Spinal Accessory Nerve Entrapment

27

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Introduction

The *spinal accessory nerve* (SAN), the 11th cranial nerve (CNXI), has a long superficial course in the neck, which makes it vulnerable to injury that is rarely recognized. SAN was traditionally believed to be a pure motor nerve, but later anatomical studies have confirmed that it has both sensory and motor components [1]. Entrapment of the SAN and/or chronic *compartment syndrome of the trapezius muscle* may cause chronic debilitating pain after *flexion-extension trauma*, without radiologic or electrodiagnostic evidence of injury. SAN palsy presents with isolated *sternocleidomastoid muscle* (SCM) and *trapezius muscle* weakness and atrophy ("*sloping shoulder syndrome*"), as well as shoulder and neck pain. The SAN is also sometimes simply called the "*accessory nerve*" [2, 3].

Clinical Presentation (Table 27.1)

Patients with SAN palsy present with ipsilateral neck, shoulder, neck, and occipital pain and headache (Figs. 27.1 and 27.2) or persistent contralateral spasm of the SCM and trapezius muscles, with asymmetric neckline, *winged scapula*, and sometimes *torticollis.* Pain is a common complaint (86 % of patients with SAN injury) [17], which may occur immediately, before the weakness and atrophy have developed. There may also be sensory changes over the angle of the jaw, the ear, the shoulder and the chest, which has been attributed to concomitant damage of the great auricular nerve (Chap. 16) [17].

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A sudden acceleration-deceleration incident (whiplash), especially with head turned (as in a motor vehicle accident in which the patient was looking in the rear view or side view mirror at impact), put the SAN at particular risk because it is stretched in this position and tethered at the level of the styloid [4, 5]. The SAN can also be injured iatrogenically during procedures such as radical neck dissections, lymph node biopsies or other *posterior triangle* interventions, often without radiologic or electrodiagnostic evidence of injury [18]. Walvekar and Li state that SAN injury occurs in 3–8 % of patients after cervical lymph node biopsy and up to 80 % of patients who have had a radical neck dissection [6].

Initially after denervation, the trapezius and SCM may be swollen; later they begin to atrophy. SAN injury causes paresis of both muscles on the ipsilateral side and unopposed contraction of the muscles on the contralateral side, leading to torticollis (Fig. 27.3), a symptom that can divert attention from the actual pathology. Often compensatory hypertrophy of the levator scapulae muscle is present on the ipsilateral side. As the patient develops weakness of the SCM and trapezius muscles, traction on the brachial plexus by the unsupported shoulder can result in a painful "*shoulder syndrome*" [8],

Table 27.1 Occupation/exercise/trauma history relevant to spinal accessory nerve entrapment

Trauma	Flexion-extension injuries [4, 5]
	Falls [5]
	Sports injuries [5]
	Superficial "love bite" on the neck [6]
Surgery	Cervical lymph node biopsy [7]
	Radical neck dissection despite nerve-sparing
	Techniques (30–40 %) [8–11]
	Carotid endarterectomy [12]
	Face lift [13]
	Rotator cuff repair [14]
	Internal jugular vein cannulation (3–8 %) [15]
Neuritis	Varicella infection [16]

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Fig. 27.1 Patient complaint of pain from spinal accessory nerve entrapment (Image courtesy of Andrea Trescot, MD)

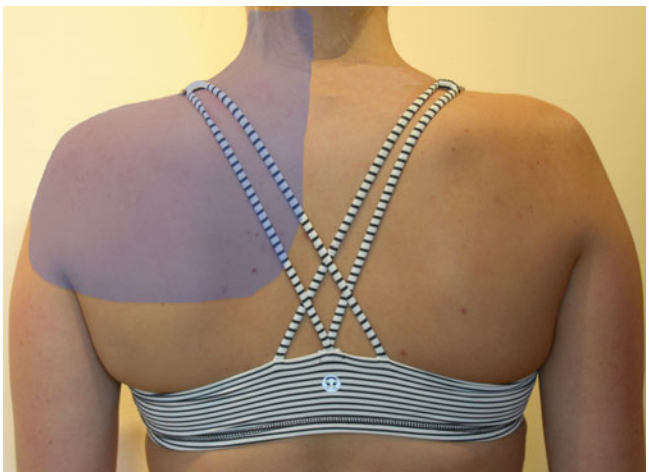

Fig. 27.2 Pain pattern associated with spinal accessory nerve entrapment (Image courtesy of Andrea Trescot, MD)

with an eventual frozen shoulder (*adhesive capsulitis)*. There may be a winged scapula (Fig. 27.4), a droop and internal rotation in the ipsilateral shoulder (Figs. 27.5 and 27.6) and

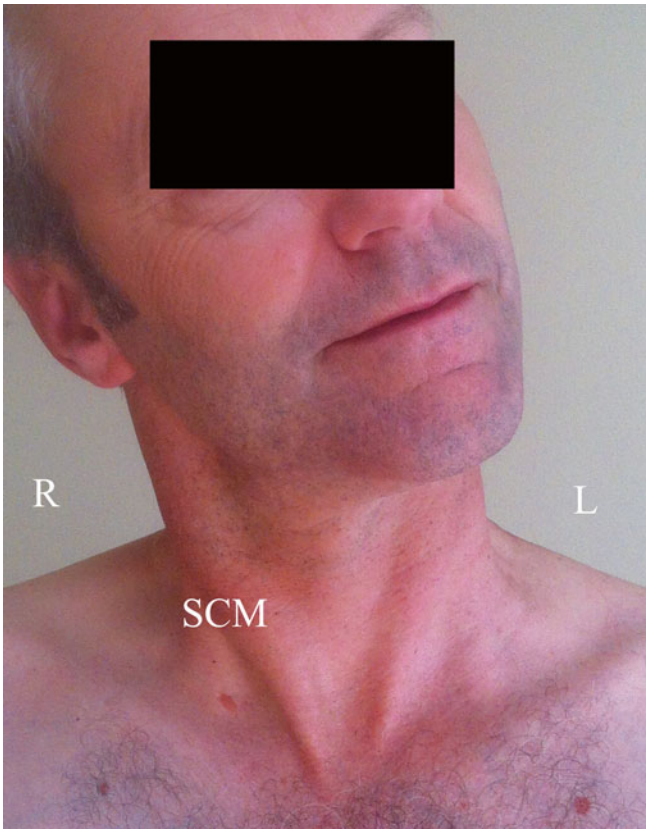

Fig. 27.3 Example of torticollis, causing shortening of the right sternocleidomastoid muscle, tilting the head to the right and turning it to the left (Image courtesy of Agnes Stogicza, MD)

atrophy of the trapezius and SCM (Figs. 27.5 and 27.6). The winging is usually most obvious when the patient actively externally rotates the shoulder against resistance. Subsequently the abnormal stresses on the clavicle can result in sternoclavicular joint hypertrophy or subluxation [19]. Pain in the shoulder will increase when its weight is not supported, and patients have decreased strength for overhead activities, such as putting away dishes. The most common sign is limited sustained abduction of the shoulder [6]. Lastly, there may be pain and spasm of the muscles on the contralateral side due to their unopposed function that diverts attention from the actual pathology.

Nystrom et al. [5] described the characteristics of 30 patients with whiplash injuries to the SAN, which included four clinical signs:

1. Asymmetric posture, typically with the shoulder elevated on the side of the greatest pain (lower on the pathologic side)
2. Decreased and painful motion in the neck and shoulder
3. Tenderness to palpation along the horizontal portion of the upper trapezius
4. Greater than 50 % reduction in pain and increased mobility following infiltration of local anesthetic into the upper trapezius

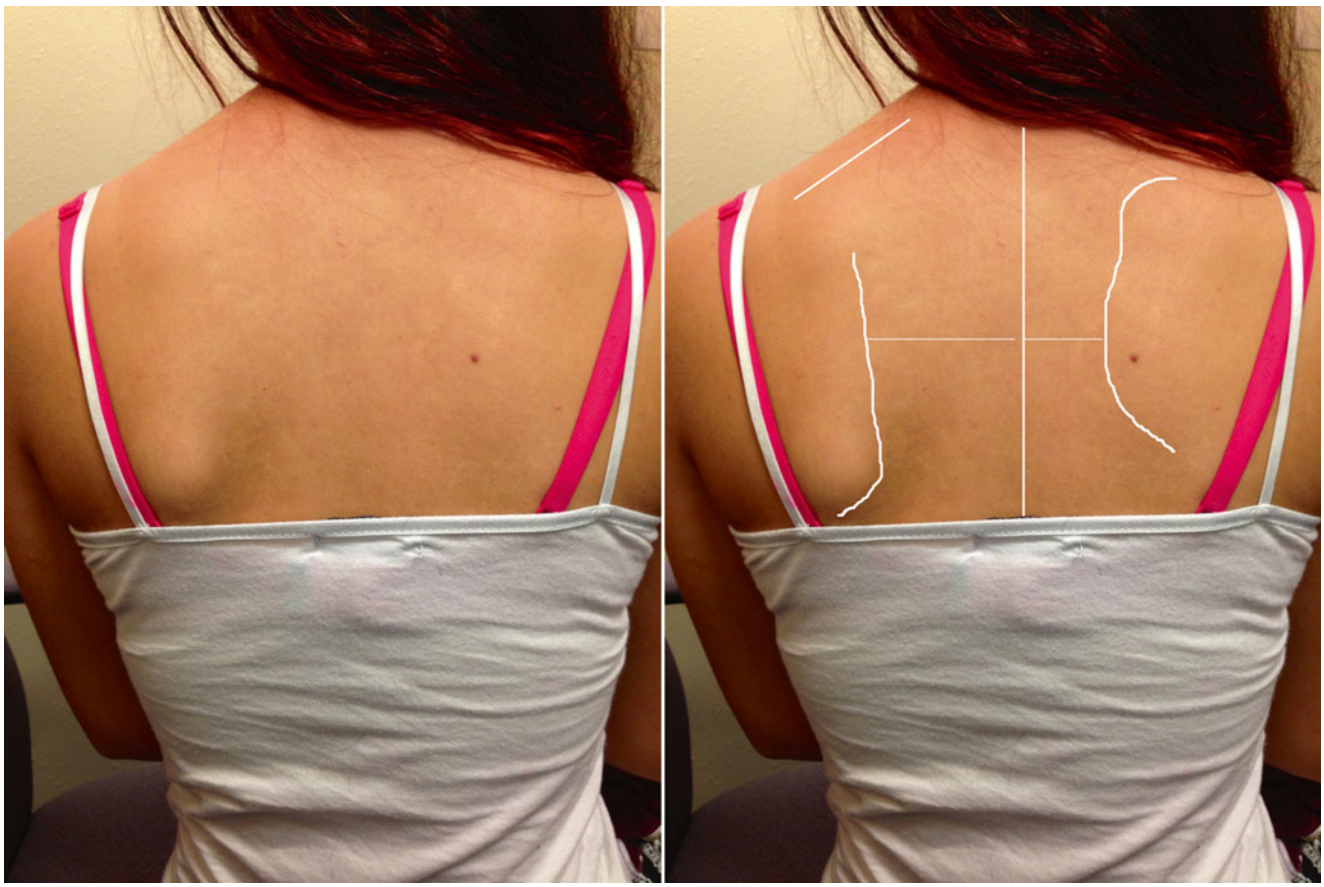

Fig. 27.4 Lateral winging of the left scapula after an MVA; winging is usually most obvious when the patient actively externally rotates the shoulder against resistance (Image courtesy of Andrea Trescot, MD)

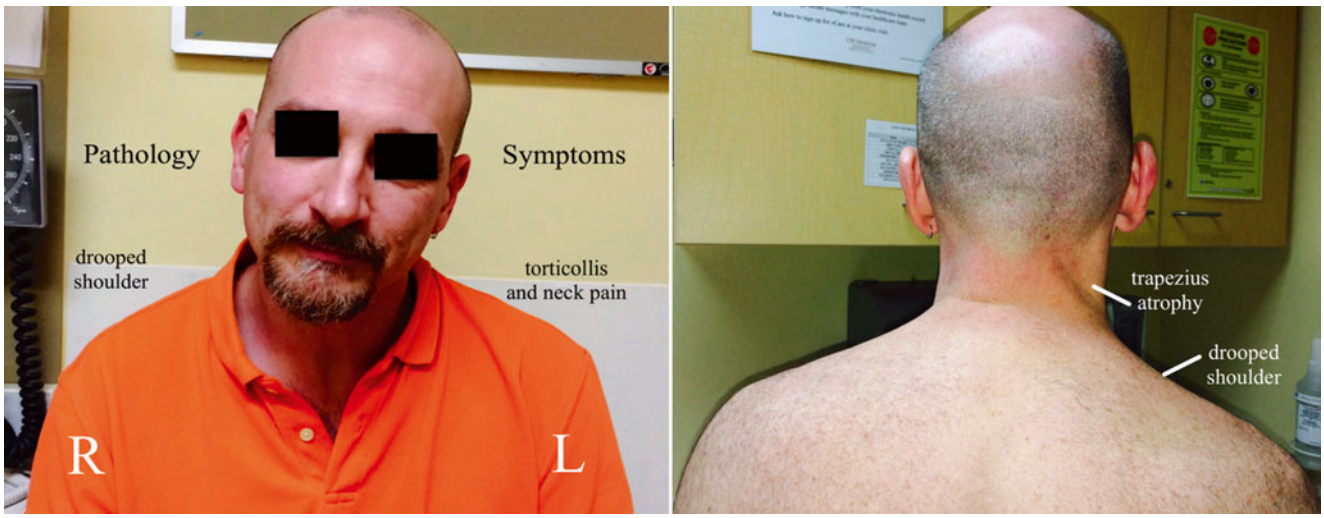

Fig. 27.5 Example of drooped shoulder from spinal accessory neuralgia. Note that the pathology (the spinal accessory neuralgia, trapezius atrophy) is on the right, but the symptoms (torticollis, shoulder pain) are on the left (Image courtesy of Agnes Stogicza, MD)

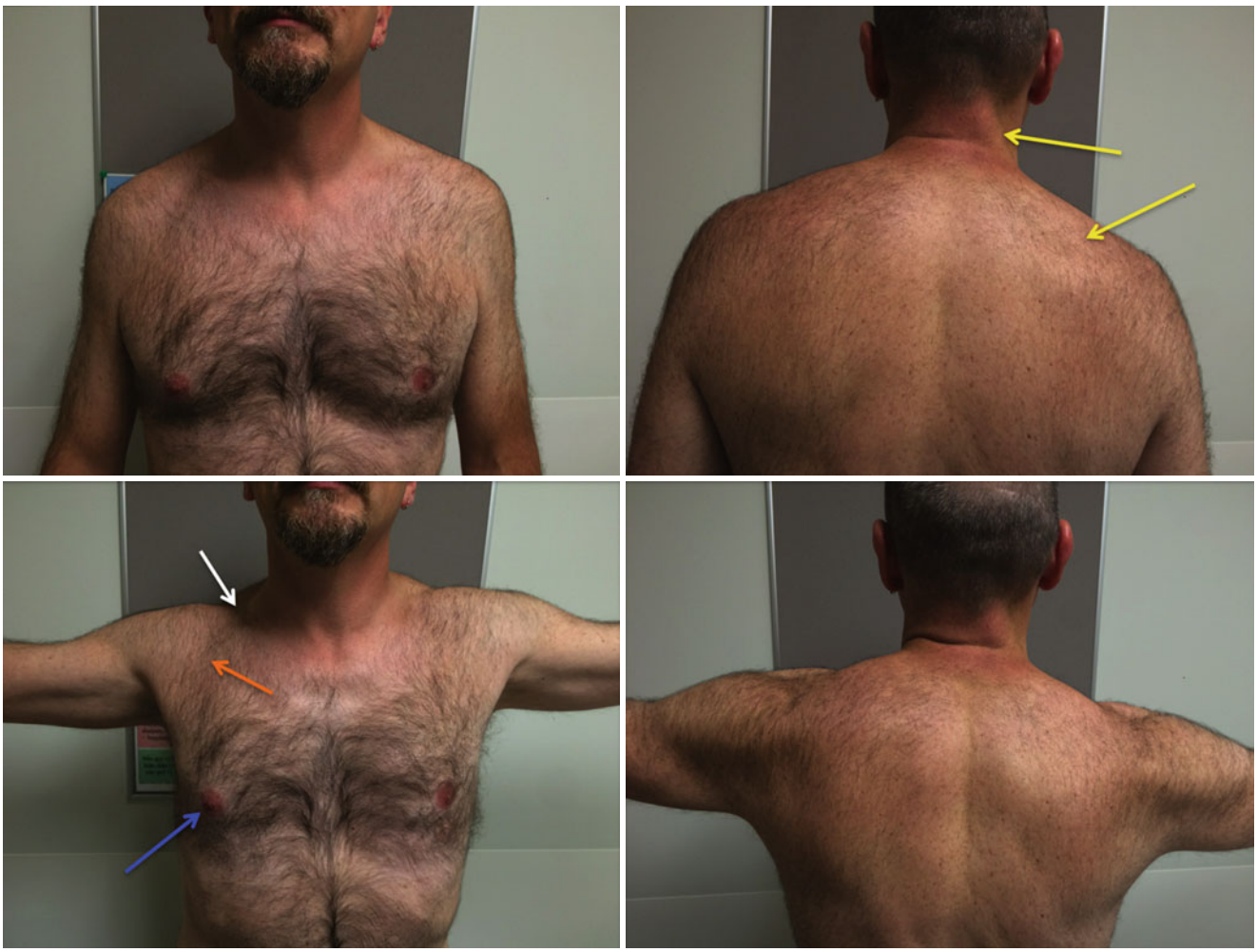

Fig. 27.6 Example of spinal accessory nerve lesion resulting in trapezius wasting, shoulder drop and asymmetry of the neck and shoulder. *Yellow arrows* point to the atrophied trapezius muscle, causing subclavicular and pectoral asymmetry. *White arrow* shows supraclavicular wasting, *orange arrow* shows subclavicular pitting and *blue arrow* shows pectoral drooping (Image courtesy of Agnes Stogicza MD)

27 of the 30 patients had head pain and headaches. After surgery to release the SAN (see *Surgery* section below), 10 of the 27 patients reported "complete relief," while 22 had 50 % relief. The authors concluded that some of the most common symptoms found in chronic flexion-extension injuries (e.g. headaches, stiffness of the neck and pain in the shoulder/neck region) may be due to either primary injury or secondary dysfunction of the spinal accessory nerve and/or the trapezius muscle.

Anatomy (Table 27.2)

The SAN is the 11th cranial nerve and had been traditionally thought to consist of two parts: spinal and cranial. The *cranial root* begins in the *nucleus ambiguous* in the medulla. The *spinal root* originates from a cluster of motor neuron cell bodies in the *accessory nucleus*, located in the lateral part of the anterior horn of the first five segments of the spinal cord, then travels cephalad through the foramen magnum where it was thought to join the cranial section. Once the two parts "join," the SAN exits the cranium through the *jugular foramen* with the *vagus nerve* and *glossopharyngeal* nerve (Figs. 27.7 and 27.8) [2], traveling behind the *styloid process* (Fig. 27.9). Even while traveling together, the spinal and cranial components make few if any distinct connections [3]; it is increasingly thought that what we commonly refer to as the SAN carries motor fibers from the cervical spinal cord and sensory fibers of unclear origin [1].

The spinal branch travels under the posterior belly of the *digastric muscle*; crosses the internal jugular vein, anterior or posterior to the occipital artery; and then passes beneath the SCM. It emerges posterior of the SCM, joins with fibers from C3 to C4, and then travels obliquely down across the floor of the posterior cervical triangle (on top of the levator scapula) to enter the trapezius muscle (Fig. 27.10); that distal section

Table 27.2 Spinal accessory nerve anatomy (CN XI)

Origin	*Cranial*: nucleus ambiguus (controversial) [3]
	Spinal: spinal accessory nucleus
General route	*Cranial*: joins spinal section inside cranium (controversial) [3]
	Spinal: travels cephalad to join cranial section
	Combined: exits cranium through jugular foramen. Cranial section joins vagus. Spinal section travels under digastric, posterior to SCM, enters trapezius
Sensory distribution	None, just communicating branches with vagus nerve, greater and posterior auricular nerve (GAN/PAN) (see Chap. 16), lesser occipital nerve (LON) (see Chap. 18)
Motor innervation	Trapezius (partial): also receives innervation from cervical plexus
	SCM (partial): also receives innervation from cervical plexus
Anatomic variability	Multiple connections to other nerves, including GAN/PAN, LON, trigeminal and hypoglossal nerves, cervical plexus, stellate ganglion, mandibular branch of the facial nerve, accessory phrenic nerve, brachial plexus
Other relevant structures	*Styloid process*
	Posterior cervical triangle (also known as the *lateral cervical region*): a region of the lateral neck bounded by the SCM anteriorly, the trapezius posteriorly and the clavicle inferiorly. It contains many blood vessels, most notably the external jugular vein, as well as lymph nodes and the trunks of the brachial plexus
	Cervical plexus: the ventral rami of C1–C4 unite and provide sensory and motor innervation to the anterior and lateral neck, including direct contributions to the SCM and trapezius muscles

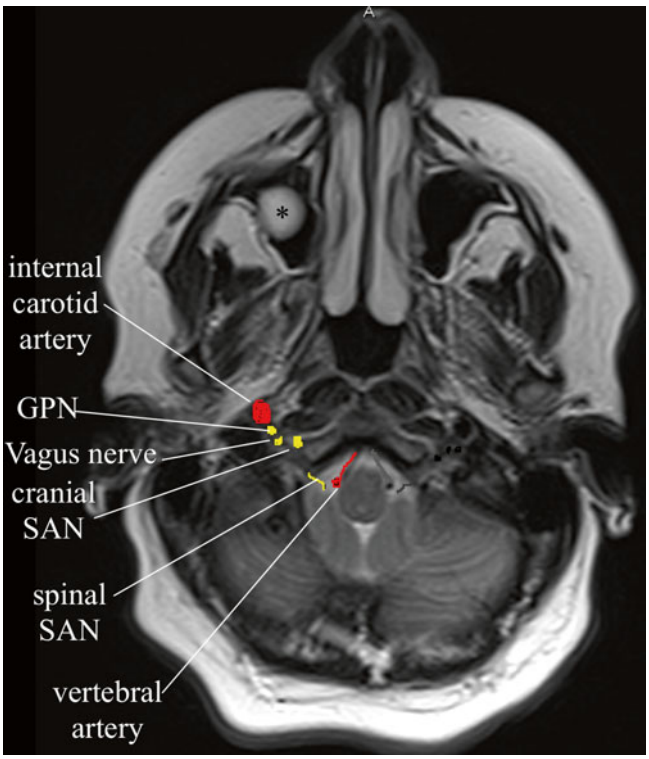

Fig. 27.7 MRI axial image at the level of the foramen magnum. *GPN* glossopharyngeal nerve, *SAN* spinal accessory nerve (cranial and spinal divisions), * = incidental maxillary sinus cyst (Image courtesy of Andrea Trescot, MD)

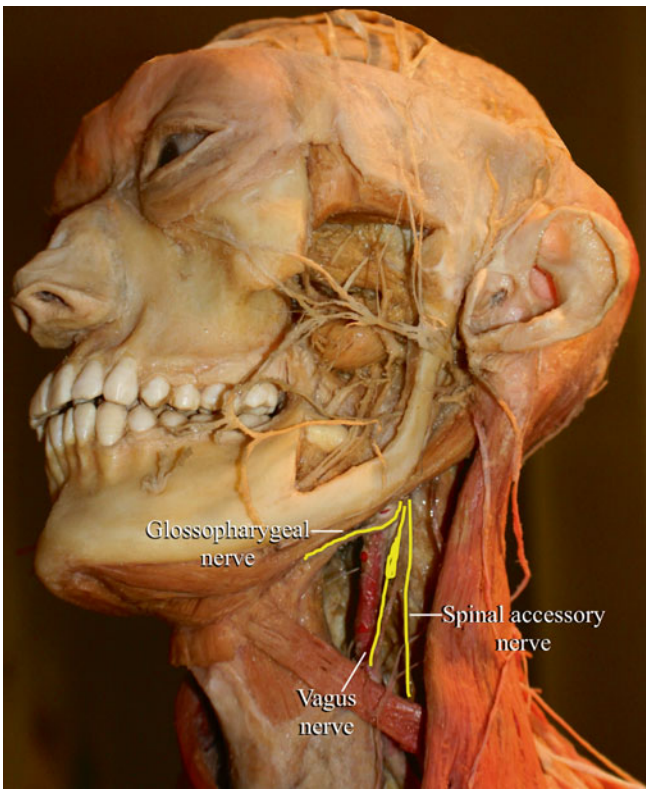

Fig. 27.8 Dissection of the head and neck, showing the relationship between the spinal accessory nerve, the glossopharyngeal nerve, and the vagus nerve in the neck next to the carotid artery (Image modified from an image from *Bodies, The Exhibition,* with permission) (Image courtesy of Andrea Trescot, MD)

is coiled and of variable length, depending on position (4–5 cm when lax with the chin pointing forward, but 9–10 cm when the chin is pointing to the opposite shoulder) [9].

The SAN innervates the trapezius and SCM muscles but has multiple connections to other nerves, likely contributing to the variable signs and symptoms associated with its injury. For instance, the *great or posterior auricular nerve* (Chap. 16) (which arises from C2 to C3) may

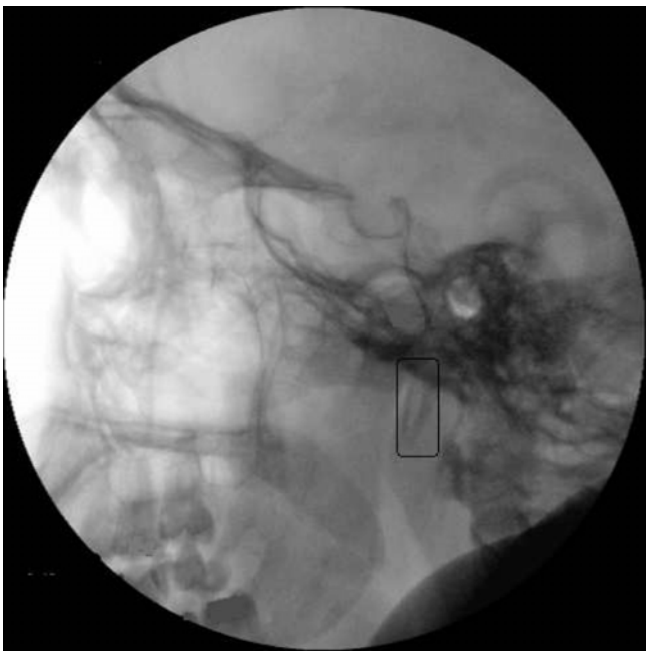

Fig. 27.9 X-ray image of the styloid process (*outlined*) (Image courtesy of Andrea Trescot, MD)

serve as a conduit between the SAN, the *lesser occipital nerve* (Chap. 18), and the lower divisions of the *trigeminal nerve* [9]. In the same way, the *cervical plexus*, which arises from C2, C3, to C4, connects the SAN to the *hypoglossal nerve* (via the ansa hypoglossi), the *stellate ganglion* and the *mandibular branch of the facial nerve* [10]. The SAN is also connected to the *accessory phrenic nerve* and the *brachial plexus* [16].

Entrapment

Because the SAN has been considered a pure motor nerve, the pain from SAN injury has been attributed to SAN entrapment caused by either trapezius compartment syndrome described below or SAN injury and entrapment at higher levels due to trauma. Patients often have ipsilateral of contralateral neck, shoulder, and occipital pain as well as headache and persistent muscle spasm. Nystrom et al. [5] described 16 patients with chronic whiplash symptoms and SAN entrapment after falls or sports injuries. At surgery,

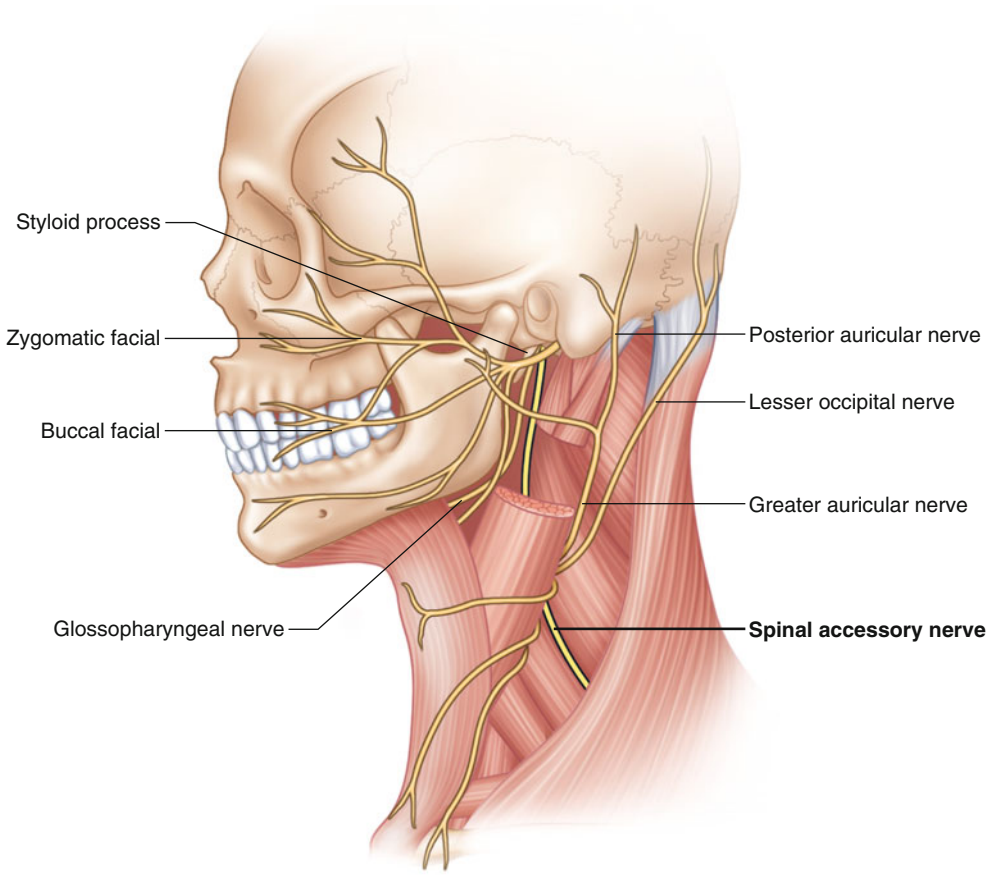

Fig. 27.10 Anatomy of the proximal spinal accessory nerve (Image courtesy of Springer)

they discovered adhesions between the SAN and underlying fascia. If the site is proximal, both the SCM and trapezius muscles will be involved; if it is at the more usual site in the posterior triangle, the trapezius alone is affected.

The *trapezius compartment syndrome* is thought to be due to mechanical drooping of the shoulder causing traction neuritis of the brachial plexus [20–23]. However, histologic evaluation of SAN samples has shown a high proportion of small unmyelinated C fibers, so that the entrapped SAN theoretically could directly carry pain signals [1].

Ewing and Martin first described SAN injury during radical neck dissections in 1952 [24]. This nerve is susceptible to injury because it is small (<2 mm diameter and therefore hard to see surgically) and has a long course across the posterior triangle of the neck, where it is intimately associated with many sets of lymph nodes [1], and postoperative scarring can contribute to entrapment symptoms. Motor neuron disorders, tumors or other neurologic pathology can be responsible for SAN entrapment. Spontaneous SAN dysfunction has also been reported [25].

Physical Exam

Physical examination focuses on assessment of the trapezius and SCM muscles. The patient is positioned standing with both scapulae visible, and areas of shoulder or neckline asymmetry, atrophy, swelling, taut bands and pain are noted (Fig. 27.6). The most consistent physical finding is weakness of arm abduction. The mechanism of injury should be considered when deciding if the muscles on one side have increased tone or the other side shows decreased activity. Tapping or applying pressure along the path of the SAN may elicit tenderness, especially where the SAN is tethered at the styloid process. The non-examining hand stabilizes the head on the contralateral side, while the examining thumb palpates the styloid process to elicit paresthesias (Fig. 27.11).

The other prominent feature of SAN palsy is lateral winging of the scapula. Have the patient stand with the arms by their side, flex the elbow to 90° and externally rotate the shoulder against the examiner's hand to elicit this sign (Fig. 27.4) [26]. If the SAN injury is proximal, SCM weakness on one side may cause unopposed contraction on the other, leading to torticollis (Fig. 27.5).

Restrepo et al. [17] noted a "*subclavicular pit*" (a concavity in the deltopectoral groove that results in a more clear outline of the clavicle) in six patients with EMG-confirmed SAN palsy; the patients were also noted to have a "*pectoral drooping,*" with a prominence and lateral deflection of the breast (Fig. 27.6).

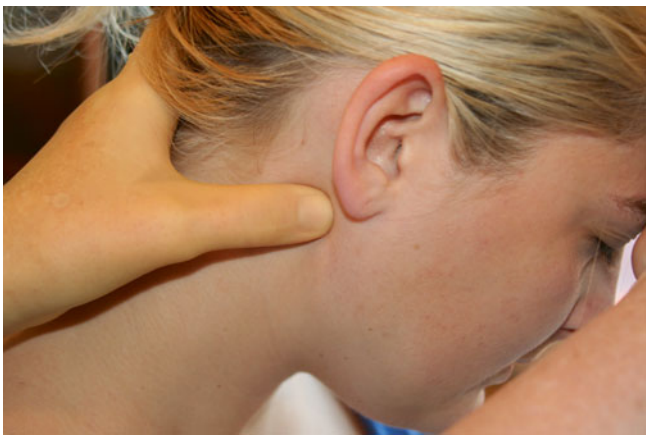

Fig. 27.11 Physical exam of the proximal spinal accessory nerve: the non-examining hand stabilizes the head on the contralateral side, while the examining thumb palpates the styloid process to elicit paresthesias (Image courtesy of Andrea Trescot, MD)

Table 27.3 Differential diagnosis of shoulder and neck pain

	Potential distinguishing features
Long thoracic nerve	Winged scapula on flexion or abduction, not external rotation
Myofascial spasms	No trapezius weakness or atrophy
Cervical facet pathology	Paravertebral tenderness, spondylosis on X-ray
Cervical radiculopathy	Dermatomal pain pattern, sensory changes, muscle weakness in a myotomal distribution, decreased reflexes

Differential Diagnosis (Table 27.3)

Diagnosis is mainly based on history (surgery, trauma or neurologic disorders) and physical exam. SAN palsy can be part of many pathologic lesions, mainly benign neoplasms that affect the 9th–11th cranial nerves. Complex 9th, 10th, and XIth cranial neuropathies (*Vernet syndrome*) usually imply a disease or tumor in the medulla, along the basal cistern, in the jugular foramen, or in nasopharyngeal carotid space; these neuropathies are also associated with aneurysms or basal skull fractures [16] (Fig. 27.7).

Lesions of the long thoracic nerve (see Chap. 30) may give similar winging of the scapula, but the lateral winging seen in long thoracic nerve pathology is elicited by forward flexion of the affected shoulder, while the medial winging of SAN palsy is accentuated by arm abduction or external shoulder rotation [2]. Myofascial trigger points in the cervical and shoulder muscles can give similar pain patterns, but they are not associated with trapezius weakness and atrophy or winged scapula [11]. The comparison of types of winging is found on Table 27.4, and the pattern of shoulder muscle atrophy due to nerve entrapments is found on Table 27.5 (Fig. 27.12).

Table 27.4 Comparison of winging from long thoracic, spinal accessory, and dorsal scapular nerve pathology

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

Table 27.5 Relationship between nerve entrapment and muscle atrophy

Nerve entrapment	Muscle atrophy
Spinal accessory nerve	Trapezius muscle and sternocleidomastoid muscle
Suprascapular nerve (Chap. 28)	Supraspinatus and infraspinatus muscle
Axillary nerve (Chap. 31)	Teres minor
Long thoracic nerve (Chap. 30)	Serratus anterior muscle
Dorsal scapular nerve (Chap. 32)	Rhomboid and/or levator scapula muscle

Diagnostic Tests (Table 27.6)

CT or MRI can help identify primary disease of SAN, e.g., neoplasm; it also helps to visualize muscle atrophy or hypertrophy of the SCM and trapezius caused SAN palsy and compensatory hypertrophy of other muscles (Fig. 27.12). Since the trapezius muscle may have dual nerve supply from the cervical plexus, it can retain some of its function in the face of SAN palsy, which might make the clinical picture less obvious. Other symptoms that result from the trapezius and SCM dysfunction, such as myofascial pain syndromes,

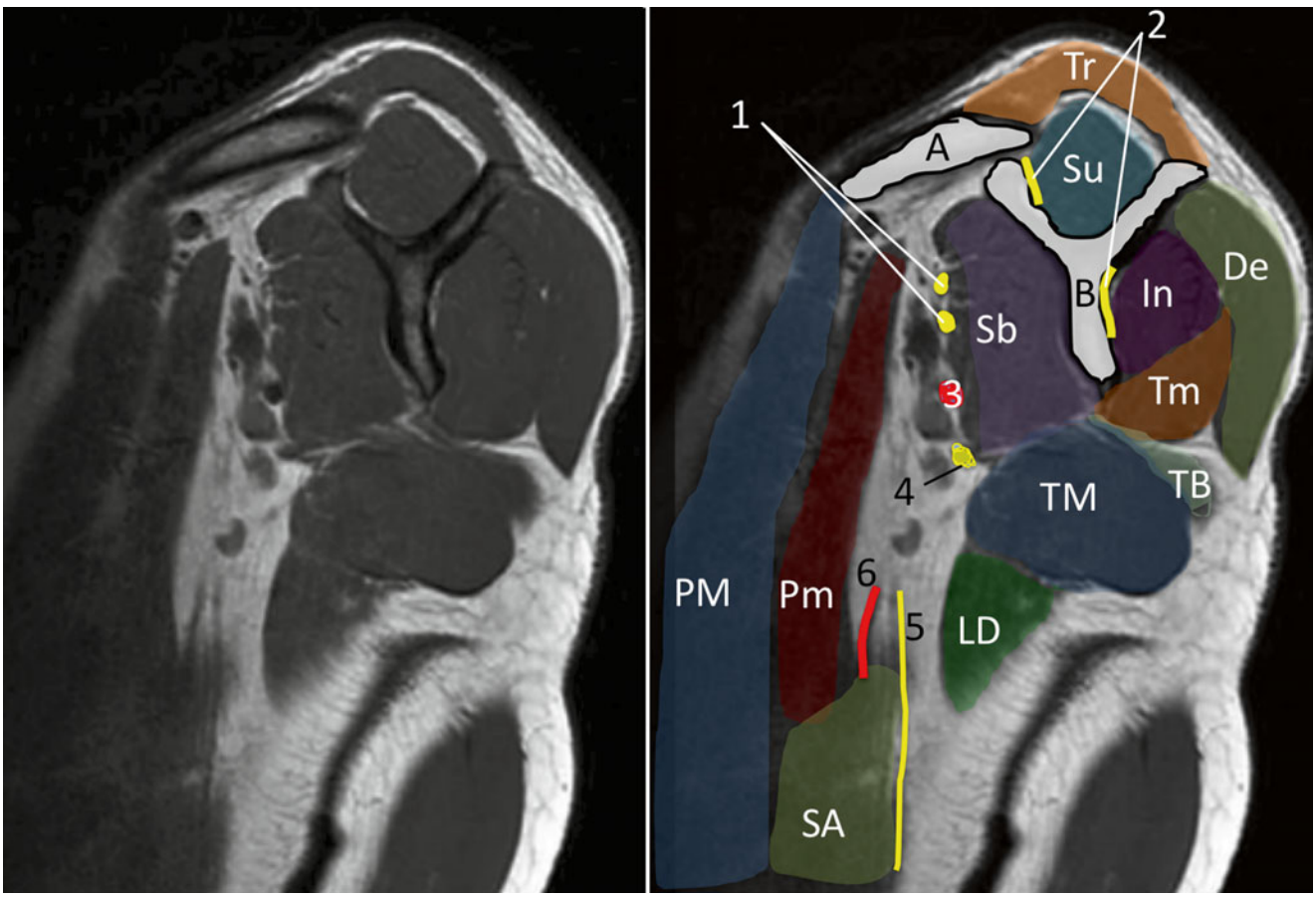

Fig. 27.12 Sagittal MRI image of the scapular structures. *A* clavicle, *B* scapula, *De* deltoid muscle, *In* infraspinatus muscle, *LD* latissimus dorsi muscle, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *Sb* subscapularis muscle, *Su* suprascapular muscle, *TM* teres major muscle, *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 (Image courtesy of Andrea Trescot, MD)

Table 27.6 Diagnostic tests for spinal accessory nerve entrapment

	Potential distinguishing features
Physical exam	Scapular winging, tenderness at styloid, SCM/trapezius spasm or atrophy
Diagnostic injection	Pain relief with early injection may be diagnostic. In the face of atrophy or torticollis, injections may be useful on the contralateral side
Ultrasound	Visualizes nerve, possibly scarring or disruption in posterior triangle
MRI	May show atrophy of trapezius and SCM, scarring or neoplasm
Arteriography	Not useful
X-ray	Not useful
Electrodiagnostic studies	Useful in prevention, somewhat useful in confirming diagnosis

contralateral paresthesias and radiculitis, can further complicate the diagnosis.

Variations in presentation can also be attributed to the specific anatomic level of SAN injury, the amount of collateral tissue damage and subjective pain thresholds. EMG can aid in diagnosis of SAN palsy, but neither the CT and MRI nor the EMG changes are necessary [7].

Clinical symptoms, including the level of shoulder dysfunction, do not necessarily correlate with the electrophysiologic integrity of the SAN [27]. However, range of motion (ROM) testing has shown a positive correlation with EMG. EMG can also be used to track trapezius recovery, plan physical therapy and to monitor SAN function intraoperatively.

Identification and Treatment of Contributing Factors

Forward head posture and posterior cervical ligamentous laxity, because of the already compromised ergonomics, may contribute to accelerated disability, brachial plexus entrapment, thoracic outlet syndrome (see Chap. 33) and misdiagnosis. Early physical therapy for postoperative shoulder dysfunction is "mandatory" [8].

Several authors have described procedures to limit intraoperative damage to SAN, including intraoperative electrophysiologic monitoring [8, 28] and attention to positioning to avoid hyperextension and traction on the SAN.

Friedenberg et al. [29] looked at 56 spinal accessory neuropathy patients (confirmed by EMG) at Mayo Clinic over a 22-year period. Good functional recovery was generally observed, regardless of the results of the electrodiagnostic studies, and no electrodiagnostic findings correlated with poor outcome. However, involvement of the dominant limb, scapular winging and impaired arm abduction were all associated with poor outcome.

Injection Technique and Results

Landmark-Guided Technique

The landmark-guided technique should only be attempted in patients with a neck thin enough to palpate the styloid process. With the head supported, palpate and straddle the styloid process with index and middle fingers of the non-examining hand (Fig. 27.13). A 27-gauge 1.5-inch needle is usually long enough to reach the styloid process; the needle should touch bone and then be redirected posteriorly. A peripheral nerve stimulator (PNS) may help to identify the nerve more accurately. 1 to 2 ccs (1 cc if using PNS) of local anesthetic with or without steroids is injected in divided doses after negative aspiration. Since there are highly vascular structures nearby (specifically, the carotid artery), serious consideration should be given to using non-particulate steroids or no steroid at all, to mitigate the risk of a steroid particle vascular occlusion. Although this complication has not been reported for this particular injection, reports of disasters after injection of particulate steroids into other blood vessels should cause the clinician great concern.

Waldman [30] described a more distal approach to the SAN. With the patient supine and head turned to the contralateral side, the patient is asked to raise their head against the resistance of the examiner's hand in order to identify the posterior border of the upper third of the SCM (Fig. 27.14). The needle is advanced through the skin to a depth of about three-fourths of an inch and 10 cc of local anesthetic with deposteroid infiltrated in a fan configuration.

Fluoroscopy-Guided Technique

With the patient positioned supine, the styloid process is identified (Fig. 27.15). Under fluoroscopic guidance, the needle is advanced to the styloid process and then directed posteriorly. The use of a 25 g 2 inch needle with

Fig. 27.13 Landmark-guided injection of the glossopharyngeal nerve at the styloid process. The non-injecting fingers straddle the styloid process (Image courtesy of Andrea Trescot, MD)

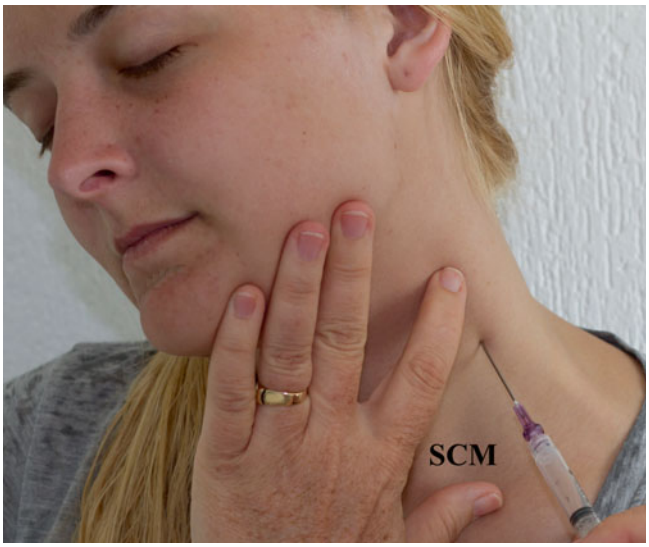

Fig. 27.14 Distal cervical injection of the spinal accessory nerve (Image courtesy of Andrea Trescot, MD)

peripheral nerve stimulator (PNS) will facilitate identification of the SAN by eliciting a scapular twitch. 1 to 2 ccs (1 cc if using PNS) of local anesthetic (with or without steroids) is injected in divided doses after negative aspiration.

Ultrasound-Guided Technique

Bodner et al. [31] originally described the US evaluation of the SAN. Ultrasound 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. The probe is placed over the posterior triangle in a horizontal fashion, so the posterior border of SCM, levator scapulae muscle and possibly the anterior border of trapezius are visualized (Fig. 27.16). The nerve is then identified as a round hypoechoic structure in the connective tissue between SCM and levator scapulae muscle. As the nerve is tracked caudally, it moves superficially and posteriorly toward trapezius.

Canella et al. [32] evaluated the SAN by US in 7 cadavers and 15 volunteers and noted that bone landmarks were not useful for the accurate localization of the SAN. More recently, Mirjalili and colleagues [33] studied 50 healthy volunteers using US; the SAN could be identified in all the subjects running superficially across the posterior triangle with either a straight (56 %) or tortuous (44 %) course at a depth of about 3 mm. They noted that 58 % of the nerves divided into two to four branches before penetrating trapezius, which could lead to confusion at surgery.

Although not yet described in the literature, the injection can be performed by either an out-of-plane approach or an in-plane approach without major risks in well-trained hands, considering the very superficial location of SAN at this level.

Neurolytic/Surgical Techniques

Cryoneuroablation/Radiofrequency Lesioning

Since the SAN is primarily a motor nerve, neurolytic procedures are less common 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.

Pulsed radiofrequency lesioning of the SAN at the styloid has been performed using a combined fluoroscopy/ultrasound technique (personal communication Dr. Christ Declerck), which allowed precise identification of the styloid (fluoroscopy), while ultrasound confirmed the lack of vascular contact (Fig. 27.17).

Chemical Neurolysis

Because of the multitude of critical nerves and blood vessels in this region, alcohol or phenol would not be recommended.

Surgical Techniques

Nystrom et al. [5] performed surgical fasciectomies on 30 consecutive patients with chronic SAN pain after flexion-extension injuries, an average of 41 months after injury.

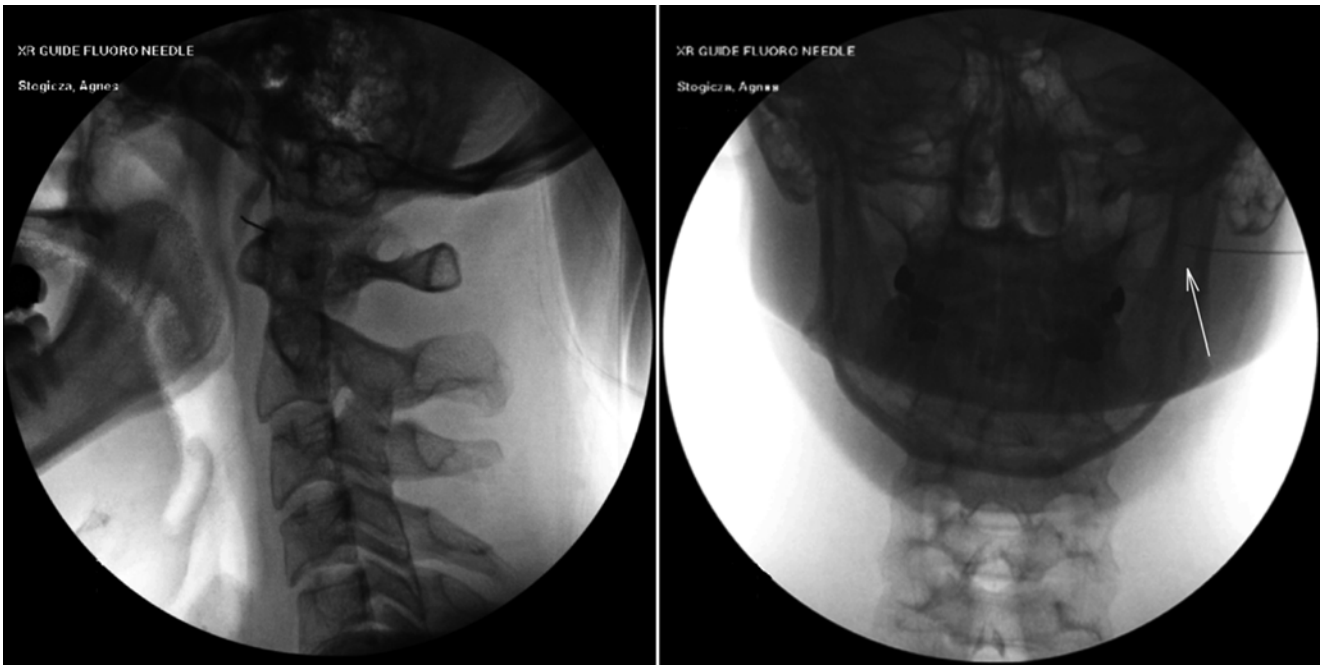

Fig. 27.15 Fluoroscopy-guided injection of the spinal accessory nerve. The lateral view shows the left and right styloid processes overlapping and a 22-gauge 4 cm needle placed onto the styloid process. After bony contact, the needle is redirected posteriorly. The anteroposterior view shows the needle placed onto the styloid process, marked by the white arrow (Image courtesy of Agnes Stogicza, MD)

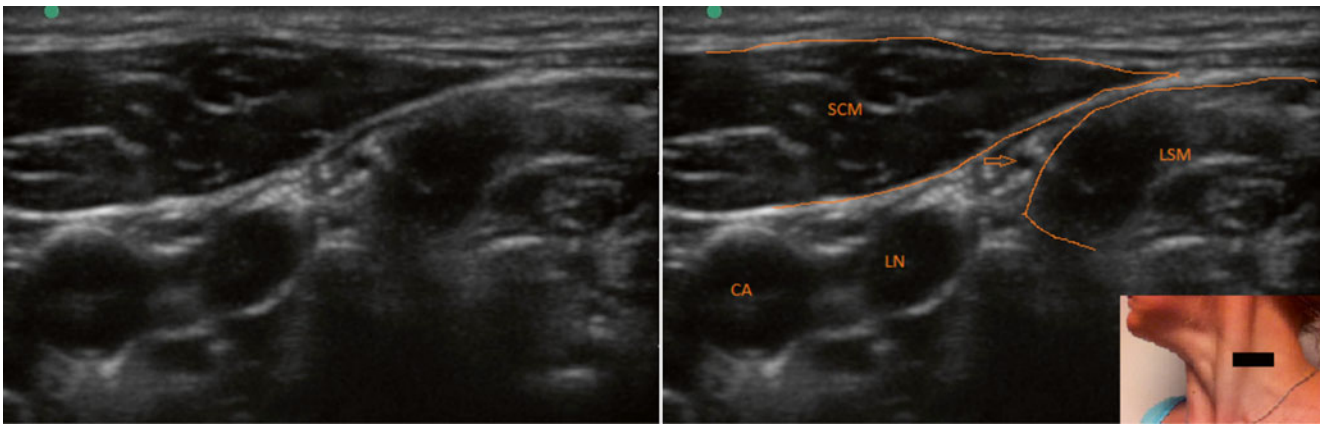

Fig. 27.16 Ultrasound image of the spinal accessory nerve (marked by the *arrow*) at the posterior triangle. *SCM* sternocleidomastoid muscle, *LSM* levator scapulae muscle, *CA* carotid artery, *LN* lymph node. Internal jugular vein is compressed (Image courtesy of Agnes Stogicza, MD)

Hagert and Christenson [34] reported that they treated chronic compartment syndrome of the trapezius and entrapment of the SAN in patients with a history of "*arm overuse syndrome,*" with a pain pattern similar to the pattern of pain seen in patients with chronic flexion-extension injuries. They described spinal accessory nerve decompression at the level of trapezius, recommending the removal of the thickened fascia, including the septa between bundles of the muscle.

Surgical repair of the spinal accessory nerve or muscle transfer is performed in patients with direct trauma of the SAN and also with spontaneous trapezius palsy [28]. Chandawarkar and colleagues [7] reported on the treatment of six patients with SAN dysfunction after cervical lymph node biopsies. Pain was the most common symptom and loss of sustained arm abduction was the most common finding. Three patients had a primary nerve repair and the other three patients had nerve grafting. All six were pain-free postoperatively, with varying degrees of motor function recovery. The authors stressed the need for prevention as well as early intervention rather than "watchful waiting."

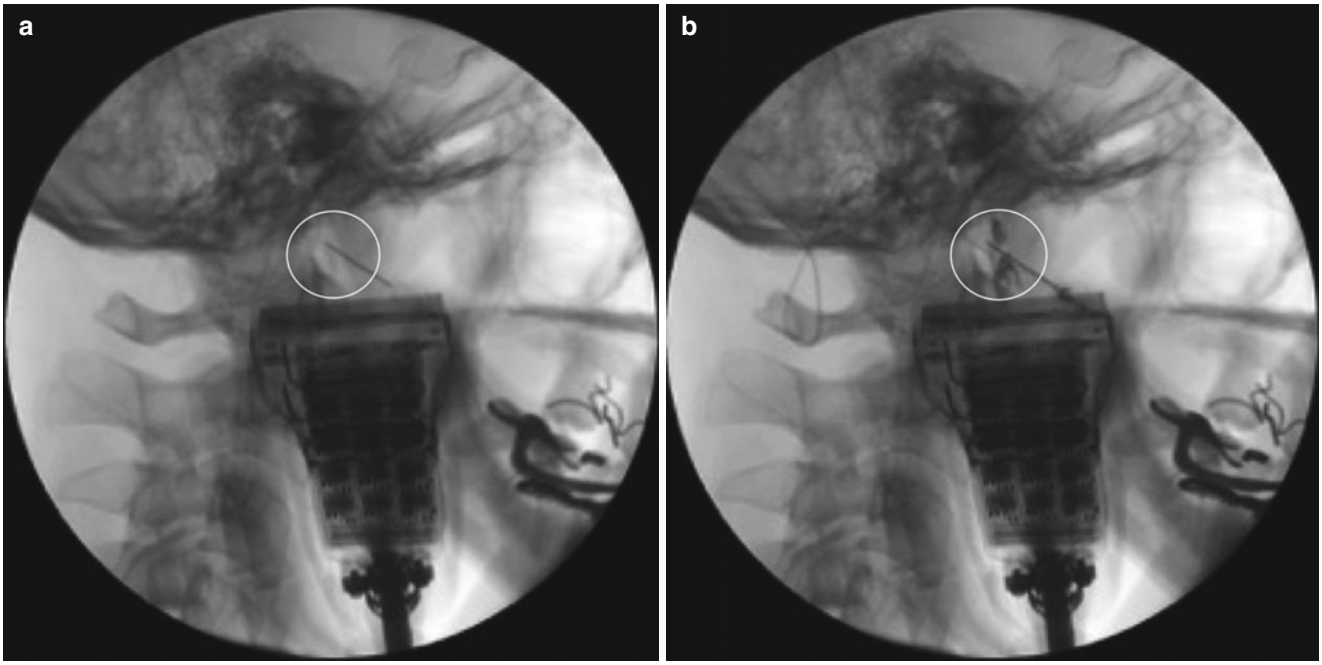

Fig. 27.17 Combined fluoroscopy/ultrasound-directed pulse radiofrequency lesioning of the spinal accessory nerve (Image courtesy of Christ DeClerck, MD General Hospital Sint Jan, Bruges AV, Belgium)

If SAN injury is diagnosed within 1 year, microsurgical reconstruction should be considered [35]. If diagnosed later, surgically repositioning the functioning shoulder muscles, known as the *Eden-Lange procedure,* can be performed; the insertions of the *levator scapulae*, *rhomboideus minor*, and *rhomboideus major* muscles are transferred, which relieves pain, corrects deformity, and improves function in patients with irreparable injury to the spinal accessory nerve [36, 37]. Treatment is less likely to succeed when the patient is older than 50 or the SAN palsy was due to a radical neck dissection, penetrating injury, or spontaneous palsy [28].

Complications

The SAN sits directly over the carotid artery and jugular vein, so intravascular injections are potentially encountered when injecting SAN at the styloid level. At the styloid level, the SAN is also in close relation with vagus, glossopharyngeal and hypoglossal nerves, and there may be unexpected anesthesia or neurolytic effect.

More distal injections (e.g., posterior triangle or in the trapezius muscle) carry significantly less risk when appropriate attention is paid to the depth, the external and internal jugular veins, the carotid artery and the apex of the lung. Ultrasound use, with constant visualization of the needle, should mitigate the risk of injuring these structures.

Summary

Injury to the SAN can be difficult to diagnose without a high index of suspicion. A careful history, identifying a flexion-extension injury or surgical trauma, as well as a careful physical exam, looking for trapezius and SCM atrophy and scapular winging, can lead to the correct diagnosis and therefore appropriate treatment.

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