REVIEW

Suprascapular Nerve Entrapment: Technique for Arthroscopic Release

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ABSTRACT

Suprascapular neuropathy can be caused by a variety of anatomic and pathologic entities as the nerve courses from the brachial plexus through the suprascapular and spinoglenoid notches to innervate the supraspinatus and infraspinatus muscles. We describe techniques for arthroscopically accessing the nerve at both the suprascapular and spinoglenoid notches and decompressing structural lesions that may be contributing to the neuropathy.
Keywords: suprascapular neuropathy, suprascapular notches, spinoglenoid notches, cyst decompression, arthroscopic release

HISTORICAL PERSPECTIVE

Isolated injury to the suprascapular nerve has long been recognized as an etiologic entity producing shoulder pain and weakness. Since Clein's 1975[1] report of open decompression in 5 patients at the suprascapular notch, the diagnosis and treatment of these lesions has undergone tremendous evolution. We describe our technique for arthroscopic nerve decompression at the suprascapular notch with release of the transverse scapular ligament, as well as the technique for spinoglenoid ligament release and nerve decompression in the spinoglenoid notch region.

Clinical Features and Evaluation

Injury to the suprascapular nerve has been associated with multiple sports, direct trauma to the neck or scapula, heavy labor, and even crutch use.[2] The patient with suprascapular nerve palsy may present with an often vague range of symptoms or even be asymptomatic.[3] Pain over the posterolateral shoulder or easy fatigability with overhead activities may be reported, or painless weakness of external rotation with or without spinatus muscle atrophy may be noted. Compression of the nerve at both the suprascapular and spinoglenoid notches are commonly reported mechanisms of injury and will be discussed in detail.

The physical examination plays a critical role in discerning the site of suprascapular nerve injury. Clinical observation of the patient's shoulder girdle is important. More proximal injury, as seen with suprascapular notch compression, may result in atrophy of both the supraspinatus and infraspinatus, whereas more distal compression at the spinoglenoid notch will result in isolated infraspinatus weakness and atrophy (Fig. 1). Tenderness over the course of the nerve may be present but is often difficult to localize. Weakness of shoulder abduction or external rotation with vague posterolateral shoulder pain may be the only significant examination finding, although a decreased range of motion, specifically adduction, may be noted due to pain.

Plain radiographs of the shoulder are routinely negative. Electromyography (EMG) and nerve conduction velocity (NCV) studies play a particularly useful role in the diagnosis and localization of a suspected suprascapular nerve injury. As with most nerve injuries, these studies are generally more useful if obtained in the subacute phase of injury, at least 3–4 weeks after onset of symptoms. However, careful clinical correlation with study results must be used, as both false-negative and false-positive nerve findings have been described.[4] Magnetic resonance imaging (MRI) may be useful in

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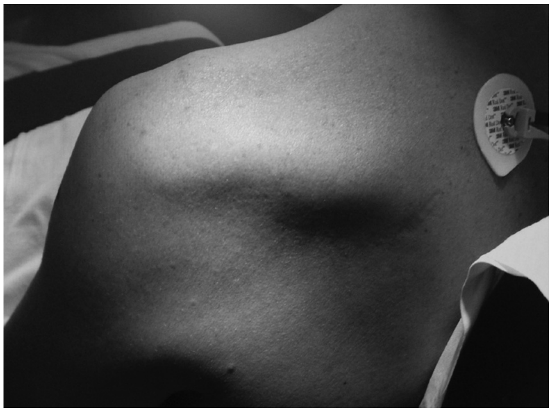

FIGURE 1. Clinical appearance of Infraspinatus and supraspinatus muscle atrophy.

demonstrating atrophic muscle degeneration of the spinatus or to reveal the presence of a compressive lesion along the course of the nerve. Most commonly, this will be ganglion cyst, often seen in association with a superior labral tear (Figs. 2A and B).

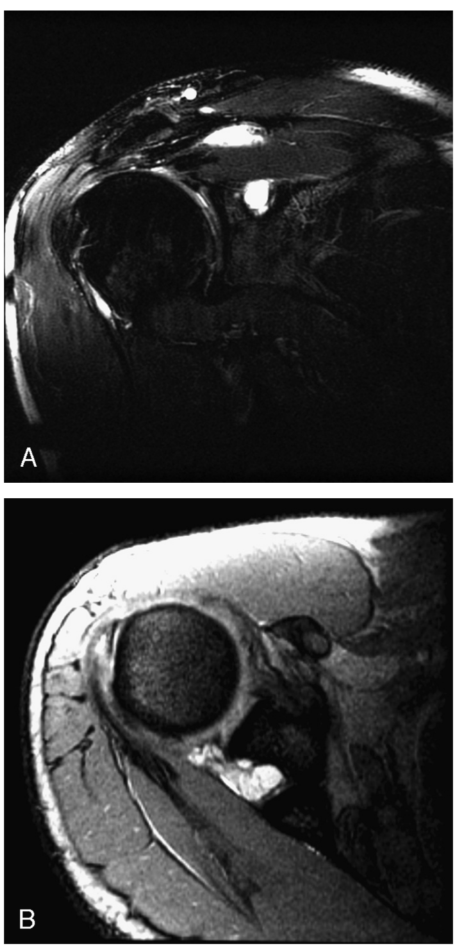

FIGURE 2. A, Coronal view of paralabral cyst. B, Axial view depicting associated labral tear.

Relevant Anatomy and Pathophysiology

At Erb point, the suprascapular nerve branches from the upper trunk of the brachial plexus, with contributions from C5 and C6. The nerve then travels below the transverse scapular ligament as it crosses the suprascapular notch to enter the supraspinatus fossa (Fig. 3), whereas the suprascapular artery usually travels above the ligament. The classic description of the superior transverse scapular ligament is a completely nonossified single band and should be expected, on average, in three fourths of the cases.[5] Partial or complete ossification and anomalous bifid or trifid bands of the superior transverse scapular ligament have been described.[5–7] The nerve then traverses the supraspinatus fossa, giving motor branches to the supraspinatus, with variable minor sensory contributions to the glenohumeral and acromioclavicular joints, and occasionally to the skin.[8] The nerve angles around the spine of the scapula at the spinoglenoid notch, traveling with the artery under the spinoglenoid ligament.[9] The motor branches to the supraspinatus are approximately 1 cm from the suprascapular notch, 3 cm from the origin of the long head of the biceps, whereas the motor branches to the infraspinatus average 2 cm from the posterior glenoid rim.[5]

Like other nerves, the suprascapular nerve is susceptible to injury from compression, traction, or direct trauma. Vascular microtrauma has also been postulated to cause

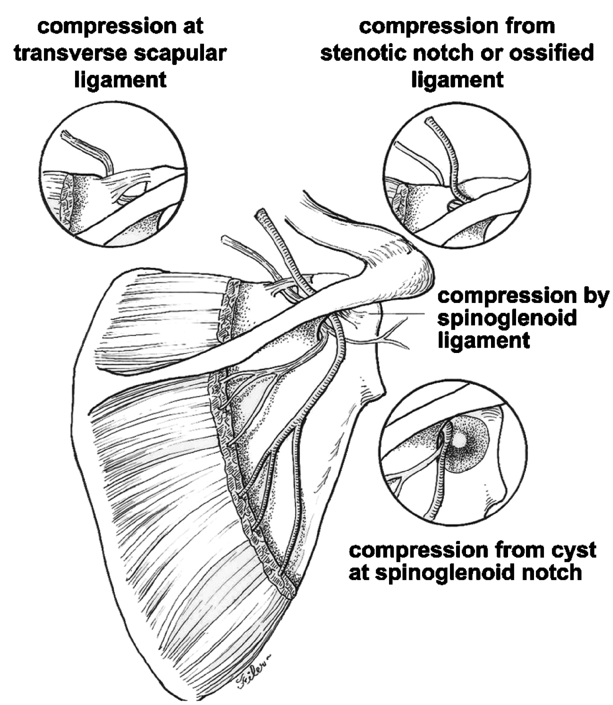

FIGURE 3. Anatomy of the suprascapular nerve as it passes through the suprascapular and spinoglenoid notches. Common compression mechanisms are depicted at each site.

nerve dysfunction. The most commonly reported mechanism of injury is compression by a ganglion cyst, usually at the suprascapular or spinoglenoid notch. The cyst is often associated with a tear in the glenohumeral joint capsule or labrum, with fluid being forced through the tear and then being trapped outside the joint. A thickened or calcified suprascapular ligament may also compress the nerve. There may also be dynamic compression from the spinoglenoid ligament as proposed recently.[8]

Nonoperative Treatment

Treatment of the acute injury to the suprascapular nerve is similar to most nerve injuries about the shoulder. Rest and pain control are followed with progressive range of motion and strengthening exercises as tolerated. More chronic cases are managed depending on the duration of symptoms and the mechanism of injury, although in some instances the exact duration of symptoms may be difficult to determine. MRI can be used to evaluate for a compressive lesion. If a compressive lesion or cyst is noted on imaging, the patient can be observed for 2–3 months, followed by surgical decompression if symptoms persist. An athlete with symptoms associated with repetitive overhead activity, as seen with volleyball, tennis, or baseball players, should have the possibility of a compressive or structural lesion excluded with an MRI, and then may be followed for 6–12 months with observation, activity restriction, and periscapular therapy to see if the symptoms abate. Periodic EMG/NCV studies can demonstrate the electrophysiological nerve recovery. Surgical intervention in individuals with this overuse mechanism of injury has demonstrated variable results at best and function usually returns by 12 months.[10] As with other painful nerve injuries about the shoulder, Parsonage–Turner syndrome must be considered, and if present, this should be managed conservatively with pain control, observation, and therapy. A neurologic consultation can also be helpful.

■ SURGICAL TREATMENT

If the nerve lesion is proximal and both the supraspinatus and infraspinatus are involved, the entire nerve should be decompressed, but most importantly the

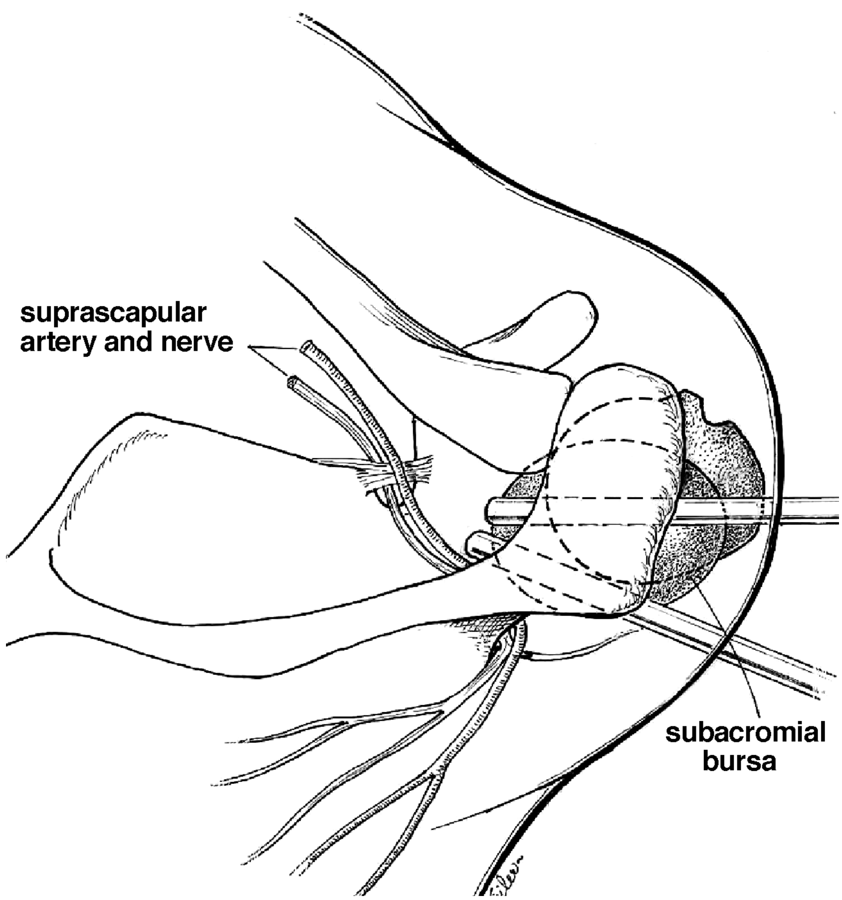

FIGURE 4. Schematic representation of portal sites in the subacromial space for nerve release at the suprascapular notch.

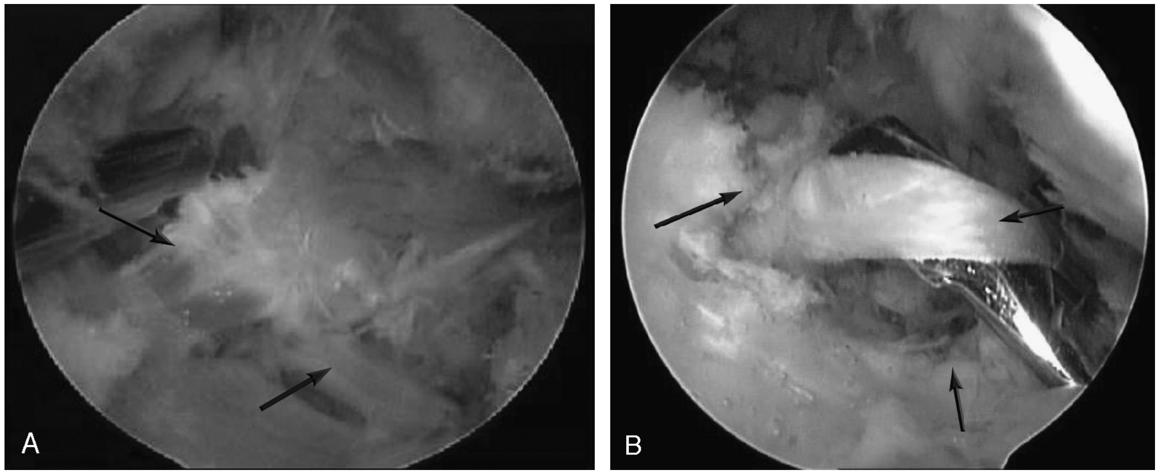

FIGURE 5. A, Arthroscopic view from posterolateral portal in subacromial space. Suprascapular nerve (bottom arrow) is depicted under the transverse scapular ligament (arrow top-left). B, Same view from posterolateral portal, elevator is in anterolateral portal exposing the nerve (right arrow) after releasing the transverse scapular ligament (left arrow) at the notch (bottom arrow).

transverse scapular ligament should be released. If only the infraspinatus is involved or if there is a structural lesion at the spinoglenoid notch, such as a paralabral cyst, then the nerve may be simply decompressed at the spinoglenoid notch.

An arthroscopic approach is a more sophisticated way of addressing the suprascapular nerve and is our preference when there is an associated intra-articular lesion, such as a SLAP tear or labral tear. It is our preferred method for treating spinoglenoid neuropathy due

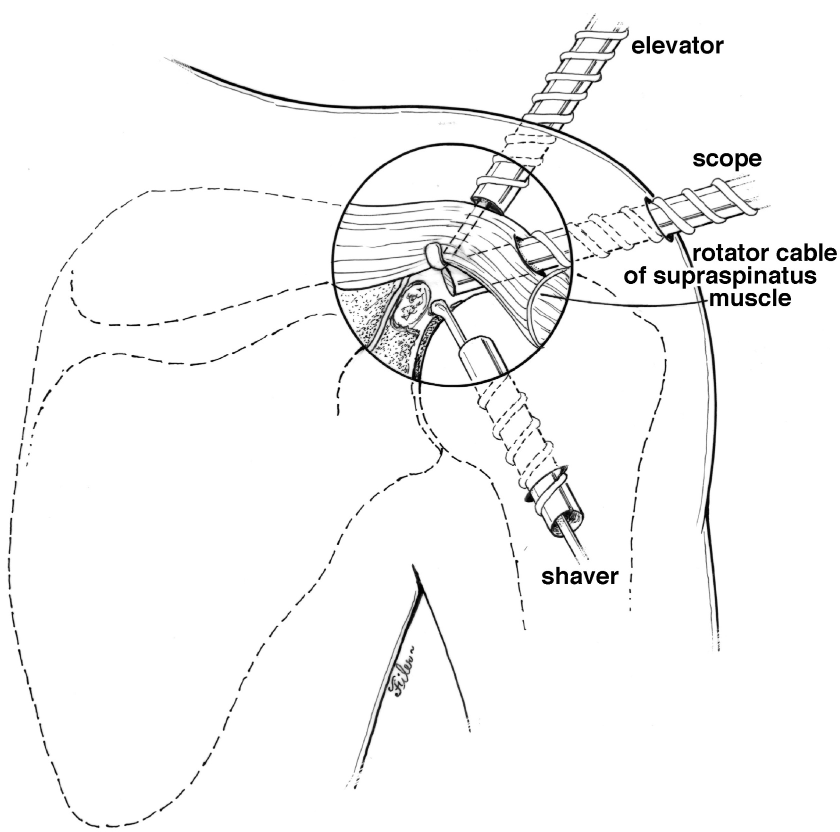

FIGURE 6. Schematic representation of portal sites in glenohumeral joint space for spinoglenoid ligament release or cyst decompression.

FIGURE 7. View from posterior portal. Instrument seen perforating the supraglenoid cyst (left arrow) from the lateral trans-cuff portal. Turbid cyst fluid (right arrow) is emanating from the perforation.

to paralabral cysts, and furthermore it is becoming our preferred method for decompressing the nerve at the suprascapular and spinoglenoid notches. It does require advanced arthroscopic skills, but it offers a less invasive and more cosmetic approach with better overall visualization and access. Moreover, concomitant intra-articular pathology can be addressed easily.

A relative contraindication to arthroscopic release is transverse scapular ligament calcification or ossification. A CT scan may help to delineate the notch, if this finding is suggested on plain films, and an open approach considered. A CT scan will demonstrate the morphologic appearance of the suprascapular notch and can exclude the possibility of an ossified notch, which is 1 morphologic variant. This is important to know preoperatively when considering arthroscopic neurolysis.

Arthroscopic Release at the Suprascapular Notch

We prefer to use the beach chair position. The arthroscope is placed in an anterolateral portal and accessory anterior and posterior portal are used (Fig. 4). The view is initially into the subacromial space. The coracoid process must be visualized and the dissection is then carried medially. Arthroscopic retractors are helpful to posteriorly retract the supraspinatus muscle belly. The dissection is carried along the posterior aspect of the coracoid process. The coracohumeral and coracoclavicular ligaments are identified, and at the medial base of the coracoid the suprascapular notch is identified. The artery is cauterized using radiofrequency ablation and the ligament is released using handheld arthroscopic tissue punches (Figs. 5A and B). The nerve can be probed to insure that there is no compression. As the nerve distally moves, it can be seen passing deep to the supraspinatus muscle.

Arthroscopic Release at Spinoglenoid Notch or Cyst Decompression

This is our preferred technique for treating paralabral cysts. Again the beach chair position is used. Standard anterior and posterior portals are created. A trans-rotator cuff portal as used for SLAP repairs is created (Fig. 6). The arthroscope is laterally placed through the trans-cuff portal.[11] This gives excellent visualization. If there is a labral tear, it is repaired with suture anchors using standard technique. Some have advocated working through the labral tear to access the cyst, but we have found this to be quite difficult and furthermore it is virtually impossible to visualize the suprascapular nerve. Therefore, we have gone to performing a capsulotomy, releasing the posterosuperior capsule at the periphery of the labrum until the fibers of the supraspinatus are identified. The supraspinatus muscle is then superiorly elevated using a retractor, which is placed from our anterior portal. With careful and meticulous dissection, the cyst itself can be invariably demonstrated and resected. The typical ganglion cyst fluid is seen when the cyst is perforated (Fig. 7). The suprascapular nerve runs 2.5–3 cm medial to the superior aspect of the glenoid at the base of the supraspinatus fossa (Fig. 8). It can be posteriorly traced from there until it passes through the spinoglenoid notch. Using handheld basket punches and arthroscopic probes, a careful neurolysis can be performed.

Fifteen patients underwent endoscopic decompression of the suprascapular nerve at the spinoglenoid notch, 13 of whom had spinoglenoid notch cysts. Three patients had endoscopic release of the suprascapular nerve at the level of the suprascapular notch. There were no complications in this series.

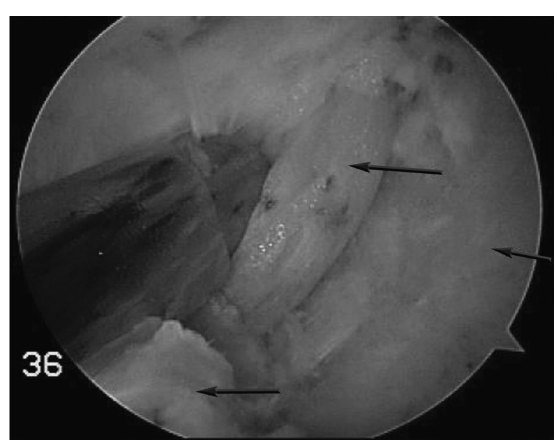

FIGURE 8. Suprascapular nerve exposed (top arrow) after releasing spinoglenoid ligament (bottom left arrow) at spinoglenoid notch (bottom right arrow).

POSTOPERATIVE REHABILITATION

Postoperatively, patients are immobilized in a sling for comfort. Early motion is encouraged. If a labral tear was repaired, then the athlete is protected for 4 weeks before resuming active motion. Strengthening begins at 6 weeks. Throwing and overhead activities generally commence at 4–5 months postoperatively.

RESULTS AND OUTCOMES

The results of both operative and nonoperative treatment of suprascapular nerve injuries are not easily interpreted. The duration of symptoms is often difficult to assess and the diagnosis may be incorrect or incomplete with respect to associated intra-articular pathology. Several studies have reported on the results of both operative and nonoperative treatment.[4,10,12] In a recent meta-analysis of the literature, Zehetgruber et al[13] found suprascapular nerve entrapment to be rare, occurring mainly in patients under 40 years. Isolated infraspinatus atrophy was most often associated with a ganglion cyst, whereas a history of trauma was usually associated with ligamentous compression of the nerve. Surgical treatment seems to give reliable pain relief, with persistent atrophy of the spinatus, a common but well-tolerated finding. We have found similar findings in our series treated arthroscopically, with less pain and faster recovery.

Fourteen of the 15 patients with releases at the spinoglenoid notch clinically improved (decreased pain and improved function), and all stated they would undergo the procedure again. All of the patients with compressive lesions showed improvement within 3 months. Six patients with spinoglenoid notch cysts had follow-up MRIs and there was no evidence of recurrence in any of these patients. One patient had transient improvement with improved infraspinatus strength and less pain but then at 2 years postoperatively developed recurrent pain. On repeat imaging, he was found to have developed significant glenohumeral arthrosis. Two of the 3 patients who underwent releases at the suprascapular notch showed marked improvement, whereas the other individual only had modest benefit with slight improvement in his pain.

COMPLICATIONS

There are no published reports on complications with either of these techniques to release the suprascapular nerve arthroscopically. We speculate that potential complications from these techniques include inadequate release of the nerve, bleeding from the suprascapular artery, further injury to the suprascapular nerve from traction, sharp injury, or thermal injury, or brachial plexus injury. In our experience, we have not seen any recurrent spinoglenoid notch cysts.

CONCLUSION

Arthroscopic release of the suprascapular nerve is a less invasive and potentially more effective way to treat suprascapular neuropathy that may occur from a variety of causes. Releasing the nerve does require advanced arthroscopic skill and detailed knowledge of arthroscopic shoulder anatomy, as there are some potential pitfalls. Nevertheless, the authors believe that with practice and meticulous technique, this technique can be mastered and will become the preferred treatment method for suprascapular neuropathy.

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