Introduction

This article includes discussion of suprascapular neuropathy, suprascapular nerve injury, suprascapular mononeuropathy, suprascapular nerve entrapment, and infraspinatus muscle atrophy. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

Suprascapular neuropathy is an underrecognized cause of shoulder girdle weakness and pain. The clinical and electrophysiologic techniques of diagnosis are easily learned. Both nonoperative and operative treatments are successful when properly chosen. Advances in correlation of nerve conduction study results with treatment choice and outcomes provide a motor-based prognostic model for suprascapular neuropathy. In this update, new data regarding diagnostic modalities, etiologies, and treatments of suprascapular neuropathy are summarized.

Key points

• Suprascapular neuropathy is 1 of the causes that should be considered in patients with shoulder pain.
• Suprascapular neuropathy could be common in some overhead sport players, especially volleyball.
• The gold standard for the diagnosis of suprascapular neuropathy is electrodiagnostic testing.

Historical note and terminology

Suprascapular neuropathy connotes injury to the suprascapular nerve anywhere in its course, from the origin at the brachial plexus to its termination in the infraspinatus muscle. Despite its relatively low prevalence, it must be kept in mind as a potential cause of shoulder pain, particularly in patients where the history, physical examination, and imaging studies do not adequately explain a patient's symptoms or disability (Moen et al 2012).

Suprascapular neuropathy is a more common cause of shoulder pain and weakness than is generally believed (Rengachary et al 1979a; Vastamaki and Goransson 1993). Though over 300 cases have been published, primarily in the surgical literature, this entity may often be clinically overlooked. Suprascapular neuropathy “deserves more than a modest place in the rare diagnoses” of upper extremity nerve injuries (Mestdagh et al 1981).

Shoulder girdle neuropathy was reported in 1879 (Joffroy 1879), but in 1909, isolated “neuritis” of the suprascapular nerve was described (Ewald 1909). In 1926, 16 cases of suprascapular neuropathy were published in a review of war injuries, and an additional case was discussed in 1936 (Foster 1926; Thomas 1936).

In Parsonage and Turner's classic publication of Neuralgic Amyotrophy in 1948, 4 cases of isolated suprascapular neuropathy are described (Parsonage and Turner 1948).

In the late 1950s, Kopell and Thompson were the first to define a suprascapular neuropathy syndrome. In a series of publications, they defined pathophysiology, clinical characteristics, and treatment approaches for suprascapular nerve entrapment at the suprascapular notch (Kopell and Thompson 1959; Kopell and Thompson 1963; Thompson and Kopell 1959).

Gassel was the first to publish nerve conduction study techniques for the suprascapular nerve, and Kraft further defined these methods (Gassel 1964; Kraft 1972). A report of isolated infraspinatus involvement in suprascapular
neuropathy was described in 1982 (Aiello et al 1982). Increasing attention to the importance of the various presentations and treatments of suprascapular neuropathy has continued to the present.

**Clinical manifestations**

**Presentation and course**

The most common presenting symptom of suprascapular neuropathy is shoulder pain. The pain is most often localized to the posterolateral shoulder girdle, though occasionally it will be described as radiating medially upward to the neck or laterally to the upper arm. Most often, the pain is described as a deep, diffuse, and dull aching. On some occasions, complaints of burning or crushing overlay to the pain are made (Post and Mayer 1987). The pain sensations are likely referred from sensory articular branches of the suprascapular nerve to the shoulder joints (Ferrick and Marzo 1999).

Often, a history of shoulder trauma or overuse is present. It is rare for the pain complaint to be fulminating, but rather the pain is gradually progressive. Often the symptoms are worsened by movements of the shoulder girdle and may eventually become more constant and severe, interrupting sleep (Vastamaki and Goransson 1993).

In those with suprascapular neuropathy involving only the infraspinatus, complaints of pain are often lacking. This is likely due to the sensory articular branches having already diverged before the motor branch to the infraspinatus (Ferretti et al 1987).

Weakness or muscle loss may also be a prominent presenting complaint in suprascapular neuropathy. The weakness is most often in external rotation (infraspinatus muscle) and in initiation of abduction of the upper extremity at the shoulder girdle (supraspinatus muscle). The pattern of weakness most often described to the clinician is difficulty with overhead use of the arm (Rengachary et al 1979a). Visible muscle loss is usually most apparent in the infraspinatus and occasionally in the supraspinatus, with the latter more difficult to visualize because of the overlying trapezius.

**Prognosis and complications**

A retrospective review of pretreatment nerve conduction study findings revealed patterns that may help frame operative versus nonoperative treatment recommendations. Thirty-four patients were interviewed at least 6 months after completing treatment for suprascapular neuropathy by 1 physician. The patients graded their motor outcomes as poor, fair, good, or excellent. Sixteen of 18 patients treated nonoperatively graded their outcome as good or excellent; 15 of 16 patients treated operatively reported good or excellent outcomes. Twelve of the 16 nonoperative good/excellent outcome group had pretreatment nerve conduction study with normal latencies but more than 50% compound motor action potential amplitude reduction when compared to the nonaffected side (p=0.005). Eleven of the 15 operative good/excellent outcome group had pretreatment nerve conduction study with prolonged latencies (greater than 1.5 millisecond) but less than 50% amplitude decrement when compared to the nonaffected side (p=0.004). Pretreatment EMG findings did not reveal statistically significant prognostic indicators per se (Smith 2004).

Though self-assessment measures do not necessarily connote highly accurate or consistently valid prognostic predictors, the outlined findings prompt further consideration.

When suprascapular neuropathy is not recognized or treated, the course is prolonged and disabling in terms of shoulder pain and weakness (Martin et al 1997). An investigation using an in vitro model of suprascapular neuropathy revealed substantial glenohumeral impairments and lingering functional shoulder deficits (Luo et al 2002). Unrecognized or untreated suprascapular neuropathy may lead to altered shoulder girdle biomechanics and secondary shoulder pathology (ie, rotator cuff dysfunction).

A review of all prior outcome reports showed that 77% of surgically treated suprascapular neuropathy patients and 92% of nonsurgically treated suprascapular neuropathy patients had good to excellent outcomes (Ferrick and Marzo 1999). The authors are quick to point out that nonoperative treatment should not be considered necessarily "better," in that most patients who were treated surgically had failed a prior course of nonoperative treatment. A review of 53 patients revealed best outcomes with surgical management of suprascapular neuropathy caused by compressive etiologies or cysts. Overuse injuries did not improve with operative treatment. Traction and direct closed traumatic injuries responded equally to surgical or nonsurgical management, but overall had worse final outcomes (Antoniou et al 2001). Almost all patients who are diagnosed with suprascapular neuropathy and treated surgically or nonsurgically with good-to-excellent results and return to full activity, including professional competition levels (Treihaft 2000; Dramis and Pimpalnerkar 2005).
Clinical vignette

A 20-year-old, left-handed woman presented with complaints of left shoulder pain and weakness of 1-year duration. In her training and competition as a Division I National Collegiate Athletic Association swimmer, she had gradually developed left posterolateral shoulder girdle deep aching, particularly noticeable to her during freestyle and backstroke maneuvers. Over the past several months, comments were made to her by teammates that she had "less muscle" over the posterior left shoulder girdle. Previous evaluation and treatment by team physicians and trainers resulted in no clear diagnosis and no sustained relief. The remainder of her history was otherwise noncontributory.

Physical examination revealed abnormalities only in the left shoulder girdle. There was marked weakness in external rotation of the left upper extremity at the shoulder. Despite apparently normal contraction of the deltoid, weakness was also noted on the left of initiation of abduction. A suggestion of loss of muscle mass was seen of the left infraspinatus. No abnormal involuntary movements or fasciculations were observed.

Previously completed roentgenograms and MRI with contrast of the shoulder and cervical spine were normal.

EMG of the left upper extremity, shoulder girdle, and cervical paraspinous muscles revealed positive sharp waves and fibrillations as well as decreased recruitment pattern in only the left supraspinatus and infraspinatus.

Nerve conduction studies were completed using surface stimulation at Erb point along with recording with surface and concentric needle electrodes over and in the supraspinatus and infraspinatus. On the left, latency to supraspinatus was 6.4 milliseconds compared to 2.3 milliseconds on the right. Latency to the left infraspinatus was 8.7 milliseconds compared to 3.2 milliseconds on the right. Compound motor action potential amplitudes on the left were 55% and 65% lower on the left compared to the right. The remainder of the nerve conduction studies was normal and symmetric.

Because the patient had already had treatment with rest, anti-inflammatory and analgesic medications, and extensive physical therapy for over 6 months without benefit, she was referred for surgical consultation. Surgery was completed with posterior approach section of the superior transverse scapular ligament at the suprascapular notch. She described immediate relief of pain following surgery.

She was seen 10 months postoperatively. She was asymptomatic. Strength on examination had returned to normal, symmetric with the right. Muscle bulk had improved substantially, with only subtle reduction of infraspinatus muscle mass compared to the right. EMG was normal. Latency to the left supraspinatus was 3.0 milliseconds (2.6 milliseconds on the right). Latency to the left infraspinatus was 4.0 milliseconds (3.7 milliseconds on the right). Amplitudes were 5% to 10% lower on the left compared to the right.

She returned to normal activities without restriction. She resumed training and competition as a collegiate swimmer and had considerable success.

She was last seen 2.5 years after surgery, and has remained asymptomatic.

Biological basis

Etiology and pathogenesis

Suprascapular neuropathy can be classified as traumatic (including repetitive micro-trauma) and non-traumatic. As with most peripheral nerve injuries, direct trauma or repetitive overuse may contribute to suprascapular neuropathy. Direct trauma may occur anywhere along the course of the suprascapular nerve, from the origin to the termination (Weaver 1983). The suprascapular nerve may be indirectly traumatized, such as in association with proximal humeral fractures (Visser et al 2001). Overuse may result in entrapment at either the suprascapular or spinoglenoid notches (Ferrick and Marzo 1999). The classic example of micro-traumatic pathology is the suprascapular neuropathy found in overhead sports, like volleyball (Lajtai et al 2009). It can also be found in other sports like baseball (Cummins and Schneider 2008). The same mechanism was blamed in a case of a paraplegic patient who developed suprascapular nerve entrapment (Facione et al 2011).

Both hereditary shoulder girdle neuropathy and hereditary neuropathy with liability to pressure palsies have been genetically mapped to chromosome 17 (Weaver and Kraft 2001). The specific genetic relationship with suprascapular neuropathy has yet to be determined. Non-traumatic causes include notch or ligamental anomalies, compression, and
neuralgic amyotrophy.

Rengachary and colleagues reported 6 different types of anatomical variations of the suprascapular notch (Rengachary et al 1979). They are:

- type I: no discrete SSN
- type II: SSN has a greater transverse diameter than vertical diameter
- type III: SSN has a greater vertical diameter than transverse diameter
- type IV: SSN with a completely formed bony suprascapular foramen
- type V: SSN with foramen and incisura

In 268 human scapulae studied in India, type II suprascapular neuropathy was the most common (50%), followed by type I, type IV, and type III (32.46%, 9.7%, and 7.84%, respectively). It was observed that type II suprascapular neuropathy has a lower chance of suprascapular nerve entrapment compared to the other types of suprascapular neuropathy (ie, types III, IV, and V) (Kumar et al 2014). Furthermore, in another study Dr. Yamakado showed that narrowing of the scapular notch was related to aging (Yamakado 2016). The study included 760 patients who underwent shoulder surgery. A 3D-CT scan of the shoulder was performed to evaluate the shape of the notch according to the Rengachary classification. The 6 types of Rengachary classification were rearranged into 3 major categories according to ossification of transverse scapular ligament and notch size. The 3 categories included wide notch (type 1 and type 2); the narrow notch (type 3 and type 4); and the ossified notch (type 5 and type 6). The categories were compared with a 1-way analysis of variance. There was statistically significant difference among the 3 categories. The narrow notch group (age: 63.4 ± 12.8 years) and the ossified notch group (age: 65.9 ± 10.6 years) were significantly older than the wide notch group (age: 57.5 ± 17.8 years) (Yamakado 2016). Hence, these studies show that narrowing of suprascapular notch occurs with aging and suprascapular neuropathy is more common in narrower suprascapular notch ie, in Rengachary type III, IV, and V.

In some cases, the variation in the suprascapular notch is accompanied by a variation of the superior transverse scapular ligament. These variations have a role to play in suprascapular nerve entrapment.

Compression, which is more common at the spinoglenoid notch, can be caused by cysts, neoplasms, and other lesions. Ganglionic cyst is a classic example, and it is usually associated with posterior capsulolabral injury (Fehrman et al 1995). Cogar and colleagues have also reported a rare anatomical variation—the subclavius posticus muscle, coursing from the rib posterolaterally to the superior border of the scapula, as a cause of compressive neuropathy of suprascapular nerve (Cogar et al 2015).

That compression at spinoglenoid notch can cause more atrophy of the infraspinatus than supraspinatus muscle was reported by Kong and colleagues (Kong et al 2016). They reported that in patients with massive rotator cuff tears, suprascapular neuropathy induced more pronounced denervation and fatty degeneration of infraspinatus muscle compared to supraspinatus muscle, partly due to the entrapment of the suprascapular nerve at the spinoglenoid notch, which is distal to the point from where the branch to the supraspinatus muscle arises (Kong et al 2016).

Le Hanneur and colleagues presented a unique case of suprascapular nerve palsy lasting for 2 years, initially considered idiopathic (Le Hanneur et al 2015). This was found to be caused by partial section of the suprascapular nerve by transverse scapular ligament during exploratory surgery. This case highlights the importance of early surgical management with an open procedure for suprascapular neuropathy of unknown etiology. According to the authors, waiting is unjustified and potentially could worsen the nerve injury by progressive strangulation of the nerve by superior transverse scapular ligament (Le Hanneur et al 2015).

In neuralgic amyotrophy or acute brachial neuritis, the suprascapular nerve was found to be involved in 97% of the cases studied by MRI (Gaskin and Helms 2006). The axillary nerve was involved in 50% of the cases.

The suprascapular nerve is a mixed sensory-motor nerve that arises from the upper trunk of brachial plexus formed by the roots of C-5 and C-6 at Erb point. In approximately 50% of people, contributing fibers are from C-4; rarely, the suprascapular nerve arises directly from the C-5 motor root (Hadley et al 1986).

The suprascapular nerve then courses laterally and downward deep to the omohyoid and trapezius muscles to the suprascapular notch. It then passes through the notch below the superior transverse scapular ligament (suprascapular ligament), when concurrently the suprascapular artery and vein pass over the ligament. In a cadaveric study of 812 specimen, ossification of the superior transverse scapular ligament was explored, and band-shaped type ossification
was suggested as a potential cause of neuropathy, as it was associated with reduced cavity size below the ligament (Polguj et al 2014). In 1 cadaver anatomical study, it was found that in 3% of the suprascapular nerves, the motor nerve to the supraspinatus branched over the suprascapular ligament, whereas the motor branch to the infraspinatus passed under the ligament (Warner et al 1992). Podgorski and colleagues conducted a cadaveric dissection study on 60 shoulders to evaluate the morphological patterns of suprascapular notch vein that occurs in addition to suprascapular vein, suggesting increased diameters as a potential etiology of nerve compression (Podgorski et al 2014).

In another study Podgórski and colleagues showed that suprascapular nerve injury can also be caused by the anterior coracoscapular ligament (ACSL) which extends below the superior transverse ligament (STSL) on the anterior side of the suprascapular notch (Podgórski et al 2015). The presence of this ligament can reduce the available space for the suprascapular nerve, causing compression and injury to the nerve. Podgórski and colleagues dissected SSN in 100 formalin-fixed, cadaveric shoulders. The ACSL was present in 52 scapulae, and in all cases the suprascapular nerve travelled superior to the ACSL. Mechanically relevant types of ACSL were found to occur significantly more often when deeper notches were present. It was concluded that mechanically efficient ACSL is more common in deep and narrow SSNs, which are associated with suprascapular neuropathy (Podgórski et al 2015). The role of ACSL is still ambiguous in the pathogenesis of suprascapular neuropathy because on one hand, it seems to narrow the opening for the suprascapular nerve, potentially increasing the risk of neuropathy (Avery et al 2002; Bayramoğlu et al 2003). On the other hand, it can provide a mechanical barrier from the subscapular muscle, protecting the nerve from compression against the STSL. It can also act as support for the nerve and protect it against hypermobility and microtrauma.

Once through the notch, the suprascapular nerve runs obliquely through the supraspinatus (supraspinous) fossa, and 2 or more motor branches penetrate the supraspinatus muscle. Isolated motor branch injury to supraspinatus muscle is extremely rare, but has been reported (Alomar et al 2011). Sensory articular branches arise to the glenohumeral and acromioclavicular joints (Mestdagh et al 1981). The sensory branch of the suprascapular nerve runs superiorly to the supraspinatus muscle towards the acromioclavicular joint (Ebraheim et al 2011). With 1 exception (Horiguchi 1980), all reports agree that the only sensory function of the suprascapular nerve is the shoulder articular sensory, with no cutaneous sensory function.

The suprascapular nerve then enters the infraspinatus (infraspinous) fossa by crossing the base of the suprascapular spine and the spinoglenoid notch. It then terminates either in arborization of fine motor branches to the infraspinatus or in 2 to 4 distinct motor penetrating branches to the infraspinatus (Warner et al 1992).

Disagreement exists regarding the presence or absence of the inferior transverse scapular ligament (spinoglenoid ligament) and its relationship to possible entrapment of the suprascapular nerve motor branch to the infraspinatus. Estimates of the presence of a spinoglenoid ligament range as low as 3% in men and women (Demaio et al 1991) to as high as 50% in women and 87% in men (Kaspi et al 1988). Additional studies indicate that the ligament is present in 36% of women and 64% of men, and the midligament width is greater in men (Demirhan et al 1998). These 2 attributes may contribute to isolated infraspinatus suprascapular neuropathy occurring more frequently in men than in women.

To understand the susceptibility of the suprascapular nerve to injury, in comparative anatomical evolutionary terms, humans have sacrificed stability in the shoulder girdle for mobility of the upper extremity at the shoulder (Clein 1975). "Shoulder joint" is a misnomer in that the shoulder girdle includes 4 joints: (1) acromioclavicular, (2) sternoclavicular, (3) scapulothoracic, and (4) glenohumeral. The muscles acting across these joints act as “force-couples” synchronously in order to help compensate for the instability (Inman et al 1944).

In a series of studies, Rengachary and colleagues have demonstrated that due to the relative fixation of the suprascapular nerve at its origin at the brachial plexus and termination in the infraspinatus, the nerve is particularly susceptible to angulation at the suprascapular notch. This results in a "sling" effect of entrapment of the suprascapular nerve at the suprascapular notch (Rengachary et al 1979b). Following reports of isolated infraspinatus involvement, entrapment of the suprascapular nerve at the spinoglenoid notch has also been postulated, perhaps even in association with excessive shoulder mobility (Witvrouw et al 2000), spinoglenoid cysts associated with superior labrum anterior posterior tear lesions (Chen et al 2003), ganglion cysts at the spinoglenoid notch (Bouzaidi et al 2005; Lee et al 2007), intraneural suprascapular ganglion cysts dissecting within the epineurium from the glenohumeral joint connections (Spinner et al 2006), or varicose veins at the spinoglenoidal notch (Van Meir et al 2011).
Further sites of entrapment that have been proposed include: (1) the level of the upper trunk of the brachial plexus in scalene muscle fascia, (2) in the fascia of the subclavius and omohyoid muscle, and (3) between the coracoid process and supraspinatus muscle. The suprascapular nerve may also be injured in full thickness rotator cuff tears (Vad et al 2003; Mallon et al 2006; Costouros et al 2007) and distal clavicle fractures (Huang et al 2005). The association between rotator cuff tears and suprascapular neuropathy, however, has remained debatable. In a review of 49 shoulders with massive rotator cuff tears, Collin and colleagues revealed low chance of association of suprascapular neuropathy with rotator cuff tears (Collin et al 2014). This showed redundancy of performing suprascapular nerve release when rotator cuff tears repair is performed. In a review of over 150,000 Finnish military conscripts focusing on backpack shoulder compression, suprascapular neuropathy was found in 7, which is approximately one third the incidence of the more commonly expected long thoracic neuropathy in backpack carriers (Makela et al 2006). Scapular winging may impose traction on the soft tissues of the shoulder region, including the suprascapular nerve (Flores 2008), which could contribute to shoulder pain in these patients.

Iatrogenic injury and suprascapular neuropathy was reported after repair of labral tears (Chan et al 2010), repair of massive rotator cuff tears (Zanotti et al 1997), arthroscopic repair of a SLAP (superior labral tear from anterior to posterior) (Kim et al 2010), arthroscopic decompression of paralabral cyst around suprascapular notch (Gupta et al 2015), and other procedures.

Bilateral suprascapular neuropathy was described following coronary artery bypass surgery, surmised to be due to surgical positioning (Hassan et al 2006). Finally, possible ischemic injury to the suprascapular nerve may occur from microemboli, originating from intimal damage in the axillary or suprascapular arteries, becoming trapped in the vasa nervorum of the suprascapular nerve (Ringel et al 1990).

**Epidemiology**

No rigorous epidemiological studies are available to define precise incidence and prevalence. The 2 case review publications that address suprascapular neuropathy incidence indicate it comprises 0.4% (Post and Mayer 1987) and 1% to 2% (Vastamaki and Goransson 1993) of all shoulder disorders. This prevalence can be as high as 33% in volleyball players according to 1 report (Holzgraefe et al 1994).

**Prevention**

A clinical strategy that may reduce likelihood of suprascapular neuropathy is inculcating proper training and technique in amateur and professional athletes in whom shoulder motions are key (eg, baseball, volleyball, and weightlifting). Educating those at risk (ie, athletes or those whose occupations involve shoulder motion) regarding avoidance of symptom-provoking motions and pursuing early evaluation and treatment at the first sign of symptoms may be of value, as would avoiding excessive loading or carrying on the shoulder (Karatas and Gogus 2003). Careful attention to the suprascapular nerve course and entrapment points during shoulder girdle injection, physical therapy, and surgery may potentially reduce injury to the nerve.

**Differential diagnosis**

The most common clinical problem to differentiate from suprascapular neuropathy is rotator cuff dysfunction.

Suprascapular neuropathy and rotator cuff disorders appear similar in terms of symptoms and signs at first glance; however, certain findings help differentiate the two. Pain in rotator cuff problems is more often anterolateral; pain in suprascapular neuropathy is posterolateral. Those with rotator cuff problems often have past history of recurrent shoulder complaints and are usually older; those with suprascapular neuropathy are often younger and rarely have past history of recurrent shoulder problems. At examination, impingement sign is often present in rotator cuff dysfunction but absent in suprascapular neuropathy. Pain with resisted abduction or external rotation is frequent in rotator cuff disorders but rare with suprascapular neuropathy (Jackson et al 1995). Electrodiagnostic evaluation and MRI can be helpful in differentiating suprascapular neuropathy and rotator cuff disorders.

Suprascapular neuropathy must also be differentiated from cervical radiculopathy. The more extensive dermatomal and myotomal patterns of complaints and findings in radiculopathy often distinguish this entity readily from suprascapular neuropathy. The same is true in those with brachial plexus problems or more diffuse peripheral polyneuropathy or muscle disease. Electrodiagnostic evaluation can distinguish these entities.
Additional shoulder girdle pathologies that may mimic symptomatology seen with suprascapular neuropathy include acromioclavicular or glenohumeral joint disease, adhesive capsulitis, biceps tendonitis, bursitis, and Pancoast tumor (Martin et al 1997). Roentgenographic or MR imaging may often help in these cases.

**Diagnostic workup**

There is no consensus on diagnostic criteria. Suprascapular neuropathy can be diagnosed by electrophysiologic testing in a subset of patients with specific clinical and radiographic findings suggestive of the pathology (Boykin et al 2011).

Thorough history and examination are the key first steps in evaluating suprascapular neuropathy. Evaluating history of shoulder trauma or repetitive overuse resulting in posterolateral shoulder girdle pain is an initial indicator raising suspicion of suprascapular neuropathy. Clinical examination findings of weakness of external rotation and initiation of abduction should point to the possibility of suprascapular neuropathy. Cross arm adduction of the affected extremity may increase suprascapular neuropathy pain. Tenderness over the suprascapular notch may indicate suprascapular neuropathy (Vastamaki and Goransson 1993).

All too often, atrophy of the infraspinatus and supraspinatus (the latter more difficult to see because of the overlying trapezius) is the reason for referral. Atrophy alone should never be a guide or catalyst; the goal is to recognize and treat suprascapular neuropathy in order to prevent atrophic stages from occurring.

Injection of the area of entrapment with local anesthetic or steroid medication for diagnostic purposes has been useful in many cases of suprascapular neuropathy (Ferrick and Marzo 1999).

Roentgenograms of the shoulder girdle may be helpful if bone or joint pathology is suspected. Standard shoulder girdle views may miss the suprascapular notch; angling the tube caudally helps fully visualize the suprascapular notch (Edeland and Zachrisson 1975). CT scan can show scapular fracture if it is not demonstrated on the plain film (Chan et al 2009).

MRI is useful not only in delineating rotator cuff tears, but also in defining ganglion cysts, tumors, hematomas, Schwannomas, or other clinically unsuspected masses that entrap or injure the suprascapular nerve (Fritz et al 1992; Sharma et al 2001). A retrospective analysis, comparing to "gold standard" EMG results, indicated that muscle edema (high muscle signal on T2-weighted fast spin-echo fat-suppressed MRI images) in the infraspinatus or supraspinatus was the most significant MRI sign of suprascapular neuropathy (Ludig et al 2001).

3T MRN (magnetic resonance neurography) has been proposed as a valuable diagnostic tool in clinically suspected cases of suprascapular neuropathy because it can directly demonstrate the nerve abnormality, as well as secondary muscle denervation changes (Chalian et al 2011). Abnormalities were detected in the suprascapular nerve in 11 of 13 cases, and denervation findings were detected in the supraspinatus or infraspinatus muscles in 12 of 13 cases studied. Currently, MRI muscle changes due to denervation are much more sensitive indicators of suprascapular neuropathy than MRI neurography. Direct visualization and characterization of abnormalities in the nerve by MRI neurography requires further optimization prior to routine clinical use. MRN, however, further helps distinguish brachial plexus, rotator cuff, and cervical spine from suprascapular nerve pathologies by allowing concurrent assessment (Ahlawat et al 2015).

A review of ultrasonography with broadband transducers of greater than 10MHz frequencies and improved near field resolution suggests this technique may also prove to be a useful adjunct in the diagnostic evaluation of suprascapular neuropathy (Martinoli et al 2004).

Electrodiagnostic evaluation is the paramount step in diagnosing suprascapular neuropathy (Treihaft 2000). Electromyography may reveal patterns of abnormality that not only help localize the lesion, but also differentiate from more widespread processes and help to define acuity. Electromyography (EMG) and nerve conduction velocity (NCV) studies can be used for the following:

- Confirm the diagnosis of suprascapular neuropathy in the setting of a suggestive history, physical examination, and imaging studies.
- Test nerve function in a patient with supraspinatus or infraspinatus atrophy, or both, with no identifiable cause.
- Evaluate for neuropathy in a patient without an identified cause of lingering shoulder pain.
- Monitor nerve function before, during, and after the treatment of any of the causes of suprascapular neuropathy.
Nerve conduction studies for the suprascapular nerve were first described in 1964 and further defined in the early 1970s (Gassel 1964; Kraft 1972). Concentric needle electrodes were used to record compound motor action potentials in the infraspinatus and supraspinatus with surface stimulation at Erb point. These and subsequent reports reveal normal latency ranges of 1.7 to 3.7 milliseconds for the supraspinatus and 2.4 to 4.2 milliseconds for the infraspinatus. Normal values for amplitudes were not initially reported. Subsequent reports have argued relative merits of concentric electrode versus monopolar electrode versus surface electrode recording (Horning et al 1972; Liveson et al 1991; Casazza et al 1998). Most agree that depth electrodes are best for determining latencies. Amplitude data, side-to-side comparisons, and recording electrode placement are well described by Casazza and colleagues (Casazza et al 1998). In light of the nerve conduction study results and treatment outcome data noted in the prognosis section, it would be advisable to use surface electrode measurements followed by depth electrode nerve conduction studies to maximize the accuracy of latency and amplitude data. The techniques are simple to perform and easily added to the electrodiagnostician's armamentarium (Kraft 1972). Nonetheless, a study found the use of surface electrodes to be a reasonable option because the normal values were comparable to the values of previous studies that used the gold standard method (surface and depth electrodes) (Buschbacher et al 2009). Side-to-side comparison of compound muscle action potential (CMAP) amplitude and latency is a valid approach to determine unilateral abnormalities. Of note, CMAP amplitude difference of up to 50% and distal latency difference of 20% in side-to-side values can be normal (Buschbacher et al 2009). These findings imply that a difference of more than 50% in CMAP amplitudes can distinguish the affected from the non-affected side.

During needle EMG, both supraspinatus and infraspinatus muscles should be examined to differentiate suprascapular notch lesions (both muscles are abnormal) from spinoglenoid lesions (just the infraspinatus muscle is abnormal) (Liveson et al 1991). The muscles innervated by C5-C6 should also be examined to rule out brachial plexus lesions and radiculopathy. According to 1 study, the sensitivity and specificity of EMG and NCV studies varies from 74% to 91% (Moen et al 2012).

Management

The first approach to suprascapular neuropathy treatment is nonsurgical if no mass lesions are identified as causative. Counseling is provided to avoid precipitating or aggravating positions and motions. A physical therapy program is initiated, with focus on gradual strengthening of the shoulder girdle, rotator cuff, and periscapular muscles as well as stabilization of the scapula. Supervised exercise and eventual self-care programs are implemented (Martin et al 1997).

Management

No studies provide evidence of medication utility beyond symptomatic relief.

Controversy exists regarding the length of time nonoperative treatment is continued. Often, if conservative management has failed or 6 months have passed, then surgical approaches are considered (Martin et al 1997; Ferrick and Marzo 1999; Cummins and Schneider 2008).

Several approaches to surgically decompress the suprascapular nerve (including posterior, anterior, and superior) have been described. Numerous variations have been described, including procedures adding bone resection around the suprascapular or spinoglenoid notch. Most reports indicate preference for posterior decompression of the suprascapular nerve without bone resection (Clein 1975; Post and Mayer 1987; Cohen et al 1997). A report describes improvement of suprascapular neuropathy by arthroscopic repair of massive rotator cuff tears (Costouros et al 2007).

A simplified summary of most surgical procedures for suprascapular neuropathy involves a section of the suprascapular (or spinoglenoid) ligament, resulting in decompression of the nerve. Surgery is usually followed by rapid postoperative mobilization to tolerance.

Suprascapular nerve transfer techniques have also been described (Merrell et al 2001). There are increasing numbers of publications outlining arthroscopic approaches to suprascapular nerve decompression, especially when cystic entrapment of the nerve is involved (Westerheide et al 2006; Lee et al 2007; Werner et al 2007).

A study conducted by Lafosse and colleagues showed that arthroscopic release of the suprascapular nerve can be performed safely and effectively (Lafosse et al 2007). All of the patients in the study had improvement in their postoperative electromyographic findings and had marked improvement in pain relief and function.

Although both open and arthroscopic approaches provide reliable pain relief and improvements in function, arthroscopic decompression has the benefits of simultaneously diagnosing and addressing intra-articular and/or
subacromial pathology with minimizing morbidity (Piasecki et al 2009).

Clavert and Thomazeau reported that arthroscopic nerve release for suprascapular nerve entrapment is a preferred procedure in patients with paralabral cysts with favorable outcomes in terms of pain, functionality, mobility, and strength (Clavert and Thomazeau 2014). Radiological cyst aspiration were, however, reported to be associated with a failure and recurrence rate as high as 75% to 100%. In a 5-year retrospective review of the outcome of suprascapular neuropathy, Hill and colleagues made an observation that presence of cyst and rotator cuff tears are important occurrences, indicating surgical treatment (Hill et al 2014).

**Special considerations**

**Pregnancy**

No reported effect of pregnancy on suprascapular neuropathy or on suprascapular neuropathy affecting pregnancy has been documented.

**References cited**


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Thomas A. La paralysie du muscle sous-épineux. La Presse Medicale 1936;44:1283-4.


**References especially recommended by the author or editor for general reading.**

**Former authors**

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**ICD and OMIM codes**

**ICD codes**

ICD-9:
Mononeuritis of upper limb unspecified: 354.9

ICD-10:
Mononeuropathy of upper limb, unspecified: G56.9

**Profile**

**Age range of presentation**

06-12 years
13-18 years
19-44 years
45-64 years
65+ years

**Sex preponderance**

male>female>2:1
male>female>1:1

**Family history**

none

**Heredity**

none

**Population groups selectively affected**

Amateur and professional athletes in baseball, volleyball, weightlifting, swimming, diving, tennis, canoeing, backpacking

**Occupation groups selectively affected**

Amateur and professional athletes
Newsreel cameramen
Occupations requiring cross body adduction

**Differential diagnosis list**

rotator cuff dysfunction
cervical radiculopathy
brachial plexus problems
diffuse peripheral neuropathy
shoulder girdle pathologies
acromioclavicular joint disease
glenohumeral joint disease
adhesive capsulitis
biceps tendonitis
bursitis
Pancoast tumor

**Associated disorders**

Shoulder rotator cuff tear
Traumatic shoulder injury

**Other topics to consider**

Neurologic injuries associated with sporting activities

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