



# Suprascapular nerve entrapment isolated to the spinoglenoid notch: surgical technique and results of open decompression

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**Background:** Entrapment of the suprascapular nerve (SSN) at the spinoglenoid notch (SGN) specifically affects the infraspinatus, and isolated external rotation (ER) weakness can result. We describe the technique of open SSN decompression at the SGN for infraspinatus involvement and report the results of a consecutive series.

**Materials and methods:** Twenty-nine shoulders underwent SSN decompression at the SGN. The mean age was 44 years (range, 15-69 years), and the mean follow-up was 4.3 years (range, 1-7 years). On manual muscle testing, ER strength was abnormal in all patients: 2/5 in 3, 3/5 in 21, and 4/5 in 5. The mean preoperative American Shoulder and Elbow Surgeons (ASES) score was 48 (range, 23-83). Atrophy of the infraspinatus was visible or palpable in 72% of shoulders. Magnetic resonance imaging showed ganglion cysts at the SGN in only 20.7% of shoulders.

**Results:** Of the patients, 19 (66%) regained full ER strength, 9 (31%) improved to 4/5, and 1 (3%) had ER strength of 3/5. The mean ASES score improved to 75 (range, 60-100) ( $P < .05$ ). Of 29 shoulders, 23 (79%) showed improved ER strength within 1 week of surgery. All ganglion cyst cases regained full ER strength within a mean of 6 weeks. In all cases, ER strength improved by at least 1 full strength grade.

**Discussion:** A ganglion cyst is not necessary to produce SSN compression at the SGN. SSN compression at the SGN can present as an isolated entity or can occur in conjunction with rotator cuff pathology or a ganglion cyst. An index of suspicion, physical examination, magnetic resonance imaging, and electromyography confirm the diagnosis. The described operative approach detaches no muscle and allows rapid recovery, and in all cases, ER strength improved to normal or by 1 full grade.

**Level of evidence:** Level IV, Case Series, Treatment Study.

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**Keywords:** Suprascapular nerve; suprascapular entrapment; infraspinatus; spinoglenoid notch

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Entrapment of the suprascapular nerve occurs primarily at 2 locations around the shoulder, the most commonly reported of these being the suprascapular notch, with entrapment also reported at the spinoglenoid notch.<sup>1,2,12,19,21,22,27,30,33</sup> Suprascapular nerve entrapment

at the shoulder was first reported in the English-language literature by Kopell and Thompson<sup>20</sup> in 1959. Entrapment at the spinoglenoid notch was first reported in the English-language literature by Ganzhorn et al<sup>15</sup> in 1981. Over the past several decades, as awareness of this pathologic entity has grown, an increasing number of case series have addressed the diagnosis and treatment of suprascapular nerve entrapment at the spinoglenoid notch.<sup>1,5-7,9,12-16,19,21-25,28,31</sup> Proximal suprascapular nerve entrapment, at the level of the suprascapular notch, affects the motor branches to the supraspinatus and infraspinatus, as well as the sensory branches to the acromioclavicular joint, subacromial bursa, and posterior glenohumeral joint capsule. Distal suprascapular nerve entrapment occurring at or near the spinoglenoid notch affects the motor branches to the infraspinatus with no involvement of sensory innervation. Multiple cadaveric studies have addressed anatomic features at both locations that may predispose to suprascapular nerve compression, but an increasing number have addressed elements of the spinoglenoid notch specifically. The inferior transverse scapular ligament, also known as the spinoglenoid ligament, has been shown to be consistently present, and this structure may generate entrapment of the nerve at the spinoglenoid notch.<sup>3,4,6,8,10,11,17,18,26</sup>

With respect to compression at the spinoglenoid notch, a wide spectrum of etiologies have been reported, including ganglia, ganglia associated with labral lesions, hypertrophic vasculature, hypertrophic spinoglenoid ligaments, and repetitive traction injuries such as those produced by the service motion in overhead or throwing athletes.<sup>1,6,9,13,15,29</sup> The majority of case reports and series involving operative treatment of spinoglenoid notch entrapment have focused on the presence of a ganglion cyst as the causative factor and reported that this cyst is the result of a labral lesion or superior labrum anterior-posterior (SLAP) lesion. Thus, the treatment emphasis has been to repair the labral lesion and decompress the cyst, and this would be curative for the nerve compression.<sup>2,7,15,16,21,23,24,28</sup>

Suprascapular nerve compression at the spinoglenoid notch caused by the spinoglenoid ligament in the absence of a mass lesion and successful treatment by release of the spinoglenoid ligament was first reported as a treatment option by Aiello et al<sup>1</sup> in 1982. This particular etiology has been the subject of multiple anatomic studies, yet clinical experience has been limited to isolated case reports.<sup>3,8,10,11,17-19,26</sup>

We encountered a series of patients with isolated infraspinatus weakness due to suprascapular compressive neuropathy at the spinoglenoid notch. The purpose of this article is to present the results of open decompression of the suprascapular nerve at the spinoglenoid notch for isolated infraspinatus weakness in a series of symptomatic patients with evidence of suprascapular neuropathy on physical examination, advanced imaging or electromyography (EMG), and nerve conduction evaluation. A secondary objective is to emphasize that nerve compression at the

spinoglenoid notch can occur as an isolated entity in the absence of any space-occupying lesion, with a ganglion cyst, or in conjunction with rotator cuff pathology and, in particular, that the spinoglenoid ligament can function as a primary causative factor for suprascapular nerve compression resulting in isolated infraspinatus weakness.

## Materials and methods

Inclusion criteria were applied by use of operative reports and patient encounters with the aim of identifying only those patients with suprascapular nerve palsy at the spinoglenoid notch. Inclusion criteria included all patients aged older than 14 years with isolated suprascapular nerve compression at the spinoglenoid notch treated with suprascapular nerve decompression through an open approach by the senior surgeon (G.P.N.). In all patients, conservative treatment consisting of physical therapy directed toward the external rotation weakness and shoulder motion and strength, as well as anti-inflammatory medications if tolerated, had failed. All patients had evidence of suprascapular nerve palsy isolated to the spinoglenoid notch with positive findings on physical examination, imaging, and/or electrodiagnostic studies. Exclusion criteria included incomplete records, follow-up of less than 12 months, and concomitant decompression of the suprascapular nerve at the suprascapular notch.

A total of 29 patients met the criteria for inclusion in the study. There were 26 male and 3 female patients. The mean age was 44 years (range, 15-69 years) at the time of surgery. The dominant extremity was involved in 75.9% of patients. Sex, extremity involvement, and hand dominance are reported in Table I. The mean follow-up was 4.3 years (range, 1-7.3 years).

## Examination and diagnostic testing

Manual muscle testing was performed on all rotator cuff muscles. Isolated weakness of the infraspinatus was identified with external rotation strength rated as 2/5 in 3 patients, 3/5 in 21, and 4/5 in 5. All patients were able to perform external rotation, but 3 patients were not able to hold this against gravity. Atrophy of the infraspinatus was visible and palpable on physical examination in 21 patients. On magnetic resonance imaging (MRI) or computed tomography scan, 13 patients had severe atrophy, 9 had mild atrophy, and 7 had no atrophy. EMG findings consistent with suprascapular nerve pathology localized to the spinoglenoid notch were noted in 21 of 29 patients (72.4%). Only 16 patients (55%) were noted to have weakness on examination, atrophy, and EMG evidence of entrapment. On MRI examination, a cyst in the spinoglenoid notch was shown in only 6 of 29 patients (20.7%). Of the 8 patients without EMG evidence of suprascapular nerve entrapment, 6 had significant infraspinatus fossa atrophy on examination and on advanced imaging, 1 had atrophy on MRI alone, and 1 had no evidence of infraspinatus atrophy on physical examination or advanced imaging but had 3/5 weakness with external rotation and no rotator cuff tear or concomitant pathology.

## Concomitant pathology

All patients in this series had glenohumeral arthroscopy. Of the 29 patients, 3 had concurrent type I SLAP tears seen at

**Table I** Patient demographics and outcomes

	Data	P value
Age [mean (range)] (y)	44 (15-69)	
Sex (male) [n (%)]	26 (89.7)	
Side (right) [n (%)]	20 (69.0)	
Dominant extremity [n (%)]	22 (75.9)	
Concomitant pathology [n (%)]	17 (58.6)	
VAS score [mean (SD)]		.017
Preoperative	4.8 (2.8)	
Postoperative	2.0 (2.6)	
SST score [mean (SD)]		.006
Preoperative	5.7 (3.6)	
Postoperative	8.8 (3.2)	
ASES score [mean (SD)]		.002
Preoperative	47.8 (23.7)	
Postoperative	75.4 (24.4)	
Functional score [mean (SD)]		<.001
Preoperative	13.3 (7.8)	
Postoperative	21.2 (7.7)	

ASES, American Shoulder and Elbow Surgeons; SST, Simple Shoulder Test; VAS, visual analog scale.

arthroscopy. There were no unstable labral lesions. No patient had a SLAP lesion that required repair. There were no visible cyst communications from the glenohumeral joint to the spinoglenoid notch either above or below a superior or posterior labral region in the 6 patients with ganglion cysts. Of the 29 patients, 11 (37.9%) had concurrent rotator cuff tears. Of these 11 rotator cuff tears, 5 were isolated to the supraspinatus and 6 (54.9%) were classified as large and involved the upper infraspinatus. Of the 11 rotator cuff tears, 5 (45.5%) were recurrent. Of the 6 rotator cuff tears that involved the infraspinatus, 5 had EMG evidence of suprascapular nerve entrapment at the spinoglenoid notch. The remaining patient had atrophy of the infraspinatus fossa on physical examination and on advanced imaging.

## Surgical technique

The patient is placed on a beanbag positioner in the lateral decubitus position with the head elevated 30° (Fig. 1). To facilitate exposure to the spinoglenoid notch, it is helpful to “over-rotate” the body 10° to 20° past parallel anteriorly. The arthroscope is always inserted into the glenohumeral joint first to perform a brief diagnostic arthroscopy and evaluate for true intra-articular pathology. This assessment is performed quickly with a syringe and not an arthroscopic pump, thus minimizing fluid extravasation that may disturb the spinoglenoid notch anatomy. The open decompression is performed, and then any other concomitant pathology can be addressed by repositioning the patient if needed and performing standard arthroscopic rotator cuff repair, intra-articular debridement, or other indicated procedures.

The spinoglenoid notch decompression is approached by making a skin incision from the level of the scapular spine, beginning approximately 3 cm medial to the posterolateral corner of the acromion and extending toward the posterior axillary skin fold. The deltoid is split in line with its fibers, beginning approximately 4 to 5 cm from the posterolateral corner of the

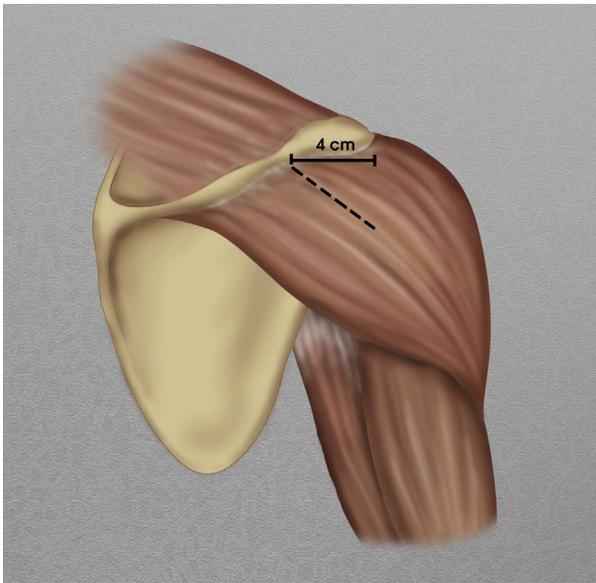


**Figure 1** Semi-lateral position, which allows the best visualization of the spinoglenoid notch using the described technique. The patient can also be maneuvered from this position into either a lateral or beach-chair position without breaking down the sterile field, depending on surgeon preference and concurrent procedures to be performed. Infraspinatus fossa atrophy can also be visualized in this photograph.



**Figure 2** Proposed incision marked along scapular spine. The skin incision begins at the scapular spine, approximately 3 cm medial to the posterolateral corner of the acromion, and extends toward the posterior axillary skin fold. A mark is also made 4 to 5 cm from the posterolateral corner of the acromion, denoting the location of the deltoid split.

acromion (Fig. 2). The deltoid fibers are almost horizontal in orientation, and the goal is to have the deltoid split centered over the spinoglenoid notch (Fig. 3). The fascia over the infraspinatus is split to expose the infraspinatus. The infraspinatus muscle belly is reflected inferiorly off the scapular spine to expose the spinoglenoid notch. There is a consistent ligamentous or fascial veil-like structure from the lateral edge of the scapular spine to the posterior capsule of the shoulder (Fig. 4). This spinoglenoid ligament is released sharply from the edge of the scapular spine, beginning superiorly on the scapular spine at a safe distance from the suprascapular nerve. It is released down to the base of the spinoglenoid notch by use of scissors proximally and blunt



**Figure 3** Graphic representation of deltoid split used to access spinoglenoid notch. The deltoid is split in line with its fibers, beginning approximately 4 to 5 cm from the posterolateral corner of the acromion. The deltoid fibers are almost horizontal in orientation, and the goal is to have the deltoid split centered over the spinoglenoid notch. The fascia of the infraspinatus should become evident after splitting of the deltoid.

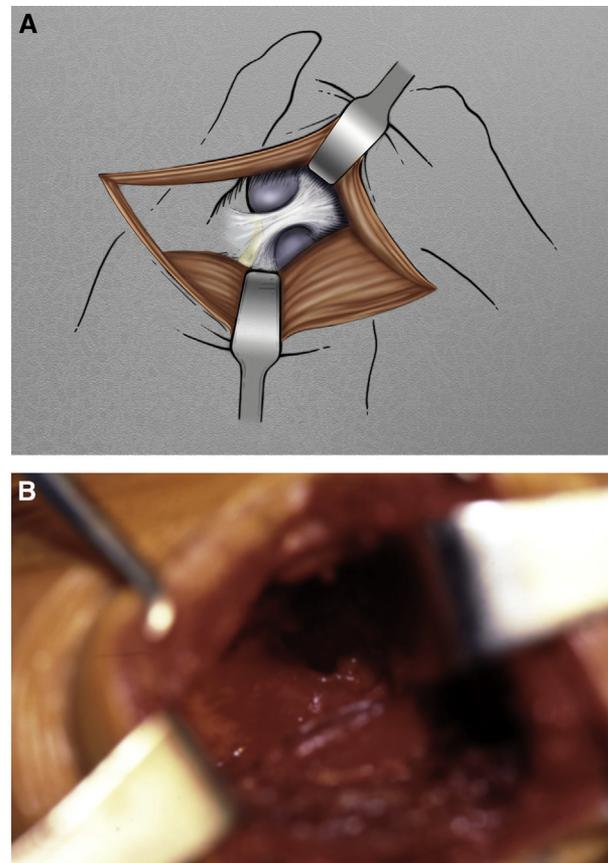
dissection with a “peanut” inferiorly to prevent nerve injury. Care is taken to avoid straying from the scapular spine to prevent iatrogenic injury to the suprascapular nerve or vessels. The suprascapular nerve and accompanying vasculature can then easily be identified as they pass through the spinoglenoid notch (Fig. 5). After ligament release, if a ganglion cyst is present, it is now well exposed and can be excised. A smooth, small periosteal elevator/freer is then used to confirm mobility of the nerve with gentle palpation. The infraspinatus is allowed to fall back up into its fossa, and the deltoid split is closed.

### Postoperative rehabilitation

Postoperative rehabilitation was dictated by concomitant pathology. In cases of isolated suprascapular nerve palsy at the spinoglenoid notch, the patients were placed in a simple sling with immediate initiation of pendulum exercises and passive range of motion. Patients were seen again at 1 week after surgery to assess strength and begin physical therapy, which consisted of passive range-of-motion and isometric exercises. If significant infraspinatus atrophy was present, electrical stimulation was used. Resistance and overhead exercises started at 4 to 8 weeks postoperatively in cases of isolated suprascapular nerve decompression at the spinoglenoid notch.

### Statistical analysis

The Student paired *t* test was performed to compare preoperative and postoperative data. Statistical significance was set at  $P < .05$ . Descriptive data were not analyzed.

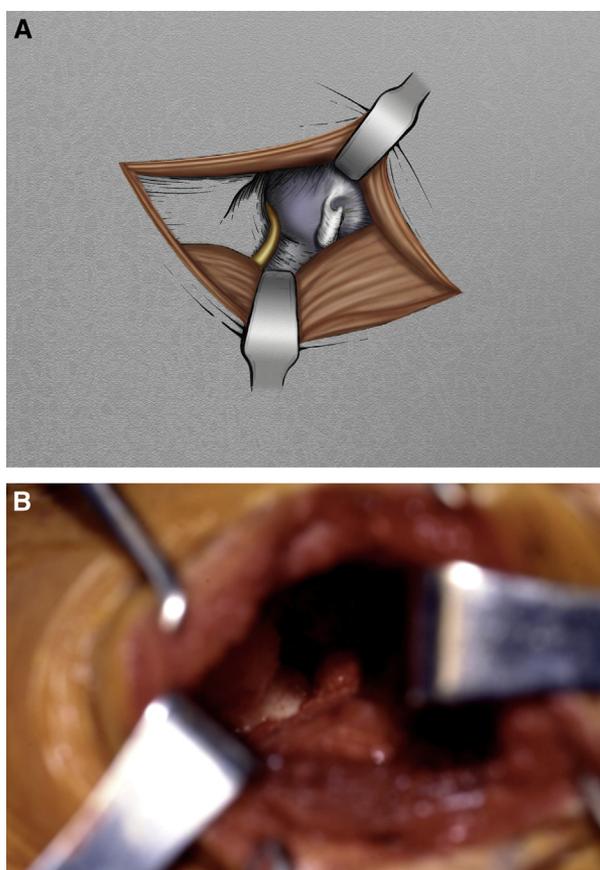


**Figure 4** Graphic representation (A) and intraoperative photograph (B) of spinoglenoid ligament, which is a consistent ligamentous or fascial veil-like structure from the lateral edge of the scapular spine to the posterior capsule of the shoulder. This spinoglenoid ligament is released sharply from the edge of the scapular spine, beginning superiorly, and then carried down to the base of the spinoglenoid notch by use of blunt dissection.

### Results

Postoperatively, external rotation was rated as 3/5 in 1 patient, 4/5 in 9, and 5/5 in 19, with a mean increase of 1.3 grades. In all patients, manual muscle strength improved by at least 1 grade. The return of external rotation strength was remarkably quick, in that 23 of 29 patients (79%) showed improved strength at the first postoperative visit (1 week). All of the ganglion cyst cases regained full external rotation strength within a mean of 6 weeks (range, 1-16 weeks). The patients who had improvement in external rotation strength but in whom no cyst was found took a mean of 10 weeks (range, 1-20 weeks) to recover.

There were no infections or other postoperative complications. There were no reoperations. Pain scores, by use of the standard visual analog scale, significantly improved from a mean of 4.8 preoperatively to 2.0 postoperatively ( $P = .017$ ). There was no difference in the preoperative or postoperative pain scores between those with concurrent pathology and those with isolated suprascapular



**Figure 5** Graphic representation (A) and intra-articular photograph (B) of course of suprascapular nerve to infraspinatus after release of spinoglenoid ligament. After ligament release, if a ganglion cyst is present, it can be completely excised.

compression. Patient-based outcome scores (Simple Shoulder Test and American Shoulder and Elbow Surgeons [ASES]) significantly improved from preoperative values to the time of final follow-up ( $P = .006$  and  $P = .002$ , respectively). The mean improvement in the ASES and Simple Shoulder Test scores was 28 points and 3.1 points, respectively ( $P < .01$ ) (Table I). Preoperative functional scores averaged 13.3 and significantly improved to 21.2 postoperatively ( $P < .001$ ) (Table I).

## Discussion

The suprascapular nerve can be compressed most commonly at the suprascapular notch or at the spinoglenoid notch. The presenting clinical picture can vary depending on the site of compression and can be variable in character. The purpose of this study was to report on a cohort of patients with isolated suprascapular nerve compression at the spinoglenoid notch, including their presenting clinical picture, concomitant pathology, and outcomes after open surgical decompression of the suprascapular nerve at the

spinoglenoid notch by use of an open technique with direct visualization of the nerve.

The anatomy of the suprascapular nerve is well described, and its course with reference to osseous landmarks of the scapula has been measured.<sup>4</sup> Shortly after passing through the suprascapular notch beneath the superior transverse scapular ligament and onto the floor of the suprascapular fossa, the suprascapular nerve gives off 1 or more motor branches to the suprascapular muscle belly and receives nociceptive fibers from the acromioclavicular joint capsule, subacromial bursa, and posterior glenohumeral joint capsule. As it passes around the base of the scapular spine, through the spinoglenoid notch and onto the floor of the infraspinatus fossa, the suprascapular nerve is purely motor in nature, dividing into multiple branches to innervate the infraspinatus muscle.

Descriptions of the spinoglenoid ligament from multiple cadaveric studies have been inconsistent. With variations in the type of cadaveric material dissected (fresh frozen vs preserved) and the definition of “ligament” used, the spinoglenoid ligament has been identified in between 3% and 100% of specimens.<sup>3,8,10,11,17,26,32</sup> Demaio et al<sup>10</sup> dissected 75 cadaveric shoulders and found a 3% prevalence of the spinoglenoid ligament and a 13% prevalence of what they termed a “thickened aponeurosis.” The type of cadaveric material was not specified in their study. Demirhan et al,<sup>11</sup> using the same anatomic definition of a ligament used by Demaio et al, dissected 23 fresh-frozen cadaveric shoulders and found a 60.8% prevalence of the spinoglenoid ligament. Demirhan et al showed lengthening of the spinoglenoid ligament with a combination of cross-arm adduction and internal rotation, generating potential suprascapular nerve compression beneath the ligament. Cummins et al<sup>8</sup> dissected 112 preserved cadaveric shoulders and found an 80% prevalence of the spinoglenoid ligament. They classified the ligament into type I and type II, with the former being defined as a “thin fibrous band” and the latter as a “distinct ligament.” Histologic analysis determined the only difference in the two to be thickness and furthermore showed that the type II ligaments had Sharpey fibers on the side attaching to the spine of the scapula and that all ligaments inserted into the periosteum of the glenoid neck. Ide et al<sup>17</sup> dissected 115 cadaveric shoulders, finding either what they termed a “ligament-type” or “membrane-type” spinoglenoid ligament in 81.7% of specimens. They also measured the distance from the ligament both to the floor of the spinoglenoid notch and to the suprascapular nerve and found a mean distance of 5.4 mm for the former and 3.1 mm for the latter. Neither the type of cadaveric preparation nor the position of the glenohumeral joint during measurement of ligament-to-bone or ligament-to-nerve distances was stated. Bektas et al<sup>3</sup> described a “spinoglenoid septum,” rather than a spinoglenoid ligament, in a series of 36 embalmed shoulders. The septum comprised a fascial thickening between the lateral third of the suprascapular and the infraspinatus muscles that

attached medially to the base of the scapular spine and laterally to the posterior capsule of the glenohumeral joint. The septum exhibited dynamic compression of the suprascapular nerve with abduction and external rotation. In a recent anatomic study, Plancher et al<sup>26</sup> dissected 58 fresh-frozen cadaveric shoulders and found a 100% prevalence of the spinoglenoid ligament. Histologic analysis was in agreement with the findings of Cummins et al, with Sharpey fibers being identified in all specimens along the scapular spine. The ligament's lateral attachment was to the bone of the posterior aspect of the glenoid neck, with fibers blending into the posterior capsule of the glenohumeral joint.

In our surgical experience, there is a consistent structure from the spine of the scapula that inserts laterally into the posterior capsular structure and periosteum of the glenoid medial to the joint line. The ligament rotates or twists in the mid portion at least 90°. When released off the scapular spine, this twist unwinds and the ligament loses all tension and bulk. The explanation behind such a wide variation in findings among these anatomic studies is difficult to provide. Differences in cadaveric preparation, the technique of dissection, the anatomic definition of the "spinoglenoid ligament," and the presence or absence of histologic analysis may all play a role. Nevertheless, whether one uses the term "ligament" or an alternate descriptor, with the exception of the study by Demaio et al,<sup>10</sup> there appears to be a consensus that the majority of shoulders studied exhibited a band of tissue that connects the base of the scapular spine to the glenoid neck, beneath which passes the distal suprascapular nerve, and that can potentiate nerve entrapment. We found a consistent fascial veil off the lateral border of the scapular spine that twists more than 90° into the posterior shoulder capsule in all patients in this series. At the medial inferior edge of this ligament is the opening that the suprascapular nerve transits up against the base of the scapular spine. This is the location where it is compressed. During the release procedure, the nerve cannot actually be visualized until the spinoglenoid ligament is released from superior to inferior, thus releasing the nerve and exposing the nerve from under the ligament.

On the basis of our clinical findings in this series, the spinoglenoid ligament can serve as the primary causative factor in suprascapular nerve entrapment at the spinoglenoid notch. The release of the spinoglenoid ligament from the scapular spine can decompress the nerve and restore external rotation strength in these patients. Furthermore, as inferred by Ide et al,<sup>17</sup> the presence of a ganglion cyst does not necessarily eliminate the spinoglenoid ligament as a causative factor in nerve entrapment. Those patients who did have a ganglion cyst that was excised in conjunction with release of the spinoglenoid ligament had a remarkably rapid relief and return of external rotation motor strength. In addition, ganglion cysts are often multiloculated, making arthroscopic decompression of the entire cyst almost impossible. Techniques in which the cyst is decompressed

through a SLAP or posterior labral tear aim to decompress the cyst through an indirect approach and can fail if the cyst is not found or if it is multiloculated. However, in this study, we found that only 20.7% of our patients had paralabral or ganglion cysts in the spinoglenoid notch. Despite this, 62% of patients showed visible infraspinatus atrophy and 72% had EMG findings of suprascapular nerve compression at the spinoglenoid notch. This finding is significant because many physicians do not consider this diagnosis without evidence of a spinoglenoid cyst. Moreover, not all patients with a spinoglenoid cyst have suprascapular nerve compression. This condition can exist with more common conditions, including rotator cuff tears, which adds complexity to the diagnosis. However, the patients diagnosed with concomitant rotator cuff pathology in this study had intact infraspinatus tendons by use of advanced imaging, which could not explain their weakness to external rotation. Thus, the diagnosis of spinoglenoid notch compression of the suprascapular nerve should be considered in patients with external rotation weakness out of proportion to their other clinical diagnoses.

Traditionally, suprascapular nerve compression at the spinoglenoid notch has been almost exclusively reported in patients who perform repetitive overhead work or in throwing athletes.<sup>5,9,12,13,30</sup> The vast majority of our patients were neither overhead workers nor throwing athletes, showing that entrapment of the suprascapular nerve by the spinoglenoid ligament—though its prevalence may be higher—is not limited to these patient populations. Patients who present with weakness in external rotation not believed to be compromised by pain alone should undergo a standard evaluation as described in this report. The pain that the patients report is typically a more vague, diffuse pain, sometimes posteriorly and sometimes more laterally in the subacromial distribution. We believe that the pain complaints are because of the rotator cuff imbalance from the weakness of external rotation due to the isolated infraspinatus weakness. This creates poor biomechanics with increased strain in the remaining rotator cuff and possibly a secondary bursitis leading to pain. We believe that those patients who have objective evidence of suprascapular nerve compression at the spinoglenoid notch and in whom appropriate nonoperative management fails have visible atrophy in the infraspinatus and that those who complain of intractable pain can benefit from open decompression of the spinoglenoid notch with release of the spinoglenoid ligament. The infraspinatus is relatively easy to locate by an experienced electromyographer, and thus, EMG findings of fibrillations and positive sharp waves may be an indication for surgery similar to other compressive neuropathies at the elbow or wrist.

Weaknesses of this study include that it was retrospective in nature. Moreover, there was no control group, and there was a lack of postoperative advanced diagnostic studies to critically evaluate return of nerve function, muscle volume, and cyst resolution where applicable. In

In addition, the need to surgically address rotator cuff pathology in 11 patients adds a potentially confounding variable to the interpretation of their results. Among the strengths of this study are that it represents the largest reported series of spinoglenoid notch decompression for treatment; involves a consistent operative technique performed by a single, experienced shoulder surgeon (G.P.N.); and includes preoperative identification of nerve entrapment in all patients by use of diagnostic testing consisting of EMG and nerve conduction studies, obvious muscular atrophy on inspection, palpation or advanced imaging, and/or clinical evidence of external rotation weakness on examination. This study questions conventional thinking, which associates suprascapular nerve entrapment at the spinoglenoid notch with a ganglion cyst causing mass effect on the nerve in this area. This study shows a novel fact in that 79% of patients had no evidence of a space-occupying lesion yet had significant improvement with release of the spinoglenoid ligament by a technically simple, reproducible open approach to spinoglenoid notch decompression that avoids muscle detachment and permits rapid rehabilitation.

## Conclusions

This study shows that suprascapular nerve compression at the spinoglenoid notch does not necessarily occur exclusively in the setting of a spinoglenoid notch cyst and actually is more frequently diagnosed in the absence of a space-occupying lesion. This finding also questions the association of glenohumeral pathology (SLAP lesions) with this diagnosis. Suprascapular nerve decompression at the spinoglenoid notch can be performed with low morbidity by a minimally invasive open technique with expedient return of infraspinatus function and statistically significant improvements in pain and shoulder outcome scores.

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