

Posterior Shoulder Pain and Arthroscopic Decompression of the Suprascapular Nerve

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Abstract

Posterior shoulder pain is more often than not mistakenly identified as rotator cuff disease or cervical disk disease. While most current thoughts may be buried in the literature as suprascapular nerve entrapment, it is hoped that with a clearer understanding of this disease entity, patients will be treated in a timely manner and receive the expected outcome with great satisfaction.

Introduction

An article in the *New England Journal of Medicine* in 1959 opened the gates for discussion about suprascapular nerve entrapment in the clinical setting (Thompson and Kopell 1959). Various authors, including the authors of this chapter, have described the transverse scapular ligament and spinoglenoid notch as two potential sites of entrapment of the Suprascapular nerve (Post and Mayer 1987; Cummins et al. 2000; Plancher et al. 2005a, 2007) (Fig. 1). This injury, when recognized, leads to a multitude of symptoms including pain and weakness with an inability to hold even a 5-kg weight in the horizontal plane. The pain and disability associated with this condition can be unduly prolonged, but it is easily cured now with advanced arthroscopic techniques. While this entity represents a small percentage of the average shoulder surgeons' practice, recent advancements as well as diagnostic testing have brought this diagnosis of exclusion to the forefront and minds of many surgeons.

While always thought as a disease for only overhead athletes, compression of the suprascapular nerve is now recognized to be associated with patients with massive rotator cuff disease especially when fat atrophy is noted, those with a labral tear with or without a paralabral cyst, patients with large space-occupying lesions in both notches, and patients whose magnetic resonance imaging (MRI) may show evidence of the enlarged nerve implying compression or even detection of weakness of external rotation of the shoulder on physical examination. Confirmation of suprascapular disease remains elusive at times because the etiology (direct trauma, indirect trauma (e.g., traction), repetitive overuse, rotator cuff disease, and anatomic variations) and the indications for decompression of this nerve remain fraught with its advocates and critics.

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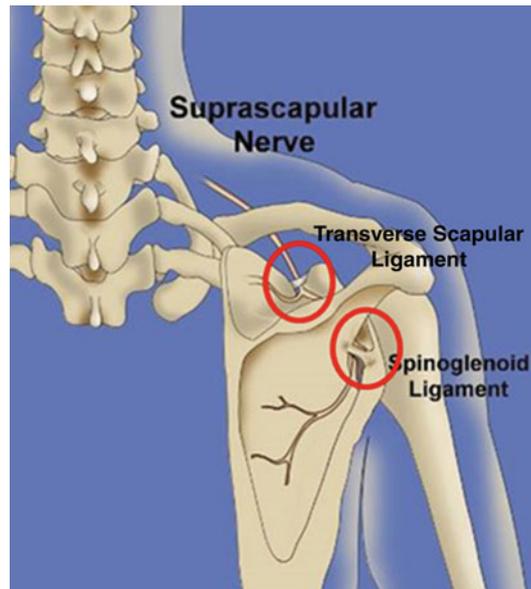


Fig. 1 Right shoulder, posterior view, artwork demonstrating the two compression sites for the suprascapular nerve (Copyright K. Plancher)

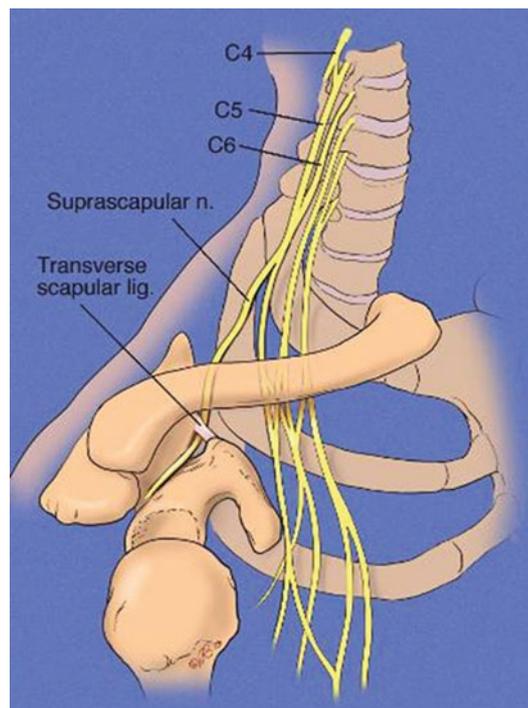


Fig. 2 Right shoulder, anterior view, artwork of the suprascapular nerve arising from the upper trunk of the brachial plexus (Copyright K. Plancher)

Anatomy of the Suprascapular Nerve

The suprascapular nerve has been classically thought to arise from the upper trunk of the brachial plexus (C5–C6) at Erb’s point. There are reports though of the nerve receiving contributions from C4

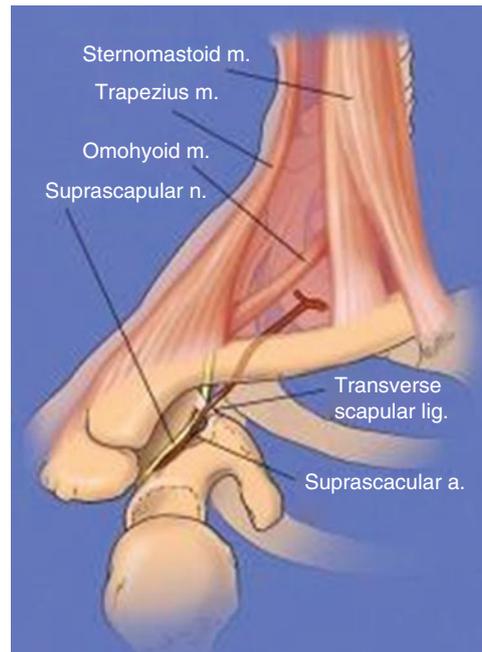


Fig. 3 Suprascapular nerve exiting the upper trunk to run parallel to the muscle belly of the omohyoid muscle along the posterior cervical triangle (Copyright K. Plancher)

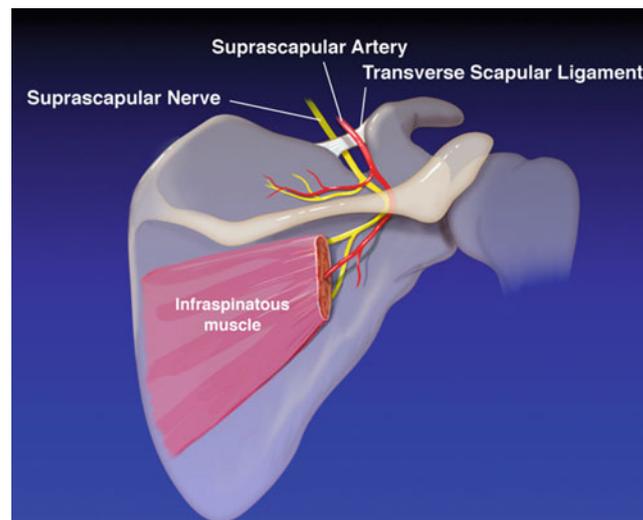


Fig. 4 The nerve travels along the posterior border of the clavicle to reach the superior border of the scapula. The nerve diverges from the artery to proceed under the transverse scapular ligament while the artery goes over the transverse scapular ligament diving into the suprascapular notch (Copyright K. Plancher)

in approximately 25 % of individuals (Rengachary et al. 1979b; Yan and Horiguchi 2000) (Fig. 2). The nerve exits the upper trunk approximately 3 cm above the clavicle to run laterally and parallel to the muscle belly of the omohyoid muscle and deep to the anterior border of the trapezius along the posterior cervical triangle (Fig. 3). As it passes through the posterior triangle, it travels with the suprascapular artery and vein. The nerve then travels along the posterior border of the clavicle to reach the superior border of the scapula. The nerve must diverge now from the artery to take a posterior approach diving into the suprascapular notch (Fig. 4). This nerve is approximately 3 cm,

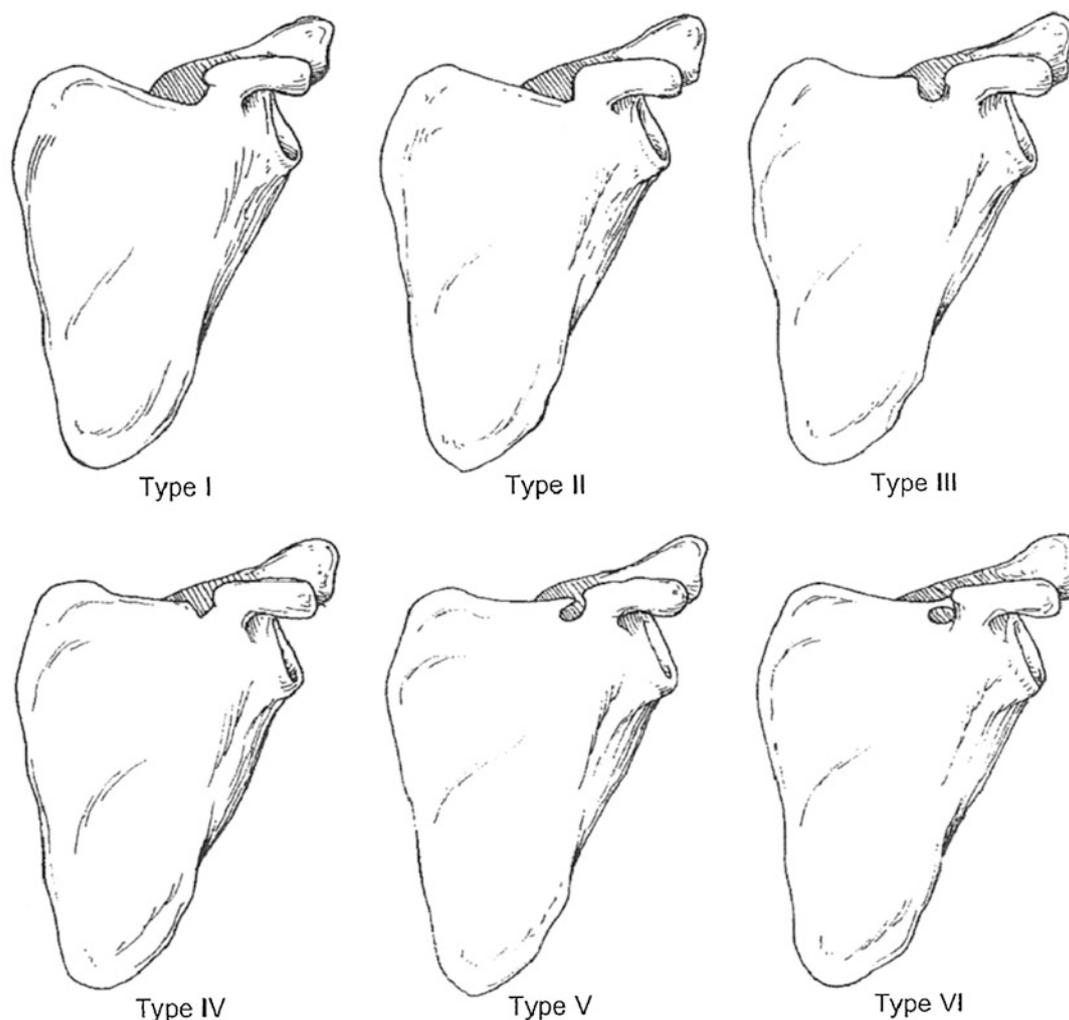


Fig. 5 Classification of abnormalities of the suprascapular notch by Rengachary et al. (1979b)

once again, away from the supraglenoid tubercle (Bigliani et al. 1990). The artery instead takes an anterior position and will enter the suprascapular notch over the ligament. Variations do occur infrequently with the artery staying with the nerve as it passes posterior to the ligament (Tubbs et al. 2003). The ligament that divides the nerve and artery in most cases, but not all, is named the transverse scapular ligament. The nerve now in the suprascapular fossa will give off two motor branches to the muscle belly. The nerve gives off sensory and sympathetic branches to two thirds of the glenohumeral joint, coracoclavicular ligament, coracohumeral ligament, subacromial bursa, as well as the posterior capsule of the AC Joint (Spinner and Spencer 1974; Aszmann et al. 1996; Ebraheim et al. 2011).

While the roof of the notch is formed by the transverse scapular ligament, which may hypertrophy leading to stenosis of the notch, it may also be the geometry of the notch that may vary leading to compression of the nerve. Classification of these variations and the six types of notches has been reported (Rengachary et al. 1979b) (Fig. 5). This narrowed notch may compress the nerve leading to a neuropraxia. The transverse scapular ligament extends from the base of the coracoid to the superior border of the scapula and can ossify in 25 % of clinical cases (Ticker et al. 1998).

The nerve travels along the suprascapular fossa heading laterally and coming within 2.0 cm of the posterior glenoid rim at the level of the spine of the scapula (Warner et al. 1992). The suprascapular

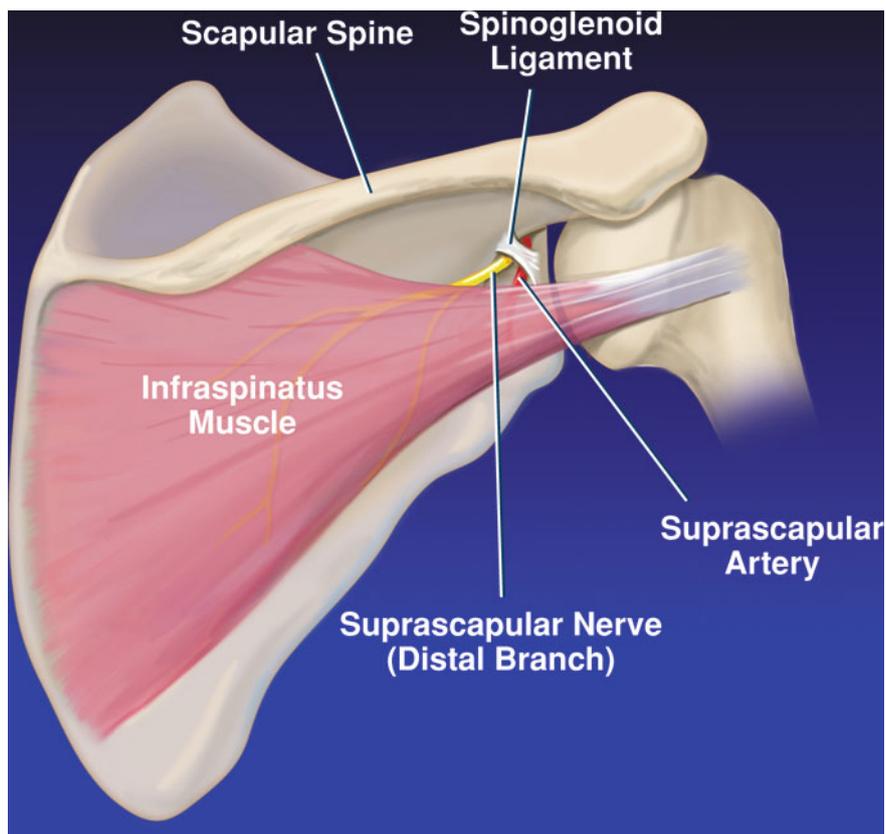


Fig. 6 The suprascapular nerve descending into the infraspinatus fossa passing under the spinoglenoid ligament (Copyright K. Plancher)

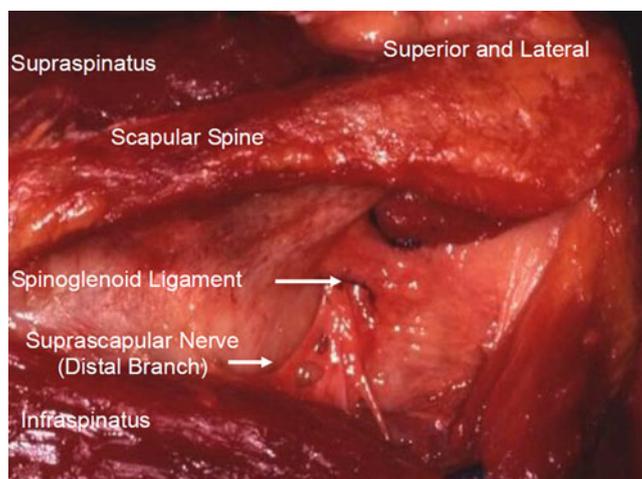


Fig. 7 The spinoglenoid ligament, quadrangular in shape, demonstrated in the posterior view of a right shoulder dissection. Note the distal branch of the suprascapular nerve compressed (Copyright K. Plancher)

nerve travels laterally around the scapular spine to descend into the infraspinatus fossa only to pass under the spinoglenoid ligament (inferior transverse scapular ligament) and give off two to four branches to this muscle belly (Fig. 6).

Some authors have described two types of ligaments: Type I, a thin indistinct band of tissue, and Type II, a well-formed ligament. The presence of this ligament was found in 100 % of cadaveric specimens and in fact with attachments to the glenohumeral joint which have larger effects upon internal rotation of the shoulder and compression of this nerve at the spinoglenoid ligament. The nerve itself is approximately 2.5 cm away from the glenoid rim and approximately 4 cm from the posterior corner of the spine of the scapula (Plancher et al. 2005a). This ligament is quadrangular in shape and extends from the posterior glenoid neck and glenohumeral capsule to insert a bilaminar ligament into the scapular spine (Fig. 7). Recent clinical studies, together with the rereading of the many articles with anatomic dissections, have convinced many of the larger amounts of sensory innervation of the shoulder by this nerve which may explain pain upon traction or compression of this nerve and perhaps upon repair of a massive rotator cuff with advancement of the tissue (Matsumoto et al. 2009).

Classification of Nerve Injuries

Correlation between the characteristic clinical symptoms and the degree of nerve injury is essential. Two different methods have been described in the literature (Sunderland 1946; Seddon 1972). Seddon introduced the triple classification of neuropraxia, axonotmesis, and neurotmesis.

Neuropraxia is known as a transient episode with complete motor paralysis with little sensory or any autonomic involvement. This diagnosis carries a good prognosis with a rapid and excellent recovery. For those students of Dr. Sunderland, neuropraxia would be equivalent to the first of the 5° of severe nerve injury. There is a strong belief that all early forms of suprascapular nerve compression comprise this type of injury. Clinical intervention is based on the presumption that the nerve is affected by this type of injury no matter what the primary mechanism. Continued compression will have the nerve act like the later descriptions below even though the integrity of the nerve is complete to the naked eye.

Axonotmesis, as described by Seddon and Sunderland, is an injury to nerve fibers that separates the proximal and distal fibers, but continuity of the nerve exists through the maintenance of the Schwann tubes. Motor, sensory, and autonomic nerve paralysis is complete and muscle atrophy becomes progressive. Recovery relies on the restoration of the muscle to nerve attachments with axon regeneration. The prognosis may be good but is dependent on the physical distance between the muscle to be reinnervated and the site of damage to the nerve as well as the length of time passed when the injury occurred. The patient with atrophy of the supraspinatus or infraspinatus gives credence for early intervention as discussed later in this chapter.

Neurotmesis as described by Seddon corresponds to Dr. Sunderland's 3rd, 4th, and 5th degrees of injury. In this type of injury, the nerve fibers are severed along with the Schwann tubes with supposed intact nerve fascicles. The recovery for this is never perfect as guiding the nerve to the correct location cannot occur without the Schwann tubes. The 4th subcategory of Sunderland has the perineurium surrounding each fascicle damaged and the endoneurium is disrupted. Finally, the 5th degree of injury is a loss of continuity of the nerve trunk. In suprascapular nerve compression, a neurotmesis is rarely seen except in a violent gunshot or knife wound. One would expect the need for tendon transfers to salvage the situation.

Classification of nerve injuries is helpful to the clinician taking care of compression of the suprascapular nerve. Electron microscopy though will show that the nerve injury is not as easily placed in one of the three categories. In reality, each nerve fiber will react where some fibers may have a neuropraxia and others an axonotmesis or neurotmesis (Sunderland 1946; Seddon 1972). In

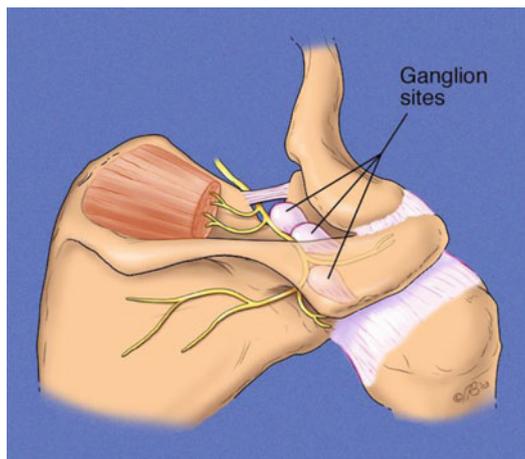


Fig. 8 Ganglion cyst causing encroachment and compression of the suprascapular notch at the transverse scapular ligament. A rarer finding seen with compression at the spinoglenoid ligament (Copyright K. Plancher)

addition, as mentioned above, increasing the time interval after the inciting event or the repetitive nature of the trauma will further damage the nerve fibers, encouraging all clinicians to identify the disease process early and consider the appropriate form of intervention to correct the compression of the nerve.

Compression at Transverse Scapular Ligament

Pathophysiology

A nerve compression lesion of the suprascapular nerve is often localized to a discrete portion of the length of the nerve, which because of its anatomical position makes it susceptible to entrapment. Early literature noted the nerve was affected with trauma such as a fracture through the scapular notch or even with a proximal humerus fracture caused by a direct blow to the shoulder. It has been reported that the suprascapular nerve is the second most common isolated nerve injury seen in shoulder dislocations second to the axillary nerve. Tumors, whether benign or malignant, are other causes with encroachment of the suprascapular notch by intrinsic or extrinsic masses.

The ganglion cyst represents one of the most common of these lesions (Fig. 8). Early authors believed as well that extremes of scapular motion would cause the nerve to kink over the edge of the scapular notch. This traction-friction theory also took hold early on in the literature.

One of the last theories utilized by the authors was the “sling theory.” Shoulder motion like hyperabduction creates an angulation against the transverse scapular ligament with resultant irritation to the suprascapular nerve (Clein 1975; Rengachary et al. 1979a). Repetitive microtrauma as seen in golf may lead to direct injury by traction to the nerve or indirect injury by affecting the vascular supply to the nerve. Iatrogenic injury to the suprascapular nerve has been reported in the literature upon distal clavicle excision or any posterior approach to the shoulder (Mallon et al. 1996). More recently the unique situation with a massive rotator cuff tear and residual discomfort could be explained by the traction of the suprascapular nerve after repair to the footprint. One study revealed a 40 % incidence of electrodiagnostic evidence of isolated suprascapular nerve dysfunction (Costouros et al. 2007).

Other authors have demonstrated that the retraction of the supraspinatus tendon leads to a reduction in the acute angle that exists between the suprascapular nerve and its first motor branch.



Fig. 9 Retraction of the supraspinatus tendon in a massive rotator cuff tear leading to reduction of the acute angle of the nerve causing increased tension of the suprascapular nerve at its fixed point in the spinoglenoid notch (Copyright K. Plancher)

On the contrary, increased tension of the suprascapular nerve is seen at the spinoglenoid notch with supraspinatus retraction. Albritton et al. demonstrated this concept in all specimens with 2–3 cm of supraspinatus retraction in the spinoglenoid notch (Albritton et al. 2003) (Fig. 9). Other investigations with EMG findings in patients exhibiting suprascapular neuropathy have been varied. Mallon et al. showed EMG findings did exist within their group of massive rotator cuff tears (Mallon et al. 2006). Others found the axillary nerve more involved when fat atrophy and a massive full-thickness rotator cuff tear exist (Vad et al. 2003). This issue is quite complex and, as presented by Albritton et al., retraction of the suprascapular nerve when a full-thickness rotator cuff tear exists can cause increased tension with a more acute angle takeoff at the spinoglenoid notch, but at the transverse scapular ligament, it is the repair of the full-thickness tear that causes increased tension on the nerve. Several cadaveric studies have shown that lateral advancement of a retracted rotator cuff tear may be between 1 and 3 cm, and with more advancement the neurovascular pedicle is placed under tension within the substance of the muscle belly (Warner et al. 1992; Greiner et al. 2003).

There are other proposed mechanisms of injury to the suprascapular nerve for compression at the spinoglenoid notch, and many are similar as stated above and many are unique to compression at this secondary point. Whatever the mechanism, compression or injury to the suprascapular nerve at the transverse scapular ligament will result in weakness and, if long term, atrophy of both the supraspinatus and infraspinatus muscles will occur.

Patient Profile

History

This is a disease more often than not of a young person. Many patients complain of a diffuse ache around the shoulder region. The pain may be localized to the posterolateral aspect of the shoulder and may radiate down the posterior aspect of the upper arm. At times, some patients complain of pain that radiates up the neck along the posterior cervical area or along the upper anterior chest wall or even night pain infrequently. A patient may complain of weakness on attempts of external rotation and abduction which may confuse the examiner because he or she may think the patient has rotator cuff disease or even cervical disk disease. A patient may describe pain when reaching across his or her body. The patient will often give a history of trauma or of playing a sport with repetitive use such as volleyball, basketball, tennis, weight lifting, and swimming. While sports activities can often lead to suprascapular neuropathy, the heavy laborer may be plagued with this disease as well because of the nature of the repetitive, overhead work they may perform daily.

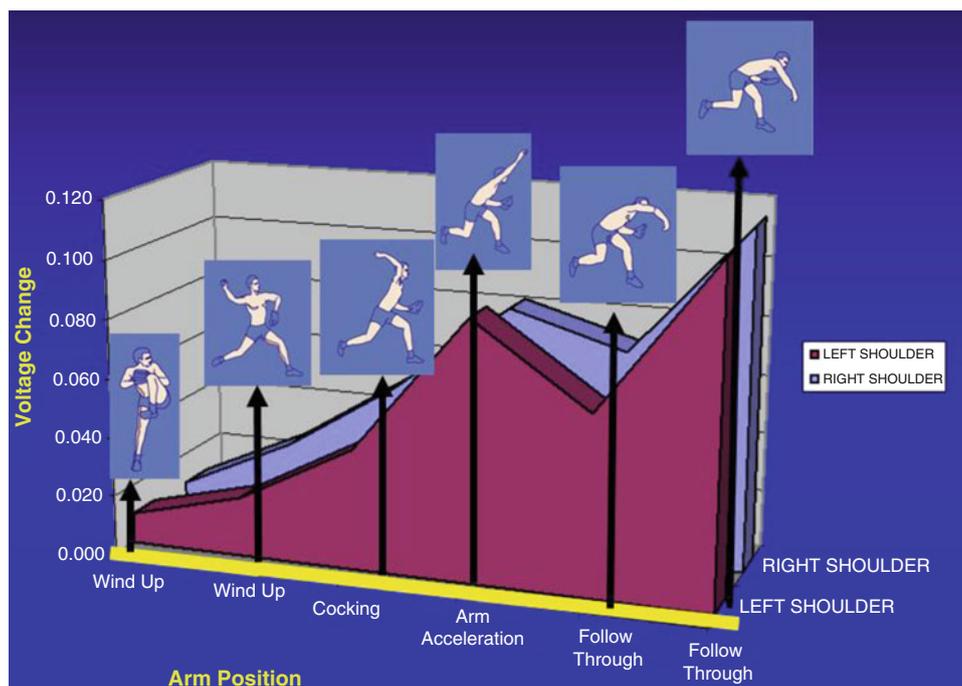


Fig. 10 The voltage change with throwing motion with intact spinoglenoid ligament. Note the follow-through or cross-arm adduction yields the highest pressure change at the spinoglenoid ligament (Copyright K. Plancher). Previously published in Plancher et al. (2007, Fig. 4)

Compression at the transverse scapular ligament is rarely insidious in onset. The symptoms patients describe are much more severe than symptoms from compression at the spinoglenoid ligament, which are usually silent. Correctly making this diagnosis is not easy, and often for this reason, patients frequently present many months after symptoms have begun and sometimes after a patient has had incorrect surgery to alleviate symptoms. The pain becomes a chronic ache and the atrophy now slowly becomes more apparent. In some chronic cases, certain scapular motions may be painful leading patients to begin to bypass or restrict certain motions. In these instances, their clinical presentation will mimic that of a patient with adhesive capsulitis. The patient will often note when performing tasks across the body that they will have an increase in their pain profile. The position of follow-through or adduction in an extended position has also been shown by our group to increase the tension and pressure within the spinoglenoid notch (Plancher et al. 2007) (Fig. 10). Pain and weakness may be more severe with compression at the level of the suprascapular notch rather than the spinoglenoid notch. Delay in diagnosis is the single biggest problem which prevents full restoration of the muscle strength.

Physical Examination

No different than any other disease entity, a full physical examination must be completed. A full neurological exam must be completed including in the cervical spine and both shoulders. Cervical discogenic disease for the most part will have a more predominant component of neck pain with radicular symptoms. Pain arising from a C3–C4 level will refer to the upper border of the trapezius, while pain from the C6–C7 area will affect the inferior border of the scapula.

The first part of the examination of the shoulder may be quite revealing. The patient when placed in a shoulder gown with the complete scapula in full view may demonstrate atrophy in both the supraspinatus and infraspinatus fossa (Fig. 11a, b). Atrophy though in a well-developed individual

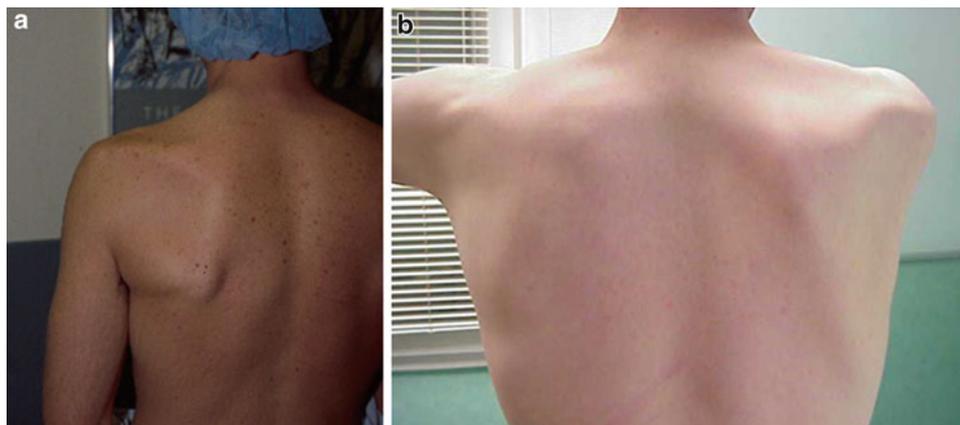


Fig. 11 (a) Clinical photo of a left shoulder demonstrating isolated infraspinatus atrophy in this young athlete. (b) Clinical photo of bilateral shoulders, right affected more than left, with supraspinatus and infraspinatus atrophy implying compression at the transverse scapular ligament (Copyright K. Plancher)



Fig. 12 Clinical photo of a weight lifter with pain, weakness, and EMG-proven compression of the suprascapular nerve at the transverse scapular ligament in the left shoulder (Copyright K. Plancher)

who participates in a weight training program may at times be misleading due to the overlying trapezius and large bulk of the deltoid muscles (Fig. 12). Range of motion must be tested and there may be only a subtle loss of external rotation and abduction strength in these young patients. In the patient with long-standing disease, the teres minor and serratus anterior muscle can compensate for weakness of the infraspinatus to obtain near normal strength. While tenderness may exist in the suprascapular notch between the clavicle and scapular spine, located 3 cm medial and anterior to Nevaizer's portal, this finding is commonly seen with many other disease entities.

Provocative tests for any labral pathology must be confirmed as labral tears may be found in conjunction with a suprascapular neuropathy. One of the best ways to help make the diagnosis of suprascapular neuropathy on physical examination is to perform the cross-arm adduction test (Fig. 13a–c). The patient puts their hand of the affected side on the opposite shoulder and lifts the elbow to the horizontal plane. The elbow is pulled by the examiner to the non-affected side and will provoke pain in the presence of a suprascapular nerve compression. The suprascapular nerve sends a branch to the acromioclavicular (AC) joint. Therefore, patients often have pain located in the AC joint with no evidence of AC joint degeneration on either x-ray or profound tenderness on palpation.

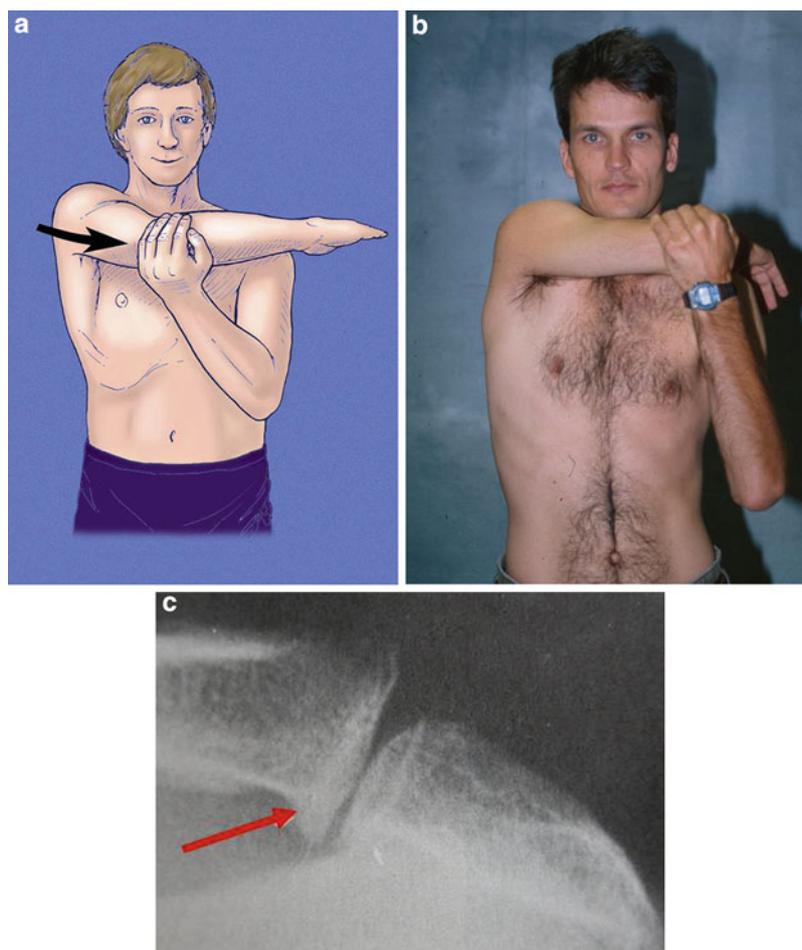


Fig. 13 (a) Artwork and (b) clinical photo of the cross-arm adduction test. (c) Zanca view of a left shoulder showing classical osteoarthritis of the acromioclavicular joint with an osteophyte which would preclude a diagnosis of suprascapular nerve entrapment (Copyright K. Plancher)

Confirmation of this disease entity is possible prior to any EMG or radiological testing with palpation in the suprascapular notch, a positive cross-body adduction test with negative plain radiographs, observation of atrophy, and when not present, a presentation of a dropping or protraction with slight winging of the scapula, and a confirmatory injection as described below when atrophy is not seen.

The differential diagnosis for suprascapular neuropathy therefore includes cervical disk disease, brachial neuritis (i.e., Parsonage-Turner Syndrome), rotator cuff tendinopathy, labral pathology with or without a ganglion cyst, a mild form of adhesive capsulitis, osteoarthritis of the glenohumeral joint, bursitis of the subacromial space with or without impingement syndrome, AC joint degeneration, posterior instability, quadrilateral space syndrome, triangular space and interval disease or thoracic outlet syndrome, and the rare Pancoast tumor.

Radiographic Examination

While many authors have suggested that the diagnosis of suprascapular neuropathy is difficult, as it is a diagnosis of exclusion, an accurate history, detailed physical examination, and appropriate diagnostic imaging can accurately diagnose this disease entity and detect any overt neoplastic disease.

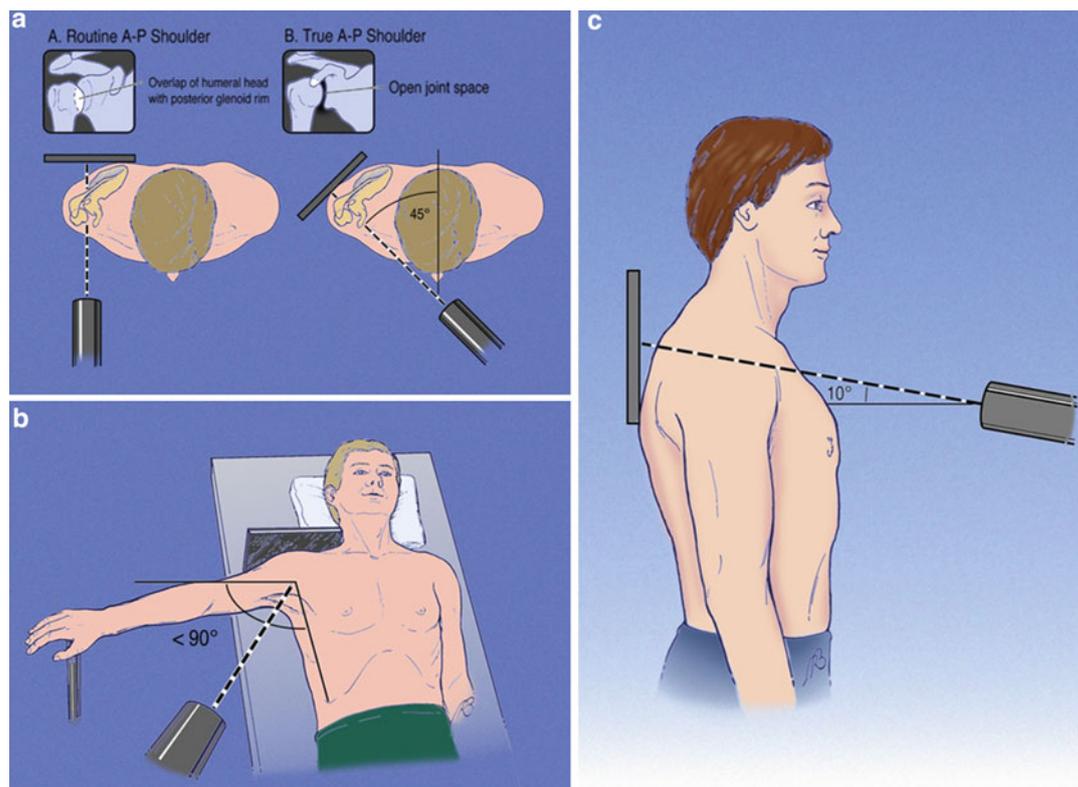


Fig. 14 (a) Artwork demonstrating the difference and correct way to obtain a true versus routine AP view of the shoulder. (b) Supine axillary view artwork demonstrated. (c) Artwork demonstrating the direction of the x-ray beam to obtain an x-ray of the acromioclavicular joint with a Zanca view (Copyright K. Plancher)

Table 1 Anatomic types of suprascapular notch configuration as described by Rengachary (Rengachary et al. 1979b)

Type 1.	Entire superior border of the scapula shows a wide depression
Type 2.	Blunted V-shaped notch occupying one-third of the superior border of the scapula
Type 3.	Symmetrical and U-shaped with parallel lateral margins
Type 4.	Very small V-shaped narrow groove, representing a bony impression of the suprascapular nerve
Type 5.	Similar to type 3, with partial ossification of the medial part of the ligament resulting in a notch with minimal diameter along the superior border
Type 6.	Ligament completely ossified

Plain radiographs should always be obtained including a true (Grashey) AP, Y view, axillary lateral, Stryker notch, and Zanca view to observe the AC joint (Fig. 14a–c). An AP scapular view with the beam aimed 15–30° cephalad obliquely at the transverse scapular ligament can also be used in the hope of seeing any calcifications, exostosis, or previous trauma in the form of callous formation at the notch of osseous notch variants (Yoon et al. 1981; Post and Mayer 1987). This plain series will hopefully catch any fracture or minute trauma to the scapula, clavicle, coracoid, or glenoid neck.

Utilization of computed tomography is valuable to detect or confirm notch variants as described by Rengachary et al. (1979a) (Table 1), fractures of the clavicle or scapula, and evidence of an ossified transverse ligament. The authors routinely use magnetic resonance imaging (MRI) as the



Fig. 15 (a–c) Three MRI slices demonstrating a classical finding of a ganglion cyst compressing the suprascapular nerve (Copyright K. Plancher)

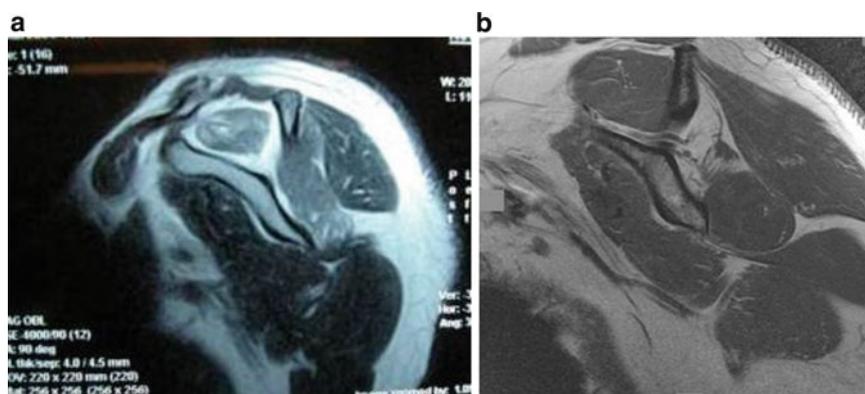


Fig. 16 (a) Sagittal oblique MRI demonstrating supraspinatus atrophy in this young male. **(b)** Sagittal oblique MRI demonstrating isolated infraspinatus atrophy in this volleyball player. Note the course of the nerve in this T2-weighted image (Copyright K. Plancher)

best imaging modality in suspected suprascapular nerve pathology because of its soft tissue resolution.

MRI and identification of soft tissue masses like a ganglion cyst have been popularized by many. Identification of these space-occupying lesions is important to identify a rare but seen cause of compression at the transverse scapular ligament. The MRI will help to identify their presence, location, and size. Fritz has described the characteristic findings in asymptomatic patients with a ganglion cyst as a homogenous signal, low T1 signal intensity with high T2 signal intensity, and rim enhancement if contrast is placed (Fritz et al. 1992) (Fig. 15a–c). The MRI will detect labral tears which may arise from the glenohumeral joint producing secondary impingement on the suprascapular nerve, rotator cuff tendinopathy, neoplastic processes whether nerve in origin or not, and osteoarthritis of the glenohumeral joint. The course of the nerve can be well seen with a T2-weighted sagittal oblique image. The presence or absence of muscle atrophy and fatty infiltration can be easily visualized of both supraspinatus and infraspinatus (Fig. 16a, b). Other authors have written about the presence of muscle edema as one of the earliest signs of suprascapular nerve entrapment (Ludig et al. 2001).

Newer modalities such as the ultrasound may be helpful as well to identify ganglion cysts. This operator-dependent test can be very helpful, not only in making a diagnosis, but in assisting surgeons

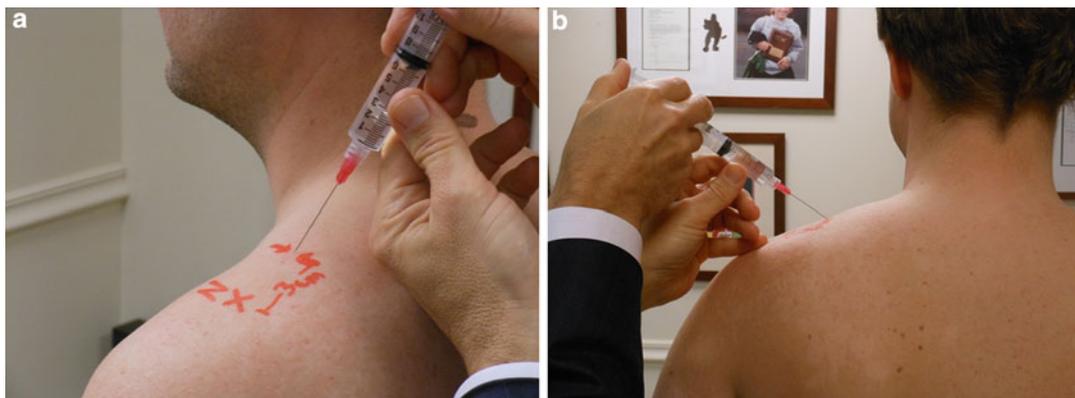


Fig. 17 (a) Clinical photo of a lidocaine injection to be placed at the transverse scapular ligament, 3 cm medial to Nevaizer's portal. (b) Posterior view. Please note the angle of the needle (Copyright K. Plancher)

to complete an ultrasound-guided aspiration of the ganglion cyst. Compression sites can be easily seen and aid in making a definite diagnosis.

Lidocaine Test Injection: How and Why

A 1 % lidocaine anesthetic injection can be immensely helpful to accurately make the diagnosis of suprascapular nerve entrapment. The needle should be placed into the suprascapular notch from a posterosuperior injection, 3 cm medial to Nevaizer's portal aiming anteriorly and aspirating first. It is important to understand the relationship of the artery to the nerve at the transverse scapular ligament. The authors use a 25-gauge, 1½-inch needle with great success, as previously described (Rose and Kelly 1969) (Fig. 17a, b). Pain relief can be dramatic and almost immediate. The cross-arm adduction test should be performed no different than when using a diagnostic injection for confirmation of impingement syndrome. The patient may not describe the absence of pain at the AC joint after this intervention, once again, helping the physician in ascertaining a definite diagnosis of a suprascapular nerve compression. The ultrasound may be used as an adjunct to guide the needle to ensure accuracy. A negative test does not rule out the disease in those patients who have a type 4–6 notch, as the ability to deliver the lidocaine is quite difficult in those situations. Diagnostic injections in other areas of the shoulder may also be helpful to rule in or rule out other disease entities.

EMG

Electrodiagnostic testing with myography and nerve conduction studies can be helpful, if positive, when the diagnosis is suspected by physical exam, imaging studies are negative (i.e., no soft tissue mass is seen), and atrophy is not present. Increased latency time often indicates impaired conductivity. The usual latency, or nerve conduction velocity, varies in a range of 1.7–3.7 ms for the supraspinatus. A value beyond 2.7 ms often indicates an abnormality. An increased latency beyond 3.3 ms (range 2.4–4.2 ms) signifies a positive result for compression to the infraspinatus. The stimulation point is typically performed at Erb's point (Khalili 1974).

Other authors have classically stated that a decrease in the amplitude or spontaneous or marked polyphasicity of the evoked potentials is significant in confirming the presence of suprascapular entrapment (Post and Mayer 1987). Patients who have a long-standing neuropathy often have a reduction in the interference pattern in denervation to the supraspinatus and infraspinatus. The presence of positive sharp waves and fibrillation potentials and absence or decreased numbers of motor unit action potentials in the infraspinatus and supraspinatus muscles are an additional or alternative finding noted on EMG that confirms a suprascapular nerve compression.

A classic, positive electrodiagnostic study that detects compression at the spinoglenoid ligament will demonstrate a motor loss to the infraspinatus without changes in the supraspinatus muscle. One expects the report to reveal a delayed terminal latency to the inferior branch of the suprascapular nerve (Ogino et al. 1991). Evaluation of the sensory velocities is less useful as the sensory innervation of this nerve is not as well defined.

Suprascapular nerve dysfunction can be present with a normal nerve conduction study and EMG. It has been shown that EMG and nerve conduction velocity may only be accurate 91 % of the time in detecting nerve injury associated with muscle weakness (Post and Grinblat 1993; Nardin et al. 2002). EMG testing of the infraspinatus is even more difficult to detect as only one branch can be affected and the rest of the muscle may be unaffected, misleading the physician to think that suprascapular nerve entrapment is not present. Nonetheless, the electromyogram may be a useful adjunct when taken as an additional piece of information with a history, physical examination, and appropriate imaging studies to confirm the diagnosis of compression of the suprascapular nerve at either the transverse scapular ligament or spinoglenoid ligament. It is essential though when ordering the test to ensure the prescription reads bilateral as the stimulating electrode to the pickup needle conduction velocity must be measured and compared to the opposite side.

Physical Therapy and Nonoperative Treatment

Most treating physicians believe that the initial treatment for an isolated suprascapular nerve compression is rest, activity modification, anti-inflammatory medications, physical therapy to maintain a normal range of motion, and strengthening of the shoulder girdle with return to sport after proprioceptive and plyometric exercises. Oftentimes, patients need sessions with their therapist to enhance scapular stability and resistive strengthening programs.

While the natural history of this disease is not known, it is therefore not known how long to pursue a nonoperative course. If there is a space-occupying lesion, nonoperative treatment should not be longer than 8 weeks. During these preoperative weeks, the patient should be improving their placement of the scapula on the trunk, and when scapular stability is regained, operative intervention should proceed. In the absence of a space-occupying lesion and a negative MRI for atrophy and negative EMG, 6 months of conservative treatment with physical therapy and activity modification should be pursued. It is important to manage the expectation of the patient and inform them that symptoms are often present for more than 6 months even with a strengthening program of the rotator cuff muscles and periscapular musculature (i.e., trapezius, rhomboids, and serratus anterior). Neuropathic symptoms, such as weakness and pain, may take more than a year to reach an improvement level satisfactory to the patient, much like the disease process of adhesive capsulitis.

All patients that present on physical examination with visible atrophy to the supraspinatus and or infraspinatus should have a minimum time of nonoperative treatment. Good results have been shown with early intervention to alleviate the pain and with release of the suprascapular nerve since this atrophy that has developed is most of the time irreversible in our young patients (Post 1999).

While many authors believe that a program of therapy that concentrates on scapular stabilization, shoulder motion, and strengthening is disease-altering, the authors have realized that this theory is incorrect and can work only to sustain a young athlete because his or her serratus anterior or teres will support the shoulder and their chronic ache and or pain may retreat. Unfortunately, when these same patients return 10 years later, they have marked atrophy of either fossa and have an irreversible muscle-damaging disease to the supraspinatus, infraspinatus, or both the supraspinatus and infraspinatus. Careful thought is necessary, and early treatment should be considered to allow these former athletes to enjoy their 30s, 40s, and 50s with the same overhead sports or even jobs they have been performing when younger (Post and Mayer 1987; Callahan et al. 1991). In newly

presenting, advanced, and long-standing cases, in our hands, spinati atrophy almost never recover completely though the shoulder pain generally improves. Prior to the arthroscopic approach because of the limited experience of many surgeons and an attitude of hesitancy because of the anatomy, a diagnosis of suprascapular neuropathy has not been pursued as vigorously. It is the hope of the authors and others writing on this topic that patients will afford the opportunity of an early diagnosis and intervention to make suprascapular compression a disease entity that no longer only sees the clinician, but the clinician sees it and can reverse this disorder that it becomes a small chapter in all patients' lives.

Arthroscopic Release of the Transverse Scapular Ligament

There are several indications for a surgical release of the transverse scapular ligament. Conservative treatment must be adequate with completion of scapular stabilization exercises. As stated above, the length of time that an individual participates in nonoperative treatment may vary. It has become clear that any sport that involves an overhead motion puts a person at risk because of the overhead motion. Surgical intervention should be considered in those patients as outlined.

If there is a soft tissue mass, we have discussed how operative intervention in our hands is superior to avoid long-standing deficits. These lipomas or ganglion cysts can be easily taken care of arthroscopically along with any labral or other intra-articular pathology that may be seen at the time of surgery.

The most controversial area that concerns release of the transverse scapular ligament is when there is a large or massive rotator cuff tear. Mallon et al. have previously demonstrated the presence of a suprascapular nerve injury by EMG in their patients (Mallon et al. 2006). The authors support that decompression of the suprascapular nerve at the transverse scapular ligament is essential with patients who have a massive rotator cuff tear to ensure viability of the repair (Albritton et al. 2003; Mallon et al. 2006). The authors hypothesized that the cuff, when it retracts, results in the tethering of the nerve at the suprascapular notch and by releasing it, the nerve may move more laterally to release its tension. Other studies have shown only a 28 % involvement of a peripheral neuropathy in patients with a full-thickness rotator cuff tear (Vad et al. 2003). Within this group, the suprascapular nerve was involved in 29 % of patients and it was the axillary nerve that was much more involved with a deficit (Vad et al. 2003). Another small series of patients found that 27 % of their patients with a massive rotator cuff tear with visual atrophy on physical examination and fatty degeneration by MRI had suprascapular dysfunction on electrodiagnostic testing (Costouros et al. 2007). All of these patients after undergoing repair of their rotator cuff had resolution of the findings to normal on EMG. Our investigations have found similar findings, and for that reason all patients with a massive rotator cuff tear that will be undergoing surgical intervention receive an EMG preoperatively. The authors routinely release the transverse scapular ligament as a part of the operation when restoring the rotator cuff architecture.

The authors have questioned, like other groups, whether the cause of suprascapular nerve dysfunction after surgical repair of a massive tear is a result of pulling on the cuff and subsequently the suprascapular nerve. Gerber et al. have studied this thought with an elegant study that revealed that the supraspinatus and infraspinatus could be advanced more than 3 cm before the motor branches of the supraspinatus are restricted and placed on tension to affect a conduction velocity (Warner et al. 1992). If a transverse scapular ligament release was not performed when the tendons were repaired, only 1 cm of lateral advancement was possible before the nerve was under tension. If the transverse scapular ligament was released, the cuff could be advanced even further before any tension arose in the nerve. This study and others have convinced, us as always, to do no harm, but to also go the extra mile to perhaps help these patients with a very difficult problem to attempt

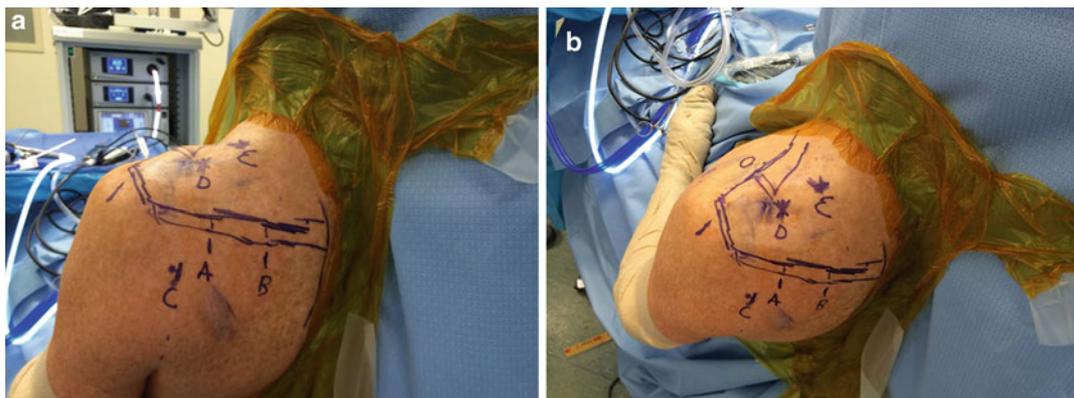


Fig. 18 (a) Intraoperative photo of a left shoulder prepped and draped in preparation for a transverse scapular ligament and spinoglenoid ligament release. The portals are labeled as follows: *A*, working portal for spinoglenoid ligament release; *B*, viewing portal with 30° arthroscope for spinoglenoid ligament release; *C*, standard posterior portal for intra-articular glenohumeral arthroscopy; *D*, Nevaizer’s portal; *E*, portal for release of the transverse scapular ligament. Note *vertical purple mark* on the lateral aspect of the shoulder approximately halfway from anterior to posterior along the lateral margin of the acromion. This is the viewing portal for release of the transverse scapular ligament. (b) View from above showing the same portal in a left shoulder. The *round circle* anteriorly is the coracoid. Anterior to portal *D* is the acromioclavicular joint. The arthroscopic shaver or thermal device will be placed at the anterolateral edge of the acromion with the viewing portal shown as the *solid purple line* laterally off the acromion to allow us to release the transverse scapular ligament (Copyright K. Plancher)

resolution of their pain and weakness. Patients with massive, chronically retracted cuffs with or without fat atrophy receive a release of the transverse scapular ligament in the hope of achieving homeostasis to the muscle tendon unit.

Technique

The advantages of arthroscopic release are well discussed by others. The ability to visualize anatomy and return to sport or activities of daily living is much faster and simpler, and the morbidity and postoperative recovery is much simpler and pleasant for the patient.

The patient is placed in the beach chair position with the arm placed at its side. It is important to prep and drape from the midsternum to the mid-posterior spine with the neck area included. The anesthesiologist should maintain a systolic blood pressure slightly below 100 mmHg. Our pump pressure is kept low at 45 mmHg to avoid unnecessary swelling.

The portals selected include the standard subacromial portals, that is, a lateral subacromial portal and an anterolateral portal. The patient more often than not because of the young age will have had a subacromial decompression with utilization of a standard posterior portal. Additional portals are necessary for success of a decompression of the transverse scapular ligament. The added portal is a portal made from outside-in first with an 18-gauge spinal needle, 3 cm medial to Nevaizer’s portal ensuring that the portal is anterior to the supraspinatus leading edge. The portal is approximately 6–8 cm medial to the anterolateral border of the acromion in between the clavicle and scapular spine (Fig. 18a, b).

Release of the transverse scapular ligament does not begin with glenohumeral inspection, and if it was to begin with a full inspection, that part of the procedure should take no more than 5 minutes to ensure a limited amount of swelling to occur in the limb. Instead, the arthroscope is introduced into the subacromial space and a subacromial decompression is completed to allow for adequate visualization. The arthroscope is moved midway to 2/3 of the way posterior along the lateral edge of the acromion or may be placed at the posterolateral corner (Fig. 19a–c). The shaver is introduced

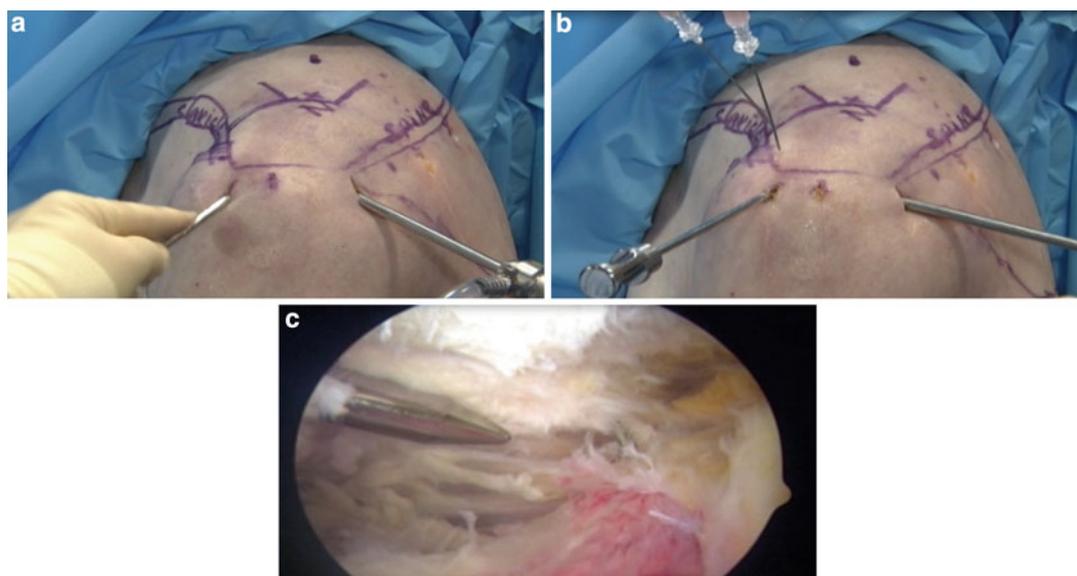


Fig. 19 (a, b) Clinical photo of a left shoulder in preparation for a transverse scapular ligament release. The arthroscope is placed in the posterolateral corner to begin a subacromial decompression with the trocar sweeping tissue and in place to be replaced with a shaver in anticipation of resecting and following the coracoacromial (CA) ligament medially to the coracoid. (c) Arthroscopic inside view of a left shoulder showing the supraspinatus, its leading edge anterior to the left, with the trocar inside pointing to the CA ligament (Copyright K. Plancher)

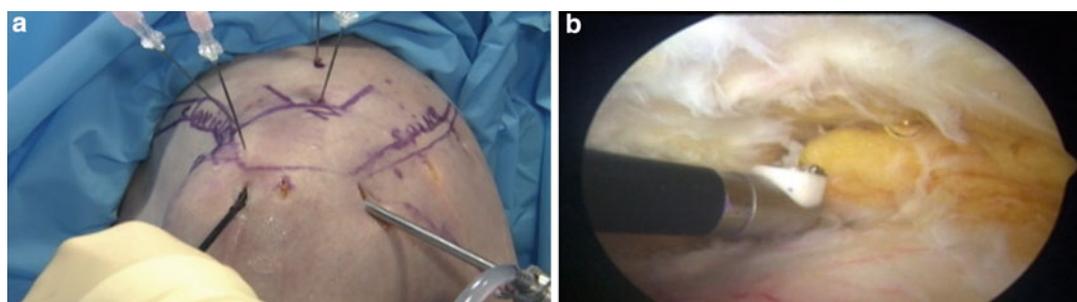


Fig. 20 (a) Left shoulder demonstrating spinal needles to accurately identify landmarks inside the subacromial space. Note the spinal needle in Nevaizer's portal. (b) Radiofrequency device clearing the soft tissue as it heads medially to identify the spinal needle coming from outside-in of Nevaizer's portal (Copyright K. Plancher)

in a new portal created at the anterolateral edge of the acromion. This portal should be placed as close to the acromion as possible. This entry point will allow for adequate clearance of all soft tissues necessary to complete this operation.

Identification of the various landmarks is completed with the aid of 18-gauge spinal needles. One spinal needle is placed in the center of the AC joint and a second needle is placed in Nevaizer's portal (Fig. 20a, b). The shaver releases the coracoacromial ligament laterally during a subacromial decompression and follows its medial side to the coracoid. The soft tissue is either ablated with a radiofrequency device or removed with a mechanical shaver, but ensuring hemostasis and perfect visualization is maintained throughout the procedure (Fig. 21). The leading or anterior edge of the supraspinatus is always maintained in view while proceeding to release the transverse scapular ligament. Upon arriving at the coracoid, the coracoclavicular ligaments are identified first, then laterally the trapezoid, and subsequently the conoid or more medial ligament. The conoid is always

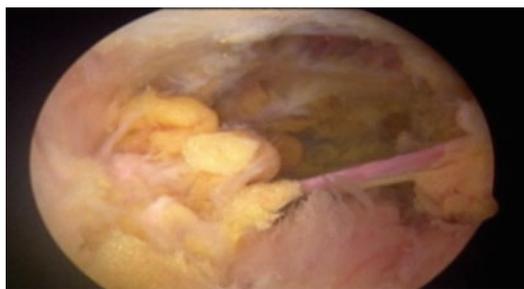


Fig. 21 Arthroscopic view of the same left shoulder demonstrating soft tissue cleared and visualization often with small tributaries of the suprascapular artery left unharmed (Copyright K. Plancher)



Fig. 22 (a) Clinical photo of left shoulder demonstrating 18-gauge spinal needle entering 3 cm medial to Nevaizer's portal to help identify the transverse scapular ligament. (b) Arthroscopic view of the same needle heading toward the transverse scapular ligament to aid in visualization of an accurate landmark (Copyright K. Plancher)

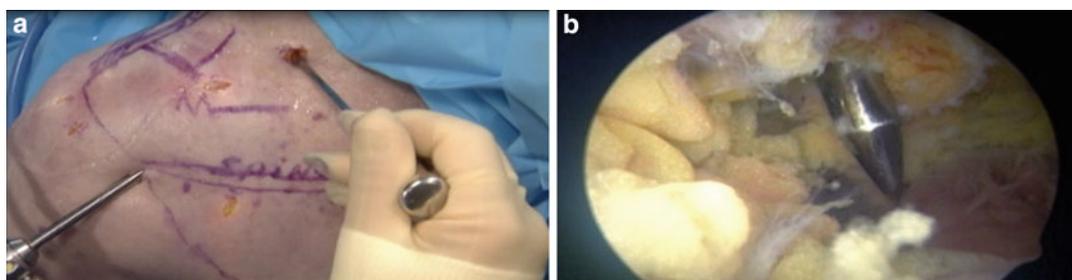


Fig. 23 (a) Clinical photo of left shoulder demonstrating a trocar entering 3 cm medial to Nevaizer's portal after a skin incision has been made. (b) Arthroscopic view of the same trocar heading toward the transverse scapular ligament to aid in retracting the artery and nerve out of harm's way (Copyright K. Plancher)

more posterior in position and there is usually an area of fat surrounding this ligament. It is recommended to clear this space with the use of a radiofrequency wand. The spinal needle placed in the AC joint will remind the surgeon of the location of the conoid ligament, and the needle in Nevaizer's portal will keep visualization in the correct orientation as the arthroscope is placed more medially as the operation continues. The key to a successful operation is understanding that the most medial border of the conoid ligament is the most lateral attachment of the transverse scapular ligament. If the surgeon, as has been already stated, stays anterior to the supraspinatus, finding the transverse scapular ligament will not be difficult, but if the arthroscope strays posteriorly, then identification becomes more difficult. When dealing with any soft tissue mass that exists in the

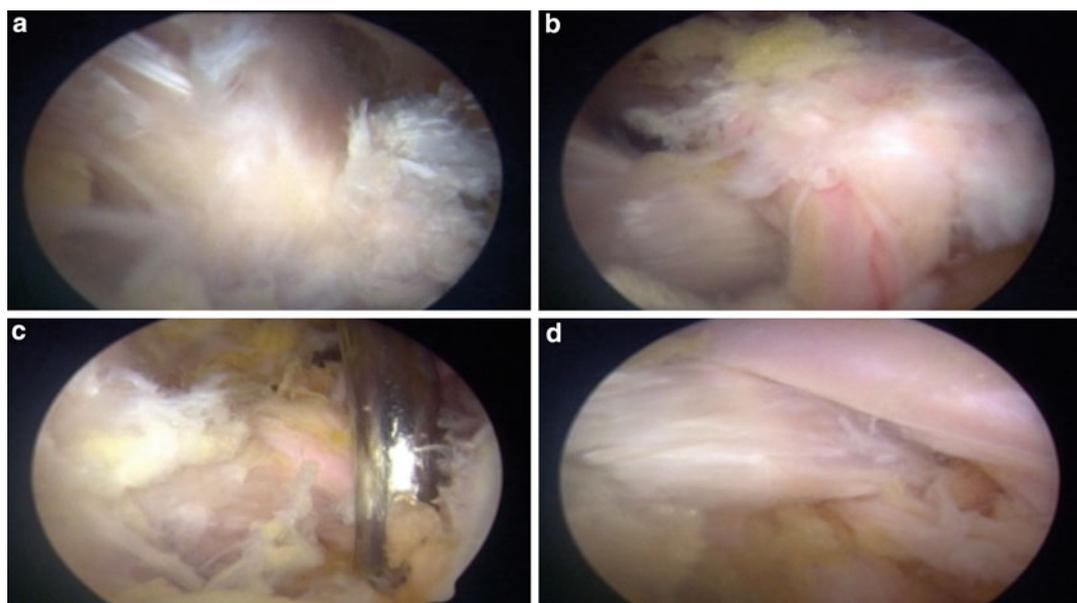


Fig. 24 (a) The conoid ligament, recognized as the most lateral attachment of the transverse scapular ligament, now with the transverse scapular ligament in sight but covered by soft tissue and the artery and nerve not protected. (b) Arthroscopic view of the suprascapular artery lying over “the transverse scapular ligament” in harm’s way. (c) A blunt obturator/trocar retracting the artery out of harm’s way revealing the suprascapular nerve adhered to the calcified and thickened transverse scapular ligament. (d) Arthroscopic view of the suprascapular nerve still not adequately retracted safely with the transverse scapular ligament in clear view (Copyright K. Plancher)

supraspinatus fossa, this must be evacuated as one continues the release and moves medially to the transverse scapular ligament. The stalk though of the soft tissue mass will almost assuredly be located alongside the transverse scapular ligament, and upon release of the ligament and protection of the nerve, the stalk may be excised.

An additional portal is now made upon recognition of the conoid ligament. The 18-gauge spinal needle is introduced 3 cm medial to Nevaiser’s portal, and the soft tissue is cleared up to this area (Fig. 22a, b). Rotation of the arthroscope to look down will identify the artery and or vein normally lying over the transverse scapular ligament. The outside-in technique allows for a safety factor, and a skin incision is made large enough to introduce the blunt obturator from the arthroscope that will aid in gently pushing away tissue to visualize the transverse scapular ligament and the suprascapular nerve (Fig. 23a, b). The blunt obturator will retract the supraspinatus muscle and fat posteriorly which will allow for an excellent view of the transverse scapular ligament, suprascapular artery, and suprascapular nerve (Fig. 24a–d). The obturator is then positioned to displace the nerve more medially so that the transverse scapular ligament is isolated. A small incision is then made in the skin, and an arthroscopic scissor is placed in the anatomic position to divide the transverse scapular ligament close to the bone (Fig. 25a–d). If the ligament is calcified, a lambotte osteotome is used through this second small incision. A 3.5-mm burr or small 3.5-mm full radius shaver may be used safely to smooth any osteophytes that may be encountered. The blunt tip trocar is utilized to assess the mobility and adequate release of the suprascapular nerve.

Our experience with this technique has been successful when a patient has failed conservative treatment, has EMG-proven compression, and has visual atrophy in the supraspinatus and infraspinatus fossa. The patient’s pain profile the next day after release is verbalized as completely gone, and while the authors have not been successful in re-insufflating the muscle belly, they have in those whose disease has not been present for more than 2 years restored some measurable strength to

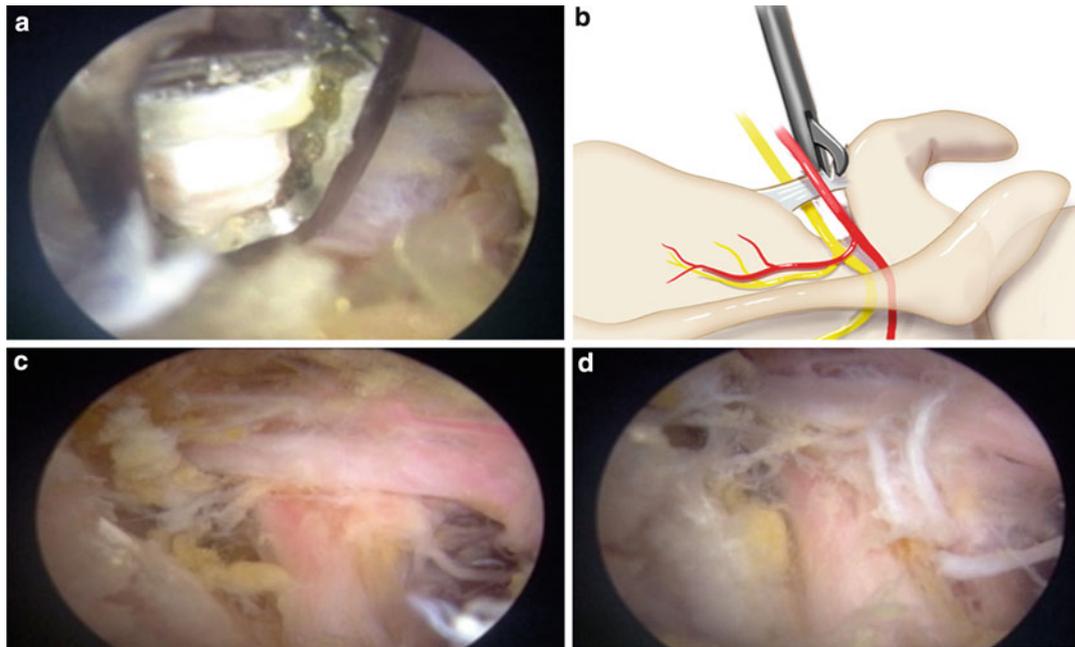


Fig. 25 (a) Arthroscopic view with the scissors attempting to cut and remove the calcified transverse scapular ligament. In this, a lambotte osteotome was introduced to help remove the calcified transverse scapular ligament. (b) Artwork illustrating release of the transverse scapular ligament corresponding to the clinical photo in (a). (c) Arthroscopic view of successful release of the transverse scapular ligament revealing the suprascapular nerve to the right and the remnant of the calcified transverse scapular ligament to the left. Note superiorly and running diagonally to the right the suprascapular artery. (d) Closeup arthroscopic view of release suprascapular nerve with remnant of calcified transverse scapular ligament on the left side of the screen (Copyright K. Plancher)

external rotation. Next, any pathology that must be dealt with in the intra-articular space is addressed.

Outcomes

Results of close to 300 reported decompressions at transverse scapular ligament have been cited in the literature although most with the open technique and many appear in the neurosurgical literature (Garcia and McQueen 1981; Hadley et al. 1986; Ringel et al. 1990; Callahan et al. 1991; Jackson et al. 1995; Fabre et al. 1999; Antoniou et al. 2001; Hazrati et al. 2003; Kim et al. 2005; Gosk et al. 2007; de Jesus et al. 2009). Recent investigations discuss outcomes with the arthroscopic technique (Lafosse et al. 2007). Within the hands of surgeons that understand the anatomy surrounding the suprascapular nerve, very few complications have been reported, although it is discouraging as previously mentioned above, the ability to restore muscle strength and reverse the muscle atrophy is very difficult if not impossible. Restoration of strength to the supraspinatus muscle has been easier to accomplish over the infraspinatus; however, the reasons are unknown. In a large series of 42 releases, 90 % restored strength of a grade 4 or better to the supraspinatus (Kim et al. 2005). Restoration of muscle atrophy on the other hand, as discussed above, is quite difficult although as reported by Fabre et al. had a resolution in 52 % of their patients with suprascapular muscle atrophy (Fabre et al. 1999).

While many studies are reported as case series without a control group for comparison of treatment options and long-term follow-up is not available, the disease entity itself is not as common. Older studies like that of Martin et al. reviewed their results of nonoperative treatment with physical

therapy in a small series of 15 patients and a 3-year follow-up (Martin et al. 1997). They stated with only 33 % with excellent results that nonoperative treatment in the absence of a well-defined lesion-producing mechanical compression is the correct clinical intervention. Larger studies like those quoted above like Callahan et al. in 1991 reported on open resection of the transverse scapular ligament and found 91 % of their patients pain-free with a long-term follow-up showing approximately 88 % survival with results unchanged at 4 years postoperatively (Callahan et al. 1991). Many other series are available that list excellent results with decompression and even without treating any pathological changes within the labrum (Post and Grinblat 1993).

Summary

The diagnosis of suprascapular nerve compression may stay elusive for some. It is our hope with this chapter that physicians and surgeons will recognize the pain and weakness suffered by many active patients and approach this diagnosis with increasing thoughts to intervene to allow our patients to have an active and healthy lifestyle.

The etiology of suprascapular nerve compression at the transverse scapular ligament can include traction by direct (e.g., soft tissue mass) or indirect means (e.g., repetitive overuse model or a retracted massive rotator cuff tear). The history will lead the physician to clues along with a complete physical examination with observation of the presence or absence of atrophy to the supraspinatus and/or infraspinatus fossa and noted weakness of external rotation of the shoulder. Utilization of diagnostic imaging may be helpful and ordering an appropriate EMG with nerve conduction velocities may assist in confirming the diagnosis. Even in the absence of a positive EMG and no mass visualized on MRI, the diagnosis must be made with nonoperative intervention commenced. Operative decompression for patients with soft tissue masses and symptoms refractory to conservative treatment will succeed to eradicate their symptoms. Decompression for patients with massive rotator cuff tears still needs further thoughtful investigation, and patients with negative EMG findings may need decompression as well with early intervention. Arthroscopic decompression of the transverse scapular ligament is now performed by many, and these results continue to show excellent long-term results in our hands. The authors encourage all physicians taking care of posterior shoulder pain to consider the diagnosis of compression of the suprascapular nerve when dealing with any patient with pain and weakness of the shoulder girdle.

Compression of the Spinoglenoid Ligament

Pathophysiology

Injury to the suprascapular nerve may occur at the spinoglenoid ligament (Fig. 26). While the usual site of suprascapular entrapment neuropathy is at the transverse scapular ligament in the suprascapular foramen, clinical presentation and diagnosis of compression at the most distal site has been well recorded (Fig. 27). Several mechanisms have been proposed and previously discussed above. While most commonly thought of in overhead athletes, injury to this nerve may occur from repetitive traction and microtrauma (Ferretti et al. 1998; Plancher et al. 2005a, 2007; Lajtai et al. 2009, 2012). The spinoglenoid ligament has also been demonstrated to tighten when the shoulder is in the position for overhead throwing, resulting in increased pressure on the suprascapular nerve (Plancher et al. 2005b) (Fig. 10). Early literature speculated from authors that injury to this nerve occurred by intimal damage from microemboli in the vasa nervorum (Ringel et al. 1990). A stenotic notch or ossified spinoglenoid ligament or even superiorly oriented fibers of the subscapularis muscle may cause a suprascapular neuropathy (Bigliani et al. 1990; Bayramoglu

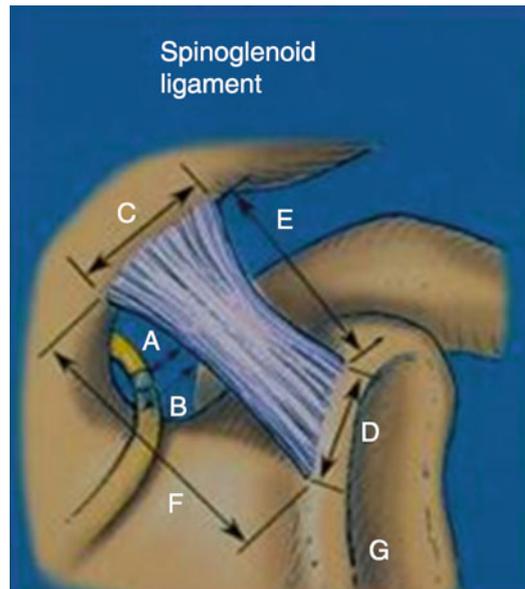


Fig. 26 Artwork demonstrating the relationship of the spinoglenoid ligament in a previous published study with investigation of space available between the suprascapular nerve and the spinoglenoid ligament. Note the attachment to the spine of the scapula (Copyright K. Plancher)

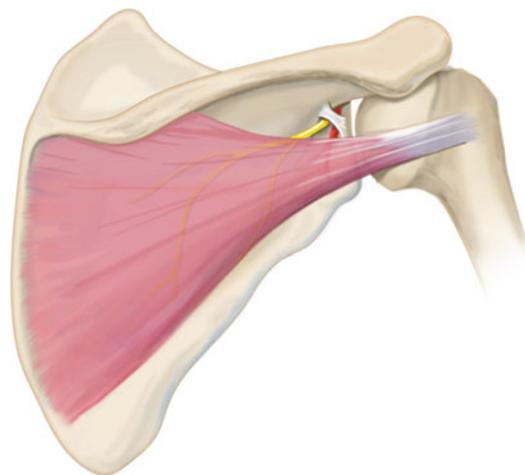


Fig. 27 Artwork demonstrating suprascapular nerve entrapment at the spinoglenoid ligament. Note the medial course of the nerve as it wraps around the spinoglenoid notch (Copyright K. Plancher)

et al. 2003). Compression of the nerve at this location has been noted by many authors to be caused by a soft tissue mass or ganglion cyst as a result of some form of a labral or capsule injury. The authors have though maintained a position of decompressing the ganglion from the posterior aspect of the shoulder and not repairing the labrum unlike others with excellent results (Abboud et al. 2006; Westerheide et al. 2006) (Fig. 28a–f). Compression by a ganglion cyst or soft tissue mass has known to occur because of the relatively fixed position of the suprascapular nerve combined with the close proximity of the infraspinatus muscle to the glenohumeral joint. These ganglia may form when a

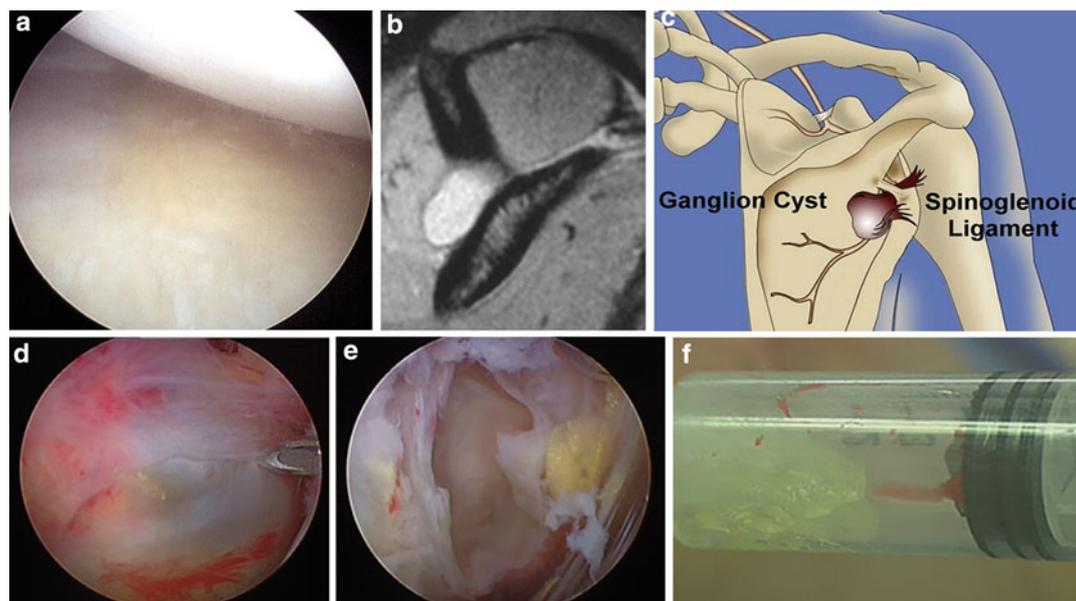


Fig. 28 (a) Arthroscopic view of a ganglion cyst decompressed from the outside emitting its contents intra-articularly through a posterior inferior perforation in the labrum. (b) Sagittal oblique MRI demonstrating ganglion cyst compressing the suprascapular nerve at the spinoglenoid notch. (c) Artwork of posterior view of right shoulder demonstrating classic ganglion compressing the spinoglenoid ligament at its notch (a-c: Copyright K. Plancher). (d) Posterior view of bulging ganglion cyst located at the spinoglenoid notch. (e) Decompressed ganglion cyst at the spinoglenoid notch prior to complete excision of its root. (f) Syringe containing the contents of the ganglion cyst commonly seen on MRI compressing the suprascapular nerve at the spinoglenoid ligament (d-f: Courtesy of Ticker et al. 1988)

capsule of labral tears and synovial fluid is forced into the tissues as a one-way valve no different than meniscal cysts known to occur in the knee (Moore et al. 1997).

While rare, a patient may have a neuropathy from a Parsonage-Turner syndrome, although it is more common for this viral neuritis to attack other nerves. Once again, whatever the mechanism, compression or injury to the suprascapular nerve at the spinoglenoid ligament will result in weakness, and, if long term, atrophy of the infraspinatus muscle with little if any probability of return to normal muscle strength will occur.

Patient Profile

History

Patients with compression of the suprascapular nerve at the spinoglenoid notch comprise a special group of more often than not overhead athletes and laborers that perform all their tasks above the shoulder. They are young, usually well-developed, and complain of a diffuse ache around the shoulder region. Their pain is more localized to 4 cm medial to the posterolateral corner of the acromion as well as near the posterior aspect of the glenohumeral joint.

A patient may complain of weakness on attempts of external rotation and abduction which may confuse the examiner because he or she may think, no different than compression at the transverse scapular ligament, that the patient has rotator cuff disease or even cervical disk disease. A patient though with compression of the suprascapular nerve at the spinoglenoid ligament has a more profound weakness on external rotation and has a longer chronic history often of missed diagnosis. There are exceptions where compression can occur because of an acute trauma as in a forced external rotation of the upper extremity required in many racquet sports. This activity when discovered on



Fig. 29 Clinical photo and posterior view of the right shoulder demonstrating severe atrophy in a 21-year-old female tennis player with chronic wasting of the infraspinatus since age 9 with no apparent diagnosis (Copyright K. Plancher)

history can produce a stretch on the suprascapular nerve and contribute to irritation at the compression point. Activities across the body are often difficult, and the motion of a follow-through whether throwing a baseball or spiking a volleyball can be quite painful at times that the athlete will avoid those movements. This position of follow-through or adduction in an extended position has been shown by our group to increase the tension and pressure within the spinoglenoid notch (Plancher et al. 2007). Common sports played by these patients include repetitive sports such as golf and others such as volleyball, basketball, tennis, weight lifting, and swimming.

While sports activities can often lead to suprascapular neuropathy, the heavy laborer may be plagued with this disease as well because of all the repetitive overhead work duties they may perform daily, no different than those laborers with compression of the suprascapular nerve at the transverse scapular ligament. Compression at the spinoglenoid ligament is often insidious in onset, and a delay in diagnosis is the single biggest problem which prevents full restoration of the muscle strength and alleviation of pain with a hope for atrophy to be eradicated.

The suprascapular nerve at the spinoglenoid notch may be compressed by a ganglion cyst because the nerve is relatively immobile as it traverses the lateral edge of the scapular spine and is in close proximity of the posterior glenohumeral joint. Diagnosis by history can be difficult because the findings overlap so much with rotator cuff and labral pathology. Certain findings though will help the clinician such as a description of weakness on external rotation activities. The patient may complain that their infraspinatus fossa appears different on comparison to the opposite side. As chronicity exists for many of these patients since their range of motion does not often decrease, the chronic ache or pain will increase, become constant, and even affect or interrupt sleeping patterns. More present with spinoglenoid compression than compression at the transverse scapular ligament is a patient complaining of catching, locking, or clicking because of the frequent association of a labral tear. Lastly, the patient who walks into the office used to be thought of as male, but with Title IX, the incidence of male to female with compression of the suprascapular nerve at the spinoglenoid ligament has an equal distribution.

Physical Examination

Clinical examination often has nonspecific findings in the early evolution of this disease entity. Symptoms are often less severe with suprascapular neuropathy at the spinoglenoid notch. Several

athletes present with painless wasting of the infraspinatus in isolation. Surprisingly so, palpation at the spinoglenoid notch can be very painful. Some patients may describe micro-instability as a part of their complaints, although confirmatory physical findings will not be found.

A cervical spine examination and both shoulders with a full neurological examination, no different than for compression of the suprascapular nerve at the transverse scapular ligament, must be completed. The patient when placed in a shoulder gown with the complete scapula in full view may demonstrate no or severe atrophy to the infraspinatus fossa (Fig. 29). Atrophy though in a well-developed individual who participates in a weight training program may at times be misleading due to the overlying trapezius and large bulk of the deltoid muscles (Fig. 12).

Range of motion must be tested and there may be only a subtle loss of external rotation and abduction strength in these young throwers. The authors have also found in long-standing disease that the teres minor and serratus anterior muscle will compensate for the loss of the infraspinatus to obtain near normal strength. Provocative tests for any labral pathology must be confirmed as labral tears may be found in conjunction with a suprascapular neuropathy and are common at the spinoglenoid ligament. The marked weakness of external rotation should be tested with the arm at the side and will be present upon testing without any significant pain. The painless finding is because the sensory portion of the suprascapular nerve may be unaffected by the spinoglenoid notch.

A cross-arm adduction test as described above must be performed and recorded and correlated with a Zanca view x-ray (Fig. 13a–c). Cross-body adduction may reproduce the patient's symptoms with the arm extended or internally rotated. The pain may be felt in the posterior aspect of the shoulder as well, but it is important to distinguish whether this pain is from the AC joint or from some other source (Fehrman et al. 1995).

The differential diagnosis for suprascapular neuropathy at the spinoglenoid notch therefore includes the same diseases as for compression of the nerve at the transverse scapular ligament (i.e., cervical disk disease, a brachial neuritis (i.e., Parsonage-Turner Syndrome), rotator cuff tendinopathy, labral pathology with or without a ganglion cyst, a mild form of adhesive capsulitis, osteoarthritis of the glenohumeral joint, bursitis of the subacromial space with or without impingement syndrome, AC degeneration disease, posterior instability, quadrilateral space syndrome, triangular space and interval disease or thoracic outlet syndrome, and the rare Pancoast tumor). The astute clinician realizes with the lack of reproducible signs on physical exam and the overlapping symptoms with other shoulder problems that compression of the suprascapular nerve at the spinoglenoid ligament may be easily overlooked.

Radiographic Examination

Plain radiographs including an AP, axillary lateral, and the Y or suprascapular outlet view should always be obtained (Fig. 14a–c). Special views such as a Stryker notch view can be ordered when necessary (Post and Mayer 1987). This plain series will identify any fracture or minute trauma to the scapula, clavicle, coracoid, or glenoid neck.

MRI and identification of soft tissue masses like a ganglion cysts have been increasingly important when evaluating compression of the suprascapular nerve at the spinoglenoid ligament (Fig. 30). The MRI can identify a ganglion with a homogenous signal, low T1 intensity with high T2 intensity, and rim enhancement if contrast is placed (Fritz et al. 1992). The MRI will also detect labral tears which may arise from the glenohumeral joint and with significance from the posterosuperior quadrant of the labrum with the ganglion cyst attached (Fig. 31). Controversy does exist with surgeons on both sides if the paralabral cyst is a secondary sign of a labral tear in patients. Those that believe that this is the case insist on treatment to the labrum to minimize

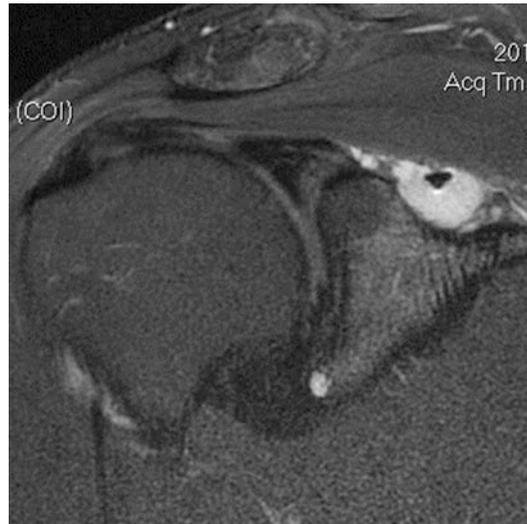


Fig. 30 MRI coronal view demonstrating ganglion cyst displacing the suprascapular nerve at the spinoglenoid notch (Copyright K. Plancher)



Fig. 31 MRI axial view demonstrating labral tear as well as ganglion cyst compressing the suprascapular nerve at the spinoglenoid notch (Copyright K. Plancher)

recurrence, while others may leave the labrum alone when the cyst has been excised or decompressed.

The presence of a soft tissue mass or ganglion cyst on MRI does not necessarily indicate suprascapular neuropathy. Abnormal signal intensity within the infraspinatus muscle can indicate



Fig. 32 Clinical photo of a lidocaine injection to be placed at the spinoglenoid ligament, 4 cm medial to the posterolateral corner of the acromion (Copyright K. Plancher)

suprascapular nerve compression at the spinoglenoid notch. Some patients will demonstrate increased signal intensity on T2 fast spin echo with fat saturation with a normal muscle mass implying subacute denervation of the nerve caused by neurogenic edema. Chronic denervation seen best on T1 spin echo with increased signal intensity within the muscle mass will demonstrate muscle atrophy with fatty infiltration (Fig. 16b).

Newer modalities such as ultrasound may be helpful as well to identify ganglion cysts. This operator-dependent test can be very helpful not only in making a diagnosis but in assisting surgeons to complete an ultrasound-guided aspiration of the ganglion cyst. Compression sites can be easily seen and aid in making a definite diagnosis no different than for compression of the suprascapular nerve at the transverse scapular ligament.

Selective Injections

A 1 % lidocaine anesthetic injection may be placed into the spinoglenoid notch to confirm the diagnosis of suprascapular nerve entrapment (Fig. 32). The needle is placed 4 cm medial to the posterolateral corner of the acromion. The patient is then asked if there is any change in the chronic ache that may have been present previously. A cross-arm adduction test is then performed and, if previously positive, should now be a non-provocative maneuver.

The authors have found pain relief to be dramatic and almost immediate. The ultrasound may be used as an adjunct to guide the needle to ensure accuracy, although unlike the injection when placed in the transverse scapular ligament, this injection is simple because one feels the spine of the scapula, drops inferior to it by 1–2 cm, and then aspirates and easily falls into the spinoglenoid notch. A negative response when there is no atrophy, a negative EMG, and no evidence of a labral tear or ganglion cyst, yet patients present with weakness and pain would require a 6-month course of nonoperative treatment before the authors consider any type of operative intervention.

EMG

Electrodiagnostic testing with myography and nerve conduction studies is the only valid way by objective evidence to confirm compression of the suprascapular nerve at the spinoglenoid notch. When the suprascapular nerve is compressed by a ganglion cyst or soft tissue mass at the spinoglenoid notch, the nerve will show decreased innervation of the infraspinatus muscle with normal innervation of the supraspinatus muscle. The stimulation point is typically performed at Erb's point. Motor distal latency and motor response amplitude at the supraspinatus and

infraspinatus muscles are measured. An increased latency beyond 3.3 ms (range 2.4–4.2 ms) confirms compression to the infraspinatus (Khalili 1974).

A classic positive electrodiagnostic study that detects compression at the spinoglenoid ligament will demonstrate a dramatic motor loss to the infraspinatus if atrophy is present without changes in the supraspinatus muscle. Patients without visible atrophy present may still have compression of the nerve to the infraspinatus and hopefully on EMG will demonstrate a delayed terminal latency to the inferior branch of the suprascapular nerve. Side-to-side measurement differences are important (Ogino et al. 1991). Evaluation of the sensory velocities is less useful as the sensory innervation of this nerve is less well defined.

Other authors have felt that the only early finding may be increased nerve conduction time. This noted finding will help the physician to understand the compression is not in the cervical spine and will be able to hopefully identify the compression point with selective injections to avoid chronic damage to the suprascapular nerve. The decrease in the amplitude or spontaneous or marked polyphasicity of the evoked potentials is significant in confirming the presence of suprascapular entrapment for many when looking at compression either at the transverse scapular ligament or spinoglenoid ligament (Post and Mayer 1987).

The authors believe suprascapular nerve dysfunction can be present with a normal nerve conduction study and electromyography. It has been shown that EMG and nerve conduction velocity may only be accurate 91 % of the time in detecting nerve injury associated with muscle weakness (Post and Grinblat 1993; Nardin et al. 2002). EMG testing of the infraspinatus is even more difficult to detect as only one branch can be affected and the rest of the muscle may be unaffected misleading the physician to think that suprascapular nerve entrapment is not present. Therefore, the authors encourage the clinician to test multiple locations. Stimulation of other periscapular muscles leads to volume interference, and perhaps needle recording is the only way of monitoring this disease in lieu of surface recordings. The suprascapular nerve, as mentioned previously, is a mixed motor and sensory nerve which makes detection of a partial compression even more difficult. The authors recommend all clinicians to communicate with the neurologist prior to allowing the patient to undergo an EMG and nerve conduction velocity testing so that the most accurate outcome is obtained.

Physical Therapy and Nonoperative Treatment

Most treating physicians believe that the initial treatment for an isolated suprascapular nerve compression is rest, activity modification, anti-inflammatory medications, physical therapy to maintain a normal range of motion, and strengthening of the shoulder girdle with return to sport after proprioceptive and plyometric exercises are completed. The authors require the therapist to enhance scapular stability and promote proper static and dynamic posture and resistive strengthening programs to the trapezius, rhomboids, and the serratus musculature prior to any operative intervention. In the absence of a lesion causing a direct compression, most neuropathies will resolve, but the symptoms of pain and weakness may take more than a year to reach full resolution.

The natural history of suprascapular nerve entrapment at the spinoglenoid notch is not known; therefore, it is not known how long to pursue a nonoperative course. If there is a space-occupying lesion, the authors would not recommend nonoperative treatment. The majority of these lesions are ganglion cysts and are often associated with labral tears. Several studies have agreed with our philosophical approach to avoid a prolonged nonoperative regime. Hawkins and his group reported that 2 out of 19 patients with a spinoglenoid cyst resolved their symptoms with conservative treatment (Piatt et al. 2002). He also found patient satisfaction was much higher with surgical

intervention. Specifically, they reported an 18 % failure rate for aspiration of the cyst and 48 % recurrence rate for those cysts which were aspirated successfully.

Ultrasound-guided aspiration of the ganglion cysts has been reported with adequate results at times. Some authors have reported recurrence rates up to 75 %, and while a safe technique, the authors do not recommend this as a disease-modifying procedure (Hashimoto et al. 1994; Piatt et al. 2002).

All patients that present on physical examination with visible atrophy to the supraspinatus or infraspinatus should have a minimum time of nonoperative treatment. The authors have found that good results only come with early intervention to alleviate the pain and with release of the suprascapular nerve since this atrophy that has developed is most of the time irreversible especially in young patients (Post 1999).

While many authors believe that a program of physical therapy that concentrates on scapular stabilization, shoulder motion, and strengthening is disease-altering, the authors have realized that this theory is incorrect and only works to sustain a young athlete because his or her serratus anterior or teres group of muscles will support the shoulder and their chronic ache will occur in young athletes in their 20s. Unfortunately, when these same patients return 10 years later, as they have now done for the last 20 years in our practice, they have even more marked atrophy of either fossa and have irreversible muscle damage to the supraspinatus or infraspinatus. The authors believe, therefore, that intervention is essential in an arthroscopic manner to arrest the disease process and allow the athlete or laborer to return to their sport or job in a very short period of “downtime” (Post and Mayer 1987; Callahan et al. 1991). In these advanced and long-standing cases with spinati atrophy that almost never recovers completely, the authors know that though the shoulder pain can improve with cessation of activity, with a resumption of the activity, the pain profile returns. Prior to the arthroscopic approach because of the limited experience of many surgeons and an attitude of hesitancy because of anatomy, the surgeon is not very familiar with the diagnosis of suprascapular neuropathy and it has not been pursued as vigorously. It is the hope of the authors and others writing on this topic that patients will afford the opportunity of an early diagnosis and intervention to make suprascapular compression a disease entity that no longer only sees the clinician but the clinician sees it.

Endoscopic Release of the Spinoglenoid Ligament Understanding Ganglion Cysts and Our Treatment Regime

The arthroscopic technique below and other methods have opened the door for treatment of ganglion cysts in an atraumatic way. Avoiding musculature detachment offers a huge benefit to the patient (Iannotti and Ramsey 1996; Piatt et al. 2002). Much debate though exists whether cyst decompression alone is sufficient or if it is more appropriate to perform cyst decompression and labral debridement and/or labral repair (Youm et al. 2006). Recently, some authors write that they do not decompress the cyst but instead treat the labrum with a repair (Schroder et al. 2008). No literature, including our technique, has a randomized study to show the efficacy of any of these four treatment modalities. This section will discuss the literature and our thoughts for effectively treating a patient with atrophy in the infraspinatus fossa, pain, weakness, and an MRI showing evidence of a ganglion cyst in the spinoglenoid notch and a labral tear.

Advocates for treating intra-articular lesions such as the labral tear believe that if you correct the one-way valve mechanism, the cyst will never return (Pillai et al. 2011). These authors at times just treat the SLAP tear and ignore the cyst as they believe it will decompress itself after correction of all intra-articular pathology. Other authors investigate the type of labral tear present and arthroscopically decompress the cyst, debride the frayed labrum, and repair and stabilize a type

2 SLAP in this young population (Black and Lombardo 1990). If the labrum is intact, these authors have in the past incised the capsule above the labrum just posterior to the biceps to decompress the ganglion cyst. Other authors who used the subacromial method to decompress the ganglion cyst find the raphe between the supraspinatus and infraspinatus which is lateral to the spinoglenoid notch, incise the capsule in this spot, and now proceed with a decompression of the ganglion cyst with an accessory posterolateral portal (Iannotti and Ramsey 1996). It appears from the literature that debridement or repair of the glenoid labrum in most patients with a spinoglenoid ganglion cyst had the best outcome with the lowest recurrence rate (Fehrman et al. 1995; Chochole et al. 1997; Westerheide et al. 2006).

The authors believe direct decompression with a posterior approach is much more efficacious. The authors routinely perform this method and have had only one patient where the pain did not resolve in a multiply-operated worker's compensation case. No recurrence of any cyst occurred in this group. It is acknowledged that every patient in this group has an investigation of any intra-articular pathology, but no one with an intact labrum receives a capsulotomy posterior and superior to the glenoid rim to decompress the stalk of the ganglion cyst. Those authors who proceed with this type of decompression understand that no dissection should proceed beyond 1 cm medial to the superior capsule attachment to the glenoid to avoid the nerve as it course through the spinoglenoid notch. The authors caution surgeons who attempt to decompress a ganglion cyst at the spinoglenoid notch to be wary of this technique to avoid its complications and consider a more direct approach. Complications to the suprascapular nerve can occur, and the average distance to the suprascapular nerve from the posterior glenoid rim is 1.8 cm and the authors have found the motor branches to be approximately 2.0 cm. The authors encouraged patients with a complication of a suprascapular nerve injury and profound external rotation weakness to consider a latissimus dorsi transfer.

The last controversy that exists is the patient treated with labral repair and no cyst decompression. These authors believe that spinoglenoid cyst excision is unnecessary and avoids undue risk of injury to the suprascapular nerve during surgery. Although good results were reported with patients without pain, the authors cannot agree since many patients had a cyst still present on repeat MRI. The presence of a cyst will continue to erode nerve conduction and ultimately irreversible muscular atrophy in the infraspinatus fossa with permanent external rotation weakness.

Recurrence of ganglion cysts with other approaches other than a posterior approach to the spinoglenoid notch has been reported. Hawkins has shown that nonoperative techniques with aspiration lead to an unacceptable recurrence rate with continued compression of the suprascapular nerve (Piatt et al. 2002). Recurrence, as reported by others, of the cyst due to failure of the SLAP repair to heal and inadequate initial resection of the cyst all give credence in our minds for a different approach (Westerheide et al. 2006). Debridement may not be adequate off the glenoid neck for fear, and appropriately so, of injury to the suprascapular nerve as visualization is so difficult. Understanding the appropriate depth of resection when working with such an oblique angle and tight space seems difficult even for the most skilled surgeon. While the cyst when working to decompress with an intra-articular method is known to be located adjacent to the posterior and superior quadrant of the glenoid at the 10:30–11:00 position on a right shoulder and at 2:00–2:30 position on the left shoulder, identification of its exact location by this method is not as simple as it may appear. Blame on the lack of healing power of the patient is also avoided with our posterior approach, as described below, although identification of the recurrence and understanding how to proceed with a road map are essential with the aid of a new MRI if the labrum is found to not heal after repair has been performed.

Rehabilitation is affected with the intra-articular technique as opposed to a posterior approach with no labrum repair. If a concomitant SLAP repair is performed, then the patient must remain in a

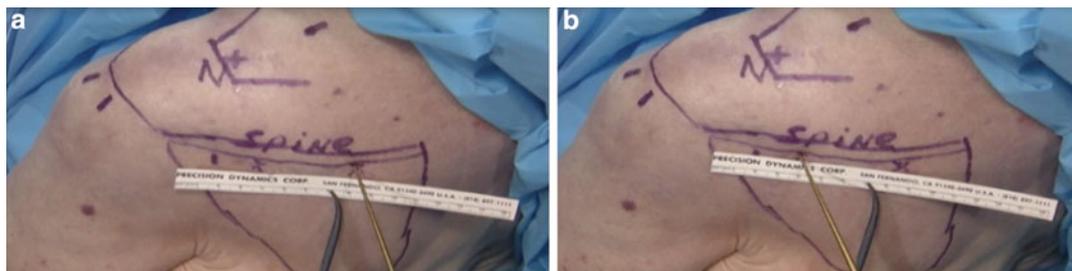


Fig. 33 (a) Clinical photo of a left shoulder posterior view. The gold probe is pointing 8 cm medial to the posterolateral corner of the acromion. This portal is the viewing portal for release of the spinoglenoid ligament compressing the suprascapular nerve at the spinoglenoid notch. (b) Clinical photo of a left shoulder posterior view. The gold probe is pointing 4 cm medial to the posterolateral corner of the acromion. This portal is the working portal for release of the spinoglenoid ligament compressing the suprascapular nerve at the spinoglenoid notch (Copyright K. Plancher)



Fig. 34 Clinical photo of a left, shoulder posterior view. The trocar is introduced in the following fashion. The tip of the blunt trocar palpates the spine of the scapula. The trocar is then moved inferiorly and gently swept to clear a space with the infraspinatus posterior and the tip of the trocar on the infraspinatus fossa. The tip of the trocar is then moved laterally toward the working portal 4 cm medial to the posterolateral corner of the acromion. The trocar as it is moved laterally sweeps the infraspinatus under the arch of its fossa to create a path for the arthroscope to allow visualization of the spinoglenoid ligament (Copyright K. Plancher)

sling for 3–4 weeks. If no SLAP repair is performed, then a sling is utilized 7 days with the patient commencing progressive range of motion exercises and strengthening with return to full overhead activities by 6 weeks. While understanding if labral repair is necessary or if isolated cyst decompression will resolve all symptoms for the patient with suprascapular nerve compression, only time will tell with future studies and meticulous follow-up.

Technique

Arthroscopic release of the suprascapular nerve at the spinoglenoid notch should be approached from the posterior shoulder. The authors utilize a posteromedial and posterolateral portal in the infraspinatus fossa (Fig. 33a, b). Others have utilized a different approach when releasing the spinoglenoid ligament as they prefer subacromial approach (Ghodadra et al. 2009). The ability to visualize anatomy and return to sport or activity of daily living is much faster and simpler than proceeding with the open technique in our opinion. The morbidity and postoperative recovery are much simpler and more pleasant for the patient as well.

The patient is placed in the beach chair position with the arm placed at its side. It is essential to prep and drape from the midsternum to the mid-posterior spine with the complete scapula included. The authors encourage the anesthesiologist to maintain a systolic blood pressure slightly below 100 mmHg. Our pump pressure is kept low at 45 mmHg to avoid unnecessary swelling.



Fig. 35 (a) Clinical photo of a left shoulder, posterior view. The 30° arthroscope is introduced into the viewing portal located 8 cm medial to the posterolateral corner of the acromion. Note the anesthesiologist is instructed to maintain a systolic blood pressure no higher than 100 mmHg mindful of the patient's health if this is not possible. We have always release the spinoglenoid ligament prior to proceeding with any intra-articular work or if needed any release of the transverse scapular ligament to avoid any undue swelling that will make this procedure more difficult. (b) Clinical photo of a left shoulder, posterior view, with the spinoglenoid portals marked out (SG). The arthroscope is in the standard posterior portal for intra-articular glenohumeral joint inspection. Note the relationship of the normal posterior portal to the spinoglenoid ligament portals. "X" represents Nevaizer's portal (Copyright K. Plancher)

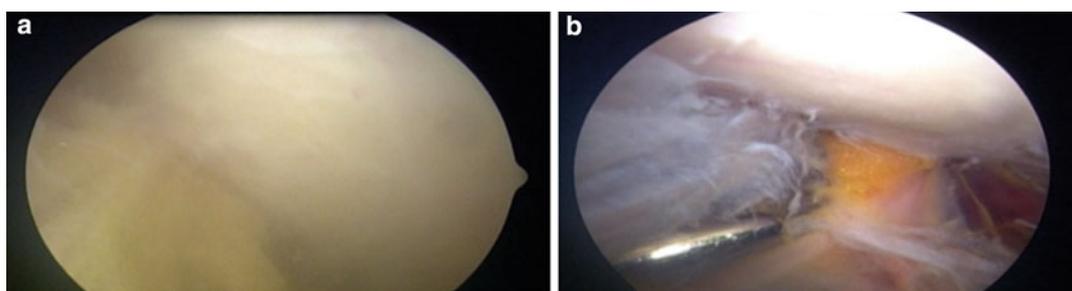


Fig. 36 (a) Arthroscopic picture of the same left shoulder after initial sweeping of the soft tissue away to expose the adipose around the spinoglenoid ligament. Clarity of the pictures occurs once the water is turned on. (b) Intraoperative photo of the same left shoulder showing perineural fat with trocar teasing the spinoglenoid ligament off the suprascapular nerve. The white above represents the spine of the scapula. The glenohumeral joint would be off to the left (Copyright K. Plancher)

The portals selected include two portals: (1) the viewing portal which is placed 8 cm medial to the posterolateral corner of the acromion just inferior to the scapular spine and (2) the working portal which is placed 4 cm medial to the posterolateral corner of the acromion just inferior to the scapular spine (Fig. 33a, b).

Release of the spinoglenoid ligament precedes any work done within the glenohumeral joint. The authors recommend that this part of the procedure should take no more than 5 min to ensure a limited amount of swelling to occur in the limb.

The blunt trocar is introduced into the viewing portal and heads straight toward the infraspinatus fossa (Fig. 34). The tissue under the spine of the scapula is swept away, and the trocar heads to the working portal passing the suprascapular nerve heading and falling into the spinoglenoid notch. The key to this step which allows for visualization is to ensure that the trocar sweeps under the roof of the infraspinatus spine feeling the curvature.

The arthroscope replaces the trocar and our first view of the spinoglenoid ligament is visualized (Fig. 35a, b). Identification of the various landmarks is completed. Success with this procedure will

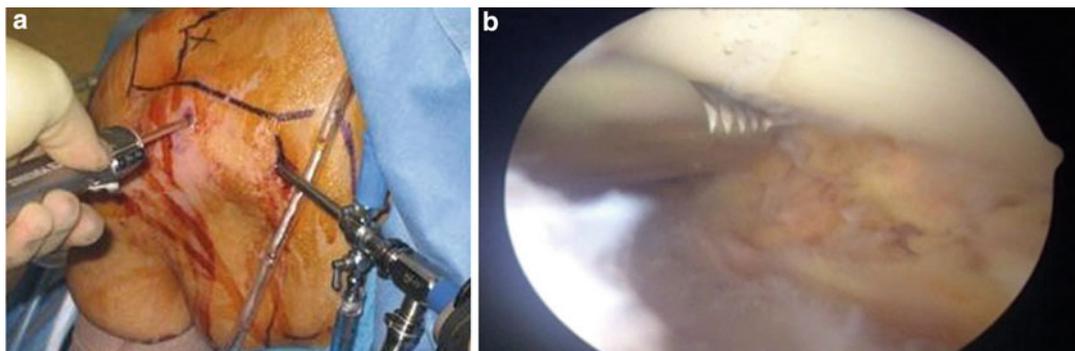


Fig. 37 (a) The arthroscope and shaver are now moved into the appropriate spinoglenoid portals for decompression of the suprascapular nerve at the spinoglenoid notch. (b) Intraoperative photo of the same left shoulder, posterior view. The spine of the scapula is above. The shaver is taking the spinoglenoid ligament directly off the spine of the scapula. All work is being completed lateral to the suprascapular nerve. No different than resecting the ligamentum mucosa/infrapatellar plica in a knee, all work is done on the bone or the notch (the knee), thereby safely avoiding injury to the nerve anterior and medially (Copyright K. Plancher)

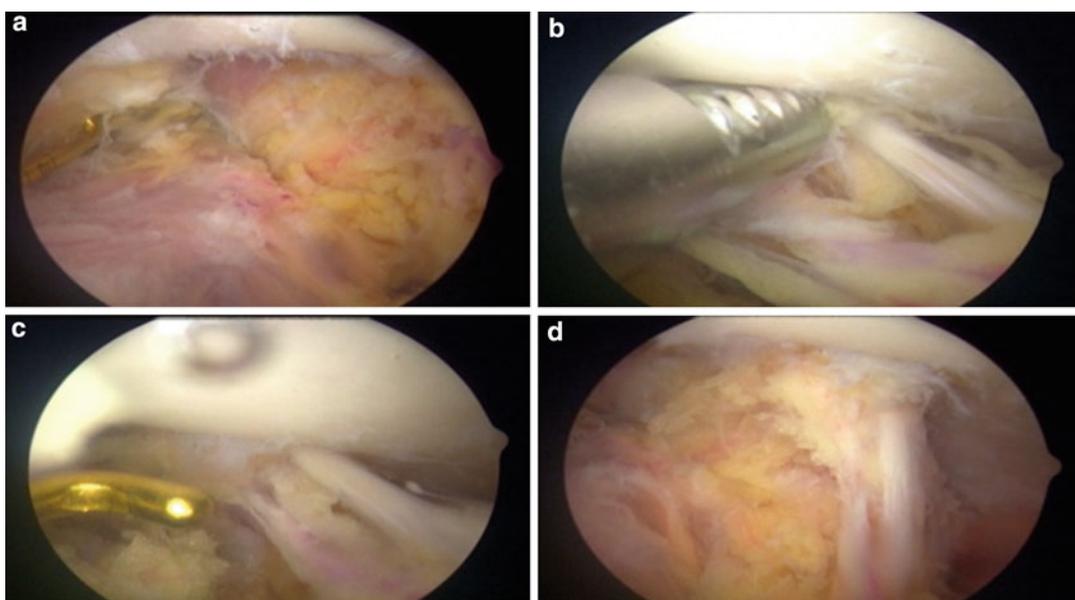


Fig. 38 Intraoperative photo of the same left shoulder, posterior view. The spine of the scapula is above (*white*). (a) The probe is teasing the spinoglenoid ligament off of the glenohumeral attachment laterally. The suprascapular nerve will reveal itself in the perineural fat with blunt dissection. (b) The dull trocar has been used to tease the tissue and expose the suprascapular nerve seen at the tip of the shaver moving obliquely to the right. (c) In this arthroscopic view, the suprascapular nerve is clearly seen off to the right and the slightly anterior to the nerve is the suprascapular artery. The *gold* probe on the left is being used to tease any remaining remnants of the spinoglenoid ligament or the tissues compressing the suprascapular nerve. (d) The suprascapular nerve is now freed and fully mobile as it exits the spinoglenoid notch to move medially now that it has been decompressed (Copyright K. Plancher)

occur with visualization of the spine of the scapula to be maintained throughout the release of the ligament and decompression of the nerve.

The trocar is now introduced into the working portal, and the soft tissue is teased away laterally as the course of the nerve can always be located in the medial side of the spinoglenoid notch (Fig. 36a,

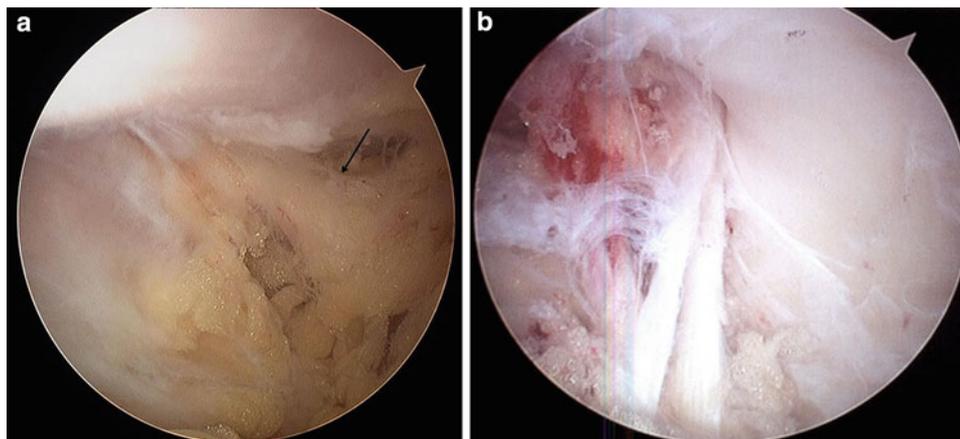


Fig. 39 (a) Arthroscopic view of the left shoulder, posterior view, with the *arrow* pointing to the suprascapular nerve heading medially. Note the bulging tissue to the left, representing a ganglion cyst not yet decompressed. The spine of the scapular (*white*) is above. (b) Arthroscopic view of a left shoulder, posterior view. Note the relationship of the suprascapular nerve as it always hugs tightly the suprascapular notch. This suprascapular nerve represents an anomaly which is yet to be described because of its bifid nature. The nerve branches will head medially toward the right. Arthroscopic decompression of the spinoglenoid ligament can be safely performed by staying lateral to the nerve which is fixed in position in the spinoglenoid notch (Copyright K. Plancher)



Fig. 40 Intraoperative photo of the same left shoulder demonstrating the most medial aspect of the spinoglenoid notch. This is the danger zone as the suprascapular nerve will always hug the most medial aspect of the notch as it heads midline giving off two to four muscular branches to the infraspinatus. Note the spine of the scapula up above (*white*). Note the curvature of the infraspinatus fossa seen to the right of the perineural fat surrounding the suprascapular nerve (Copyright K. Plancher)

b). A radiofrequency wand of small radius nonaggressive shaver with the suction turned off can be utilized at this point to clear the tissue and more specifically the spinoglenoid ligament (Fig. 37a, b). The ligament can be resected by staying on the spine of the scapula to avoid any bleeding. The ligament can be followed to the glenohumeral joint at its insertion to understand and visualize the complete resection of the ligament.

The blunt tip trocar is utilized now to assess the mobility and adequate release of the suprascapular nerve (Fig. 38a–d). Then the spinoglenoid notch is inspected to note any aberrations in anatomy such as a ganglion cyst or a bifid nerve that now may be compressing the suprascapular nerve (Fig. 39a, b). Decompression of the ganglion and excision of the stalk can now be easily completed. It is important to understand that the ganglion root may be heading toward the posterior inferior quadrant of the glenohumeral joint. Observation of the released suprascapular nerve with the artery can now be seen hugging tightly as it wraps around the notch and heads medially giving its two to

four muscular branches to the infraspinatus (Fig. 40). Upon completion and full inspection, the equipment is removed from the body and the portals are closed in routine fashion. The patient should wear a sling for 7 days for comfort to start. Thereafter, all activities can be resumed but are dependent on any other work that may have been performed to this same shoulder.

Our experience with this technique has been successful when a patient has failed conservative treatment, has EMG-proven compression, and has visual atrophy in the infraspinatus fossa. The patient's pain profile the next day after release is verbalized as completely gone, and while the authors have not been successful in re-insufflating the muscle belly, they have in those whose disease has not been present for more than 2 years restored some measurable strength to external rotation. This technique is safe and effective as it approaches the anatomy directly without taking down any nonessential or essential muscular planes. The authors have also used this approach successfully in the last 20 patients who did not exhibit any infraspinatus wasting but had a chronic ache and a positive adduction test on physical exam with immediate success and return to overhead sport and activities of daily living.

Outcomes

Literature on this topic is not plentiful. There are very few series with long-term follow-up including the author's series. The authors have waited for at least a 3-year average follow-up before reporting to ensure accuracy that the ganglion cyst has not returned and that the athlete or laborer has in fact returned to all activities without pain. Understanding what to do with chronic atrophy is a difficult issue which at this time has no perfect answer.

Warren et al. reviewed, as discussed above, their results with nonoperative treatment. They recommended that if no ganglion cyst or soft tissue mass was present and no compression of the suprascapular nerve was detected, then no intervention should proceed. This chapter did not focus though on the spinoglenoid notch solely (Martin et al. 1997). Post reported on open surgical decompression without evaluation of the labrum and felt he could expect excellent or good results in 88 % of the patients (Post and Grinblat 1993). Fehrman reported in a small series after nonoperative treatment great success with complete pain relief with intervention both in the intra-articular lesions combined with an open resection of the ganglion (Fehrman et al. 1995). Chen in one report and Lichtenberg in another both reported on a small series with repair of a SLAP and excision of the ganglia in an arthroscopic approach (Lichtenberg et al. 2004; Chen et al. 2006). All patients in both series had complete pain relief and improvement in strength and excellent function at their reported follow-up.

The last group of labral repair alone without decompression of the cyst is discussed above with the study of Schroder (Schroder et al. 2008). Curiously, there is a case report of a debridement of a labral tear with radiographic evidence of resolution of a spinoglenoid notch cyst and reinnervation shown by EMG after this procedure (Chochole et al. 1997). The most recent reports are yet to come from our group with direct posterior decompression and others with nerve decompression performed arthroscopically with limited follow-up, data, although as presented in many meetings across the globe, the results are very promising.

Conclusion

Compression of the suprascapular nerve at the spinoglenoid ligament is a disease of a young overhead laborer or avid athlete. This chapter will hopefully make the reader aware of its existence as this disease entity has seen clinicians but clinicians have not seen it readily because of its less

frequent appearance. The patients' complaints can often be confused with rotator cuff disease, but by following the guidelines written above, it is the hope that all physicians will identify the disease and perhaps consider after practicing in a learning environment how to endoscopically release the ligament and decompress the suprascapular nerve to return the patient to all activities in a short period of time.

Cross-References

- ▶ [Anatomy and Portals in Shoulder Arthroscopy](#)
- ▶ [Arthroscopic Repair of Rotator Cuff Disorders](#)
- ▶ [Nerve Decompressions: Principles and Different Techniques](#)
- ▶ [Pathogenesis of Rotator Cuff Tears, Implications on Treatment](#)
- ▶ [Rehabilitation and Return to Sports After Conservative and Surgical Treatment of Shoulder Injuries](#)

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