Summary: Limitation of internal rotation has been reported in conjunction with impingement syndrome of the shoulder. A group of 9 patients was identified who had discrete, painful loss of internal rotation associated with refractory impingement syndrome. The duration of symptoms averaged 18 months (range, 11 to 33 months), and all patients failed a course of physical therapy specifically addressing loss of internal rotation. Six patients reported traction as the mechanism of injury, and 3 developed motion loss and pain following a posterior capsular shift procedure. All patients underwent arthroscopy, and were observed to have a thickened posterior capsule. An arthroscopic release of the posterior capsule improved motion in all patients, with substantial relief of pain. At an average of 19 months follow-up (range, 11 to 35 months), internal rotation in 90° of abduction improved from 10° preoperatively to 47° postoperatively, and there were no complications related to the procedure. We conclude that chronic loss of internal rotation secondary to posterior capsular contracture may be an explanation for refractory pain in some patients with an initial diagnosis of impingement syndrome. This condition appears to be amenable to arthroscopic posterior capsular release. Key Words: Shoulder—Impingement—Adhesive capsulitis—Arthroscopy—Capsular release—Kinematics.

The diagnosis of impingement syndrome is identified in the typical patient with pain localized over supraspinatus insertion on the greater tuberosity and pain with forward flexion.1,4 Although associated loss of internal rotation has been described, motion loss is usually not considered to be a common feature of this condition.2 Adhesive capsulitis is considered to be a separate and different condition.5-10 Nevertheless, it has been the experience of the senior author that many patients with stiff shoulders will have pain with flexion of the arm, and their complaints may resemble impingement symptoms. Recent biomechanical work11 has shown that contracture of the posterior or anterior capsule can alter normal glenohumeral kinematics, resulting in anterior-superior translation of the humeral head during arm elevation. This can cause a form of nonoutlet impingement as the humeral head is forced into the coracocromial arch. Although Matsen and Arntz2 have emphasized the importance of stretching a stiff shoulder during physical therapy for impingement syndrome, there has been no clear description of these types of patients and their treatment. We have identified a subset of patients with chronic painful loss of internal rotation who were found at arthroscopy to have a posterior capsular contracture. Although there are recent reports2,10,12-16 of techniques for arthroscopic capsular release for adhesive capsulitis and refractory loss of external rotation, there are no such reports for treatment of isolated loss of internal rotation. We have developed a technique of posterior capsular release that is successful in treating these kinds of patients. The purpose of this article is to describe the clinical
presentation of these patients and report our technique and results for arthroscopic posterior capsular release for refractory posterior capsular contracture of the shoulder.

METHODS

Patient Selection
Over a 3-year period, the senior author surgically treated 281 patients with chronic and refractory pain that was attributed to either impingement syndrome or adhesive capsulitis. A subset of 9 patients was identified who had discrete, painful loss of internal rotation, particularly evident with the arm in 90° of abduction, and also associated with painful limitation of forward flexion. None of the patients had motion loss associated with advanced arthritis, fracture, or diabetes mellitus, and none had a full-thickness rotator cuff tear. The dominant arm was involved in 7 patients; there were 5 male and 4 female patients. The average age was 40 years (range, 20 to 57 years). The duration of symptoms averaged 18 months (range, 11 to 33 months) and all patients failed a course of at least 4 months of supervised physical therapy addressing loss of motion in the affected planes. Although absolute motion loss was not a prerequisite for surgery, all patients had painful and marked limitation of internal rotation when measured in 90° of abduction, and 6 of the 9 patients had a positive impingement sign on their preoperative physical examination.

Eight of the 9 patients had undergone a total of 15 operative procedures before their initial presentation. Six patients described a sudden pull on the affected arm in a traction mechanism, and reported that this event initiated their shoulder pain. Three of these patients had a prior arthroscopic subacromial decompression, but continued to have pain, and 1 also underwent 2 open acromioplasties. A fourth patient had an open Bankart procedure, and afterward underwent an open distal clavicle resection. A fifth patient had an open repair of a SLAP lesion with a metal staple, then an open hardware removal and, finally, a manipulation under anesthesia. The sixth patient who described a traction mechanism of injury had no prior surgery. Three patients had a posterior capsular shift after which they developed painful loss of internal rotation and flexion; 1 of these patients had an unsuccessful attempt of closed manipulation.

Clinical Assessment
All patients were evaluated preoperatively and postoperatively by the senior author, and they also answered a questionnaire about their subjective complaints. Preoperative, intraoperative, and postoperative motion was documented photographically in all cases. Both active and passive motion was assessed goniometrically. Forward flexion, external rotation in adduction (0° abduction) and abduction (90°), and internal rotation in abduction (90°) were assessed with the patient supine. Internal rotation in adduction (0° abduction) was measured with the patient in a seated position. While all patients had limitation of both active and passive internal rotation, passive measurements were used in this study (Fig 1). In addition, all patients had painful forward flexion and a partial relief of their pain with an impingement test performed by injection of 1% lidocaine into the subacromial space.

Radiographic evaluation consisted of anteroposterior and axillary radiographs taken in the plane of the scapula. In addition, the supraspinatus outlet view and caudal tilt view were obtained to define acromial morphology. Five patients were found to have a type I (flat) acromion, and 4 had a type II (curved) acromion. None of the patients were found to have a type III (hooked) acromion.

OPERATIVE TECHNIQUE

Anesthesia and Postoperative Analgesia
General or interscalene anesthesia using bupivacaine was used. Three patients had general anesthesia, 5 had regional interscalene anesthesia, and 1 had a combination of general and regional anesthesia. Two of the patients with an interscalene block had an...
indwelling catheter that was used for postoperative analgesia. As it is our impression that regional interscalene anesthesia gives better postoperative pain control and allows more aggressive immediate therapy, we counseled our patients to have this form of anesthesia. The interscalene block was performed using approximately 30 mL of 0.5% bupivacaine with a 1:200,000 concentration of epinephrine. This gave adequate anesthesia during the procedure and provided 5 to 6 hours of postoperative analgesia, which allowed for immediate postoperative range of motion and stretching. The patients who had an interscalene block for anesthesia had repeat blocks on the morning of postoperative days 1 and 2. This allowed the physical therapist to perform both a morning and afternoon session for passive range of motion, in addition to the patient performing exercises.

When regional anesthesia and postoperative analgesia was performed through an interscalene catheter, a continuous infusion of 0.25% bupivacaine at 6 mL/h was administered for 48 hours postoperatively. All patients had self-administered patient-controlled analgesia through an intravenous pump set to administer 1 mg of morphine every 8 minutes as needed by the patient, up to a maximum of 30 mg in 4 hours.

Posterior Capsular Release Technique

An examination under anesthesia was performed first to confirm the degree of motion loss in each plane. The beach-chair position was used for arthroscopy. A diagnostic glenohumeral arthroscopy was performed with the arthroscope in the posterior portal, and, in all cases, the anterior capsular structures and rotator cuff were normal. The arthroscope was then placed through the anterior-superior portal and a disposable operative cannula was placed in the posterior portal, over a switching stick. Thus, the posterior aspect of the joint was viewed through an anterior portal. In all cases, the posterior capsule was observed to be thickened and shortened.

An electrocautery device with a hooked tip was then placed through the posterior portal cannula. The posterior capsule was divided adjacent to the glenoid rim beginning just posterior to the biceps origin on the superior glenoid rim at about the 11 o’clock position and continuing inferiorly to about the 8 o’clock position (for a right shoulder) (Fig 2A and B). Division of the posterior capsule was performed along the glenoid rim because the muscle of the cuff tendons are superficial to the capsule at this level. Therefore, the depth of the capsular division is completed when one visualizes the muscle fibers (Fig 2C). An arthroscopic shaver was then inserted to remove the ragged edges of the capsule to clearly identify the capsular edge and rotator cuff muscle. If the capsule is divided more laterally, there is risk of injuring the rotator cuff tendons, which become conjoined with the capsule in this location. By using a shaver to remove the divided and cauterized edges of the capsule, a wider gap is created to avoid recurrence from scarring of the capsule.

After removal of the arthroscope, a gentle manipulation completed the release of any remaining capsular fibers to restore internal rotation and flexion. This occurred through a gradual yielding of tissue, like a rubber band stretching. This is in contrast to our experience with arthroscopic release of the anterior capsule in patients with anterior capsular contractures. In these cases, a discreet audible improvement of motion has been observed.

Arthroscopic Impingement Test

All patients had an arthroscopic subacromial bursoscopy performed, at which time they were found to have an inflamed, thickened bursa. Following removal of only the inflamed bursal tissue, and without removing any bone, an arthroscopic impingement test was performed. This is done by placing the arthroscope through the lateral arthroscopic portal and then flexing the shoulder anterior to the scapular plane through an arc of 140°, observing the relationship of the humeral head to the acromion. In a normal shoulder, the rotator cuff will pass underneath the acromion, and the interval between the acromion and the rotator cuff will be maintained in all positions. Before posterior capsular release in this series of patients, the humeral head was observed to move superiorly during flexion with the rotator cuff contacting the undersurface of the acromion, eliminating the subacromial space. After the posterior capsular release, the arthroscopic impingement test was repeated. In all cases, the rotator cuff tendon was observed to pass underneath the acromion with the subacromial space maintained.

Additional Surgery

Two patients had full-thickness cartilage loss on the humeral head. One of these patients also had full-thickness cartilage loss on the glenoid, and the cartilage lesions in this patient were a result of intra-articular placement of a metallic staple. The cartilage injuries in both patients were debrided and a microfracture arthroplasty was performed.
Postoperative Treatment

On the morning of the first postoperative day, passive motion was begun by a physical therapist, with therapy sessions in both the morning and afternoon. In addition, the patient was instructed in self-assisted motion exercises. Analgesia was by the methods described previously, and cryotherapy (Aircast, Summit, NJ) was used in all cases. The patients were discharged in the afternoon of the second postoperative day after the second postoperative physical therapy session of that day. For the first 2 weeks, the patient attended daily sessions of supervised therapy, as well as performing a home program. Supervised physical therapy was then reduced to 3 times per week for the next 4 weeks, and the home program was advanced. The rehabilitation was then individualized according to each patient’s progress. Patients were not given a sling for support, and they were encouraged to use their operated arm for activities of daily living as soon as possible after surgery. Strengthening was begun as

**Figure 2.** Technique of arthroscopic posterior capsular release. (A) The posterior capsule is released along the glenoid rim and the electrocautery device introduced through the posterior portal. The arthroscope is introduced through the anterior-superior portal. (PC, posterior capsule; RC, rotator cuff. Right shoulder with humeral head removed). (B) The release is performed adjacent to the glenoid rim. The depth of release is complete with visualization of the posterior rotator cuff muscle fibers. (C) After the capsular release, the thickened edges of the posterior capsule and the underlying muscle can be seen.
soon as postoperative pain and active shoulder motion allowed, and patients were encouraged to swim in a pool at 2 to 4 weeks after surgery.

Statistical Analysis

Statistical analysis was performed using a t test for paired samples (SPSS-Statistical Package for the Social Sciences Release-5.0 for VAX/VMS; SPSS Inc, Chicago, IL). Comparison of postoperative motion was made to both preoperative motion and motion of the contralateral, asymptomatic shoulder.

RESULTS

The average postoperative follow-up was 19 months (range, 11 to 35 months), and there were no operative complications. In particular, there was no posterior instability noted postoperatively. As motion loss was primarily limited to internal rotation in these patients preoperatively, there were no significant differences between the preoperative and postoperative values for external rotation in adduction and abduction.

The average preoperative internal rotation in abduction was 10° (range, 0° to 40°) for the involved side, compared with 58° (range, 50° to 80°) for the contralateral side. Postoperative internal rotation in abduction increased by an average of 37° (range, 30° to 50°) to an average motion of 47° (range, 30° to 80°), which was statistically significant (P < .01) (Fig 3A). For forward flexion, the average preoperative motion for the involved shoulder was 133° (range, 95° to 150°) and for the noninvolved shoulder was 156° (range, 150° to 170°). The average improvement in forward flexion was 15° (range, −20° to 45°) to an average motion of 148° (range, 130° to 160°). Although there was a trend toward gains in forward flexion, these were not statistically significant compared with postoperative values (Fig 3B).

All patients noted substantial relief of pain, except for 1 who had the articular cartilage changes due to prior hardware placement that became intra-articular. In addition, this was the only patient to lose motion in any plane after the procedure. This patient lost 20° of forward, but a gain of 30° of internal rotation in abduction was measured postoperatively.

DISCUSSION

We have identified and successfully treated a group of patients with chronic painful shoulder motion that was refractory to conservative treatment and, in some cases, both arthroscopic and open acromioplasty. Although some of our patients developed pain after a

![Figure 3](image-url)
posterior capsular shift procedure, all other patients reported a specific event that they described as a traction injury to the affected arm. We recognize that prior surgical procedures may be a factor in this condition. In 5 cases, surgical procedures were performed to treat a variety of diagnoses, including classic outlet impingement. In all of these cases with prior procedures, the operative approaches failed. All of the patients in this series had painful loss of internal rotation in abduction as a consistent feature of their condition, as well as a trend toward loss of forward flexion. Furthermore, all were observed at the time of arthroscopy to have a contracted and thickened posterior capsule. It is unclear why the posterior capsule became scarred and contracted, although we would postulate that in the cases of injury associated with a traction mechanism, trauma to the posterior capsule resulted in localized and excessive scarring. In the patients who had a posterior capsular shift procedure, it would appear that either the repair was too tight or there was excessive scarring in this region following the repair.

In all cases, we documented arthroscopically that glenohumeral kinematics were altered and abnormal, with superior translation of the humeral head during flexion such that the rotator cuff was compromised by the overlying coracoacromial arch prior to release. This observation is consistent with the biomechanical data from Harryman et al.11 showing that experimental posterior capsular shortening will cause anterior and superior translation of the humeral head. This results in a condition that Neer has termed “non-outlet” impingement.8 The impingement that occurs is not a result of pathology of the coracoacromial arch,26 but a dynamic translation of the humeral head, which is forced superiorly by a tight posterior capsule during flexion (Fig 4A).11 After arthroscopic posterior capsular release, we observed that this superior translation was reduced (Fig 4B). The finding of subacromial bursitis in these patients is further support of this “non-outlet” form of impingement, and, in all cases, we excised the inflamed bursal tissue, but did not perform an acromioplasty.

While the concept of motion loss primarily affecting internal rotation and flexion is not new,2,27,28 to our knowledge there are no prior reports of release of the

**Figure 4.** (A) A tight posterior capsule causes obligate anterior-superior translation of the humeral head during forward flexion, resulting in “nonoutlet” impingement. (B) When the capsule is loose, as in a normal shoulder or following arthroscopic posterior capsular release, the humeral head remains centered on the glenoid during forward flexion.
posterior capsule to treat this condition. Loss of motion following posterior capsular shift for instability is considered to be a rare occurrence. However, the senior author has successfully treated several cases by an open release, and the posterior capsule was found to markedly thickened in these patients. Therefore, we were interested in determining the effectiveness of an arthroscopic release technique as well. In the current arthroscopic series, as well as in the open release series, the posterior capsule was found to be markedly thickened in all cases.

Postoperative pain control has been shown to be critical to maintaining motion gains after closed manipulation treatment of adhesive capsulitis. The use of interscalene anesthesia, preferably with an indwelling catheter, during the in-patient hospitalization, was felt to be an essential part of our technique and is now used routinely. We have found, and others have reported, that this technique is safe, well tolerated, and significantly reduces the need for narcotics, while allowing aggressive passive range of motion in the immediate postoperative period.

There were no complications related to this procedure in our series. Although some surgeons have recommended against the insertion of an arthroscope into a stiff shoulder because of concerns about articular injury, our technique allowed insertion of the standard size arthroscope without any injury to the articular surfaces. We accomplished this by injecting saline from posterior to distend the joint and carefully guiding the blunt trocar and cannula sheath into the posterior portal, through the thickened posterior capsule and over the humeral head. If there is a bevel on the arthroscopic cannula, the longer portion is placed superior to decrease any chance of damaging the articular cartilage. Arthroscopic division of the posterior capsule was performed at the level of the glenoid rim where the muscle of the infraspinatus and teres minor are superficial to the capsule, thus avoiding injury to their tendinous portions found more laterally. Our release did not extend into the inferior rim where the muscle of the infraspinatus and teres minor inserts.24,25,26

In conclusion, we believe that there is a small subset of patients who may have an initial diagnosis of impingement syndrome, but remain refractory to both nonoperative and traditional operative treatment secondary to posterior capsular contracture. In all cases where impingement is suspected, a careful examination of both passive and active motion in all planes is necessary. In those patients who are found to have limitation of internal rotation and flexion, a therapy program should be directed at improving these motion planes. When nonoperative treatment fails and painful motion limitation persists, this technique for an arthroscopic posterior capsular release in conjunction with subacromial bursectomy appears to be a reliable treatment with minimal morbidity. Finally, some patients who develop this condition following a posterior capsular shift can also be successfully treated with this arthroscopic technique.

REFERENCES


