17 Unusual Nerve Entrapments and Neuropathic Syndromes of the Neck and Shoulder

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Abstract

There are multiple peripheral nerve problems that can mimic or co-exist with thoracic outlet syndrome (TOS), including primary or secondary pathology related to the suprascapular, axillary, dorsal scapular, long thoracic, and spinal accessory nerves. Failure to recognize these problems can lead to inadequate or failed TOS treatment. This chapter discusses the anatomy, entrapment, diagnosis, and types of treatment of the suprascapular, axillary, dorsal scapular, long thoracic, and spinal accessory nerves. It should be noted that this chapter deals with the diagnosis and treatment of primary entrapment syndromes encountered during the workup of patients with cervicobrachial syndrome.

Critical Take Home Points

1. Several specific nerve entrapment syndromes can mimic NTOS.
2. There is a significant potential for multiple, concurrent nerve entrapments.
3. For those patients who do not respond to standard treatment or have a presentation that is not classic or clear cut, then consider one of these other pathologies.

17.1 Introduction

There are many causes of neck and shoulder pain, with facet spondylosis, herniated cervical discs, and thoracic outlet syndrome (TOS) being some of the most common. However, there are a group of uncommon (or perhaps just less commonly identified) peripheral nerve entrapment syndromes that can easily be missed. The resultant failure of facet injections, cervical discectomy, or thoracic outlet interventions is frustrating, expensive, and potentially worsens the underlying condition. Knowledge of these syndromes will improve outcomes by improving the diagnosis and therefore the treatment plan. Patients may see a multitude of providers (chiropractors, physical therapists, neurologists, spine surgeons, physiatrists, and pain specialists) as they seek care, but effective treatment requires an accurate diagnosis. Discussed in this chapter are five nerves (suprascapular, axillary, dorsal scapular, spinal accessory and long thoracic) that, when abnormally affected, have similar symptoms to TOS, and may occur concurrently. The description of these nerve problems, combined with the response of workup and diagnostic injections, will direct treatment.

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17.2 Anatomy and Relationships to Thoracic Outlet Syndrome

Although each nerve entrapment syndrome will be discussed separately below, it is useful to review the relationship of these nerves to the brachial plexus and the thoracic outlet (Fig. 17.1). The suprascapular nerve is a branch of the brachial plexus itself, and often entrapped by the scalene muscles [1, 2], while the dorsal scapular and long thoracic nerves arise more proximally and pass through the middle scalene muscle. The axillary nerve is a branch of the radial nerve (also part of the brachial plexus), while the spinal accessory nerve, the 11th cranial nerve, is also separate and can be entrapped by the trapezius and sternocleidomastoid muscle and is often traumatized by flexion/extension injuries [3, 4].

Compression of the brachial plexus proximally can create a condition called "double crush syndrome", where the presence of a lesion anywhere in the course of a nerve renders the nerves elsewhere more vulnerable to compression, and often with a degree of pain and dysfunction greater than that expected from either entrapment alone. Originally described by Upton and McComas [5], they observed that 81 of 115 (70%) patients with carpal or cubital tunnel syndrome also had electrophysiological evidence of a nerve injury in the neck. They postulated that this was due to the effect of compression on anterograde axoplasmic flow, which was later confirmed by other investigators [6, 7]. As an example, the surgical outcome of carpal tunnel release is poorer in patients with lesions elsewhere in the nerve tract, suggesting that both entrapments likely require treat-

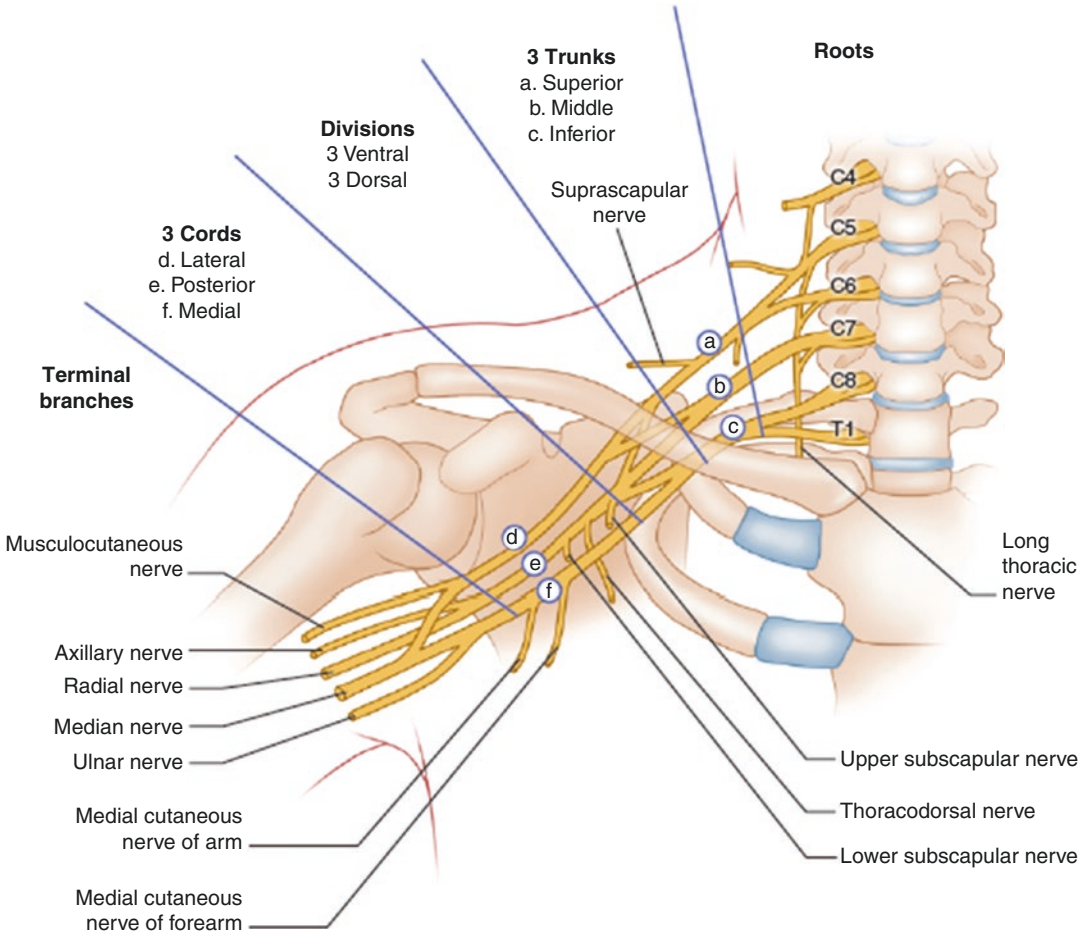

Fig. 17.1 Relationship between the brachial plexus and several peripheral nerve entrapments that mimic thoracic outlet syndrome. (Image courtesy of Springer)

ment for optimal results [8]. It is thus critical to recognize and treat both problems for optimum improvement.

17.3 Suprascapular Nerve (SSN)

17.3.1 Symptoms

The signs and symptoms of suprascapular nerve (SSN) entrapment depend on the location of nerve compression. Entrapment at the suprascapular notch usually presents as a sudden onset of a a dull ache in the posteriolateral aspect of the shoulder and scapular regions [9] that can radiate into the ipsilateral shoulder, arm, or neck, often with limitations in abduction and external rotation of the shoulder. It can be triggered by an acute event, such as a lifting injury with the arm internally rotated (like carrying a heavy suitcase), but the onset is typically insidious, involving the dominant arm. Patients can also develop a "frozen shoulder," (adhesive capsulitis) because of an unwillingness to move the shoulder joint due to pain [9]. If the entrapment occurs more distally, at the *spinoglenoid notch*, the patient will have isolated atrophy and weakness of the infraspinatus muscle, which is usually not painful [9].

17.3.2 Anatomy

Thomas first described suprascapular neuralgia in 1936 [10]. Once thought to be a purely motor nerve, the SSN is a mixed motor and sensory nerve arising from C5 and C6 (and, in 15–22% of the cases, also C4). The SSN provides motor innervation to the supraspinatus and infraspinatus muscle, as well as the subacromial bursa, the acromioclavicular joint, and the glenohumeral joint. The SSN arises from the brachial plexus, crossing over the posterior cervical triangle, under the trapezius muscle, and through the suprascapular notch (Fig. 17.2), and then heads caudally to pass through the spinoglenoid notch (Fig. 17.3).

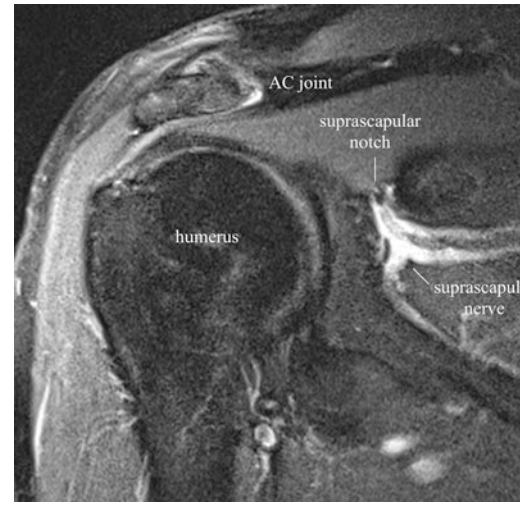

Fig. 17.2 MRI anatomy of the suprascapular notch

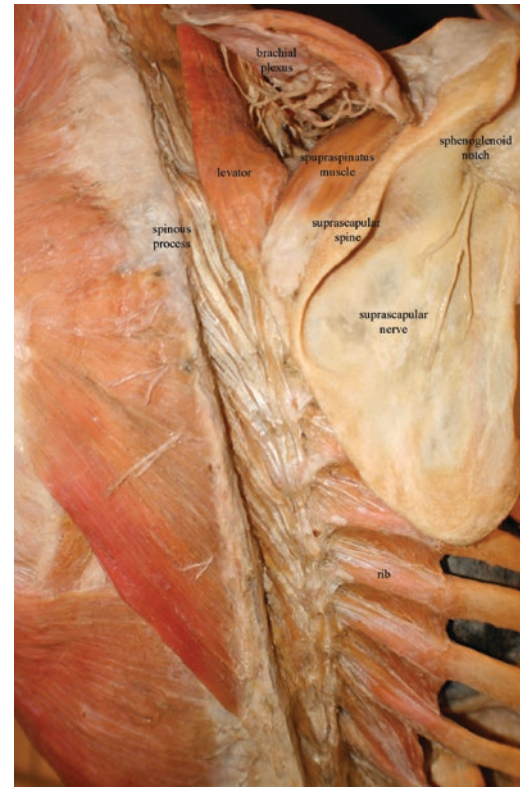

Fig. 17.3 Suprascapular anatomy, modified from an image from *Bodies, The Exhibition*, with permission

17.3.3 Entrapment

There are three sites where entrapment of the SSN can take place: at its origin, at the suprascapular notch, and at the spinoglenoid notch. The SSN can be injured at the level of the neck by flexion/extension injuries, especially if the head was turned at impact, in a manner similar to the mechanism of traumatic TOS. Direct blows to the base of the neck (such as injury from a high-riding seatbelt during a motor vehicle collision), backpacking, or weight lifting can also injure the SSN. At the suprascapular notch, microtrauma from repetitive movements as well as a small or calcified notch can lead to entrapment. The nerve also can be traumatized at the spinoglenoid notch during forced adduction and internal rotation (movements used during the follow through of a throwing motion) [11]. Labial or mucoid cysts and tumors have been identified as the cause of compression at the spinoglenoid notch, as have dilated veins [12].

17.3.4 Diagnosis

Patients with SSN entrapment may complain of a sudden onset of shoulder pain, though more commonly patients note a chronic, deep shoulder pain, increased with arm abduction [13]. Entrapment of the SSN is most commonly seen in athletes, especially those playing volleyball [14], baseball, and tennis, [15].

Atrophy of the supraspinatus muscle may be hard to identify on physical exam, because the overlying trapezius muscle can mask it, but infraspinatus atrophy is usually relatively easy to spot (Fig. 17.4). There can be as much as 75% loss of abduction and external rotation with pathology at the suprascapular notch, [13] though with isolated infraspinatus pathology at the spinoglenoid notch, the posterior deltoid and teres minor may be able to compensate, masking some of the weakness [15]. Edema of the denervated supraspinatus and/or infraspinatus muscle may

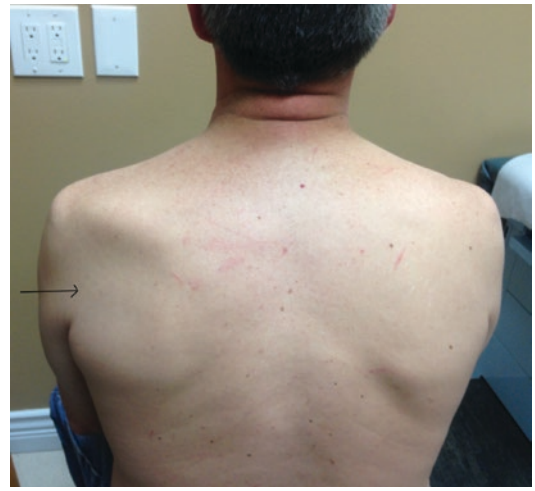

Fig. 17.4 *Arrow* shows left infraspinatus atrophy

be the surest and earliest characteristic sign of SSN entrapment [15], and can help to differentiate SSN entrapment from pure rotator cuff tears (Fig. 17.5).

The SSN can be injected using landmarks, [16] peripheral nerve stimulator, fluoroscopy, CT, [17] or US, targeting the nerve at the brachial plexus [18], the suprascapular notch, or the spinoglenoid notch [19].

17.3.5 Types of Treatment

Since diagnostic injections of the SSN usually contain a steroid, diagnostic injections can be therapeutic as well. However, if the diagnostic injection gives only temporary relief, there are a variety of other treatments. If the nerve is scarred down because chronic irritation, then hydrodissection using local anesthetic, saline, or platelet lysate may be useful [20]. If there is only temporary relief, cryoneuroablation can be useful [21]. Pulsed or conventional radiofrequency lesioning has been used [22], as has surgical resection. Fluoroscopy or ultrasound can be used to place a peripheral nerve stimulator (Fig. 17.6).

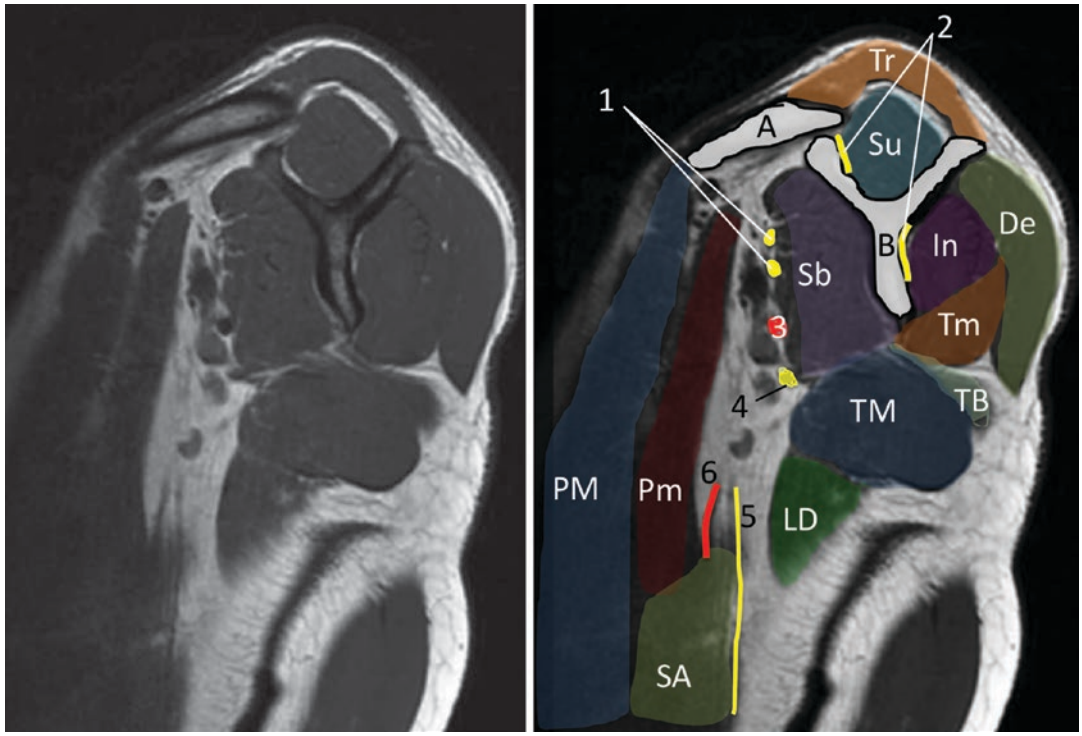

Fig. 17.5 Labeled sagittal MRI image of the scapular structures. A = clavicle; B = scapula; De = deltoid muscle; In = infraspinatus muscle; LD = latissimus dorsi muscle; PM = pectoralis major muscle; Pm = pectoralis minor muscle; Sb = subscapularis muscle; Su = suprascapular muscle; TM = teres major muscle; Tm = teres minor muscle; Tr = trapezius muscle; TB = triceps brachii muscle; 1 = brachial plexus; 2 = suprascapular nerve; 3 = axillary artery' 4 = suprascapular nerve; 5 = long thoracic nerve; 6 = long thoracic artery

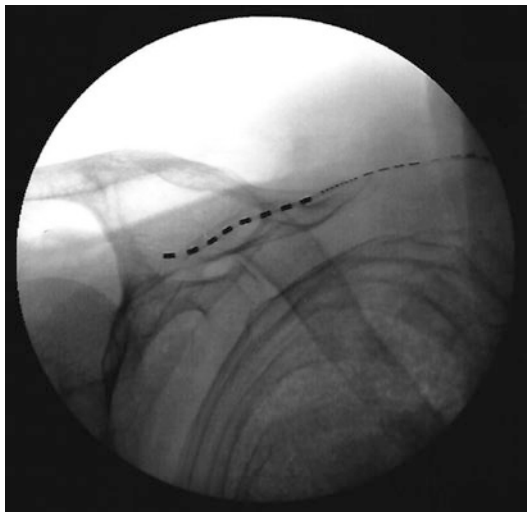

Fig. 17.6 Suprascapular peripheral nerve stimulator

17.4 Axillary Nerve (AN)

17.4.1 Symptoms

Patients suffering from an axillary nerve (AN) entrapment or injury are mostly young adults presenting with diffuse, poorly localized shoulder and upper arm pain in the dominant arm that may have an insidious onset. Athletes involved in overhead sports such as tennis are at particular risk [13], though this can also be a work-related pathology as seen with the arm in abduction for activities such as window cleaning [8]. Patients often complain of arm fatigue with overhead activity or throwing, and pain aggravated by sustained arm flexion, and they may report a non-dermatomal pattern of dull ache, burning,

or paresthesias of the lateral and posterior upper arm, especially in the deltoid region, that may wake them from sleep [13]. Anterior dislocation of the shoulder can cause stretching of the nerve in up to 55% of patients, and a proximal humeral fracture causes AN pathology in up to 58% of patients [13].

17.4.2 Anatomy

Axillary nerve entrapment occurs at the level of the quadrilateral space (the space between the teres major, teres minor, the long head of the triceps, and the surgical neck of the humerus) [23] (Fig. 17.7); this syndrome was first described by Cahill in 1980 [24]. The axillary nerve is the terminal branch of the posterior cord of the brachial plexus, usually composed of fibers from C5 and C6. The nerve leaves the brachial plexus, runs across the inferiolateral border of the subscapularis muscle, and then passes through the *quadrilateral space* (also called the lateral axillary hiatus) with the circumflex radial artery to the humeral shaft. The nerve then divides into the anterior (superior) branch, which wraps around the humerus and supplies motor function to the anterior and middle deltoid muscle, and the posterior (inferior) branch, which supplies motor to the subscapularis, teres minor, and posterior deltoid muscles as well as sensory innervation to the posterolateral shoulder [25].

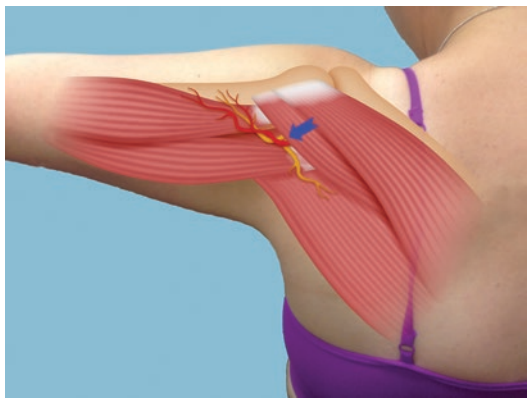

Fig. 17.7 A posterior view of the quadrilateral space with the arm in abduction, showing entrapment of the axillary nerve and artery (*arrow*) by the teres major and teres minor. (Image courtesy of David Trescot and Steve McGuire)

17.4.3 Entrapment

Entrapment of the axillary nerve occurs with spasm or fibrous bands from the muscles that compose the quadrilateral space [24] or by posteroinferior labral cysts [26]. The nerve can also be injured acutely during anterior shoulder dislocation or humeral head fracture [27].

17.4.4 Diagnosis

Patients with axillary nerve pathology are often athletes involved in throwing sports who present with subacute shoulder pain and paresthesias, especially with abduction and external rotation. On physical exam, there is tenderness of the teres minor, with paresthesias with palpation over the quadrilateral space. The diagnosis of axillary nerve entrapment is dependent on a high level of suspicion from the clinician. Axillary or suprascapular entrapment should be considered when there appear to be several shoulder muscles involved [9].

Injections of the axillary nerve have been described using landmarks, fluoroscopy, and US (Fig. 17.8) [28, 29]. The axillary nerve can also be anesthetized to provide anesthesia for painless reduction of upper extremity fractures [30].

17.4.5 Types of Treatment

Lysis of the fibrous bands by hydrodissection may offer relief. Botulinum toxin can be used to relax the spasms of the muscles. Cryoneuroablation may be useful in recalcitrant cases (personal experience). Surgery has been use to section the fibrous bands, [15] and mucoid cysts can be managed by excision of the cyst or repair of the offending labral tear [13]. Peripheral nerve stimulation may be useful (Fig. 17.9).

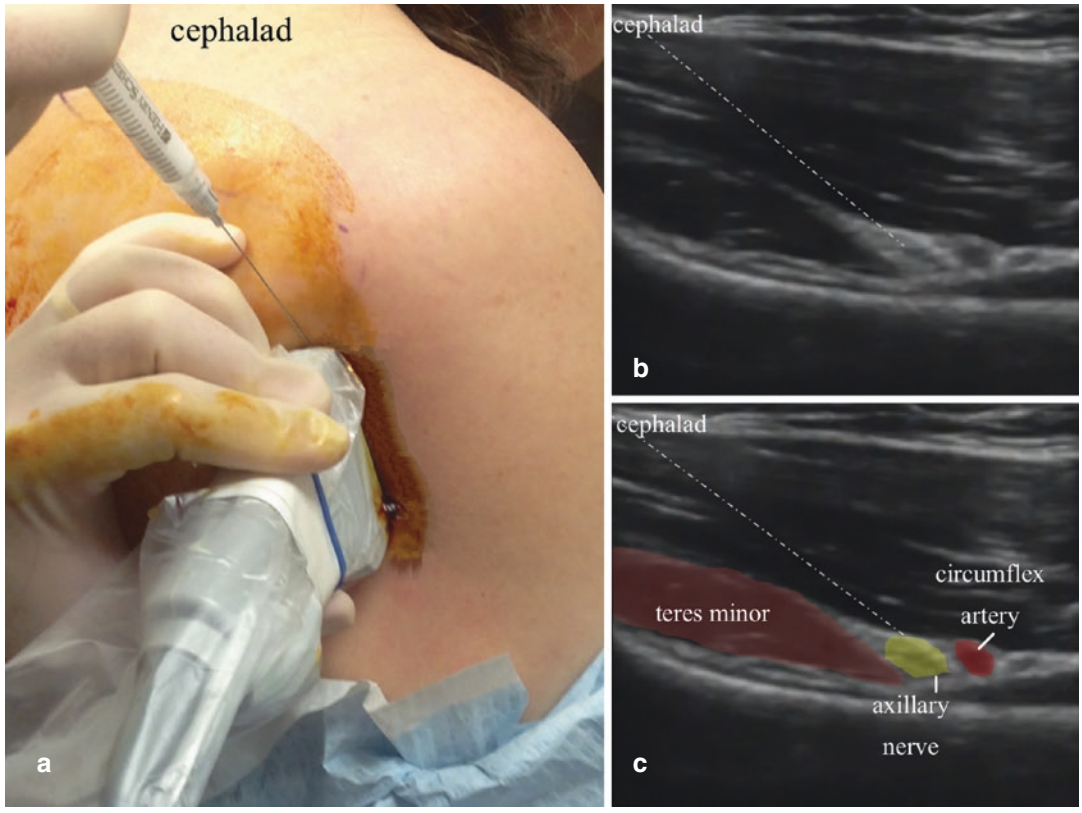

Fig. 17.8 Ultrasound-directed injection of the axillary nerve. (**a**) Location of probe and needle direction. (**b**) Ultrasound image of axillary nerve with simulated needle path. (**c**) Labeled ultrasound image with simulated needle path

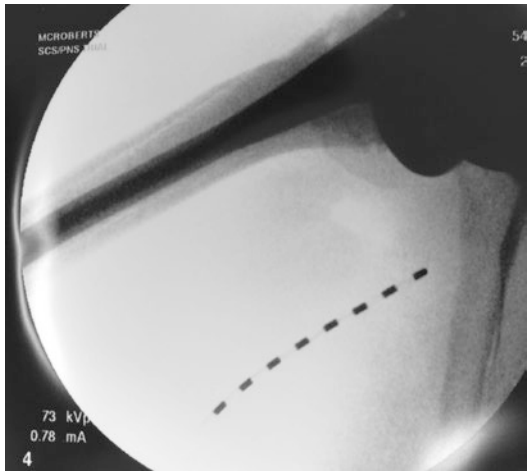

Fig. 17.9 Axillary nerve stimulation (Image courtesy of Porter McRoberts, MD)

17.5 Dorsal Scapular Nerve

17.5.1 Symptoms

Patients with dorsal scapular nerve (DSN) entrapment will present with pain in the interscapular region, as well as shoulder and arm pain, weakness of arm abduction, and/or winged scapula [31]. They may complain of sharp or knife-like medial scapular pain, with lateral arm pain, as well as a concomitant anterior chest wall pain and tenderness over the T4 sternocostal border, leading to hospitalization for a possible heart attack. There may also be an itching sensation in the ipsilateral interscapular region. The scapular winging often goes undiagnosed [32], and the

subsequent shoulder pain can result in unnecessary or unsuccessful shoulder surgeries. The onset of pain can be sudden or develop slowly over time.

17.5.2 Anatomy

The DSN arises from C5 (rarely C5 and C6), and frequently shares a common nerve trunk with the long thoracic nerve (LTN) (see below). The DSN passes through the middle scalene, an average of 3 cm from its origin, [33] and then separates from the LTN. As it leaves the brachial plexus, the DSN has a variable relationship with the levator scapula; in one study, the DSN gave branches to the levator muscle in only 11 of 35 necks studied [34]. It then travels along the medial border of the scapula, and innervates the rhomboid major and minor muscles (Fig. 17.10) [35].

17.5.3 Entrapment

Entrapment of the DSN occurs at three sites—within the middle scalene, under the levator scapula, and at the rhomboid fascia. DSN entrapment has been described due to a hypertrophied middle scalene [36]. The DSN may also be injured during brachial plexus blocks; Kim et al. looked at 70 patients undergoing posterior brachial plexus block under US and noted that the DSN was encountered in 44 cases (62.8%) and the long thoracic nerve (see below) was encountered in 15 cases (21.4%) [37].

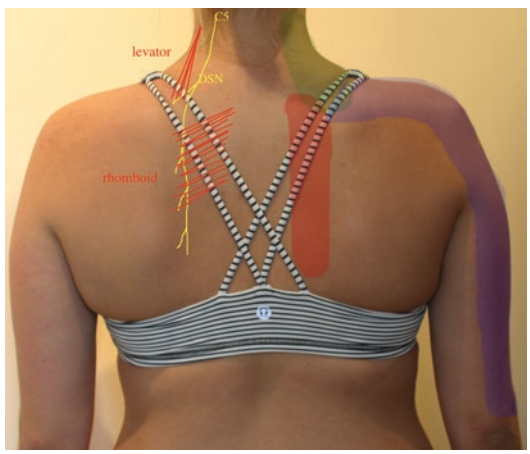

Fig. 17.10 Dorsal scapular nerve path and pain pattern associated with dorsal scapular nerve entrapment. *Red* = interscapular pain; *green* = posterior neck and "levator" pain; *purple* = lateral arm pain

17.5.4 Diagnosis

DSN pathology has been underrecognized as a pathologic process. Patients describe dull or sharp, acute or chronic pain at the neck, medial top of the scapula, and medial border of the scapula. Occasionally, they will also or instead complain of an ipsilateral anterior chest pain at the costosternal junction, usually about T4. Because of tenderness in this region, especially with deep breathes or movements, the pain may be ascribed to "Tietze syndrome"; because the pain may radiate down the ipsilateral arm, the patient may receive a workup for cardiac disease. Most of the time, there is not a specific injury, though there have been case reports of trauma to the neck causing DSN pathology [38].

Patients will have palpable tenderness along the medial border of the scapula, almost always at about the level of T4; however, this area is difficult to find if the shoulders are not rolled forward. There is usually minimal scapular winging, though it was a prominent feature of at least one case report [32]. The best way to test rhomboid weakness may be to have the patient lower their arms from a forward elevated position; if the muscles are weak, the examiner can easily place several fingers underneath the medial border of the scapula [39]. DSN entrapment can present as an atypical scalene entrapment, since it has a scalene pain pattern, as well as an atypical TOS [40].

Diagnostic injections can be done at any of the three entrapment sites (middle scalene, levator, and rhomboid regions), usually either landmark- or US-guided.

17.5.5 Types of Treatment

Hydrodissection of the DSN in the middle scalene muscle has not been reported but can be quite effective (as an example, see https://www.youtube.com/watch?v=t6atN2wfYBE). For the "Trescot approach" injection, the shoulder is rotated anteriorly to move the scapula laterally, and a reproducible area of tenderness at the medial scapula can be palpated. Straddling this region with the non-injecting hand, with the fingers parallel to the rib, the needle is introduced under the rib at an acute angle to avoid the lung [41].

Haim and Urban described temporary relief with injection of 100 units of botulinum toxin [42]. They also described the potential use of cryoneuroablation at the levator site. Chen et al. [40] described 24 surgical resection of the middle scalene muscle on 22 patients to release the DSN; "20 sides of 19 patients were completely or partially relieved". There has not been a peripheral nerve stimulation technique described.

17.6 Long Thoracic Nerve (LTN)

17.6.1 Symptoms

Patients with long thoracic nerve (LTN) dysfunction often have a deep, non-specific ache in the shoulder radiating "through the neck to the scapula" [43], and lateral chest wall [44], or it may be near the inferior scapula, with a burning pain at the lower pole of the scapula, which was a "striking feature, present in each patient" [45]. There may be weakness with forward elevation and overhead activities. Athletic activities cause many injuries to the LTN, but it can also be injured during breast surgery (specifically mastectomy), or even carrying a heavy bag over the shoulder for a long time.

17.6.2 Anatomy

The LTN (also known as the Charles Bell nerve) [15] is a branch of the supraclavicular portion of the brachial plexus, arising from C5, C6, and C7, passing across or within the middle scalene, [46] and then traveling behind the clavicle and over the first rib to innervate the serratus anterior muscle. In the neck, the LTN is usually a flat structure seen under US within the body of the middle scalene muscle, and it becomes round after it leaves the muscle.

17.6.3 Entrapment

There are multiple sites of entrapment of the LTN - between the middle and posterior scalene muscles, at the first rib, between the clavicle and the second rib (under the subclavicularis muscle), between the second rib and the coracoid (under the pectoralis minor muscle), the inferior angle of the scapula, or by inflamed bursa along the course of the LTN [47, 48].

17.6.4 Diagnosis

There is often a history of trauma to the chest or shoulder girdle (26% of cases) [45] or sports injuries such as volleyball, weightlifting, and archery (35% of cases) [49]. Surgical dissection in the axilla, as is seen with radical mastectomies or resection of the first rib, is associated with LTN damage, but there might also be a history of abduction of the arms during general anesthesia, as seen with median sternotomy surgeries [50]. There is at least one case report of LTN paresis after massage therapy [51]. In 17% of the cases, there is not a clear cause [52].

During the physical examination, there will be a winged scapula (see Table 17.1), with the shoulder dropping inferiorly and the inability to actively abduct the arm >90°, or forward flex the arm past the horizontal.

MRI imaging may show signs of edema or atrophy of the serratus anterior, consistent with denervation changes (Fig. 17.5). EMG may show denervation changes of the serratus anterior; however, according to Friendenberg et al., electrodiagnostic studies did not predict the functional outcome [53].

Diagnostic injections of the LTN are relatively uncommon among interventional pain physicians,

Table 17.1 Comparison of winging from long thoracic, spinal accessory, and dorsal scapular nerve pathology

Nerve	Muscles involved	Type of winging	Provocative maneuvers
Dorsal scapular (a rare cause of subtle winging)	Rhomboid and levator scapulae	Scapula shifted laterally and dorsally	Slowly lowering arm from forward elevation
Long thoracic	Serratus anterior	Medial winging	Forward elevation and pushing with outstretched arms (wall push-up)
Spinal accessory	Trapezius	Lateral winging with drooping shoulder	Resisted arm abduction or external rotation

Modified from *Peripheral Nerve Entrapments*, Trescot A. M. (ed), New York, Springer, 2016

and there is relatively little literature on the techniques. Because the LTN travels through the middle scalene nerve, it is often anesthetized during scalene injections [46]. Ramamurthy et al. [54] reported nerve stimulator-guided LTN injections to treat lateral chest wall pain thought to be due to SAM spasm. That injection can be particularly useful for LTN injuries from chest wall injuries.

17.6.5 Types of Treatment

There are no reported techniques of cryoneuroablation or radiofrequency lesioning of the LTN. However, the author has used cryoneuroablation to treat LTN dysfunction, directing the cryo probe on the chest wall from inferior to superior, parallel to the nerve. Surgery may be necessary to stabilize the scapula [48]. There are no described peripheral nerve stimulation techniques.

17.7 Spinal Accessory Nerve (SAN)

17.7.1 Symptoms

Patients with pathology of the spinal accessory nerve (SAN) can present with ipsilateral neck, shoulder, neck, and occipital pain. However, they can also present with persistent contralateral spasm of the SCM and trapezius muscles, with asymmetric neckline, winged scapula, and sometimes torticollis, because of palsy of the nerve on the other (ipsilateral) side. Pain can be a common complaint, occurring in as much as 86% of patients immediately after a SAN injury, before any weakness and atrophy have developed [55]. There may also be sensory changes over the ear and lateral neck, as well as angle of the jaw, the shoulder, and the anterior chest wall.

17.7.2 Anatomy

The spinal accessory nerve (SAN) is the 11th cranial nerve. It exits the skull through the jugular foramen (with the vagus and glossopharyngeal nerves), travels behind the styloid process to pass below the posterior belly of the digastric muscle, and then enters the superior sternocleidomastoid muscle (SCM) at the level of the internal jugular vein (Erb's point). It stretches across the posterior cervical triangle to the trapezius muscle, which it innervates.

17.7.3 Entrapment

Because the SAN has been considered a pure motor nerve, the pain from SAN injury has been attributed to SAN entrapment caused by trapezius compartment syndrome (mechanical drooping of the shoulder causing traction neuritis of the brachial plexus) or SAN injury, as well as entrapment at higher levels due to trauma. Nystrom et al. [56] described 16 patients with chronic whiplash symptoms and SAN entrapment after falls or sports injuries; at surgery, they discovered adhesions between the SAN and underlying fascia. If the site was proximal, both the SCM and trapezius muscles were involved; if it was at the more usual site in the posterior triangle, the trapezius alone was affected [57]. Surgeries in the posterior cervical triangle (such as radical neck surgery or lymph node biopsy) can also injure the LTN. Patients with SAN injury may have a history of trauma (blunt or surgical) to the pos-

terior cervical triangle, as well as flexion/extension injuries. The pathology may also show up in wrestlers, weightlifters, and hockey players.

17.7.4 Diagnosis

The physical exam of patients with complete paresis of the SAN shows severe dysfunction of the shoulder girdle, with a dropped shoulder, trapezius atrophy, and lateral displacement of the shoulder (with the superior angle of the scapula more lateral than the inferior scapular angle) (see Table 17.1), but partial denervation or entrapment may be more subtle. Winging can be accentuated by abduction and external rotation of the arm against resistance [48]. This nerve tends not to be in the field of dissection for thoracic outlet decompression, but can be injured during carotid endarterectomy if the SCM is dissected too deeply.

Neurodiagnostic testing, such as EMG, can be difficult to interpret, so much emphasis is placed on the MRI findings of atrophy, edema, and denervation changes of the trapezius without changes in the serratus anterior muscle (to rule out confounding LTN pathology) (Fig. 17.5).

17.7.5 Diagnostic Injections

The landmark-guided technique should only be attempted in patients with a neck thin enough to palpate the styloid process. Using fluoroscopic guidance, the needle is advanced to the styloid process (Fig. 17.11), and then directed posteriorly; use of a 25 g 2inch needle with peripheral nerve stimulator (PNS) will facilitate identification of the SAN by eliciting a scapular twitch. Bodner et al. [58] originally described the US evaluation of the SAN; US is less efficient visualizing the SAN at the level of the styloid process or above, but it easily shows the SAN in the posterior triangle (Fig. 17.12).

17.7.6 Types of Treatment

Since the SAN is primarily a motor nerve, neurolytic procedures are less commonly reported,

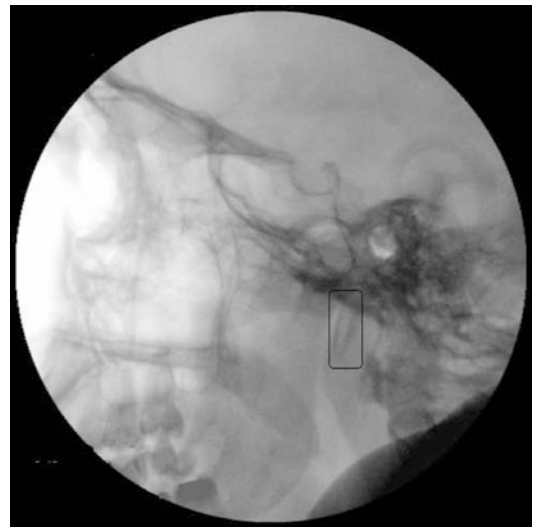

Fig. 17.11 Xray image of the styloid process (outlined)

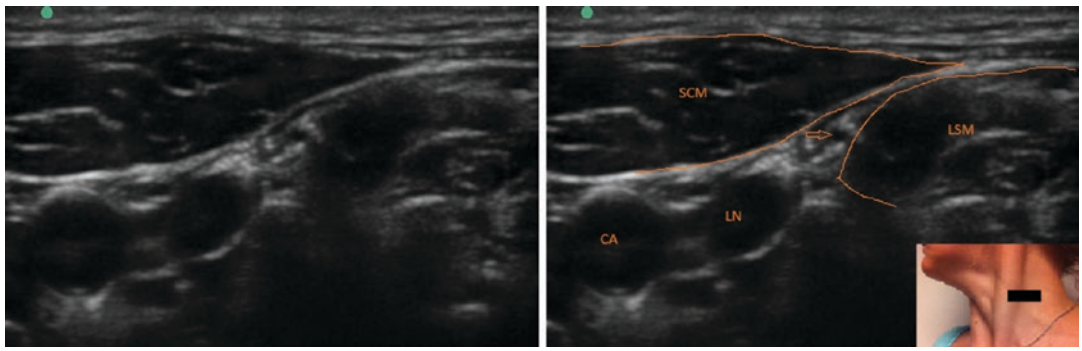

Fig. 17.12 Ultrasound image of the spinal accessory nerve (marked by the *arrow*) at the posterior triangle. SCM = sternocleidomastoid muscle; LSM = levator scapulae muscle, CA = carotid artery, LN = lymph node. Internal jugular vein is compressed. (Image courtesy of Agnes Stogicza, MD)

but may be appropriate for pain or to complete a partial denervation. There is most likely a potential for ultrasound-guided cryoablation or pulsed radiofrequency of SAN at the level of the posterior triangle, but no report has been published so far.

Nystrom et al. [56] performed surgical fasciectomies on 30 consecutive patients with chronic SAN pain after flexion-extension injuries, an average of 41 months after injury. There has been no peripheral nerve stimulation described.

17.8 Winged Scapula

For the arm to properly abduct, the lower trapezius and the serratus muscles, working together, are paired with the upper trapezius and rhomboid muscles [59]. Winged scapula describes dysfunction of the scapula, usually as a consequence of nerve injury, which causes weakness of one or more of those scapular muscles, causing the scapula to have less connection with the posterior chest wall (Fig. 17.13). Probably the most common cause of scapular winging is injury to the long thoracic nerve, resulting in weakness of the serratus anterior muscle. However, it is important to note that a winged scapula is not "just" due to long thoracic nerve dysfunction - other causes of scapular winging include injury to the spinal accessory nerve, causing trapezius nerve weakness, and damage to the dorsal scapular nerve, causing rhomboid weakness (Table 17.1).

A winged scapula can affect the patient's ability to lift, pull, or push heavier objects, and can infer with the ability to perform activities of daily living, [60]. Winging may be subtle, but the change in function of the scapula causes increased work for the non-affected muscles, leading to spasm and pain. It is sometimes the secondary spasm that becomes the focus of treatment (myofascial release, trigger point injections, botulinum toxin) and not the underlying cause (nerve entrapment and subsequent weakness). It is interesting to note that Jordan et al. [61] treated 245 TOS patients with image-guided (fluoroscopy or ultrasound plus EMG) botulinum toxin injections; all of them received additional botulinum toxin into the "painful and dystonic shoulder muscles, including the trapezius and levator scapula", suggesting the potential that the botulinum was actually for concomitant (or misdiagnosed) treatment of one of these "unusual" entrapments.

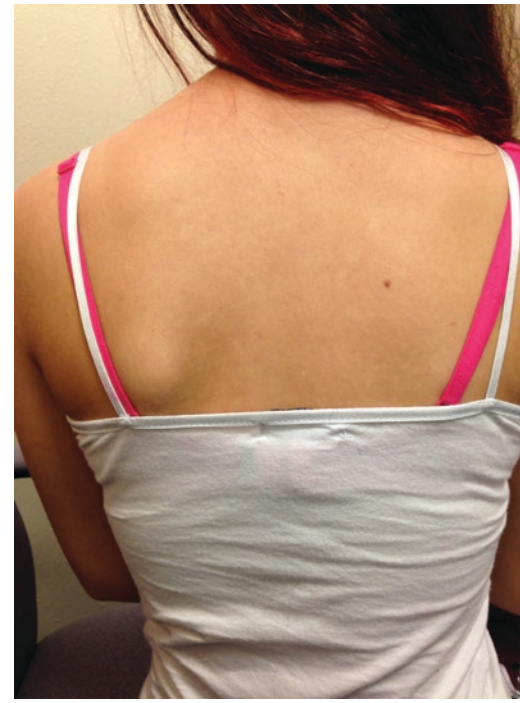

Fig. 17.13 Example of scapular winging after a motor vehicle collision

17.9 Who Diagnoses and Treats These Problems, and How to Identify When Referral Is Needed?

Diagnosis of these unusual entrapments requires a high index of suspicion, but these entrapments should come to mind when patients are not responding to standard TOS treatment, especially when the TOS clinical presentation is not "classic". Patients will often "drift" into pain management when pain is the presenting symptom; however, early referral to an interventional pain management physician (especially one skilled

in ultrasound -guided injections) may speed the diagnosis and treatment by the use of diagnostic and therapeutic injections. Electrodiagnostic testing (by a neurologist or physiatrist) may help to guide diagnosis, but requires a higher level of skill, since these nerves are not part of a standard EMG evaluation. There must, therefore, be a working diagnosis that includes these possibilities.

17.10 Conclusion

There are multiple causes of neck and arm pain, some of which can be confused (or co-exist) with NTOS. For example, NTOS would not be expected to cause winging of the scapula, so that physical finding should make the clinician explore other diagnoses. In addition, patients who are not responding to standard therapy may warrant a consideration of these less common nerve entrapments. An interventional pain physician with knowledge of peripheral nerve entrapments may be the best resource for diagnosis and treatment.

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