

# Suprascapular Neuropathy

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## Abstract

Suprascapular neuropathy is a relatively uncommon but significant cause of shoulder pain and dysfunction. The suprascapular nerve follows a tortuous course from the neck to the posterior shoulder. There are several potential causes of nerve entrapment along this path, particularly at the vulnerable suprascapular and spinoglenoid notches, where nerve excursion is limited by bony and ligamentous constraints. Additional extrinsic compression may be caused by glenohumeral joint–related ganglion cysts or soft-tissue masses. Traction neuropathy may occur following excessive nerve excursion during overhead sports or as a result of massive, retracted rotator cuff tears in older patients. Diagnosis is based on a careful history, physical examination, focused imaging, and electrodiagnostic studies. In the absence of a clear structural compression or overstretching of the nerve, treatment initially should be nonsurgical, with activity modification and physical therapy. Discrete nerve compression or failure of nonsurgical measures warrants early surgical intervention. Arthroscopic alternatives to the traditional open suprascapular and/or spinoglenoid notch decompressions have the benefit of simultaneously diagnosing and addressing intra-articular and/or subacromial pathology while minimizing morbidity. In most patients, both open and arthroscopic approaches provide reliable pain relief and improvements in function; return of strength and muscle bulk is less predictable.

Suprascapular neuropathy was first described by Kopell and Thompson in 1959.<sup>1</sup> Although it is a relatively uncommon cause of shoulder pain and dysfunction, latent suprascapular neuropathy is common in overhead athletes,<sup>2</sup> and in some scenarios, nerve entrapment and/or injury can contribute to significant disability. From the cervical nerves root to innervation of the infraspinatus muscle along the posterior aspect of the scapula, the suprascapular nerve is vulnerable to various insults at and between the narrow suprascapular and spinoglenoid notches. The supraspinatus muscle will not be affected by spinoglenoid notch neuropathy, whereas both supra- and in-

fraspinatus atrophy will occur with suprascapular notch involvement, making the distribution of muscle involvement helpful in localizing the site of injury. And because traction injuries typically respond well to nonsurgical management whereas mass compression usually responds best to surgical decompression, the cause of nerve dysfunction should be determined.

## Anatomy

The suprascapular nerve provides sensory fibers to the coracoacromial ligament, acromioclavicular and glenohumeral joints and, in 15% of the

Figure 1

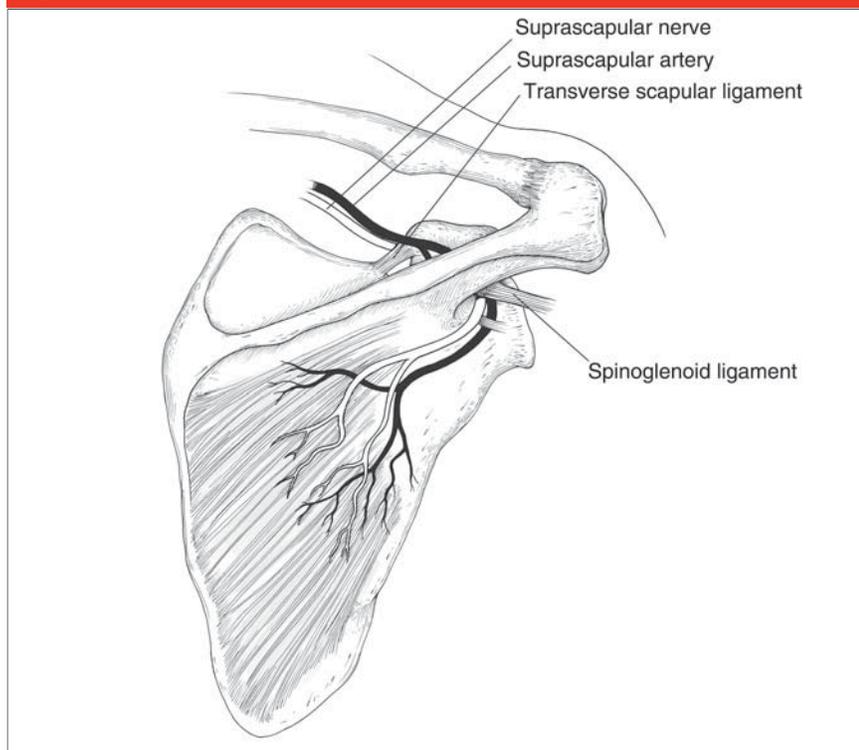


Illustration of suprascapular anatomy. After exiting the posterior cervical triangle, the suprascapular nerve passes through the suprascapular notch, across the floor of the supraspinatus fossa, and past the spinoglenoid notch. The suprascapular notch is roofed by the transverse scapular ligament, and the spinoglenoid notch is covered by the spinoglenoid ligament; both structures have the potential to compress the nerve. (Reproduced with permission from Safran MR: Nerve injury about the shoulder in athletes: Part 1. Suprascapular nerve and axillary nerve. *Am J Sports Med* 2004;32: 803-819.)

population, may have a cutaneous branch supplying sensation in the lateral arm. Its chief function is that of a motor nerve, innervating the supraspinatus and infraspinatus muscle bellies.<sup>3</sup> The nerve originates from the upper trunk of the brachial plexus (ie, C5, C6, occasionally C4), from which it courses laterally

through the posterior cervical triangle to reach the suprascapular notch (Figure 1).

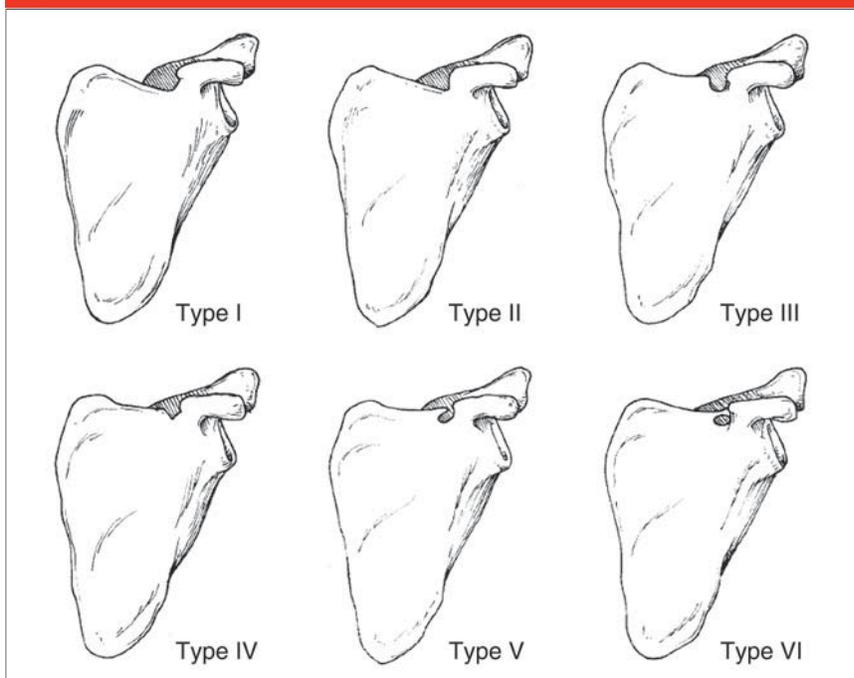
The suprascapular notch is a bony depression medial to the base of the coracoid process. Its superior aspect is enclosed by the transverse scapular ligament. Various morphologies have been described for the bony portion

of the notch, ranging from a subtle depression to pure bony tunnel with ossification of the overlying ligament<sup>4</sup> (Figure 2). Although the suprascapular artery and vein usually pass above the transverse scapular ligament, the nerve passes beneath, limiting its potential excursion within the narrow fibro-osseous tunnel. On average, the nerve is 3.0 cm medial to the supraglenoid tubercle as it passes through the suprascapular notch.<sup>6</sup> On exit, it travels posterolaterally across the floor of the supraspinatus fossa, providing several motor branches to the overlying supraspinatus muscle and receiving sensory fibers from the acromioclavicular and glenohumeral joints.<sup>3,6</sup> These various tethers limit excursion of the nerve to  $\leq 3$  cm within the supraspinatus fossa.<sup>3</sup>

At the posterolateral corner of the fossa, the nerve reaches the spinoglenoid notch, a depression at the lateral base of the scapular spine that occurs 1.8 to 2.1 cm medial to the glenoid rim.<sup>3,6</sup> A spinoglenoid ligament has been identified with variable frequency coursing over the top of the notch<sup>5</sup> (Figure 3). Recently, Plancher et al<sup>7</sup> demonstrated the ligament's presence in all 58 study specimens and attributed the variable presence reported in prior studies to differences in specimen preparation. Histologic analysis confirmed true ligamentous insertional features, and the distance from nerve to ligament was well-conserved across specimens at a mean of 4.6 mm.<sup>7</sup> Because portions of the ligament insert into the

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Figure 2



The six types of suprascapular notch: type I, depression (8%); type II, shallow V-shaped (31%); type III, U-shaped (48%); type IV, deep V-shaped (3%); type V, type III with partial ossification of the ligament (6%); and type VI, complete ossification of the ligament (4%). (Reproduced with permission from Rengachary SS, Burr D, Lucas S, Brackett CE: Suprascapular entrapment neuropathy: A clinical, anatomical, and comparative study. Part 3: Comparative study. *Neurosurgery* 1979;5:452-455.)

posterior capsule, the spinoglenoid ligament becomes taut in positions of adduction and internal rotation,<sup>8</sup> which corresponds to the follow-through phase of throwing. It is just beyond the spinoglenoid notch that the nerve makes an acute medial turn around the base of the scapular spine, traveling along the scapular body and sending two or more motor branches into the infraspinatus muscle.<sup>6</sup> These motor branches split off the main nerve and travel along the muscle's undersurface 2.0 to 2.2 cm medial to the glenoid rim.<sup>9</sup>

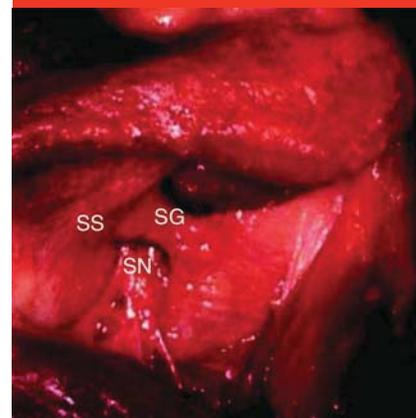
### Pathophysiology

Suprascapular neuropathy may occur secondary to a variety of mechanisms that vary depending on the local anat-

omy and nature of the insult. Focal nerve entrapment may occur at any location along the nerve's course but is most common where mobility is already limited. Once the nerve reaches the confines of the suprascapular notch, it is particularly vulnerable. Anything that narrows the notch may injure the nerve. Fractures involving this region have been reported to cause neuropathy,<sup>10</sup> and although nerve injury has been attributed to supraglenoid cysts at the spinoglenoid notch, paralabral cysts can extend more medially and may affect the nerve at the suprascapular notch.<sup>11</sup>

Anatomic variations also play a role. The transverse scapular ligament,<sup>3</sup> a recently described anterior coracoscapular ligament,<sup>12</sup> and fascial extensions from the subscapu-

Figure 3



Posterior photograph of a cadaver right scapula demonstrating the scapular spine (SS), spinoglenoid ligament (SG), and suprascapular nerve (SN). The spinoglenoid notch is bounded by the glenoid neck inferiorly, the scapular spine medially, and the spinoglenoid ligament superolaterally. (Adapted with permission from Plancher KD, Peterson RK, Johnston JC, Luke TA: The spinoglenoid ligament: Anatomy, morphology, and histological findings. *J Bone Joint Surg Am* 2005;87:361-365.)

laris muscle<sup>13</sup> may all contribute to suprascapular notch entrapment. As the nerve passes into the suprascapular fossa, mobility is greater but is still limited by the nerve's adherence to the periosteum and by its motor branches to the supraspinatus.<sup>3</sup> A scapular fracture malunion, or any other space-occupying fossa lesion, may compress the nerve in this region. Likewise, as the nerve turns medially at the spinoglenoid notch and beneath the spinoglenoid ligament, it is particularly susceptible to localized compression. Entrapment caused by paralabral ganglion cysts is now commonly recognized,<sup>14</sup> but other mass-effect scenarios have been reported in recent years, including prominent hardware<sup>15</sup> and enlarged veins.<sup>16</sup>

In addition to focal nerve entrapment, neuropathy can follow more

Figure 4

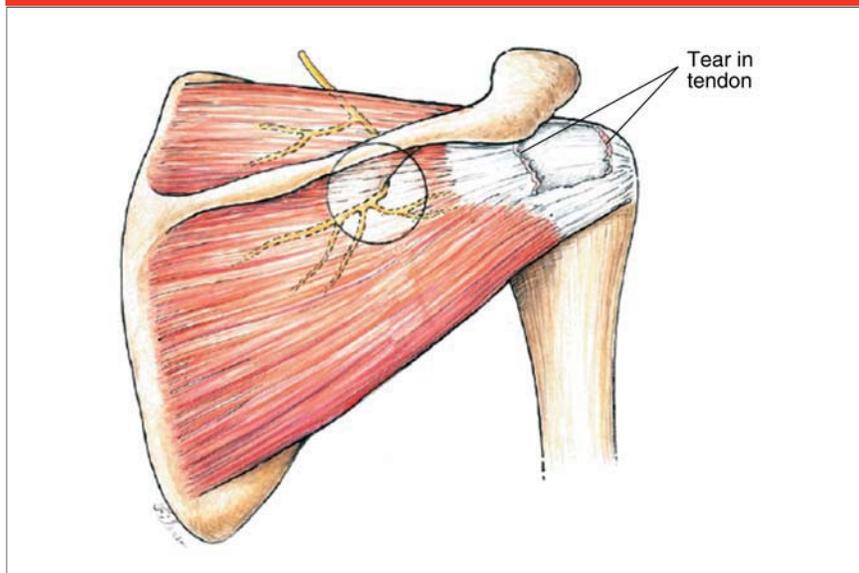


Illustration of a proposed mechanism for the association between suprascapular neuropathy and massive, retracted rotator cuff tear in which retraction of the torn tendon bowstrings the nerve against the scapular spine (circled area). (Reproduced with permission from Costouros JG, Porrmatikul M, Lie DT, Warner JJ: Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy* 2007;23:1152-1161.)

dynamic insults. The identification of suprascapular neuropathy in elite volleyball players<sup>17</sup> and pitchers<sup>18</sup> has suggested that a combination of traction, friction and/or kinking of the nerve at points of tethering may induce nerve injury. This may be particularly true at the spinoglenoid notch, a site at which anatomic studies have demonstrated an increase in spinoglenoid ligament tension against the nerve in positions that correspond to the follow-through phase of throwing.<sup>8</sup> Combined scapular protraction and infraspinatus contraction during this phase may further bowstring the nerve against the scapular spine, with acute and/or chronic injury resulting.<sup>17</sup> Sandow and Ilic<sup>2</sup> have speculated that the superior aspect of the infraspinatus may impinge on the nerve at the spinoglenoid notch in positions of abduction and external rotation. In some cases, overuse traction forces

may affect the nerve indirectly by inducing intimal damage to the axillary or suprascapular arteries, resulting in microemboli and secondary ischemia of distal nerve branches.<sup>18</sup>

Suprascapular neuropathy may also accompany other primary shoulder pathology. Nerve compression frequently occurs at the spinoglenoid notch as the result of paralabral ganglion cysts that develop after a primary labral tear establishes a one-way synovial valve.<sup>11,14</sup> Additionally, de Laat et al<sup>19</sup> reported a 29% rate of suprascapular nerve injury in association with shoulder dislocations and proximal humerus fractures. Recent focus has also turned toward the association of suprascapular nerve injury with massive, retracted rotator cuff tears. Albritton et al<sup>20</sup> simulated supraspinatus retraction in a cadaver model and noted that up to 5 cm of retraction markedly altered the course and tension of the first su-

praspinus motor branch at the suprascapular notch. Costouros et al<sup>21</sup> reported a 38% rate of isolated suprascapular neuropathy in a series of 26 massive rotator cuff tears and speculated that the nerve may be further tethered at the scapular spine by infraspinatus retraction (Figure 4). This report documented nerve recovery after attempted rotator cuff repair.

These relationships have led some authors to speculate that suprascapular neuropathy may be a significant contributor to the pain and muscle atrophy of retracted tears,<sup>20</sup> and some now perform routine electromyography (EMG) testing in these patients.<sup>21</sup> Iatrogenic suprascapular neuropathy has also been reported following distal clavicle resection, spinal positioning, transglenoid stabilization, and procedures requiring an open posterior approach to the shoulder.<sup>9,22</sup>

## Diagnosis

### History

Suprascapular neuropathy causes pain and spinal weakness in 80% to 100% of patients.<sup>23-25</sup> Pain is often described as a dull, sometimes burning, ache in the posterior and lateral shoulder regions,<sup>25</sup> with occasional radiation to the neck or lateral arm. The pain may be worsened by arm positions involving cross-body adduction and internal rotation<sup>8</sup> (Table 1). This distribution may be explained by the nerve's sensory contribution to the shoulder and acromioclavicular joints and the known tensioning of the spinoglenoid ligament during adduction and internal rotation maneuvers.<sup>8</sup> Subjective weakness during external rotation and/or abduction is more variable and is dependent on the degree and level of nerve compromise. Isolated infraspinatus involvement may not be functionally limiting because the

**Table 1****Common History and Physical Examination Findings for Suprascapular Neuropathy****History**

Posterolateral and/or superior shoulder pain  
 Mild subjective weakness (abduction, external rotation)  
 Chronic overhead sports or labor  
 History of shoulder trauma or surgery  
 Massive, retracted rotator cuff tear

**Physical Examination**

Supraspinatus and/or infraspinatus atrophy  
 Tenderness posteromedial to the acromioclavicular joint and/or posterosuperior joint line  
 Weakness of external rotation and/or abduction

**Table 2****Differential Diagnosis for Suprascapular Neuropathy**

Rotator cuff pathology (ie, tendinitis/bursitis, tears)  
 Acromioclavicular arthritis  
 Adhesive capsulitis  
 Labral pathology (eg, SLAP tears, Bankart lesions)  
 Glenohumeral arthritis  
 Cervical radiculopathy  
 Brachial plexitis (ie, Parsonage-Turner syndrome)

SLAP = superior labral anterior-posterior

posterior deltoid and teres minor can often compensate.<sup>17</sup>

By contrast, nerve compression at the suprascapular notch more often causes functional deficits, with up to 75% loss of abduction and external rotation strength.<sup>26</sup> Symptoms may develop gradually, with or without a preceding injury, and may worsen over time to become constant.<sup>27</sup> A sense of instability is also frequently reported,<sup>14</sup> likely in association with

**Figure 5**

Photograph of a patient with infraspinatus atrophy. The posterior aspect of the scapula demonstrates atrophy in the region of the infraspinatus fossa.

labral pathology. An initial traumatic event is reported in 40% of patients.<sup>25</sup> The typical scenario is that of a chronic, traction-type injury in the younger athlete or laborer who places repetitive overhead demands on the upper extremities during activity (eg, volleyball, basketball, weightlifting, swimming).<sup>17</sup> However, suprascapular neuropathy should be considered in older patients with rotator cuff tears<sup>28</sup> and in any patient with otherwise unexplained shoulder symptoms after a shoulder operation or traumatic shoulder injury. The described symptoms may overlap with several other potential shoulder pathologies (Table 2). The presence of gradually progressive, dull shoulder pain with isolated weakness in forward elevation and/or external rotation may help distinguish isolated suprascapular neuropathy from most of these other conditions.

**Physical Examination**

Patients with suspected suprascapular neuropathy should first be examined for signs related to prior surgery and trauma (Table 1). Evidence of penetrating injury along the course

of the nerve or of surgical incisions consistent with prior spine procedures, open posterior shoulder approaches, rotator cuff repair, or distal clavicle resection should raise suspicion for the possibility of suprascapular nerve injury. Atrophy of the supraspinatus and/or infraspinatus muscle belly is often evident<sup>24</sup> (Figure 5). Isolated infraspinatus atrophy suggests spinoglenoid notch entrapment, whereas the presence of both supraspinatus and infraspinatus atrophy indicates a more proximal injury, typically at the suprascapular notch. Atrophy of other periscapular musculature with or without humeral head subluxation may suggest a more global neurologic injury pattern. Active and passive range of motion along with careful evaluation of scapular mechanics should be performed in all patients to determine the contribution of capsular contracture and/or scapular dyskinesia to the primary complaint. With suprascapular notch entrapment, the acromioclavicular joint and supraspinatus fossa may be tender to palpation, whereas spinoglenoid notch compression may cause pain at the pos-

**Table 3****Imaging Studies for Suprascapular Neuropathy****Plain Radiographs**

Rule out scapular fracture, glenohumeral dislocation

Consider humeral head position as an indicator of massive rotator cuff tear

Stryker notch view: evaluate suprascapular notch type, patency

**MRI**

Trace nerve's course, with emphasis on both notches

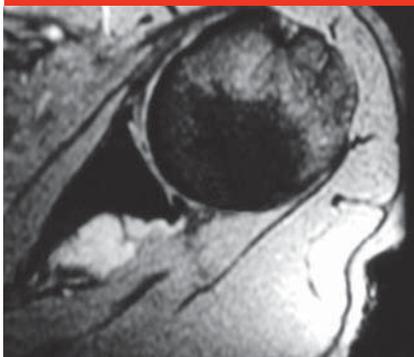
Assess degree of supraspinatus and infraspinatus atrophy

Look for mass compression (ganglion cyst)

Evaluate rotator cuff and labrum

**CT**

Thin cuts: evaluate potential bony sites of compression

**Figure 6**

Axial T2-weighted MRI scan of a paralabral ganglion cyst. Cysts typically appear loculated with an increased signal on T2-weighted images. An associated labral tear may also be seen.

**Table 4****Additional Diagnostic Tests for Suprascapular Neuropathy****Suprascapular Nerve Block**

Suprascapular or spinoglenoid notch injection of local anesthetic

**EMG/NCV Studies**

Must request suprascapular nerve evaluation

Conduction delays (increased latency)

Signs of denervation (fibrillations, sharp waves, decreased motor potentials)

Often can localize the site of compression

EMG = electromyography, NCV = nerve conduction velocity

terosuperior joint line.<sup>24</sup> Weakness to resisted external rotation and/or abduction will be present in most cases.<sup>24</sup> Cross-body adduction and internal rotation may elicit pain in the posterior shoulder, probably resulting from tensioning the spinoglenoid ligament,<sup>8</sup> but the diagnosis should be differentiated from primary acromioclavicular pathology. Likewise, given the overlap and frequent coexistence of rotator cuff and labral pathology, careful evaluation for superior labral anterior-posterior tears, glenohumeral instability, and rotator cuff function should be performed. Finally, a careful neurovascular examination to test specific distal cervical root function at C5-T1 is critical for ruling out neurologic injury that is more proximal at the cervical root level or more generally involving the brachial plexus.

**Imaging**

Routine radiographs should be obtained to rule out potential osseous

causes of nerve entrapment and to assess comorbid shoulder conditions, such as glenohumeral subluxation or arthrosis (Table 3). The addition of a Stryker notch view allows visualization of the suprascapular notch and may demonstrate complete ossification or near obliteration of its foramen. Scapular body or neck fractures with malunion or fracture callus at either notch and the presence of hardware impingement along the known course of the nerve should be considered. Proximal humeral head migration suggests chronic rotator cuff deficiency and should also be recognized. In situations in which osseous abnormalities are thought to be the primary cause of nerve injury, a CT scan may be helpful in appreciating specific regions of likely nerve compression.

An MRI study is particularly useful for quantifying the degree of supraspinatus and infraspinatus atrophy, for discerning potential soft-tissue causes of nerve entrapment, and for determining the presence of associated pathology, such as labral or rotator cuff tears. In addition to visualizing the course of the suprascapular

nerve, sagittal images at the level of the lateral scapular spine can be used to quantify the degree of muscle atrophy.<sup>29</sup> Compressive lesions such as paralabral ganglion cysts are easily identified with increased signal on T2-weighted images, along with the frequently associated labral tears<sup>30</sup> (Figure 6). Other soft-tissue masses may be seen on MRI as well, including lipomas and enlarged spinoglenoid notch veins.<sup>16,31</sup>

**Additional Diagnostic Tests**

When a clinical evaluation and routine imaging studies do not demonstrate a clear focus of nerve entrapment, the orthopaedist may consider additional diagnostic tests (Table 4). An injection of local anesthetic into the suprascapular notch helps to localize the region of entrapment if pain is relieved.<sup>10</sup> Likewise, EMG and nerve conduction velocity (NCV) studies, with specific attention paid to the suprascapular nerve, are routinely obtained to establish a baseline before treatment and to localize the site of nerve entrapment. Increased latency, fibrillation potentials, and diminished amplitude suggest nerve compression

and denervation, although EMG and NCV studies may be falsely negative or positive.<sup>18</sup>

## Treatment

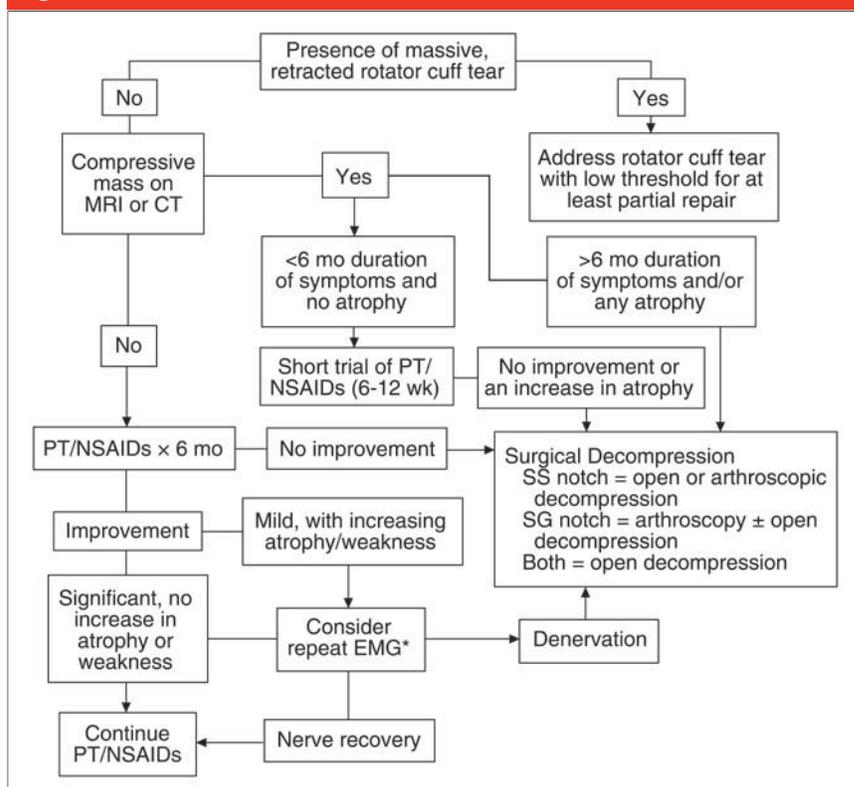
Once the diagnosis of suprascapular neuropathy is made, several treatment options are available. Most patients with an overuse type of neuropathy and no focal mass compression of the nerve will benefit from nonsurgical management. Those with neuropathy secondary to a space-occupying lesion or with massive, retracted rotator cuff tears usually benefit most from immediate surgical intervention to prevent further nerve injury. Additional considerations include the duration of symptoms, degree of muscle atrophy, and associated shoulder pathology. Identifying the region of compression is very helpful for surgical planning, for which open and arthroscopic techniques may be considered (Figure 7).

## Nonsurgical

Although there are limited data to guide the clinician, most authors agree that an initial nonsurgical approach is prudent for most patients with suprascapular neuropathy. The approach includes activity modifications, nonsteroidal anti-inflammatory drugs, and a comprehensive program of rotator cuff, deltoid and periscapular stretching and strengthening.<sup>25</sup> Depending on the duration and circumstances of nerve compression, the recommended length of treatment ranges from 0 to 12 months, underscoring the need to individualize a given regimen.<sup>25,32</sup>

To this end, the duration of symptoms and the etiology of entrapment are important considerations when determining the length of any initial nonsurgical treatment. Although pain relief and restoration of function are reliably achieved in most patients,<sup>25</sup> once significant atrophy has occurred, mus-

Figure 7



Treatment algorithm for suprascapular neuropathy. EMG = electromyography, NSAIDs = nonsteroidal anti-inflammatory drugs, PT = physical therapy, SG = spinoglenoid, SS = suprascapular spine  
\* An EMG would have been obtained initially to confirm the diagnosis.

cle bulk and motor strength may be irreversibly lost.<sup>32</sup> The patient presenting with long-standing (ie, >6 months) symptoms and early muscle atrophy may therefore miss an opportunity to regain full function if surgery is delayed. Likewise, the patient with structural compression of the nerve has been shown to do best after surgical decompression, whereas the patient with overuse neuropathy typically does not improve with surgery.<sup>33</sup> Nonsurgical management is therefore indicated in most patients who present with suprascapular neuropathy, particularly in those with an overuse-type etiology.<sup>25</sup> However, the patient with a long duration of symptoms, muscle atrophy, entrapment by a mass lesion, and/or associated massive rota-

tor cuff tears is unlikely to benefit from a prolonged nonsurgical approach.<sup>21,33</sup>

The frequently cited scenario of a paralabral ganglion cyst is worth special mention, given that some authors have recommended immediate decompression for this condition to prevent further nerve injury.<sup>32</sup> Although this step may be reasonable in many cases, an initial nonsurgical approach is justified in the patient with a short duration of symptoms, because in some instances a cyst may be incidental or transient. Some authors have reported spontaneous regression of ganglia, although it is rare.<sup>24</sup> Prior to surgical intervention, additional consideration may also be given to CT- or ultrasonography-guided cyst aspiration. Although

Figure 8

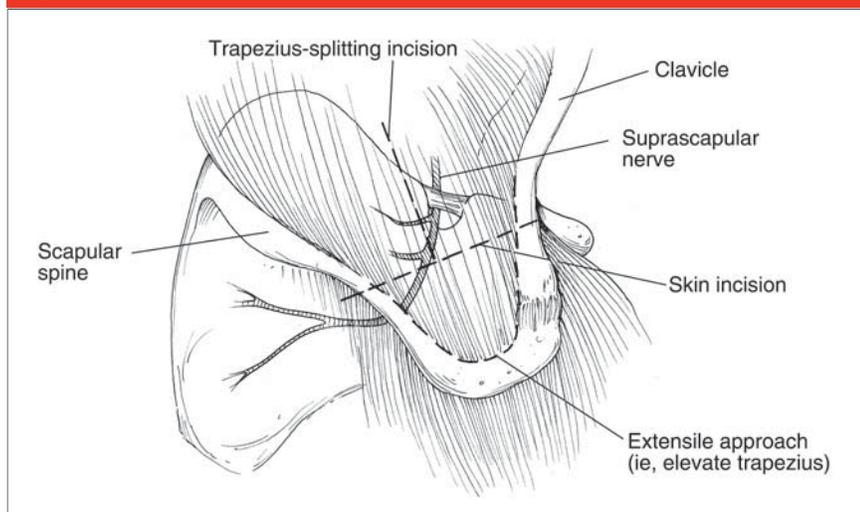


Illustration of open suprascapular notch decompression. The most frequently used open approaches to the suprascapular notch involve a superior, trapezius-splitting exposure or one that elevates the trapezius off the scapular spine. (Reproduced with permission from Romeo AA, Rotenberg DD, Bach BR Jr: Suprascapular neuropathy. *J Am Acad Orthop Surg* 1999;7:358-367.)

roughly half of cysts can be expected to recur following this intervention,<sup>14</sup> symptom relief after aspiration can be helpful diagnostically in the patient who has multiple potential sources of pain.

## Surgical

In the patient for whom nonsurgical treatment fails, particularly the patient with a reversible, structural cause of entrapment, surgical intervention is warranted, with the goal of eliminating compression of the nerve. Ideally, the surgeon would appreciate a specific locus of nerve entrapment such that surgical decompression may be targeted.

The most common sites of nerve compression are the suprascapular and spinoglenoid notches. The suprascapular notch has traditionally been decompressed through an open trapezius-splitting approach,<sup>32</sup> using either a saber or transverse incision along the scapular spine (Figure 8). Following mobilization of subcutaneous flaps, the trapezius muscle is

split in line with its fibers, the supraspinatus is retracted posteriorly, and the notch is identified medial to the coracoid base. The transverse scapular ligament is localized, extending over the top of the notch, after which the suprascapular vessels (above the ligament) and nerve (below the ligament) are exposed and protected as the ligament is released. If the nerve continues to be constrained by the bony notch, its medial border may be carefully widened with a burr. Dissection can be performed posterior to the supraspinatus muscle when simultaneous spinoglenoid notch decompression is desired. After adequate decompression, repair of the trapezius is followed by a standard closure.

Several authors have described arthroscopic techniques for suprascapular notch decompression.<sup>34,35</sup> In these reported procedures, the patient is positioned in the beach-chair position with<sup>34</sup> or without<sup>35</sup> traction. After a diagnostic glenohumeral arthroscopy through a standard poste-

rior portal, the arthroscope is redirected into the subacromial space, and a subacromial bursectomy is performed. Following bursectomy, the arthroscope is repositioned into a lateral portal and a shaver or radiofrequency device is introduced through an accessory anterolateral portal to expose the coracoacromial ligament medially to the coracoid process.<sup>34</sup> Further medial dissection is performed to the coracoclavicular ligament origins on the coracoid base, 15 mm medial to the acromioclavicular joint.<sup>36</sup>

Just medial to these ligaments, the lateral margin of the transverse scapular ligament can be identified. To expose and instrument the suprascapular notch, additional portals are required between the clavicle and the scapular spine. Bhatia et al<sup>35</sup> describe placing two portals along a line bisecting the angle created by the clavicle and scapula (Figure 9). An initial medial portal is placed 30 to 35 mm medial to the angle, through which a probe and/or elevator is used to bluntly expose the transverse scapular ligament and subsequently retract the suprascapular artery, vein, and nerve. A "suprascapular portal" is then established 5 to 10 mm lateral to the medial portal, through which arthroscopic scissors are used to perform the actual decompression (Figure 10). A similar approach is described by Lafosse et al,<sup>34</sup> with the addition of occasional bony notch resection in cases in which ligament release does not adequately mobilize the nerve.

Open decompression of the spinoglenoid notch is usually accomplished through a posterior approach, using a longitudinal incision 3 cm medial to the posterolateral corner of the acromion (Figure 11). After subcutaneous flaps are developed, the deltoid fascia is divided and the muscle split in line with its fibers, with care taken to avoid dissection more than 5 cm below the

Figure 9

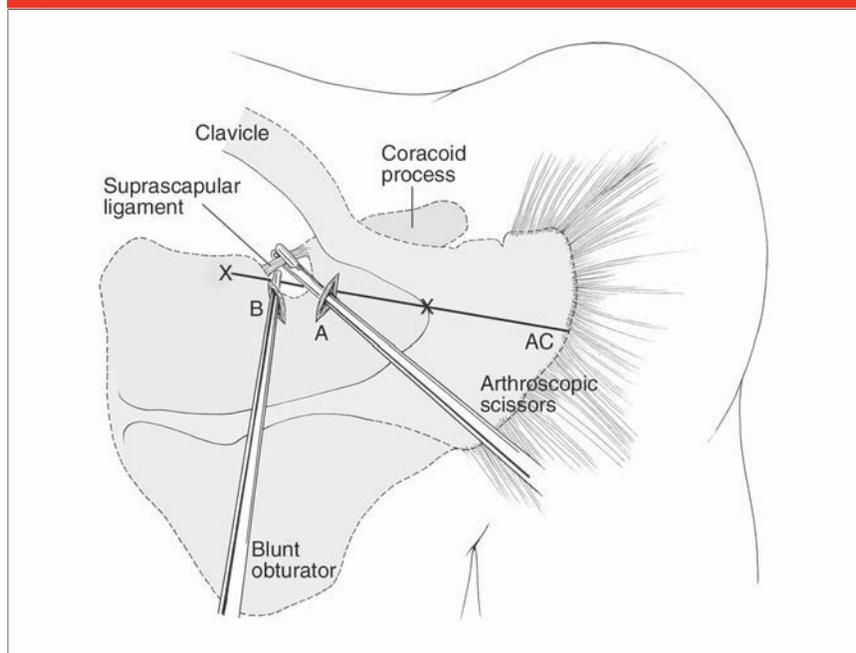


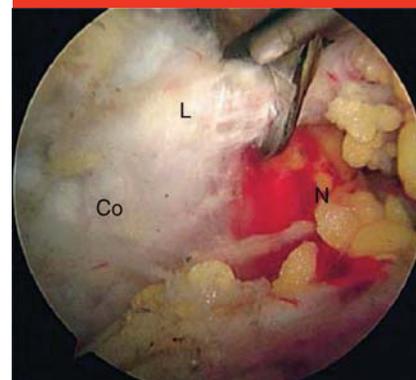
Illustration demonstrating technique for arthroscopic suprascapular notch decompression, requiring additional portals for exposing and instrumenting the notch. The angle that the scapular spine and acromion (AC) makes with the clavicle is bisected (line x-x). An initial medial portal (B) and a "suprascapular portal" (A) are placed along line x-x. (Adapted with permission from Bhatia DN, de Beer JF, van Rooyen KS, du Toit DF: Arthroscopic suprascapular nerve decompression at the suprascapular notch. *Arthroscopy* 2006;22:1009-1013.)

acromial border to prevent axillary nerve injury. Retraction of the deltoid facilitates identification of the superior border of the infraspinatus, which is subsequently mobilized inferiorly to reveal the scapular spine. Dissection is then carefully performed above the lateral extent of the spine to release the spinoglenoid ligament and underlying nerve.

Arthroscopic spinoglenoid notch decompressions have been reported, usually in association with the management of paralabral ganglion cysts.<sup>37</sup> The high frequency of labral tears in association with ganglion cysts at the spinoglenoid notch has led many authors to suggest the addition of glenohumeral arthroscopy to the management of this scenario.<sup>11,14</sup> Arthroscopy allows identification and repair of labral pathology

combined with effective arthroscopic decompression of the cyst through the tear<sup>37</sup> (Figure 12). When no labral tear is present, the cyst may be decompressed via a small capsulotomy just posterior and medial to the posterosuperior labrum. Other authors have described intra-articular labral repair followed by a more complete cyst decompression from the subacromial space.<sup>37</sup> When a single locus of compression is not obvious but clinical and MRI evidence of both supra- and infraspinatus denervation is seen, it is not clear if both notches should be decompressed, although some authors have reported good results after doing so. Sandow and Ilic<sup>2</sup> reported excellent results in eight volleyball players treated with an open posterior approach, elevating the trapezius off

Figure 10



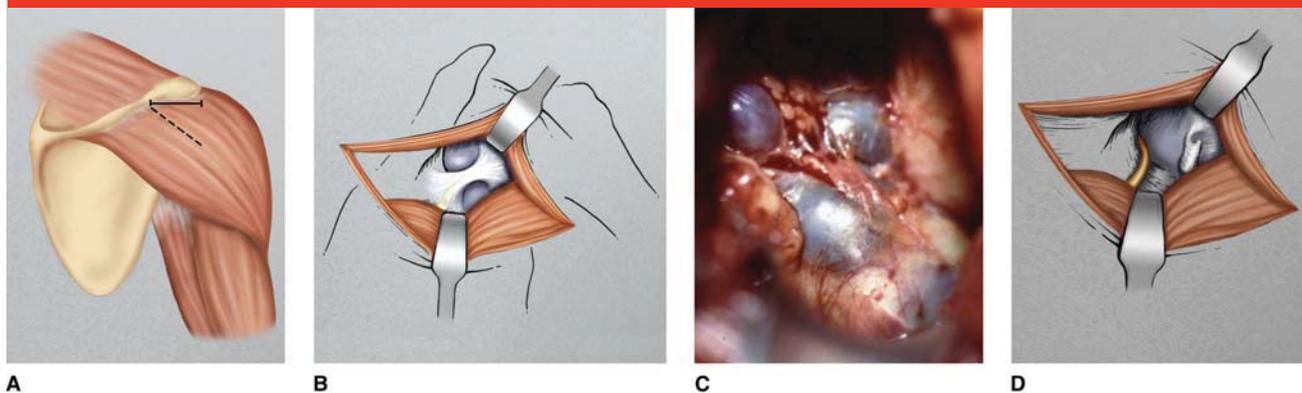
Arthroscopic view of the suprascapular notch at the shoulder. Scissors have been introduced through one of the accessory suprascapular portals to resect the ligament. L = superior transverse scapular ligament; Co = coracoid, N = suprascapular nerve. (Reproduced with permission from Bhatia DN, de Beer JF, van Rooyen KS, du Toit DF: Arthroscopic suprascapular nerve decompression at the suprascapular notch. *Arthroscopy* 2006;22:1009-1013.)

the scapular spine and retracting the supraspinatus posteriorly to access the suprascapular notch, then anteriorly to access the spinoglenoid notch.

One preliminary report describes an all-arthroscopic technique for decompressing both notches. Soubeyrand et al<sup>38</sup> successfully decompressed both notches using a series of arthroscopic portals along the scapular spine, through which the plane beneath the supraspinatus was developed. Preliminary results were described as good in three patients, but further study is needed to justify the potential morbidity associated with this approach.

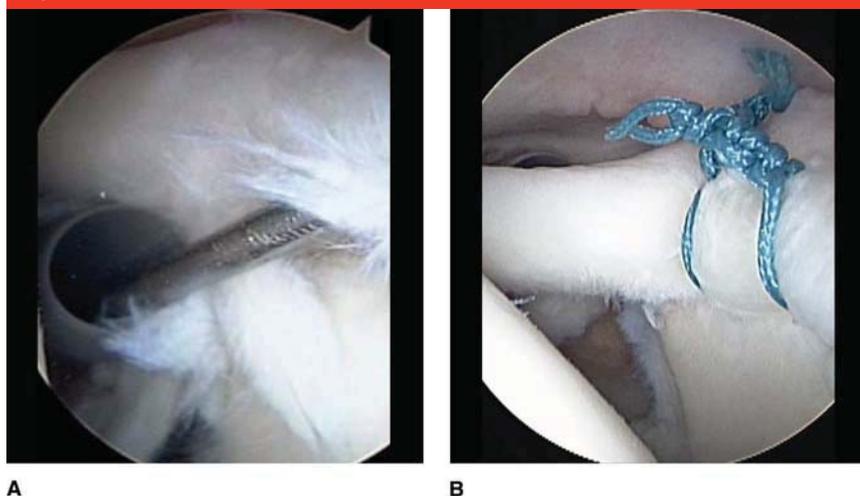
The coexistence of massive retracted rotator cuff tears has recently received increased attention. Several recent preliminary studies documented the association of suprascapular neuropathy in 28% to 100% of massive, retracted rotator cuff

Figure 11



Open decompression of the spinoglenoid notch. **A**, Illustration of an oblique incision starting 4 cm medial to the posterolateral corner of the acromion. **B**, Exposure of the notch is then possible after splitting the deltoid and retracting the infraspinatus inferiorly. **C**, In this intraoperative photograph, after the notch is exposed, a spinoglenoid ganglion is visualized. **D**, Finally, the spinoglenoid ligament is released.

Figure 12



Arthroscopic images of surgery for spinoglenoid notch compression caused by a ganglion cyst. The procedure should include an evaluation of the joint. Because of the frequency of associated labral tears, most authors recommend cyst decompression through the labral tear, followed by labral repair. **A**, Labral tear. **B**, Appearance following cyst decompression and labral repair. (Reproduced with permission from Youm T, Matthews PV, El Attrache NS: Treatment of patients with spinoglenoid cysts associated with superior labral tears without cyst aspiration, debridement, or excision. *Arthroscopy* 2006;22:548-552.)

tears.<sup>21,28</sup> Nerve recovery was seen after attempts at tendon repair, and was correlated with clinical improvement, suggesting that tendon retraction (presumably bowstringing of the nerve at the two notches) might be causing the neuropathy and could be

contributing to muscle atrophy. Although further study is needed to clarify the potential role of isolated nerve decompression in the setting of irreparable tears or the addition of focused neurolysis at the time of attempted rotator cuff repair, the find-

ing of suprascapular neuropathy in patients with massive rotator cuff tears may lower the threshold for surgical intervention.

## Results

### Nonsurgical

Martin et al<sup>25</sup> reported on a series of 15 patients with suprascapular neuropathy. MRI studies were obtained in only a small subset; no mass lesions were seen. Treatment consisted of activity modifications and physical therapy to improve range of motion and strengthen the rotator cuff and deltoid. After a minimum of 6 months, 80% of patients had achieved a good or excellent clinical result with improvement in pain and function. However, although most patients were unrestricted in the use of their pathologic shoulder, persistent atrophy and mild weakness on isokinetic testing was common. In the subset of patients who had posttreatment EMG studies, persistent deficits were seen in roughly half. This would suggest that nonsurgical management provides benefit mostly through compensatory muscle action, and less so through nerve recovery.

## Surgical

The reporting of outcomes following surgical management is limited. Most published studies are case reports or small case series in which it is difficult to ascertain the extent of denervation before treatment and/or the potential coexistence of other sites of compression. However, most appropriately selected patients can expect an improvement in pain and function, although preoperative muscle atrophy, particularly in chronic situations, may never resolve.<sup>32</sup>

Kim et al<sup>23</sup> reported long-term pain relief and strength improvements in nearly 90% of 31 patients at a mean 18-month follow-up after open suprascapular notch decompression. Good preliminary results have also been reported arthroscopically. A case series on arthroscopic suprascapular notch decompression by Lafosse et al<sup>34</sup> demonstrated excellent results in a prospective cohort of 10 patients. At a mean 15-month follow-up, all patients had significant improvement in pain and function, with normalization of EMG findings in seven of the eight tested.

Open spinoglenoid notch decompression has been reported in concert with arthroscopic management of associated labral tears. Fehrman et al<sup>39</sup> noted complete pain relief in a series of six patients with spinoglenoid notch entrapment from a ganglion cyst. Similar good to excellent results have been reported in most patients with ganglion cyst–related spinoglenoid notch entrapment after arthroscopic cyst decompression with<sup>37</sup> or without<sup>40</sup> labral repair.

Mallon et al<sup>28</sup> reported on a series of eight patients with suprascapular neuropathy and massive rotator cuff tears. Of the four patients who underwent attempts at repair, two had follow-up EMG studies that demonstrated significant nerve recovery. And among the six patients who un-

derwent partial repair in the series of Costouros et al,<sup>21</sup> EMG/NCV studies performed 6 months after surgery demonstrated significant nerve recovery that correlated with pain relief and functional improvement.

## Summary

Suprascapular neuropathy is an uncommon but potentially significant cause of shoulder pain and dysfunction that can result from a variety of static and dynamic insults along the path of the suprascapular nerve from neck to shoulder. It should be suspected in patients with recurrent shoulder symptoms after more common conditions have been ruled out, particularly in overhead athletes and those with traumatic shoulder injuries and/or massive, retracted rotator cuff tears.

Most patients with an overuse type of neuropathy will benefit most from extended nonsurgical management; those with reversible, structural causes of nerve compression will improve with surgical intervention. Given evidence that chronic denervation may be irreversible, the duration of symptoms and degree of muscle atrophy may be important considerations in choosing treatment options. Nerve decompression can be performed effectively through open approaches. Recent reports describe arthroscopic techniques, which may be particularly effective in cases of spinoglenoid notch ganglion cysts that allow the simultaneous management of coexistent labral tears. The association of suprascapular neuropathy with massive, retracted rotator cuff tears is a recent finding that may affect future management. Although outcome studies are limited, when treatment is appropriately individualized, significant improvements in pain and function can be expected in most cases.

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*Evidence-based Medicine:* References 19, 26, 29, and 32 are level II studies. References 5-9, 12, 13, 20, 24, 27, 36, and 40 are level III studies. The remaining references are level IV studies.

Citation numbers printed in **bold type** indicate references published within the past 5 years.

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