of the contracted arthroscopic triangle in a left shoulder. The vast majority of cases, regardless of etiology, exhibited similar pathology, which included gelatinous synovial hyperplasia, capsular thickening, and a contracted rotator interval. LHB, long head biceps; G, glenoid; HH, humeral head.

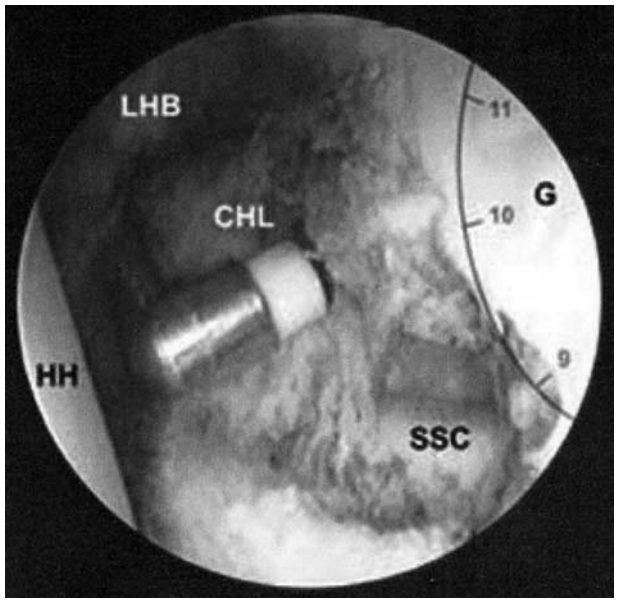

FIGURE 2. The bipolar cautery releases the thickened, contracted coracohumeral ligament, opening the rotator interval. (The clockface overlay orients the reader to position within the joint.) LHB, long head biceps; G, glenoid; HH, humeral head; CHL, coracohumeral ligament; SSC, subscapularis.

The cautery was then used to release the anterior capsule beginning just below the biceps origin. The capsule was released just off the glenoid rim, preserving the labrum. The cautery tip was always oriented parallel to the anterior glenoid neck and placed between the labrum and capsular attachment (Fig 3). The goal was to create an extralabral capsular release from the glenoid. The subscapularis tendon was not released or violated. By lowering the arthroscope into the joint parallel to the articular surface, the surgeon could gently distract the joint, facilitating exposure as the release moved into the anteroinferior area. To

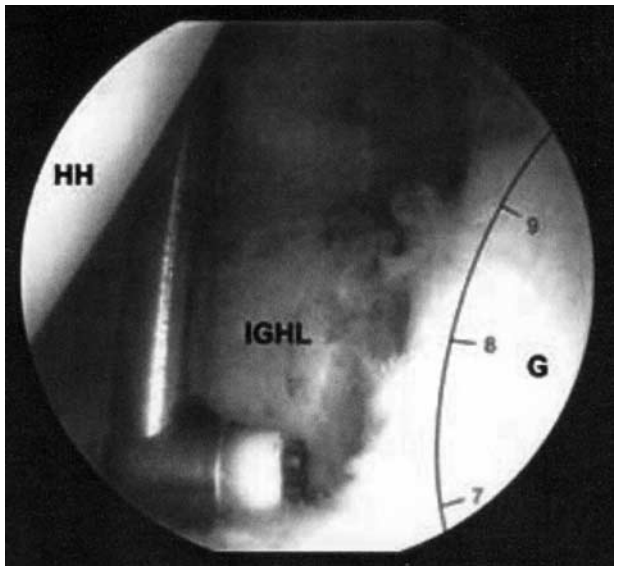

Figure 3. The release must begin superiorly to allow mobility to proceed with the inferior capsular release. The thickened, contracted capsule is released in an extralabral fashion. HH, humeral head; IGHL, inferior glenoid humeral ligament; G, glenoid.

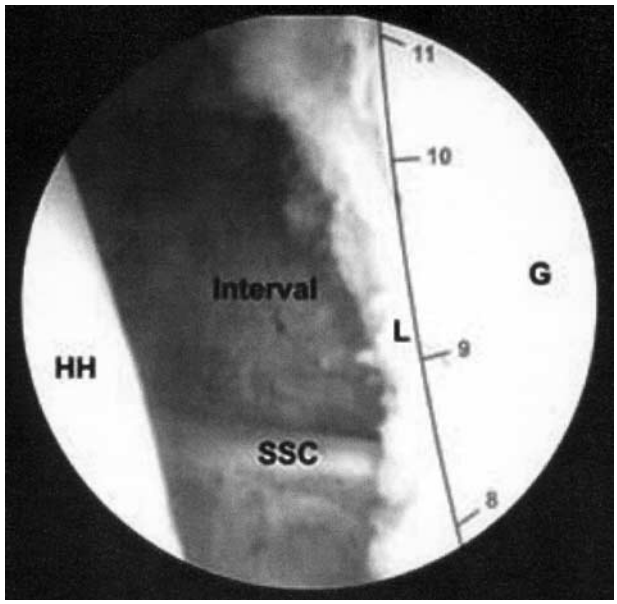

Figure 5. After complete release, the interval is wide open and the subscapularis is freely mobile. HH, humeral head; SSC, subscapularis; L, labrum; G, glenoid.

release the inferior capsule, the 90° bipolar Arthrowand tip was oriented up or away from the axillary nerve and placed in the axilla of the capsular release anteroinferiorly (Fig 4). The capsule was released from the inferior glenoid rim and carried down around the 6 o'clock position.

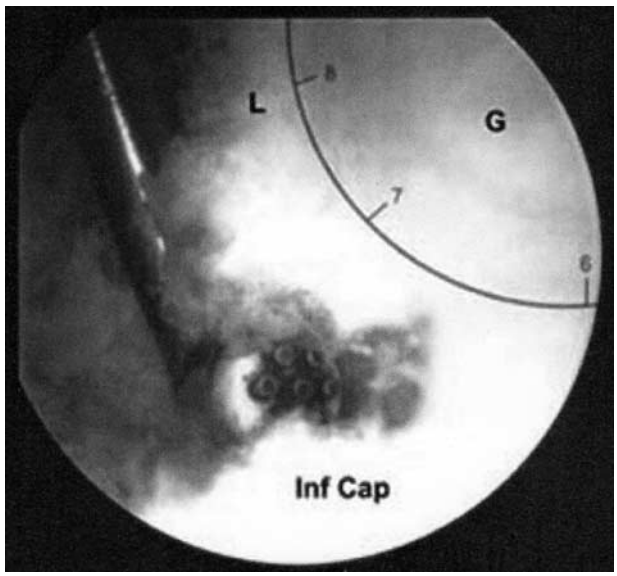

Figure 4. To release the inferior capsule, the 90° tip of the bipolar cautery is rotated up or away from the axillary nerve. The capsular release of the inferior capsule then proceeds along the inferior glenoid. L, labrum; G, glenoid.

The arthroscope was then positioned in the anterior portal. The posterior capsule was assessed for involvement. Forty-five patients (66%) in this series required posterior capsular release. The cautery was placed through the posterior portal. The release began over the posterosuperior recess, where the disease can obliterate this recess and tether the supraspinatus tendon to the glenoid rim. The release was carried down the posterior capsule to meet the previous anterior release. Posteriorly, muscle fibers of the infraspinatus were seen as the thickened capsule was released.

After the complete circumferential release was completed, the shoulder was put through a gentle range of motion with proximal humeral pressure. Abduction, scapular elevation, ER at the side, ER and abduction, and then internal rotation in abduction were performed. Typically, there was a small feeling of giving way, rather than the sudden snap or release feeling during traditional manipulations.

The arthroscope was reintroduced into the glenohumeral joint. Typically, there was minimal to no bleeding. The subscapularis tendon was now visible and was freely mobile (Fig 5), and the arthroscope could now navigate easily through the joint.

The subacromial space was evaluated in all patients. In patients with previous impingement syndrome,

trauma, postsurgery, or nondisplaced greater tuberosity fractures, the subacromial space required evaluation, debridement, and, in some cases, acromioplasty. In this series, 24 patients required subacromial debridement and acromioplasties, including all patients with an impingement etiology. An additional 11 patients required subacromial adhesiolysis and debridement without formal acromioplasty. Therefore, 51% of cases required subacromial work. In the idiopathic etiology group, however, only 3 (18%) required subacromial debridement or acromioplasty. If the subacromial space was without adhesions or was not inflamed, the subacromial space was not debrided. This was to minimize the surgical trauma, bleeding, and postoperative pain and swelling. If there was preoperative clinical acromioclavicular (AC) joint arthralgia, arthroscopic AC resection was performed. Five patients in this series had arthroscopic AC resection. The portals were closed in a routine fashion. A sling and swathe were applied to support the arm. We used a derotation wedge of our own design attached to the sling. This wedge places the forearm and thus the shoulder in a neutral rotation position instead of internal rotation. A passive cold wrap device was applied to the shoulder.

Regardless of etiology, the majority of patients showed qualitatively remarkably similar glenohumeral pathology. Gelatinous, red, synovitic proliferative material around the long head of biceps tendon and down the anterior capsule was seen in all but 1 patient (Fig 6). The glenohumeral joint volume was contracted in all patients. The capsular structures themselves were noted to be thickened, firm, and lacking pliability. The humeral head was tightly opposed to the glenoid, and distraction away from the glenoid was difficult. The arthroscopic anatomy of the glenohumeral ligaments was altered into the appearance of a wall of collagen, and the axillary pouch was contracted in volume. No intra-articular surface adhesions were seen. The posterosuperior recess, a reflection of synovium under the supraspinatus tendon over the glenoid rim, was noted to be obliterated with adhesive synovitis in all patients.

The arthroscopic capsular release procedures were performed as the first cases of the day so that physical therapy could be performed twice that day and then once in the morning before discharge. All patients were admitted for a 23-hour observation stay. The long-acting scalene block allowed the patient to go the ward and begin range of motion exercises without pain. This illustrated to the patient that their shoulder could be moved in a more normal range without pain. After the block resolved, intra-muscular and oral narcotics were used, as was intra-muscular ketorolac. Oral ketorolac was used for 4 days, with oral narcotics as needed. No in-dwelling scalene catheters or shoulder continuous passive motion devices were used in this series. Patients were encouraged to use the shoulder out of the sling and wedge device for dressing, eating, and activities of daily living. Pendulums, pul-

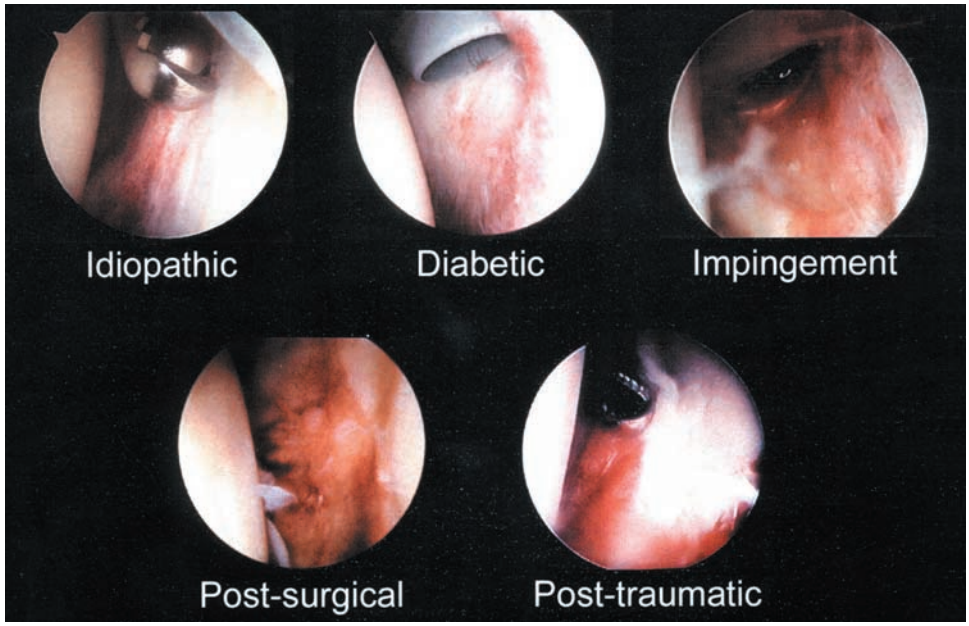

FIGURE 6. The initial arthroscopic view of the anterior capsule and interval from a representative case from each of the 5 identified etiologies in this series. Remarkably similar pathology was seen regardless of etiology. Note the reddish, proliferative synovium, thickened capsule, and reduced joint volume in these left shoulders.

Table 3. *Etiology and Outcome Values*

	ASES†		SST*		VAS*		AFE†		AER†		AIR*	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Idiopathic	36.9	94.4	4	11	7	0	83	170	10	57	Buttock	T10
Posttraumatic	36.3	95.5	3	11	8	0	82	162	10	56	Buttock	T11
Postsurgical	37.9	91.1	3	10	5	0	107	163	16	60	Buttock	T11
Diabetic	39.2	88	2.5	9	4.5	1	85	154	12	45	Trochanter	T12
Impingement	26.0	93.3	3	10.5	7	0	110	172	20	56	Buttock	L1
Study Population	35.5	93	3	10	6	0	92	165	12	56	Buttock	T12

Abbreviations: ASES, American Shoulder and Elbow Surgeons Score; SST, Simple Shoulder Test; VAS, Visual Analog Score for pain; AFE, active forward elevation; AER, active external rotation of side; AIR, active internal rotation (measured to spinal segment); Pre, preoperative; Post, postoperative.

*Studied using Wilcoxon test.

†Means tested with Student *t* test.

ley exercises, elevation in the scapular plane, passive ER with a stick and IR were emphasized. Patients were instructed to do their exercises at home 3 to 4 times a day, with each session lasting only 15 to 20 minutes. Warm moist heat was used before and ice after the sessions.

Patients underwent outpatient physical therapy 3 times per week for the first 3 weeks, and 2 times per week for the next 3 weeks. Only range of motion was emphasized; no machines, resistive exercises, or weights were allowed until pain-free motion had been restored. After that, usually at 6 to 8 weeks, light resistive strengthening could begin.

RESULTS

The average preoperative ASES for the study population was 35.5 (range, 10 to 77), of a possible 100 points. The median preoperative SST was 3 (0 to 10) of a possible 12 points. The median preoperative VAS pain score was 6.0 (0 to 10), with 10 being the most severe pain. The average preoperative active range of motion measures were AFE was 92° (40° to 140°); AER at side was 12° (−10° to 45°); and median AIR was to the buttock (trochanter – buttock). Statistical analysis of preoperative outcome measures and range of motion values between etiologic groups revealed only 1 category of any difference. In the impingement group, the average preoperative ASES score was marginally statistically lower ($P = .046$) than the other etiologic groups, 26.0 versus 37.3.

Overall results for the 68 shoulders showed significant improvements in outcome parameters and all range of motion measures. Average and median postoperative outcome scores with the magnitude of improvement were: ASES = 93 (+57.5); SST = 10 (+7); and VAS pain = 0 (-6). Average post-operative active motion with improvement was: AFE = 165° (+73°), ER at side = 56° (+44°); and IR to T12 (+7 spinal segments). All improvements were significant ($P < .0001$). (See Table 3)

Analysis of each etiologic group with regard to outcome measures and range of motion was performed. All 5 etiologic groups had significant improvement in ASES, SST, AFE, and AER at the side ($P < .0001$). Shoulders with post-traumatic and idiopathic etiologies showed significant improvement ($P < .005$) in all outcome measures. The VAS for pain was significantly improved for all groups ($P < .007$) except the diabetic group. However, this group's improvement was close to significance ($P = .0554$). The postoperative improvement in active IR for the impingement, postsurgical, and diabetic etiologies did not reach significance (Table 3). This was due primarily to the lower number of subjects in these categories and the use of the Wilcoxon test for nonparametric statistics, which the AIR data were subjected to.

Analysis of all postoperative measures (ASES, SST, VAS, AFE, AER, AIR) between etiologic groups was then performed. Each etiology was compared with the other 4 grouped etiologies. The idiopathic and postsurgical shoulders showed no difference in outcome measures or range of motion when compared with shoulders from all other groups. There were few significant differences noted: the impingement group had a higher average AFE (172 *v* 163; $P = .004$), and the post-traumatic group had a higher ASES score (95.5 *v* 92.3; $P = .03$) when compared with all other etiologies.

The patients with diabetic etiologies, although significantly improved in ASES, SST, AFE, and AER and satisfied with the outcome, showed the most post-

operative differences in comparison with patients with the other etiologies. The SST was lower (9 *v* 10; $P = .009$); the VAS for pain was higher (1 *v* 0; $P = .0176$); the AER at the side was lower (44° *v* 58°; $P = .004$) and the ASES score was marginally lower (88 *v* 93.6; $P = .056$).

Time to regain pain-free final active range of motion was documented, and the time in formal outpatient physical therapy was documented. The average time in formal physical therapy was 2.3 months (range, 2 to 20 weeks), with no difference among etiologic groups. The average time to final pain-free range of motion was determined by the time the patient was pain free and had reached a plateau in motion gains. This time averaged 2.8 months (range, 1.6 to 5.8 months). All patients did, however, continue on home stretching, motion, and strengthening for an average of 4 months. There was no statistical difference among etiologic groups in time to pain-free final motion after arthroscopic capsular release. The need for subacromial debridement or acromioplasty had no statistical effect on results.

Complications

There were no complications from the surgical procedures or scalene regional anesthesia blocks and no axillary nerve injuries. No shoulders developed recurrent stiffness or required remanipulation or repeat surgery. Patients' postoperative course did vary, however, in regard to ease of regaining motion and pain level. Fifteen patients (22%) developed increased pain and a plateau in their motion progress between 3 weeks and 6 weeks after surgery. These patients had experienced initial improvement and were afraid they were "stiffening up" again. However this flare was transient, and by 6 to 8 weeks postoperatively, all these patients experienced a decrease in pain and an increase in motion and were not behind in progress compared with the other patients. There were no identifiable factors in these 15 patients that could be associated with this transient stiffening effect.

DISCUSSION

Unfortunately, a cogent, concise discussion of the literature on the diagnosis, pathogenesis, and treatment of frozen shoulder is not possible. The variety of treatment modalities employed speaks to the fact that we still know very little about the inciting etiology or etiologies, the pathogenesis, natural history, and response to treatment of frozen shoulder. It does appear, however, that this entity does follow a prolonged but probably self-limited course.[1-3,5,6,9] The time course reported has been up to 2 years, and there is evidence that deficits in both motion and pain relief may persist.[3,5,7,19,20]

However, some patients do not respond to conservative treatment and experience significant pain and functional limitations. Diabetes has been associated with a difficult treatment course,[16,17,19] but other factors associated with a more refractory or recalcitrant frozen shoulder have yet to be identified. Patients in this series had symptoms for an average of 7 months and had undergone some type of therapy for their condition for an average of 3 months. We could not identify any factors that could be conclusively associated with recalcitrant disease course in these patients. However, they were reluctant to repeat a regimen that had not shown any progress.

Glenohumeral capsule involvement has been documented as the primary pathology in adhesive capsulitis,[4,21,23,30-32,34] but differing areas of the shoulder joint have also been implicated. The coracohumeral ligament,[25] the axillary recess,[16] and the subacromial space have all been reported as areas in which frozen shoulder can be initiated or sustained.[6,26,27,34] The glenohumeral capsular pathology of shoulder stiffness observed in this series was remarkably similar, regardless of etiology. The subacromial space was involved infrequently in idiopathic and diabetic frozen shoulder etiology, but was more commonly involved in post-surgical, post-traumatic, and impingement etiology groups.

Recent arthroscopic investigations using biopsy specimens have shown that frozen shoulder does involve a generalized phase of synovitis, fibrosis, and then contracture. There appears to be an early hypervascular synovitis that may progress to subsynovial thickening and collagen deposition. Fibroplasia, thickening, and contracture of the capsular structures may then occur.[4,8,29] The pathologic synovium may be involved in local hormonal mediation of the process.[4,8,29] From our clinical and arthroscopic experience and the literature, we believe that frozen shoulder may represent a common expression of a variety of causal factors. The pathologic process could be caused by, sustained by, or be involved in any or all of the components of the glenohumeral capsule, coracohumeral ligament, and subacromial space. The arthroscope allowed us to debride the pathologic proliferative synovium and address all potential areas of stiffness pathology. The capsular release with the bipolar cautery device ablated a 3-mm swathe of capsule

and allowed for a controlled, less traumatic, and fuller release than if achieved by closed manipulation alone. Less force was needed for the final manipulation itself.

In a recent and similar study to ours, Watson et al.[34] found a similar capsular pathology to that documented in this study. The resultant rapid clinical improvement and patient satisfaction with the arthroscopic capsular release technique was also similar to results in the current study. These authors also documented the early postoperative transient flare-up of pain and stiffening seen in a minority of patients.[34]

We do not advocate this arthroscopic approach for patients presenting initially with a frozen shoulder. A concerted effort at conservative management is mandatory. This allow the surgeon to evaluate the patients' personality, functional demands, compliance with therapy, and progress with therapy. We do not know the optimal time for arthroscopic intervention in recalcitrant cases, but we have been successful with the following general guidelines: Before our evaluation, patients had been symptomatic for an average of 5 months and in some form of therapy for an average of 2 months. After 6 to 8 weeks of treatment under our care and showing no progress, patients had now been symptomatic for a substantial amount of time. They were unwilling to repeat or continue a regimen that had been unsuccessful. With this clinical course and with pain and shoulder dysfunction that affected occupation, recreation, or sleep, we proposed the arthroscopic capsular release.

Using consistent technique and aftercare, this arthroscopic capsular release approach allowed assessment and treatment of stiff shoulder pathology regardless of etiology. Treatment failures were not seen, which may be due to the ability to debride the pathologic synovium, release the capsule in a balanced fashion, and evaluate and address subacromial involvement. The natural history of this poorly understood condition was possibly shortened because patients attained final pain-free motion within an average of 3 months. We were mildly surprised that the etiology of the shoulder stiffness pathology did not have a more profound effect on preoperative scores and postoperative outcome scores and motion or time to regain motion parameters. The arthroscopic capsular release was an effective means to treat stiff shoulder pathology regardless of the etiology.

Stiff shoulder pathology can result from a variety of etiologic factors, but the adhesive capsulitis pathology of the glenohumeral joint was qualitatively consistent regardless of etiology. A frozen shoulder may represent a common expression of a variety of causal factors. Arthroscopic capsular release was an excellent treatment option for shoulders resistant to conservative management. It was equally effective across the 5 identified etiologic groups, and provided significant pain relief, restoration of motion, and function.

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