

CURRENT CONCEPTS REVIEW

Suprascapular Neuropathy

By Robert E. Boykin, MD, Darren J. Friedman, MD, Laurence D. Higgins, MD, and Jon J.P. Warner, MD

Investigation performed at Massachusetts General Hospital and Brigham and Women's Hospital, Boston, Massachusetts

- Suprascapular neuropathy has often been overlooked as a source of shoulder pain.
- The condition may be more common than once thought as it is being diagnosed more frequently.
- Etiologies for suprascapular neuropathy may include repetitive overhead activities, traction from a rotator cuff tear, and compression from a space-occupying lesion at the suprascapular or spinoglenoid notch.
- Magnetic resonance imaging is useful for visualizing space-occupying lesions, other pathological entities of the shoulder, and fatty infiltration of the rotator cuff.
- Electromyography and nerve conduction velocity studies remain the standard for diagnosis of suprascapular neuropathy; however, data on interobserver reliability are limited.
- Initial treatment of isolated suprascapular neuropathy is typically nonoperative, consisting of physical therapy, nonsteroidal anti-inflammatory drugs, and activity modification; however, open or arthroscopic operative intervention is warranted when there is extrinsic nerve compression or progressive pain and/or weakness.
- More clinical data are needed to determine if treatment of the primary offending etiology in cases of traction from a rotator cuff tear or compression from a cyst secondary to a labral tear is sufficient or whether concomitant decompression of the nerve is warranted for management of the neuropathy.

History and Epidemiology

Suprascapular neuropathy results from an injury to the suprascapular nerve and is typically due to compression or traction in association with other injuries. This injury leads to a spectrum of clinical symptoms, including pain and weakness in forward flexion and external rotation of the shoulder. Schilf documented the first evidence of compression of the suprascapular nerve¹. The clinical entity of suprascapular neuropathy was further described by Thompson and Kopell². Aiello et al.³ differentiated between entrapment of this nerve at the suprascapular notch and entrapment at the spinoglenoid notch. The condition has been assumed to be a rare cause of shoulder pain and dysfunction, and was often considered a diagnosis of exclusion, but there have been recent advancements that relate to anatomic variants, etiology, electrodiagnostic findings, and treatment options for suprascapular neuropathy. Nonetheless, the

etiology of suprascapular neuropathy and the indications for operative decompression of this nerve remain controversial.

The incidence and prevalence of suprascapular neuropathy remain largely unknown. The largest reported case series included only fifty-three patients⁴, while a meta-analysis revealed only eighty-eight published reports on suprascapular neuropathy from 1959 through 2001⁵. The number of articles relevant to suprascapular neuropathy has increased over the past ten years. Most reports deal with high-level overhead athletes, and the prevalence of suprascapular neuropathy in professional male volleyball players has ranged widely from 12.5% (twelve of ninety-six) to 33% (twenty-two of sixty-six)⁶⁻⁹. Recent reports have suggested an association between retracted rotator cuff tears and suprascapular neuropathy. Vad et al. reported that suprascapular neuropathy was associated with 8% (two) of twenty-five massive rotator cuff tears with muscle

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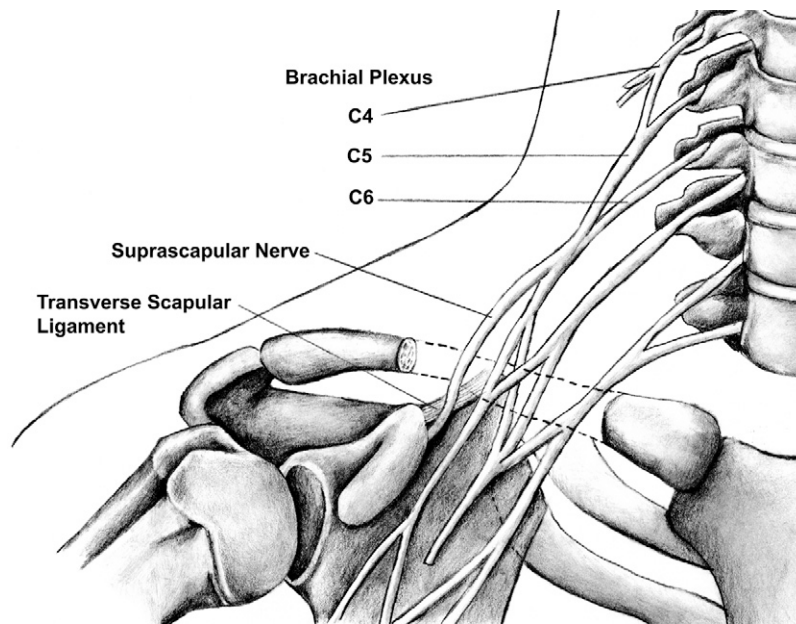


Fig. 1

The origin of the suprascapular nerve from the upper trunk of the brachial plexus.

atrophy¹⁰, Mallon et al. reported that all eight individuals with a massive rotator cuff tear (>5 cm) in their study had fatty infiltration of the muscle¹¹, and Costouros et al. reported that 27% (seven) of twenty-six patients with a massive rotator cuff tear and fatty infiltration had suprascapular neuropathy¹². Other authors have suggested that suprascapular neuropathy accounts for 1% to 2% of all shoulder pain¹³, although this remains conjectural at this time. As a result of heightened awareness of this condition, suprascapular neuropathy is being diagnosed with increasing frequency in groups thought to be at highest risk: i.e., athletes engaging in substantial overhead activity, individuals with a massive rotator cuff tear or those with a tear associated with fatty infiltration and/or atrophy of muscle, individuals with a labral tear and resultant paralabral cyst formation, and those with a space-occupying lesion in the suprascapular or spinoglenoid notch. The recent perception of a higher incidence of suprascapular neuropathy is likely due to increased interest and more careful clinical evaluation of overhead athletes, preoperative electrodiagnostic testing of patients with a massive rotator cuff tear, and better assessment of patients with marked weakness with external rotation of the shoulder.

Anatomy

The suprascapular nerve arises from the upper trunk of the brachial plexus with contributions from the C5 and C6 nerve roots and occasionally the C4 nerve root. The nerve initially runs posterior to the clavicle and travels obliquely across the superior border of the scapula, coursing into the suprascapular notch (Fig. 1). In most cases, the nerve traverses the suprascapular notch under the transverse scapular ligament while its associated artery travels over the ligament. The suprascapular notch is highly variable in shape and has been

classified into six types¹⁴. Forming the ceiling of the suprascapular notch, the transverse scapular ligament may hypertrophy and lead to stenosis within this notch. There are several reports of partial or complete ossification of the transverse scapular ligament¹⁴⁻¹⁶ (Figs. 2-A, 2-B, and 2-C). The nerve then runs roughly 3.0 cm medial to the supraglenoid tubercle and 1.8 cm medial from the posterior glenoid rim at the base of the scapular spine¹⁷. The distance from the palpable posterolateral corner of the acromion to the base of the scapular spine is roughly 4.5 cm¹⁸. The nerve most commonly supplies two motor branches to the supraspinatus muscle and proceeds to the spinoglenoid notch of the scapula, where it passes under the spinoglenoid (inferior transverse scapular) ligament, supplying at least two branches to the infraspinatus muscle^{17,18} (Figs. 3-A and 3-B). The spinoglenoid ligament has been classified into two types: Type I, which is a thin, indistinct band of tissue, and Type II, which is a well-formed ligament¹⁹. This ligament has been demonstrated to be present in 3% to 100% of human cadavers, although the definition of what was considered a ligament was not standardized in the reports¹⁹⁻²². One cadaveric study demonstrated tightening of the spinoglenoid ligament with internal rotation of the glenohumeral joint in any position of the arm²³.

The suprascapular nerve has been historically thought to be predominantly a motor nerve. However, cadaveric studies have demonstrated nerve branches to the glenohumeral joint, acromioclavicular joint, and coracoacromial ligament as well as to the skin^{17,18}. Some evidence indicates that the nerve may supply up to 70% of the sensation of the shoulder²⁴, which is supported by the clinical findings in studies by Ritchie et al.²⁵ and Matsumoto et al.²⁶, who reported less postoperative pain after a suprascapular nerve block

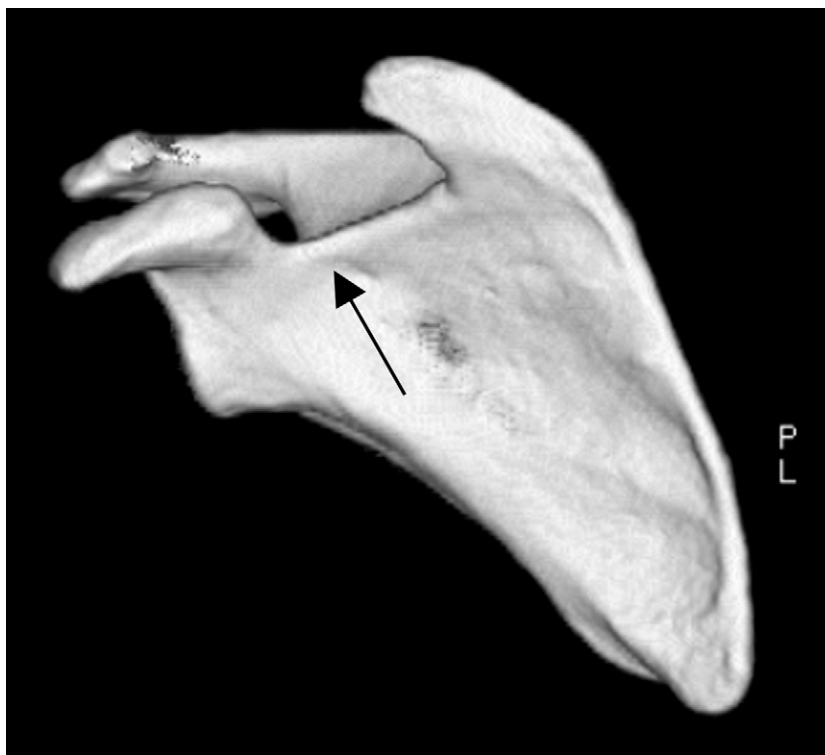


Fig. 2-A

Figs. 2-A, 2-B, and 2-C Images from three-dimensional reformatted computed tomography scans demonstrating variations in the anatomy of the suprascapular notch. **Fig. 2-A** Complete absence of the notch (arrow).

in patients who had had shoulder surgery. Vorster et al. recently demonstrated a glenohumeral sensory branch in 87% (twenty-seven) of thirty-one cadavers and an acromial sensory branch in 74% (twenty-three)²⁷. The evolving anatomic understanding of the sensory contributions of the suprascapular nerve helps explain the pain associated with injury of or traction on the nerve.

Etiology

A number of mechanisms of injury to the suprascapular nerve have been proposed. It has been thought that suprascapular neuropathy in overhead athletes is secondary to repetitive traction and microtrauma^{6,8,19}. The spinoglenoid ligament has been demonstrated to tighten when the shoulder is in a position for overhead throwing, resulting in increased pressure on the suprascapular nerve²³. Another proposed mechanism of suprascapular neuropathy is intimal damage to the suprascapular or axillary artery, leading to microemboli in the vasa nervorum²⁸. Individuals with a stenotic suprascapular or spinoglenoid notch, an ossified transverse scapular ligament or spinoglenoid ligament, or superiorly oriented fibers of the subscapularis muscle may be at increased risk for suprascapular neuropathy^{3,7,14-16,19}. The nerve may also be compressed at either the suprascapular or the spinoglenoid notch by a soft-tissue or bone tumor, a cyst secondary to labral or capsular injury, or anatomic variants that may lead to nerve

constriction²⁹⁻³². Numerous radiographic and clinical studies have confirmed the association between labral tears and paralabral cysts causing suprascapular neuropathy³³⁻³⁷ (Figs. 4-A and 4-B).

The suprascapular nerve is also at risk for traction injury when a retracted superior or posterior rotator cuff tear exists secondary to traction at the suprascapular notch or around the base of the scapular spine (Figs. 5-A and 5-B). In a cadaveric study, Albritton et al. demonstrated that increasing retraction of the supraspinatus tendon led to a reduction in the angle between the suprascapular nerve and its first motor branch and increased tension on the nerve³⁸. The first motor branch became taut in all specimens with 2 to 3 cm of supraspinatus tendon retraction. While Mallon et al. demonstrated electrodiagnostic findings of suprascapular neuropathy in eight patients with a massive rotator cuff tear¹¹, Vad et al. found only an 8% rate of suprascapular neuropathy (two of twenty-five) in patients with a full-thickness rotator cuff tear and muscle atrophy; they noted that a brachial plexopathy affecting the axillary nerve was more common (seen in 16%)¹⁰ (see Appendix).

Cadaveric studies have indicated that the maximum lateral advancement of a retracted rotator cuff tear is between 1 and 3 cm; with more advancement, the neurovascular pedicle is placed under tension^{18,39}. A study by Warner et al. demonstrated tension on the motor branches of



Fig. 2-B

A narrow notch (arrow).

the suprascapular nerve with rotator cuff advancement of >3 cm¹⁸, while a separate study by Greiner et al. demonstrated tension on the medial motor branches of this nerve with only 1 cm of advancement³⁹. In a clinical study, Hoellrich et al. did not find any electrodiagnostic findings of suprascapular neu-

ropathy after repairs of massive rotator cuff tears in nine patients with an average advancement of 2.5 cm (range, 2.0 to 3.5 cm) and suggested that the tendon can be mobilized and advanced up to 3.5 cm without risk to the nerve⁴⁰ (see Appendix).

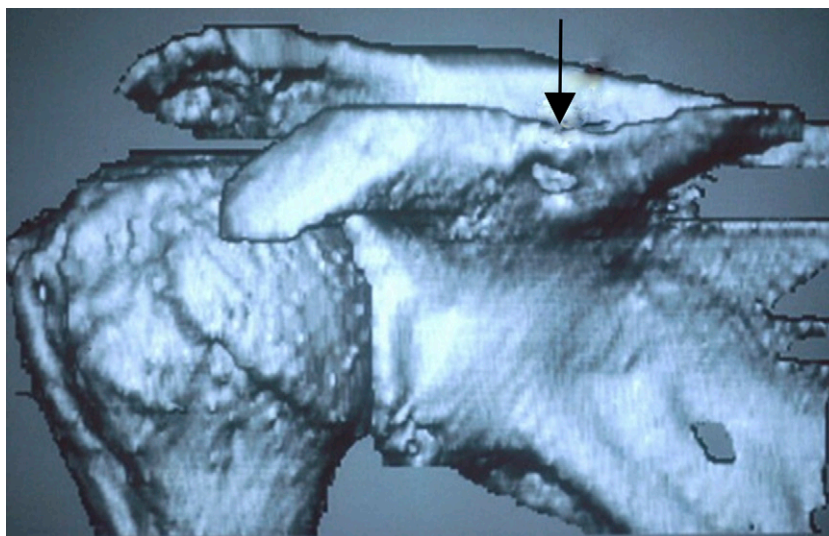


Fig. 2-C

An ossified notch (arrow).

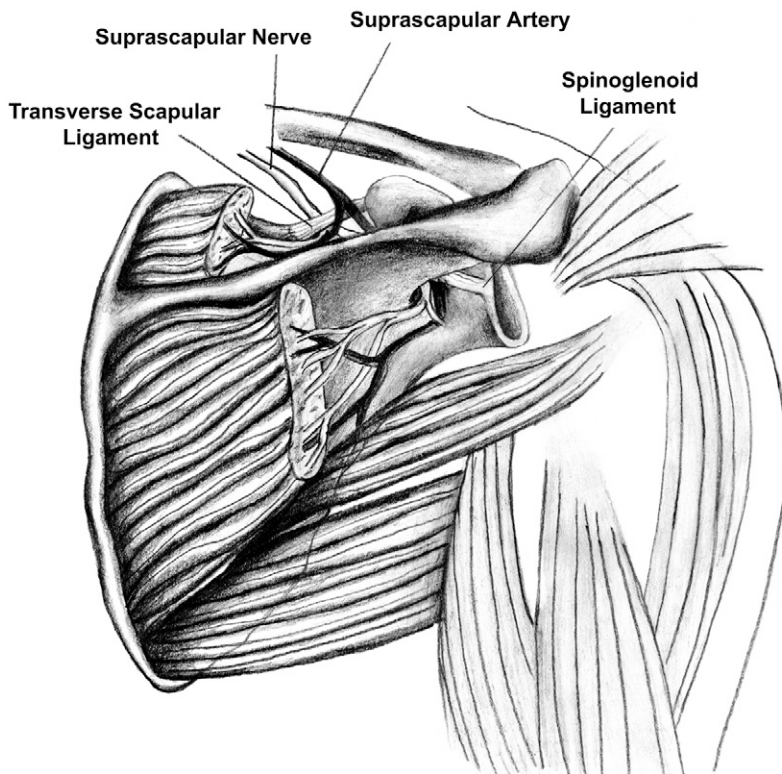


Fig. 3-A

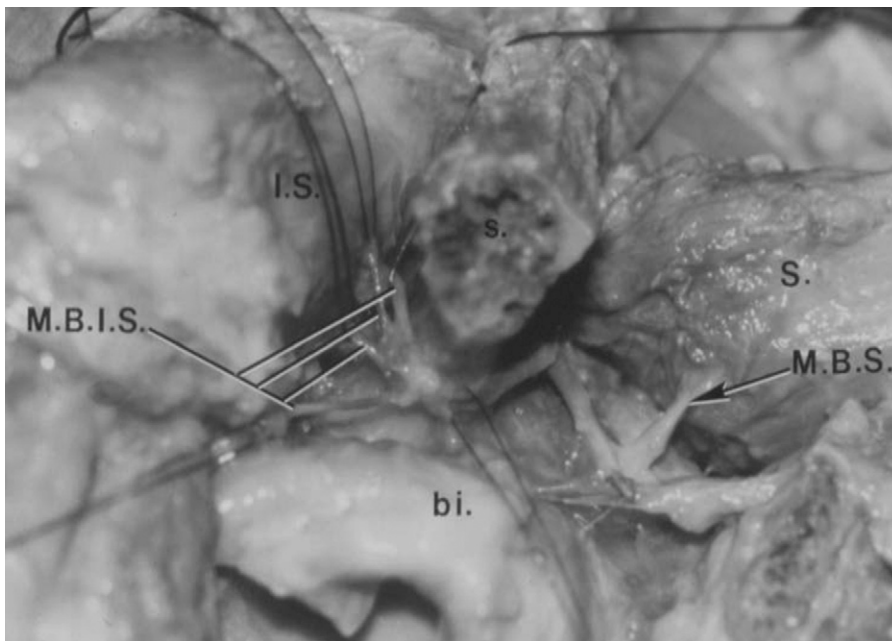


Fig. 3-B

Fig. 3-A Anatomy of the course of the suprascapular nerve passing under the transverse scapular ligament and the spinoglenoid ligament with motor branches to the supraspinatus and infraspinatus. Note the suprascapular artery, which passes above the transverse scapular ligament. **Fig. 3-B** Anatomy of the suprascapular nerve. This photograph faces medially and is of a right shoulder. The deltoid and acromion have been removed, and the supraspinatus (S.) and infraspinatus (I.S.) have been divided from their respective insertions and reflected proximally. The base of the scapular spine (s.) and the origin of the biceps tendon (bi.) are shown. There is a single motor branch to the supraspinatus (M.B.S.) and four branches to the infraspinatus (M.B.I.S.). A suture has been placed around the suprascapular nerve at the base of the scapular spine. (Reprinted from: Warner JP, Krushell RJ, Masquelet A, Gerber C. Anatomy and relationships of the suprascapular nerve: anatomical constraints to mobilization of the supraspinatus and infraspinatus muscles in the management of massive rotator-cuff tears. *J Bone Joint Surg Am.* 1992;74:38.)

Suprascapular neuropathy may also result from Parsonage-Turner syndrome, a viral neuritis with a predilection for affecting the suprascapular nerve, although other nerves of the shoulder are usually also involved⁴¹. Suprascapular neuropathy has been demonstrated after glenohumeral dislocation⁴²⁻⁴⁴, fractures about the shoulder girdle⁴⁵⁻⁴⁷, penetrating injury to the

shoulder, and surgical procedures requiring a posterior approach to the scapula⁴⁸.

Physical Examination and Diagnostic Studies

Patients with a suprascapular nerve injury typically have an insidious onset of dull, aching pain localized to the superior or



Fig. 4-A

Sagittal oblique (Fig. 4-A) and axial fast-spin-echo (Fig. 4-B) magnetic resonance images demonstrating a large paralabral cyst (arrow) about the spinoglenoid notch.

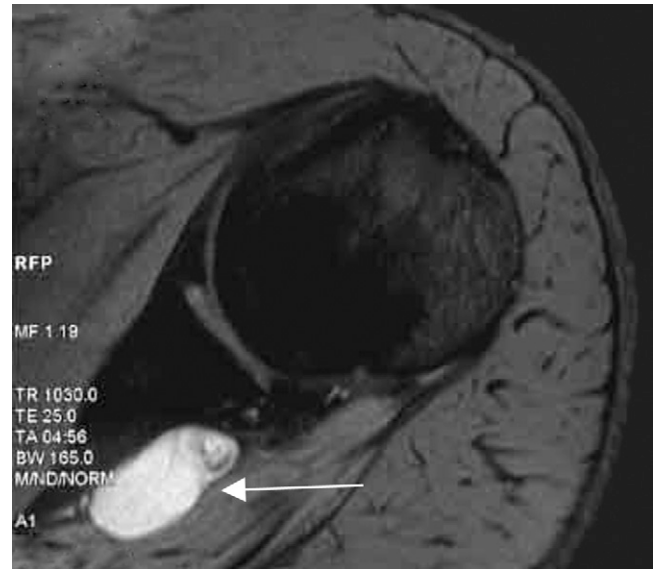


Fig. 4-B

posterolateral aspect of the shoulder. Complaints of shoulder weakness and fatigue with overhead activities are common, whereas the presence of night pain is variable. While a history of trauma or repetitive overhead activity can often be obtained, it is difficult to diagnose a suprascapular nerve lesion on the basis of the history alone as suprascapular neuropathy is often associated with other pathological entities in the shoulder⁴⁹.

A complete physical examination of the cervical spine and both shoulders is needed to evaluate the patient for a possible rotator cuff tear, labral injury, and other abnormal shoulder or neck conditions. Some patients may have few symptoms associated with the physical finding of atrophy of the supraspinatus and/or infraspinatus fossa⁵⁰ (Fig. 6). Individuals with nerve injury about the suprascapular notch may have tenderness to palpation posterior to the clavicle in the region between the clavicle and the scapular spine, weakness with resisted abduction and external rotation of the shoulder, and atrophy of the supraspinatus and infraspinatus fossa. With injury about the spinoglenoid notch there may be tenderness deep and posterior to the acromioclavicular joint, pain with cross-body shoulder adduction secondary to tightening of the spinoglenoid ligament²³, infraspinatus muscle atrophy with sparing of the supraspinatus muscle, and decreased strength in external rotation of the shoulder. In long-standing cases, the teres minor muscle may compensate for the loss of the infraspinatus muscle and maintain nearly normal strength of the shoulder in external rotation⁶.

Patients with a history or physical examination findings suggestive of suprascapular neuropathy should undergo further testing. With a history of trauma and the new onset of

nerve symptoms, radiographs are recommended to assess for fracture, exuberant callus formation, osseous dysplasia, bone tumor, and osseous variants of the suprascapular notch. In addition to standard shoulder radiographs, a suprascapular notch view (with the beam directed 15° to 30° cephalad) allows evaluation of osseous notch variants, and a Stryker notch view will show the suprascapular notch (Fig. 7). Computed tomography scans may better define fractures and anatomy but are generally of limited utility for the majority of patients with suprascapular neuropathy. However, computed tomography may be the imaging study of choice to document an ossified transverse scapular ligament or characterize a fracture. Magnetic resonance imaging is the best study for visualizing the course of the nerve and for detecting space-occupying lesions^{35,51,52}. Magnetic resonance imaging provides excellent evaluation of the labrum, associated cysts, rotator cuff tendons, and muscle quality including fatty infiltration and atrophy⁵³ (Fig. 8). Combining magnetic resonance imaging with arthrography increases sensitivity for identifying labral abnormalities⁵⁴.

Although magnetic resonance imaging may demonstrate an apparent cause for suprascapular neuropathy, electrodiagnostic studies including electromyography and nerve conduction velocity studies remain the standard for diagnosis and confirmation of suprascapular nerve injury. Indications for electromyography and nerve conduction velocity studies include unexplained continued persistent shoulder pain, atrophy and weakness with no evidence of a rotator cuff tear, or magnetic resonance imaging findings of fatty infiltration and edema of rotator cuff muscles with no evidence of a rotator cuff tear^{55,56}. If a patient has a massive retracted rotator cuff tear or a labral tear with an associated cyst, preoperative electrodiagnostic studies

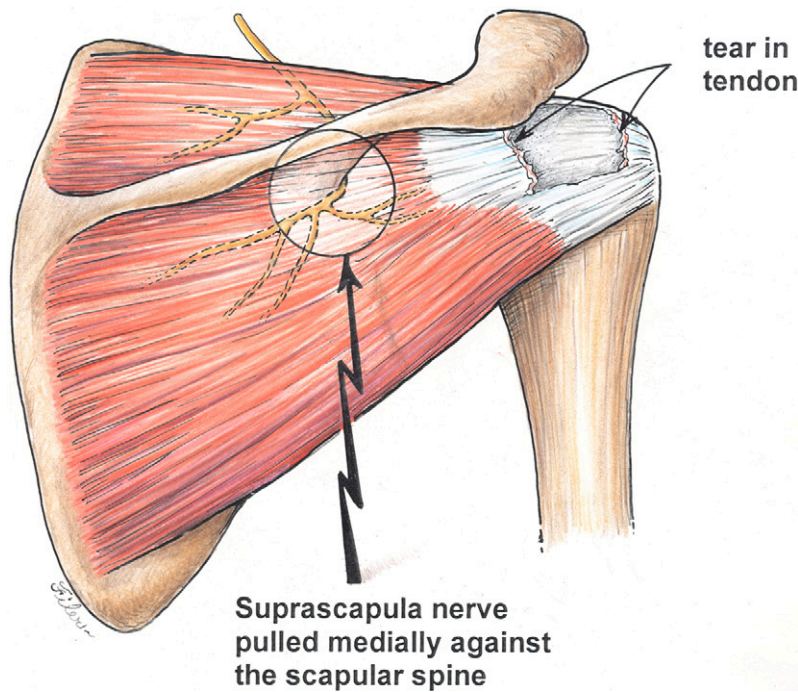


Fig. 5-A

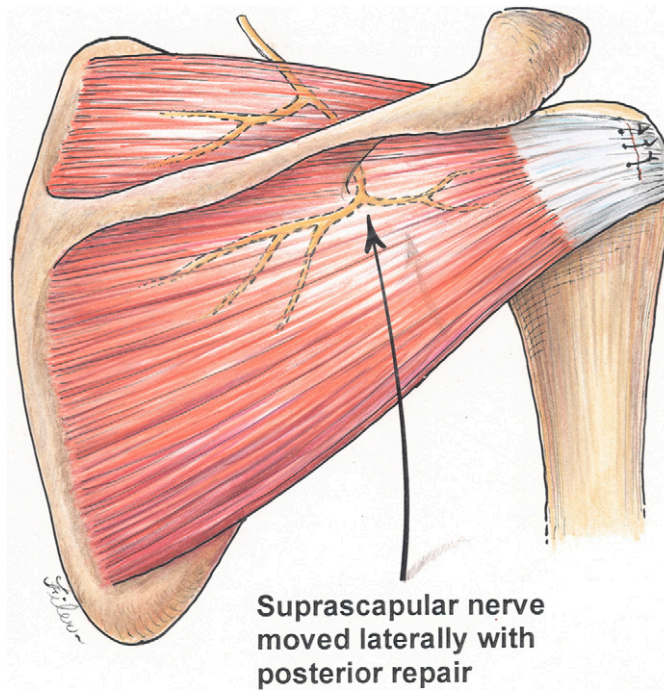


Fig. 5-B

Fig. 5-A Representation of suprascapular nerve traction around the base of the spine of the scapula following medial and inferior retraction of a massive tear involving the supraspinatus and infraspinatus tendons. (Reprinted, with permission of Elsevier, from: Costouros JG, Porramatikul M, Lie DT, Warner JJP. Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy*. 2007;23:1152-61.) **Fig. 5-B** Representation that postulates the mechanism for resolution of suprascapular neuropathy by relieving traction around the base of the scapular spine with infraspinatus tendon repair. (Reprinted, with permission of Elsevier, from: Costouros JG, Porramatikul M, Lie DT, Warner JJP. Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy*. 2007;23:1152-61.)



Fig. 6
Infraspinatus atrophy noted on physical examination secondary to compression of the suprascapular nerve at the spinoglenoid notch.

may help to rule out suprascapular neuropathy¹⁰, although this remains controversial. Electromyography may demonstrate denervation of the supraspinatus or infraspinatus muscle with resultant fibrillations and sharp waves. With nerve conduction velocity studies, the motor conduction velocities of the suprascapular nerve provide a latency value from the Erb point to the supraspinatus and infraspinatus muscles as well as a latency value between the muscles. Evaluation of sensory velocities is less useful as the sensory innervation of this nerve is less well defined. Normative standards for electrodiagnostic studies have been established, although variability remains⁵⁷⁻⁶⁰, and the specific criteria for interpreting the study may vary somewhat between centers. Electromyography and nerve conduction velocity studies have been shown to be accurate (91%; seventy-two of seventy-nine) in detecting nerve injury associated with muscle weakness⁶¹. The overall sensitivity and specificity of electromyography and nerve conduction velocity studies have been debated and are highly variable depending on the condition being tested. For example, the sensitivity of electrodiagnostic testing in patients with other peripheral neuropathies such as carpal tunnel syndrome was shown to be 74% (thirty-two of forty-three) to 91% (126 of 138)^{62,63} in prospective studies. Because of this potential variability, a fluoroscopy-guided injection of local anesthetic in the region of the suprascapular nerve may be useful to evaluate for pain relief in patients for whom the findings on electromyography or nerve conduction velocity studies are negative for suprascapular neuropathy but who continue to have unexplained symptoms. The request for the electromyography or

nerve conduction velocity studies must specify evaluation of the suprascapular nerve as general upper-extremity studies may not provide all necessary information. It has been suggested that bilateral studies may be useful to enable a comparison of values.

Treatment and Results

Nonoperative Treatment

The initial treatment for most isolated suprascapular nerve lesions not associated with a space-occupying lesion or a rotator cuff tear is activity modification, nonsteroidal anti-inflammatory drugs, and physical therapy⁶⁴⁻⁶⁶. Avoidance of overhead activities and a program of therapy focusing on shoulder motion and muscle strengthening, including scapular stabilization and mechanics, should be initiated⁶⁷⁻⁶⁹. Drez found that four patients with isolated suprascapular neuropathy had improvement with therapy and recommended six to eight months of nonoperative treatment⁶⁷. Walsworth et al. found that three of their five patients with suprascapular neuropathy had improvement with therapy, one required an operation, and one was lost to follow-up⁶⁹. Black and Lombardo reported four cases of suprascapular neuropathy involving only the infraspinatus, with all patients having improvement with use of nonoperative modalities over six months to one year⁷⁰. In a study of fifteen patients with isolated suprascapular neuropathy managed nonoperatively and followed for an average of almost four years, five patients had an excellent result and seven had a good result, while three patients required surgical treatment⁶⁴. The



Fig. 7
A Stryker notch radiograph of the left shoulder, made with the beam aimed 15° cephalad to provide visualization of the suprascapular notch (arrow).

authors recommended nonoperative treatment except in the setting of a space-occupying lesion or persistent pain. Callahan et al. detailed twenty-three cases of suprascapular neuropathy for which nonoperative management had failed and that were treated with surgery⁷¹. While most authors have agreed that an initial course of nonoperative treatment in the absence of a space-occupying lesion is prudent and may result in the resolution of pain and weakness, the rate of success of nonoperative treatment is still not clear. In fact, some authors have suggested that operative intervention should be undertaken as soon as suprascapular neuropathy is confirmed to prevent further muscle damage, claiming therapy in the setting of a compressed nerve may be contraindicated⁷². However, currently there is a lack of data linking a delay in surgery for suprascapular neuropathy with irreversible nerve damage. Nonoperative treatment of suprascapular neuropathy has been demonstrated to lead to poorer outcomes when the nerve is being compressed by a mass or a cyst. Piatt et al. reported that ten (53%) of nineteen patients with pain secondary to a spinglenoid cyst were satisfied with the outcome after nonop-

erative management as compared with twenty-six (96%) of twenty-seven patients who were satisfied with the outcome after operative treatment⁷³. Of these forty-six patients, fourteen had had suprascapular neuropathy proven by electrodiagnostic studies preoperatively, four had had normal findings on electrodiagnostic studies, and the rest were not evaluated with electrodiagnostic testing (see Appendix).

Operative Treatment

The decision to recommend operative treatment must be tailored to the individual patient and is based on clinical and electrodiagnostic findings, the cause of the injury, and the location of the lesion. We are not aware of any prospective studies comparing operative and nonoperative treatment for suprascapular neuropathy. Operative treatment may include decompression of the suprascapular nerve with or without repair of associated shoulder abnormalities. While some authors have recommended concomitant decompression of the suprascapular notch with repair of a massive rotator cuff tear and concomitant decompression of labral cysts with labral repair^{37,74-76}, others have shown resolution of suprascapular neuropathy with isolated rotator cuff or labral repair^{12,77,78}. Isolated suprascapular neuropathy that has not responded to a trial of nonoperative treatment is best managed with suprascapular nerve decompression^{73,79,80}.

Operative Treatment at the Suprascapular Notch

Isolated nerve injury at the suprascapular notch may be caused by repetitive shoulder motion, constriction due to anatomic variants, traction from a rotator cuff tear, or a space-occupying lesion. In most cases, operative treatment consists of release of the transverse scapular ligament with excision or decompression of any associated mass. Release of the transverse scapular ligament may be undertaken through an open or arthroscopic technique. The traditional open approach is performed through a transverse incision just cephalad to the scapular spine or alternatively through a vertical incision 4.5 cm from the posterolateral edge of the acromion. The trapezius muscle is elevated from the scapular spine, the supraspinatus muscle is reflected, and dissection is carried down to the suprascapular notch posteriorly. The transverse scapular ligament is identified and released, but caution must be used to not damage the overlying vascular structures⁷² (Figs. 9-A, 9-B, and 9-C). A notchplasty may also be undertaken to release a restrictive suprascapular notch or to decompress a notch with complete osseous bridging⁸¹.

The results of 267 open decompressions of the suprascapular nerve at the suprascapular notch by release of the transverse scapular ligament have been reported^{14,13,28,29,45,64,67,68,71,79,80,82-98}. Few complications were noted, and most patients had resolution of pain and improvement of muscle strength, although reversal of the muscle atrophy was not always observed. The largest series included thirty-nine patients: thirty-one had profound muscle weakness, and 90% (twenty-eight) of them showed improvement in supraspinatus muscle strength to grade 4 or better, although improvement in infraspinatus muscle strength occurred less often⁸⁰. Of eight patients with severe pain



Fig. 8

Sagittal oblique fast-spin-echo magnetic resonance image demonstrating fatty atrophy of the supraspinatus (white arrow) and infraspinatus (black arrow) in association with suprascapular neuropathy at the suprascapular notch secondary to a massive rotator cuff tear. In addition, there is atrophy of the teres minor (asterisk).

in that series, seven had improvement. Another study, of twenty-five patients, demonstrated improvement in the Constant score and an 89% rate of good, very good, or excellent results after open suprascapular nerve decompression. However, in this group, only 52% (thirteen) of the twenty-five patients had resolution of their muscle atrophy⁷⁹.

Endoscopic exploration of the brachial plexus and suprascapular nerve was described by Krishnan et al.⁹⁹. The all-arthroscopic approach for release of the transverse scapular ligament was proposed by Bhatia et al., who recommended additional portals, nerve stimulation, and utilization of a 70° arthroscope¹⁰⁰. Arthroscopic decompression of an ossified suprascapular notch with use of a Kerrison rongeur was described in a case report⁸¹, and a technique was described for arthroscopically assisted percutaneous release of the transverse scapular ligament with a 14-gauge needle¹⁰¹. More recent reports on arthroscopic techniques have detailed common portals from which the transverse scapular ligament and the spinoglenoid ligament can be decompressed^{102,103}. Lafosse et al. described an all-arthroscopic technique that includes the use of a suprascapular nerve portal, which is positioned between the clavicle and the scapular spine approximately 7 cm medial to the lateral border of the acromion⁷⁵. The transverse scapular ligament is initially identified as the medial continuity of the conoid ligament above the suprascapular notch. A spinal needle is used to guide the placement of the suprascapular nerve portal at an angle orthogonal to the suprascapular fossa and slightly anterior toward the trans-

verse scapular ligament. A blunt trocar is placed through the suprascapular nerve portal and positioned lateral to the suprascapular nerve within the notch for protection. A second portal 1.5 cm lateral to the suprascapular nerve portal is created to allow introduction of arthroscopic scissors for release of the transverse scapular ligament (Figs. 10, 11-A, and 11-B).

Lafosse et al. recently reported the early results of the arthroscopic approach in ten patients with suprascapular neuropathy secondary to compression at the suprascapular notch⁷⁵. At a mean of six months postoperatively, seven of the ten patients had complete normalization of electromyographic findings; two electromyograms showed partial recovery of the nerve. Nine patients graded their outcome as excellent with complete relief of pain. Although the arthroscopic technique is novel and outcomes have been reported in only a small number of patients, the early results are encouraging. These procedures require advanced arthroscopic skills, and care must be taken to not damage the surrounding neurovascular structures. Reineck and Krishnan reported on three patients with a subligamentous suprascapular artery noted during arthroscopic release; these patients represented 3% of the 100 patients undergoing the procedure¹⁰⁴. The authors recommended a medial arthroscopic approach to aid in identification of anatomic variations that may predispose to iatrogenic injury.

There is limited information regarding the treatment of suprascapular neuropathy secondary to traction as a result of a massive rotator cuff tear. Mallon et al. reported that, of four

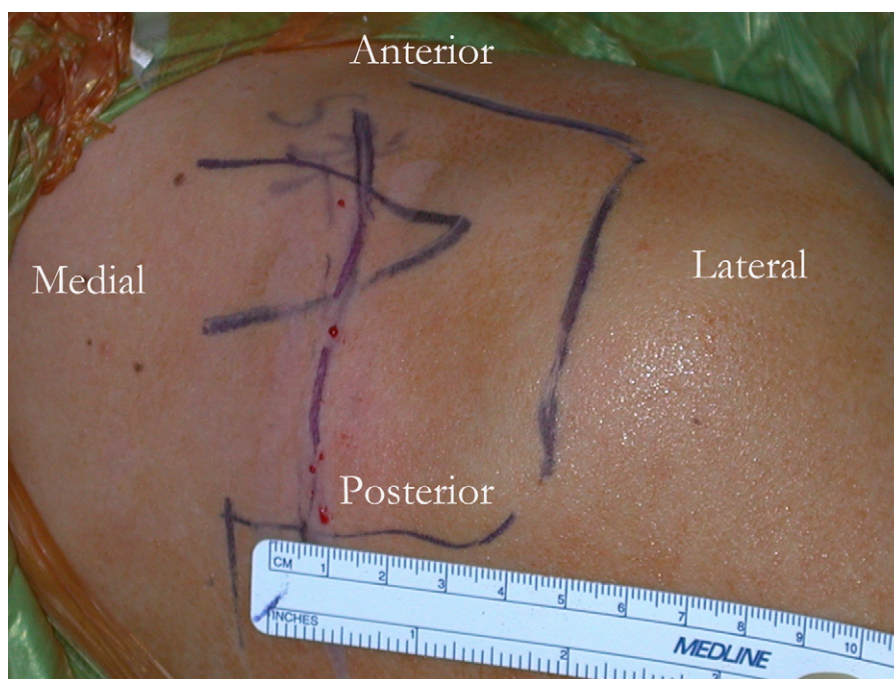


Fig. 9-A

Figs. 9-A, 9-B, and 9-C Technique for open decompression of the suprascapular nerve. **Fig. 9-A** A vertical incision is made 4.5 cm from the posterolateral edge of the acromion extending superiorly.

patients with suprascapular neuropathy and a massive retracted rotator cuff tear, two who had postoperative electromyography showed reinnervation potentials after partial arthroscopic rotator cuff repair¹¹. Costouros et al. found that six of six patients with electrodiagnostically confirmed suprascapular neuropathy who had undergone partial or complete rotator cuff repair were seen to have partial or full resolution of the suprascapular neuropathy on postoperative electromyography and nerve conduction velocity studies¹². These reports suggest that suprascapular neuropathy secondary to traction from a rotator cuff tear may partially or completely resolve with rotator cuff repair alone. We know of no published studies comparing rotator cuff repair alone with rotator cuff repair combined with suprascapular nerve release in the setting of suprascapular neuropathy.

Operative Treatment at the Spinoglenoid Notch

Suprascapular neuropathy at the spinoglenoid notch is typically secondary to compression of the nerve by a space-occupying lesion. The majority of these lesions are cysts, often secondary to a concomitant labral tear with communication to the joint, or lipomas or other benign tumors in the region^{29,33}. Surgical management of a benign tumor often requires an open approach^{96,105}, with resection of the lesion. Operative treatment of periarticular cysts associated with other shoulder abnormalities remains much more controversial. Patients with suprascapular neuropathy due to extrinsic compression about the spinoglenoid notch have been reported to have poor results with nonoperative treatment^{4,33,35}. Piatt et al. reported that only two

of nineteen patients with a spinoglenoid notch cyst became pain-free with conservative treatment, and the authors highlighted a much higher rate of patient satisfaction with surgery⁷³. They suggested nonoperative management only for patients who are minimally symptomatic and recommended that those with more symptoms have aspiration or arthroscopic decompression of the cyst with labral repair⁷³. It is important to distinguish a true spinoglenoid notch cyst from distended veins, which may also cause compression at the spinoglenoid notch and which can lead to complications if aspiration or excision is attempted¹⁰⁶. A spinoglenoid ligament, which may lead to compression of the nerve, was observed in 3% to 100% of cadavers^{19,22}. Decompression of the spinoglenoid ligament may be done through an open or arthroscopic approach. If an arthroscopic approach is used, the surgeon utilizes posteromedial and posterolateral portals in the infraspinatus fossa²³ or employs a sub-acromial approach¹⁰³.

Sonographically guided needle aspiration of cysts has been reported, and the results have been mixed^{107,108}. Several authors have reported good pain relief in all patients and low recurrence rates^{35,109-111}, while others have observed recurrence rates between 45% and 75%^{73,112}. It appears that sonographically guided aspiration is a safe technique, but the results vary widely. The variable risk of recurrence is likely secondary to the fact that aspiration does not address the underlying intra-articular pathological condition.

Surgical management of a spinoglenoid notch cyst may consist of open or arthroscopic decompression of the cyst with or without labral repair, or it may consist of labral repair alone.

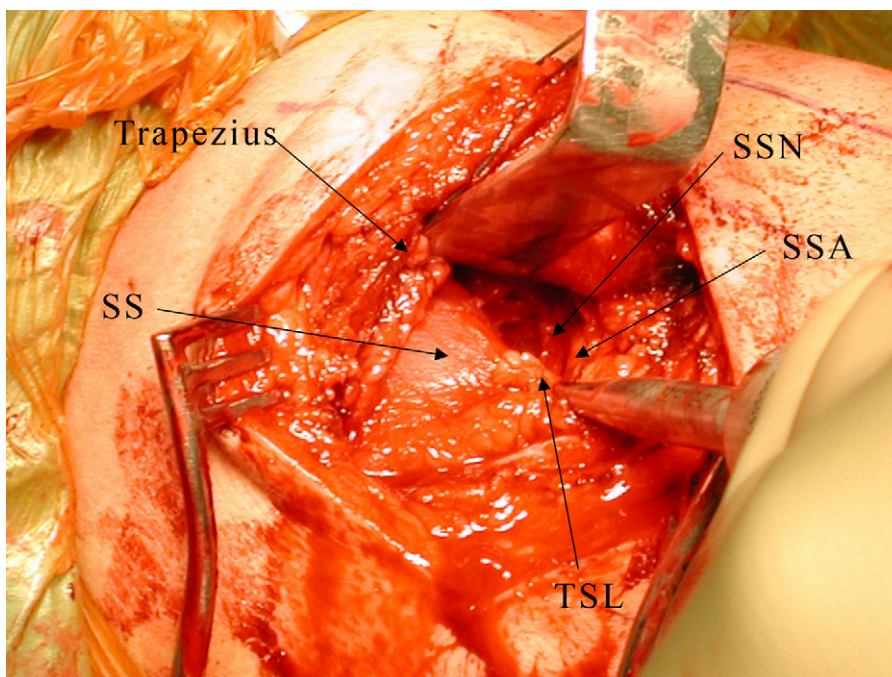


Fig. 9-B

The trapezius is elevated off the scapular spine, and the supraspinatus (SS) is retracted inferiorly and posteriorly, revealing the transverse scapular ligament (TSL), suprascapular artery (SSA), and suprascapular nerve (SSN).

Open decompression may be undertaken through a posterior approach to the shoulder; however, this allows limited visualization of the labrum and other associated intra-articular

pathological conditions⁴. Arthroscopic management is a more versatile technique that allows cyst decompression in addition to visualization and treatment of labral lesions. Some authors

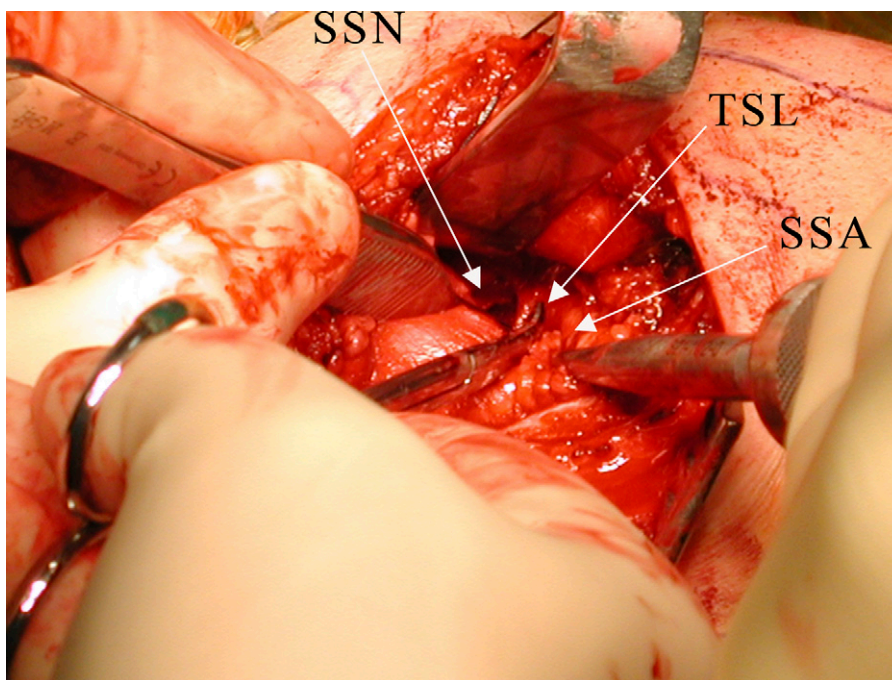


Fig. 9-C

The suprascapular nerve (SSN) is retracted with a blunt instrument while the transverse scapular ligament (TSL) is sharply divided, with care taken to protect the suprascapular artery (SSA)^{96,105}.

have suggested that the risk of recurrence is lower with arthroscopic methods^{73,113}. Fehrman et al. evaluated six individuals with suprascapular neuropathy secondary to a spinoglenoid notch cyst and found that five of them had complete resolution of pain and one had some relief of pain after a combined open and arthroscopic approach³³. Westerheide et al. evaluated the results of fourteen patients treated only with arthroscopic decompression of the cyst and, at a mean of fifty-

one months postoperatively, no patient had had a recurrence and all had improved shoulder function, decreased pain, and increased strength in external rotation of the shoulder³⁶. Chen et al. reported that three of three patients demonstrated resolution of the cyst on magnetic resonance imaging and resolution of suprascapular neuropathy on postoperative electromyography after arthroscopic cyst decompression and repair of a superior labral anterior-posterior (SLAP) tear¹¹⁴. Lichtenberg et al. evaluated eight patients with suprascapular neuropathy; all of those with an associated labral lesion (six of the eight) were treated with arthroscopic decompression of the cyst and labral repair¹¹⁵. All patients had pain relief and improvement in strength and function at the time of follow-up. Antoniou et al. evaluated fifty-three patients with compressive suprascapular neuropathy and noted that those who underwent operative decompression of a spinoglenoid notch cyst responded better than did those in whom such a cyst was treated nonoperatively; the results of open and arthroscopic decompressions were similar⁴.

Some authors have reported that labral repair alone without decompression of the cyst is sufficient treatment for suprascapular neuropathy secondary to a spinoglenoid notch cyst. In a study in which ten patients with a spinoglenoid notch cyst and a concomitant labral tear were treated with labral repair alone, eight of eight patients available for follow-up at an average of 10.2 months demonstrated resolution of the cyst on postoperative magnetic resonance imaging⁷⁸. Preoperatively, four of the ten patients had electrodiagnostic findings of suprascapular neuropathy, and repeat nerve conduction velocity studies performed postoperatively demonstrated resolution of the suprascapular neuropathy in all patients. A case report documented radiographic evidence of resolution of a spinoglenoid notch cyst and reinnervation on electromyography after debridement of a SLAP lesion¹¹⁶. Schroder et al. evaluated forty-two patients with a labral tear and a spinoglenoid notch cyst who had been treated with labral repair alone⁷⁷. Eighty-eight percent (thirty-seven) of the patients were found to have complete resolution of the cyst on postoperative magnetic res-

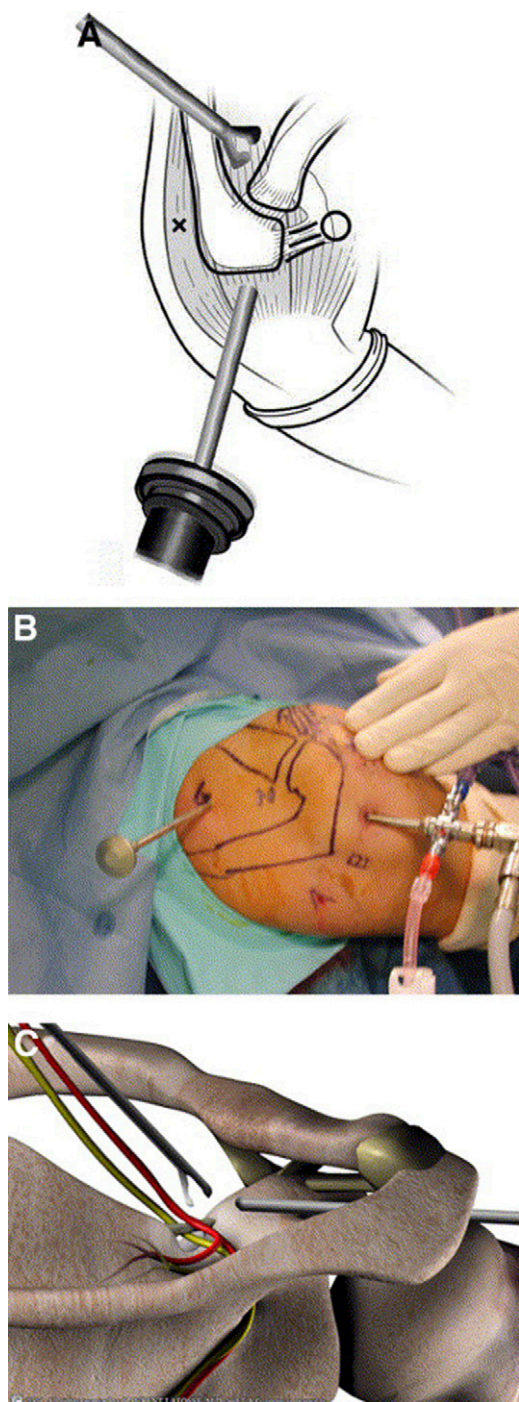


Fig. 10

A: Superior view of portals for arthroscopic decompression of the suprascapular nerve. A blunt trocar is introduced through the suprascapular nerve portal approximately 2 cm medial to the Neviaser portal¹¹⁷. The arthroscope is visualizing through the lateral portal (x = standard posterior portal). B: Portals on a right shoulder. The arthroscope is placed in the lateral portal, and the trocar shows the position of the suprascapular nerve portal medial to the Neviaser portal¹¹⁷. The trocar is used to dissect the suprascapular nerve and protect it during ligament release. A portal approximately 1.5 cm lateral to the suprascapular nerve portal (N) is used to transect the transverse scapular ligament with use of arthroscopic scissors. C: Illustration of arthroscopic release. The blunt trocar used to protect the suprascapular nerve is not shown. (Reprinted, with permission of Elsevier, from: Lafosse L, Tomasi A, Corbett S, Baier G, Willems K, Gobezie R. Arthroscopic release of suprascapular nerve entrapment at the suprascapular notch: technique and preliminary results. *Arthroscopy*. 2007;23:34-42.)

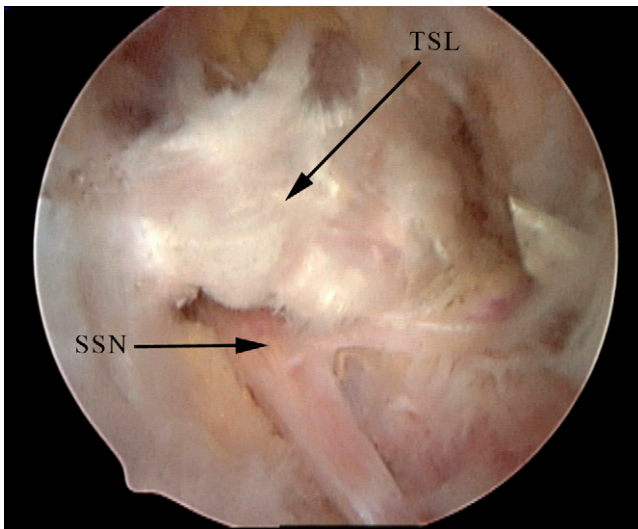


Fig. 11-A

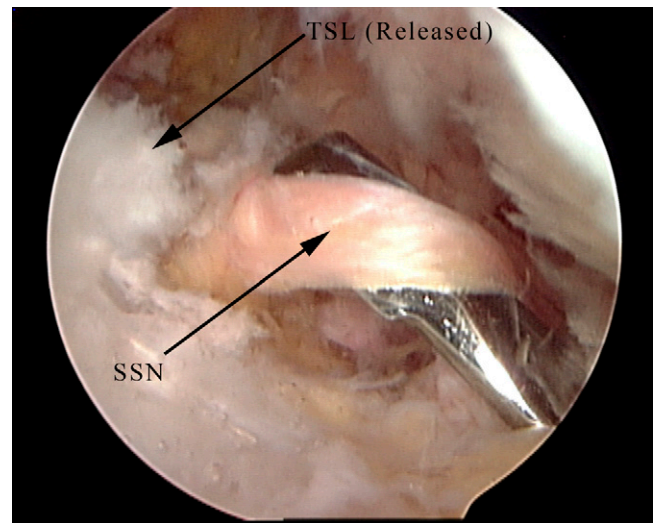


Fig. 11-B

Intraoperative arthroscopic images of the transverse scapular ligament (TSL) and suprascapular nerve (SSN) before (Fig. 11-A) and after (Fig. 11-B) release of the ligament.

onance imaging, with the other 12% (five) showing a decrease in cyst size. In a retrospective study, seventy-three patients with suprascapular neuropathy secondary to a spinoglenoid notch cyst were divided into four treatment groups: (1) nonoperative, (2) needle aspiration, (3) arthroscopic treatment of the labrum only, and (4) open or arthroscopic decompression of the cyst with concomitant labral fixation⁷³. Individuals in Group 4 demonstrated the highest satisfaction rating, and Groups 3 and 4 (operative treatment) combined had a higher satisfaction rating than Groups 1 and 2 (nonoperative treatment).

Overview

The diagnosis of suprascapular neuropathy has historically been considered a diagnosis of exclusion. In recent years, many reports have highlighted the fact that suprascapular neuropathy may cause substantial pain and weakness both in patients with and in those without concomitant shoulder pathology. Causes of suprascapular neuropathy include traction injury or direct compression of the nerve. The association of ganglion cysts from a labral tear with compressive suprascapular neuropathy has been well established, while more recent studies have demonstrated that traction injury to the nerve occurs with retraction of a large rotator cuff tear. Physical examination may reveal atrophy of the supraspinatus and/or infraspinatus muscles with resultant weakness in forward flexion and/or external rotation of the shoulder. Magnetic resonance imaging (or magnetic resonance imaging arthrography) is the preferred modality with which to assess for atrophy of the rotator cuff and causes of suprascapular nerve compression, while electromyography and nerve conduction velocity studies remain the standard for confirmation of the diagnosis of suprascapular neuropathy. Initial treatment is usually nonoperative, consisting of activity modification, physical therapy, and nonsteroidal anti-inflammatory drugs. Operative intervention is considered for patients with nerve compression by a discrete

structural lesion, with symptoms refractory to more conservative measures, and those with coexistent shoulder injuries requiring surgical treatment. Decompression of the suprascapular nerve may be accomplished through an open surgical approach, with good results reported overall. More recent reports have described nerve decompression performed arthroscopically and, although limited follow-up data are available, the early results appear promising. More clinical studies are needed to determine if treatment of the offending pathology in cases apparently caused by traction with a rotator cuff tear or compression from a cyst secondary to a labral tear is sufficient or whether concomitant decompression of the nerve is warranted for optimal management of this neuropathy.

Appendix

eA Tables summarizing studies of suprascapular neuropathy associated with rotator cuff tears and of the results of nonoperative management of suprascapular neuropathy are available with the electronic version of this article on our web site at jbjs.org (go to the article citation and click on "Supporting Data"). ■

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Robert E. Boykin, MD
Jon J.P. Warner, MD
Harvard Shoulder Service,
Massachusetts General Hospital,
55 Fruit Street,
Yawkey Center for Outpatient Care,
Suite 3200,
3G, Room 3-046, Boston,
MA 02114.
E-mail address for J.J.P. Warner: jwarner@partners.org

Darren J. Friedman, MD
Department of Orthopaedic Surgery,
New York University School of Medicine,
Hospital for Joint Diseases, 301 East 17th Street,
New York, NY 10003

Laurence D. Higgins, MD
Department of Orthopaedic Surgery,
Brigham and Women's Hospital,
75 Francis Street, Boston,
MA 02115

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