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Current Concepts in the Management of Neurogenic Thoracic Outlet Syndrome: A Review

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Background: Thoracic outlet syndrome is a constellation of signs and symptoms due to compression of the neurovascular bundle of the upper limb. In particular, neurogenic thoracic outlet syndrome can present with a wide constellation of clinical manifestations ranging from pain to paresthesia of the upper extremity, resulting in a challenge to correctly diagnose this syndrome. Treatment options range from nonoperative treatment, such as rehabilitation and physical therapy, to surgical correction, such as decompression of the neurovascular bundle.

Methods: Following a systematic review of the literature, we describe the need for a thorough patient history, physical examination, and radiologic images which have been reported to correctly diagnose neurogenic thoracic outlet syndrome. Additionally, we review the various surgical techniques recommended to treat this syndrome.

Results: Postoperative functional outcomes have been shown to be more favorable in arterial and venous thoracic outlet syndrome (TOS) patients when compared with neurogenic TOS patients, likely due to the ability to completely remove the site of compression in cases of vascular TOS as compared with incomplete decompression in neurogenic TOS.

Conclusions: In this review article, we provide an overview of the anatomy, etiology, diagnostic modalities, and current treatment options of correcting neurogenic TOS. Additionally, we offer a detailed step-by-step technique of the supraclavicular approach to the brachial plexus, a preferred approach for decompressing neurogenic TOS. *(Plast Reconstr Surg Glob Open 2023; 11:e4829; doi: 10.1097/GOX.0000000000004829; Published online 3 March 2023.)*

INTRODUCTION

The term thoracic outlet syndrome (TOS) was first coined in 1956 by Peet et al[1] to collectively describe the various signs and symptoms caused by compression of the neurovascular bundle exiting an area above the first rib and behind the clavicle, also known as the thoracic outlet.[2] In order of most clinically prevalent, TOS can be classified as either neurogenic (nTOS), venous (vTOS), or arterial (aTOS) based on the compression of the brachial plexus, subclavian vein, or subclavian artery, respectively. Due to its different etiologies, TOS can manifest in a number of diverse clinical presentations.

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When compared with vascular TOS, which encompasses vTOS and aTOS, neurogenic TOS is the more prevalent subtype, accounting for approximately 90%–95%[3,4,10] of cases. Commonly, in nTOS, the brachial plexus is compressed at the interscalene space or retropectoralis space.[5] Symptoms of nTOS can include upper extremity weakness, tingling, numbness, paresthesia, and pain in a nonradicular distribution and may even be further categorized as secondary to upper, lower, or combined plexus compression.[6,7] In a majority of nTOS patients (85% to 90%),[8] combined plexus pathology is seen. Due to the diversity of presentations seen in patients with nTOS,[9] the clinician is often faced with a diagnostic conundrum, and nTOS is frequently the diagnosis of exclusion.

Although the understanding of TOS has improved since it was first identified, the diagnosis and treatment options remain controversial. We seek to review and

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evaluate current published diagnostic and treatment options to manage nTOS and to discuss our approach for appropriate diagnosis and surgical decompression of nTOS.

Takeaways

Question: What are the current methods to properly diagnose and manage neurogenic thoracic outlet syndrome (TOS)?

Findings: This article discusses the need for an understanding of the clinical signs and symptoms of TOS for an accurate diagnosis. We also go into deep discussion of the varying approaches to perform a decompression of the brachial plexus and advocate for a specific approach.

Meaning: Neurogenic thoracic outlet syndrome is a constellation of signs and symptoms that warrants a general knowledge of the clinical signs, symptoms, and treatment options for appropriate management.

ANATOMY

To fully understand the pathophysiology and management of TOS, the relevant anatomy of the thoracic outlet should be appreciated. The thoracic outlet compromises the space from the supraclavicular fossa to the axilla that passes between the clavicle and the first rib (Figs. 1, 2).[3,5,11,12] The thoracic outlet contains three important compartments that are involved in the pathophysiology of TOS: the (inter)scalene space, the costoclavicular space, and the retropectoralis minor space (Figs. 1, 3). (**See Table, Supplemental Digital Content 1**, compartment contents, **http://links.lww.com/PRSGO/C419**.)

The interscalene space is the most medial compartment in the thoracic outlet and is comprised of the anterior scalene muscle (anteriorly), middle scalene muscle (posteriorly), and first rib (inferiorly) (Fig. 1B).[3] Its contents include the subclavian artery and the upper, middle, and lower trunks of the brachial plexus. Distal to the interscalene space, the costoclavicular space is formed by the clavicle (superiorly), subclavius muscle (anteriorly), first rib (inferiorly), and anterior scalene muscle (posteriorly) (Figs. 1, 3). The contents of this space include the divisions of the brachial plexus, subclavian artery, and subclavian vein. The lateral most space in the thoracic outlet is the retropectoralis minor space (also known as the subcoracoid space), which is formed by the coracoid (superiorly), ribs 2–4 (posteriorly), and pectoralis minor muscle (anteriorly) (Figs. 1, 3). Its contents include the cords of the brachial plexus, axillary artery, and axillary vein. Compression of the brachial plexus, subclavian artery, or subclavian vein in any of the compartments aforementioned can result in signs and symptoms associated with thoracic outlet syndrome.

ETIOLOGY

Compression of the neurovascular bundle exiting the thoracic outlet can be due to repetitive overhead movements, injuries, and/or developmental abnormalities.[4] Abnormalities in the thoracic outlet, which can either be congenital or acquired, can be further subdivided into soft tissue (70%) or osseous etiologies (30%).[6]

Soft-tissue abnormalities include variation in the scalene origin and insertion, hypertrophy of the scalene musculature, or congenital anomalous ligaments or bands. The scalenus minimus muscle, found posterior to the lower portion of the anterior scalene, can be found in 30%–50% of patients with TOS.[13] Osseous abnormalities associated with TOS include the presence of a cervical rib, prominent C7 transverse process, and clavicular malunions. One of the most cited causes of TOS due to an osseous abnormality is compression by a cervical rib,

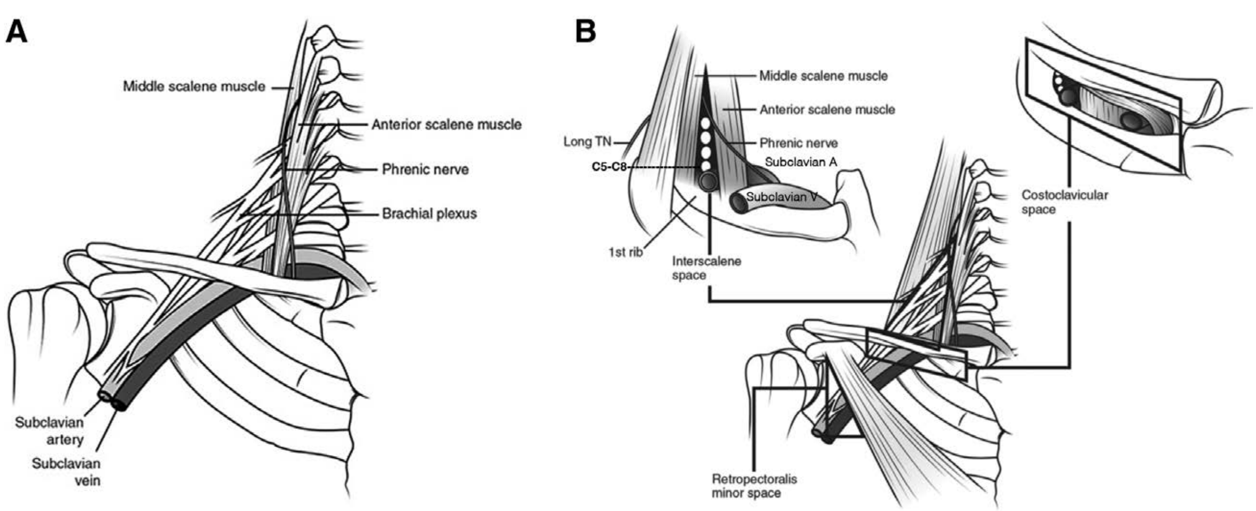

Fig. 1. Normal and pathologic thoracic anatomy. A, Normal thoracic anatomy. B, Anatomy of thoracic outlet, identifying the three potential regions of compression: interscalene triangle, costoclavicular space, and retropectoralis minor (subcoracoid) space. Adapted with permission from Wolters Kluwer Health, Inc.: Kuhn J, Lebus G, Bible J, Thoracic Outlet Syndrome, *Journal of the American Academy of Orthopaedic Surgeons.* 2015;23(4). doi: 10.5435/JAAOS-D-13-00215.

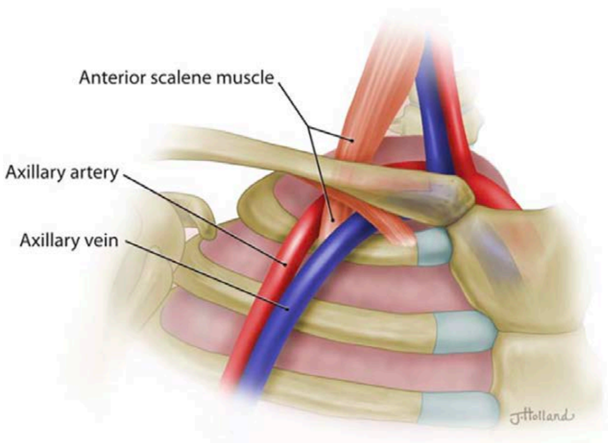

Fig. 2. Schematic depicting the thoracic outlet and the relationship of the axillary vein, anterior scalene, and axillary artery from anterior to posterior. Adapted with permission from John Wiley and Sons: Loukas M, Shirk S, Shah R et al. Thoracic outlet syndrome: a neurological and vascular disorder. *Clinical Anatomy*. 2013;27(5):724–732. doi: 10.1002/ca.22271.

with up to 20% of nTOS cases attributable solely to the presence of a cervical rib.[14] A study that examined 200 patients undergoing surgical correction of TOS discovered that 8.5% of patients had a cervical rib articulating with the first thoracic rib, 10% had supernumerary scalene muscles, and 43% had variations in scalene muscle attachments.[15]

Repetitive overhead movements can lead to hypertrophy of the scalene muscles, swelling, hematomas, and subsequent fibrosis, causing compression of the neurovascular bundle.[3] For example, cases of nTOS have been reported in young, active individuals, who participate in athletic activities that involve repetitive overhead upper extremity motion and heavy lifting.[9]

Posttraumatic causes of TOS most commonly include falls and hyperextension-flexion (whiplash) injuries, which may account for approximately 35% of all cases of TOS.[16] Mid-shaft clavicular fracture malunion may also cause compression of the thoracic outlet, resulting in nTOS.[17–20] Additional causes of TOS include malignancies, such as Pancoast tumors (superior sulcus or apical lung tumors), which can invade and compress the brachial plexus.[21]

DIAGNOSIS/DIAGNOSTIC MODALITIES

A thorough history, physical examination, and radiologic imaging remain instrumental in differentiating neurogenic TOS from other conditions such as cervical radiculopathy, carpal tunnel, or cubital tunnel syndrome. All three conditions can present similarly to nTOS with vague complaints of numbness and tingling in the unilateral or bilateral upper extremity.[22,23]

During the physical examination, the clinician should initially evaluate the patient for the presence of unilateral or bilateral signs of swelling, cyanosis, or pallor, in the upper limbs as well as atrophy of the hands, specifically the thenar and hypothenar muscles.[24] The Gilliatt-Sumner hand (Fig. 4), a characteristic finding of nTOS, is described as atrophy of the abductor pollicis brevis, the hypothenar musculature, and the interossei.[25] Palpation of the supraclavicular region may demonstrate tenderness, masses, the existence of a cervical rib, or other osseous abnormalities.[26] Strength and sensation should be assessed, as prolonged compression of the plexus may cause weakness and paresthesias of the affected muscles. Several provocative tests[27–31] may also be performed to help narrow-in on the diagnosis and cause of TOS, as described in Table 1. Images depicting proper performance of the upper limb tension test and the scratch collapse test have been included (Figs. 5, 6).

Following a thorough history and physical examination that may indicate a diagnosis of TOS, chest and cervical spine radiographs should be obtained to assess for the presence of a cervical rib, prominent C7 transverse processes, clavicular malunion, or other osseous abnormalities that may be compressing the thoracic outlet. Ultrasonography can also be used to detect the "wedge-sickle sign," a hyperechoic fibromuscular structure at the medial edge of the middle scalene muscle, which indents the lower trunk of the brachial plexus and may be found in some cases of nTOS.[32]

CT and MRI may be effective in the diagnosis of nTOS when it is suspected to be due to a congenital anomaly, metastatic disease, space-occupying lesions, or malunited fractures of ribs or the clavicle. These imaging modalities are also important for presurgical planning to more precisely localize areas of compression to be targeted intraoperatively. Additionally, MRI of the brachial plexus is often performed and can demonstrate compression of the neurovascular elements when the patient is in the ABER (abduction and external rotation) arm position and can aid in diagnosis.

Electrodiagnostic (EDX) studies, such as nerve conduction studies and electromyography, can be useful methods in ruling out other conditions mimicking nTOS as mentioned above. In the case of nTOS, most EDX studies are normal. On rare occasions though, they can highlight which areas of the plexus are affected. Conversely, radiculopathy would produce EDX changes in dermatomal and myotomal patterns, while carpal and cubital tunnel syndromes would produce EDX changes in the territories of the affected peripheral nerves.[22,23] Ultrasound-guided intramuscular scalene injections using local anesthetics have also been used in diagnosing TOS and predicting response to surgery.[31] We heavily rely on this diagnostic and prognostic modality in our practice, and it must be performed before considering surgery.[33,34]

In 2013, the Consortium for Outcomes Research and Education on TOS developed preliminary diagnostic criteria for nTOS (Table 2).[32] While this list is the first of its kind to help diagnose nTOS, it does not assign a level of importance to each component of the criteria, nor does it guide treatment. As a result, diagnosis of TOS is currently based on a combination of history, physical examination, and EDX and imaging studies, rather than a specific set of criteria.

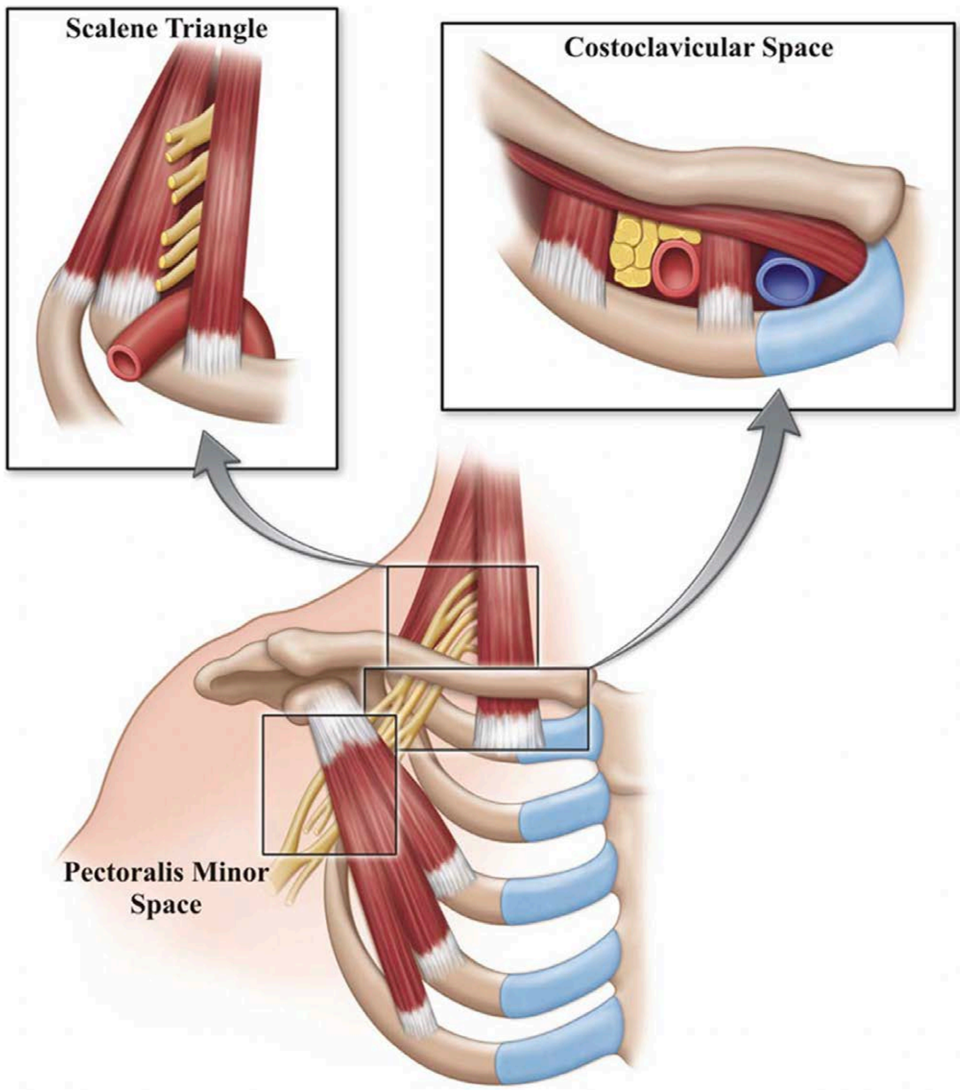

Fig. 3. Schematic depicting the compartments of the cervicoaxillary canal and the course of the brachial plexus, demonstrating three possible sites for compression and nTOS. Adapted with permission from John Wiley and Sons: Loukas M, Shirk S, Shah R et al. Thoracic outlet syndrome: a neurological and vascular disorder. *Clinical Anatomy*. 2013;27(5):724–732. doi: 10.1002/ca.22271.

TREATMENT OPTIONS

Nonsurgical

In neurogenic TOS that is activity-dependent and/or presenting with intermittent symptoms, nonsurgical treatment is usually the preferred first choice of management, which includes rehabilitation and thoracic outlet-specific physical therapy that focuses on range-of-motion exercises and targeted muscle strengthening.[24] A study demonstrated that 25 of 42 patients with nTOS experienced symptomatic relief after at least 6 months of physical therapy.[35] However, if symptoms do not improve with physical therapy, botulinum toxin A injections into the anterior and middle scalene muscles have also provided patients with short-term relief.[6,36] The effectiveness of botulinum toxin A injections into the anterior scalene, middle scalene, and pectoralis minor has been improved when performed under ultrasound guidance, as shown in Figure 7.[37] If there is no improvement in symptoms following 6 months of nonoperative treatment, surgical options are discussed.

Surgical

Surgical intervention for patients with nTOS is warranted for any patient who does not have an appropriate response to nonsurgical management, has muscle atrophy on examination, or has sustained or persistent neurological dysfunction.[37,38] There are three main surgical approaches for decompression of the thoracic outlet: transaxillary, supraclavicular, and posterior. Although there is much conflict regarding the optimal surgical approach reported in the literature, the surgeon will

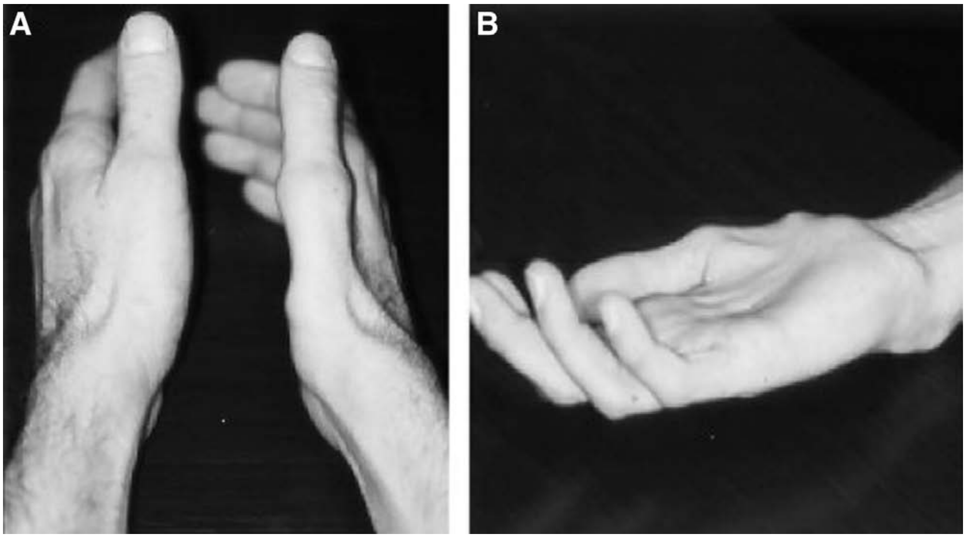

Fig. 4. Photographs demonstrating right-sided Gilliat-Sumner hand due to nTOS, as demonstrated by atrophy of the interossei dorsally (A) and the hypothenar and abductor pollicis brevis volarly (B). Adapted with permission from Wolters Kluwer Health, Inc.: Huang J, Zager E. Thoracic outlet syndrome. Neurosurgery; 2004:55(4):897–902; discussion 902–903.

Table 1. Common Provocative Maneuvers for Diagnosing TOS

Test	How to Perform	Indicates
Adson test	Radial pulse palpated with arms at sides. Patient then inhales, holds breath, and rotates neck toward side being tested. Positive test if pain/parasthesia or changes in radial pulse elicited.[7]	Compression of brachial plexus at thoracic outlet (pain/parasthesia elicited) Compression of subclavian artery (change in radial pulse)
Roos test	Patient places both arms in the 90° abducted position with the elbows flexed to 90° while opening and closing his or her hand. Positive test if the patient has pain/parasthesia that limit completion of the test.[8]	Compression of the brachial plexus at thoracic outlet
Wright test	Radial pulse palpated with arms at sides. Arm placed into abduction over patient's head and held for 1–2 min. Positive test if pain/parasthesia elicited or change in radial pulse.[7]	Compression of brachial plexus at thoracic outlet (pain/parasthesia elicited) Compression of subclavian artery (change in radial pulse)
Costoclavicular test	Radial pulse is first recorded while the patient sits straight with his or her arms by their side. Patient then retracts and depresses his or her shoulders while the chest is protruded for 1 min. Positive test if there is a change in radial pulse and/or pain/parasthesia in the upper limb.[7]	Compression of brachial plexus at thoracic outlet (pain/ parasthesia elicited) Compression of subclavian artery (change in radial pulse) or subclavian vein
ULTT	Three positions Abduct both arms to 90° with elbow straight. Dorsiflex both wrists. Tilt head to either side. Positive test if pain/parasthesia elicited.[9]	Compression of brachial plexus at thoracic outlet
Tinel's test	Patient sits upright with his or her arms by their side. The supraclavicular fossa is palpated with a reflex hammer. Positive test if pain or parasthesia in the arm and/or tenderness is elicited.[29]	Compression of the brachial plexus at thoracic outlet
Scratch collapse test	While the patient is exerting bilateral external shoulder rotation, a stimulus is asserted over the suspected nerve compression.[30]	Can be used to localize the site of peripheral nerve compression

ULTT, upper limb tension test.

choose one or a combination of approaches based on the underlying etiology and surgeon preference.

The transaxillary technique was first described in 1966 by Roos[39] and offers a good exposure for resection of the first rib, cervical rib, or proximal fibrous bands.[40] However, the transaxillary approach is a relatively limited approach to the brachial plexus and requires significant retraction on the neural elements, placing the patient at increased risk for iatrogenic injury.[41–45] For many, especially vascular surgeons, the transaxillary approach is the preferred technique when performing first rib and costoclavicular ligament resection, scalenectomy, and C7-T1 neurolysis.[46] However, many brachial plexus and peripheral nerve reconstructive surgeons favor a supraclavicular approach, especially in the setting of reoperation.[47]

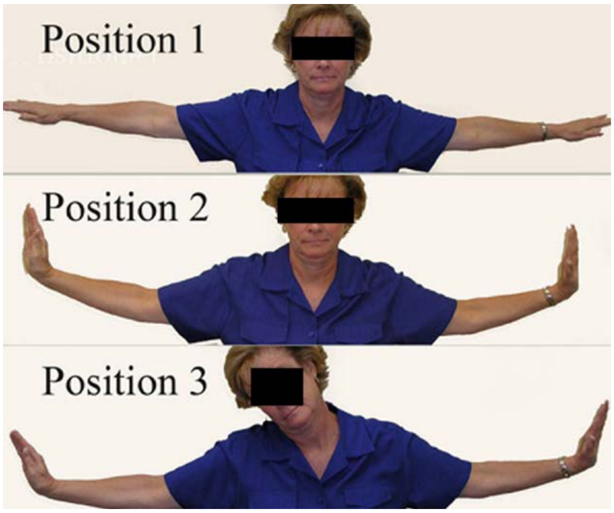

Fig. 5. Proper demonstration of the upper limb tension test. Adapted from *J Vasc Surg.* 2007;46:601–604. Adapted with permission from Elsevier: Sanders RJ, Hammond SL, Rao NM. *Journal of Vascular Surgery*. 2007;46:601–604.

For a more favorable exposure of the brachial plexus, the first rib, and the surrounding vascular structures, the supraclavicular approach is recommended. The supraclavicular approach provides access to the superior, middle, and inferior trunks, as well as the roots of the brachial plexus and scalene musculature. In isolated scalenectomies and cervical rib removal for nTOS, the supraclavicular approach is ideal.[48] The supraclavicular technique has been shown to have superior outcomes and fewer complications when performed for first rib resection as compared with the transaxillary approach.[49]

The posterior approach was initially described in 1962 by Clagett[49] and provides exposure of the proximal elements of the brachial plexus for neurolysis. This approach is more invasive, with increased risk of postoperative shoulder morbidity and scapular winging,[50] and has fallen out of favor.

OUTCOMES

Nonsurgical treatment through education and physical therapy demonstrated success in 59% to 88% of TOS cases after a 2-year follow-up.[51] Although nonsurgical therapy is recommended as the first line of treatment, surgical intervention is often advocated to achieve long-term symptom relief.

Postoperative functional outcomes for vascular TOS, such as aTOS and vTOS, have been reportedly better than those for nTOS. In a meta-analysis study, postoperative symptomatic improvement was excellent or good in 90% of cases of aTOS and vTOS, but only 56%–89% in nTOS cases.[52,53] Furthermore, Al Rstum et al[54] documented that out of 105 patients who underwent a paraclavicular approach to decompression for TOS, good or excellent results were reported in 85% of those with vTOS when compared with only 67% of nTOS patients reporting good or excellent results. In the same study, patients with

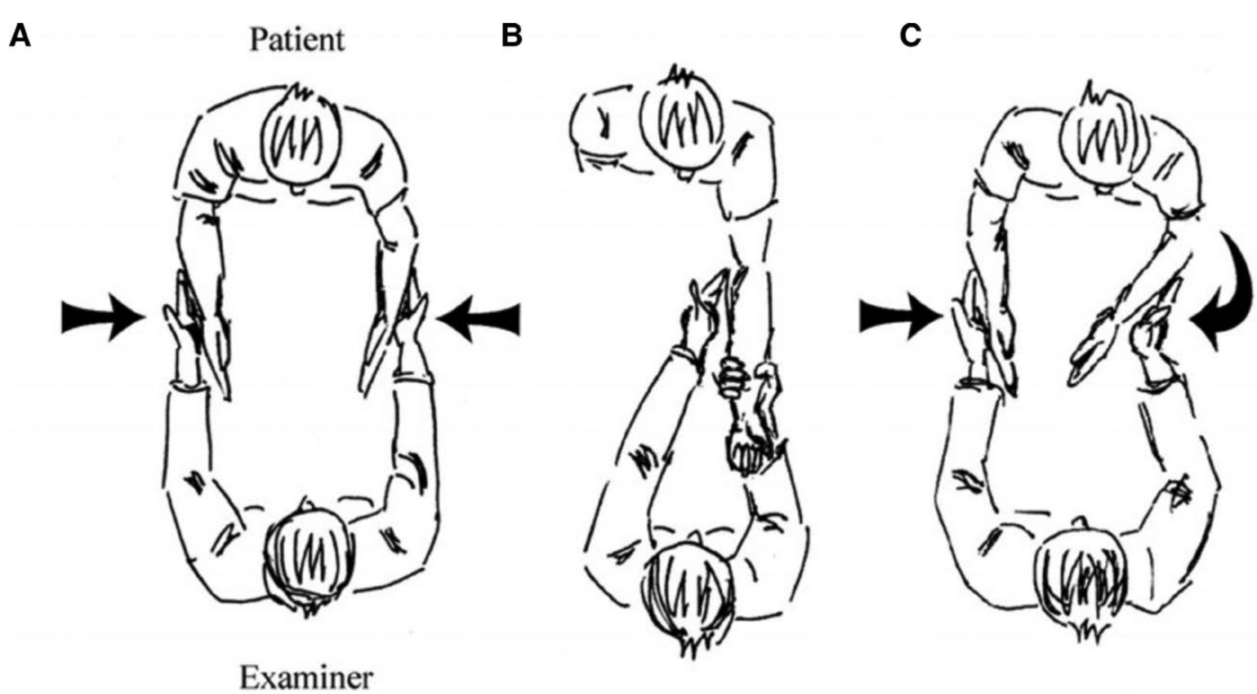

Fig. 6. Illustration of the scratch collapse test. A, The patient resists bilateral shoulder adduction/internal rotation to the forearms applied by the examiner. B, Next, the examiner "scratches" or swipes with fingertips over the course of the compressed nerve (ulnar nerve at elbow illustrated). C, Step A is immediately repeated. Brief temporary loss of the patient's external resistance tone is considered a positive scratch collapse test. Adapted with permission from Elsevier: Montgomery K, Wolff G, Boyd KU. Evaluation of the scratch collapse test for carpal and cubital tunnel syndrome—a prospective, blinded study. *J Hand Surg Am*. 2008;33:1518–1524.

Table 2. Diagnostic Criteria for Neurogenic TOS

To diagnose nTOS, the following must be true:	
Upper extremity symptoms extend beyond the distribution of a single cervical nerve root or peripheral nerve root	
Symptoms are present for at least 3months	
Symptoms are not explained by another condition	
Meet at least one criterion in at least four of the following five categories:	
1. Principal symptoms	1A: pain in neck, upper back, shoulder, arm, and/or hand
	1B: numbness, paresthesia, and/or weakness in arm, hand, or digits
2. Symptom characteristics	2A: pain/paresthesia/weakness exacerbated by elevated arm positions
	2B: pain/paresthesia/weakness exacerbated by prolonged or repetitive hand/arm use
	2C: pain/paresthesia radiating down the arm from the supraclavicular or infraclavicular spaces
3. Clinical history	3A: symptoms began after occupational, recreational, or accidental injury of the head, neck, or upper extremity
	3B: prior ipsilateral clavicle or first rib fracture, or known cervical rib
	3C: prior cervical spine or ipsilateral peripheral nerve surgery without sustained improvement in symptoms
	3D: prior conservative or surgical treatment for ipsilateral TOS
4. Physical examination	4A: local arm tenderness on palpation over the scalene triangle and/or subcoracoid space
	4B: arm/hand paresthesia on palpation over scalene triangle and/or subcoracoid space
	4C: objectively weak hand grip, intrinsic muscles, or thenar/hypothenar atrophy
5. Provocative maneuvers	5A: positive upper limb tension test
	5B: positive elevated arm stress test

Adapted from the work by Thompson 2013.

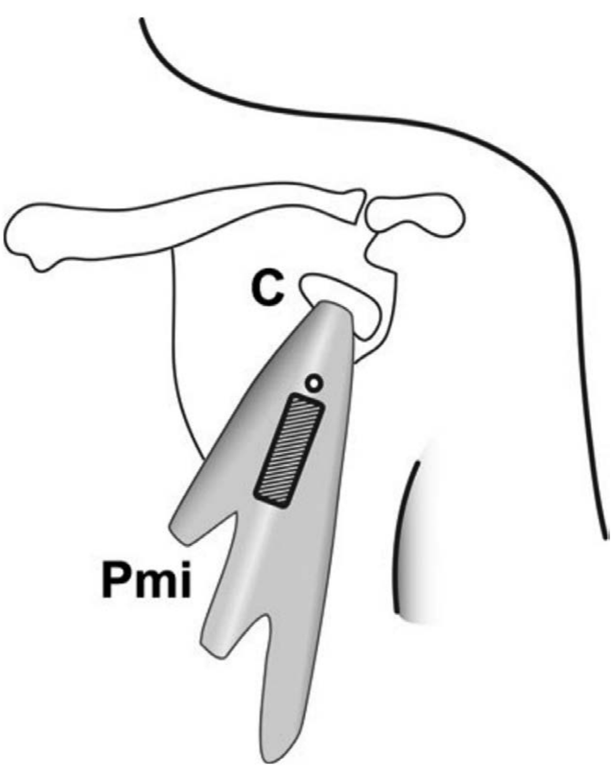

Fig. 7. Schematic depicting technique for botulinum toxin injection of the pectoralis minor under ultrasound guidance, with placement of the ultrasound transducer obliquely over the axis of the muscle belly and the optimal site for needle injection. Adapted from Springer Nature: Torriani M, Gupta R, Donahue DM. Botulinum toxin injection in neurogenic thoracic outlet syndrome: results and experience using a ultrasound-guided approach. *Skeletal Radiol.* 2010;39:973–980.

nTOS reported significantly lower mental health Quality of Life scores than patients with vTOS preoperatively and throughout follow-up.[54]

As surgical approaches for the management of TOS involve decompression surrounding delicate, important neurovascular structures, complications can be severe and even life-threatening. Injury to the subclavian vein or artery, brachial plexus, thoracic duct and long thoracic, intercostobrachial, recurrent laryngeal, and phrenic nerves have been reported.[55] In one study analyzing the surgical removal of the first rib via a transaxillary approach, the most common complications included apical pneumothorax occurring in 25% of patients, followed by wound infections (3%) and lymphatic or nerve injury (<1%).[40] Recurrent TOS, often requiring additional operations,[46] is due to failure to completely decompress the brachial plexus due to residual sites of compression.

We advocate for a thoughtful and thorough supraclavicular approach to relieve TOS. In addition to the upper plexus structures, the lower trunk and C8 and T1 nerve roots can be easily identified and protected. Additionally, we find it simple to resect the first rib as well as any cervical ribs or prolonged transverse processes.

SURGICAL TECHNIQUE

During exposure of the brachial plexus, we utilize a supine approach and eschew a beach chair position. The affected upper extremity, neck, arm, and chest are prepared and draped in the standard sterile fashion. The sternal notch is included in the draping so, in an emergent situation, proximal vascular control can be achieved. Our supraclavicular approach begins in the supraclavicular fossa, in a neck crease parallel to and 2 cm above the clavicle. To prevent postoperative causalgia, the supraclavicular nerves are identified just beneath the platysma, mobilized, and retracted.

The omohyoid is divided, and the supraclavicular fat is elevated and pedicled off of the transverse cervical artery and preserved as a vascularized adipofascial flap for later use. The lateral portion of the clavicular head of the sternocleidomastoid is divided, gaining access to identify the phrenic nerve on the anterior surface of the anterior scalene muscle. This, as well as the long thoracic nerve at the posterior aspect of the middle scalene, serves as the lighthouse to the brachial plexus. In addition, these two nerves must be preserved. The long thoracic nerve can often run within the middle scalene, so careful identification is necessary. Moreover, a vessel loop should not be passed around the phrenic nerve as it is quite sensitive to retraction. An intraoperative nerve stimulator is used throughout the procedure to confirm appropriate identification of the parts of the brachial plexus and can be helpful in confirming the presence of the phrenic and long thoracic nerves.

Next, the anterior scalene is divided from the first rib, noting the subclavian artery immediately deep to it. With resection of the anterior scalene, the upper, middle, and lower brachial plexus trunks are easily identified and retracted to permit division of the middle scalene muscle (Fig. 8). **(See Video 1 [online],** which displays the intraoperative illustration of decompression of the brachial plexus following resection of the anterior and middle scalene muscles.) This permits mobilization of the brachial plexus to easily identify the lower trunk and the C8 and T1 nerve roots resting above and below the first rib. Any congenital bands or thickenings in Sibson's fascia can also be divided now. The rib can then be encircled, carefully protecting the pleura, and it can be divided. By reflecting the nerve roots anteriorly, the posterior segment of the divided first rib can be rongeured back to its spinal attachment. By reflecting the nerve roots posteriorly, the anterior segment of the first rib can be likewise removed. We advocate for complete removal of the first rib to prevent new bone formation from remnants that could cause recurrent compression and demonstrate increased normal conduction as compared with before neurolysis. (**See Video 2 [online]**, which displays the intraoperative presentation of the brachial plexus following removal of the cervical rib.)

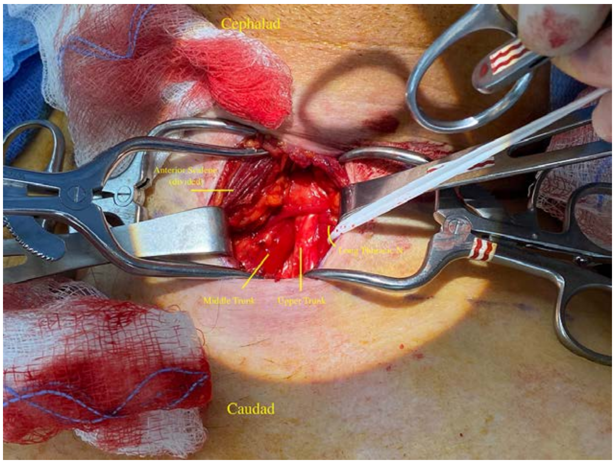

Fig. 8. The upper, middle, and lower brachial plexus trunks are shown following resection of the anterior scalene muscle.

If hemostasis is insufficient, then we use a technique described by Nelems[56] to open the pleura and facilitate drainage of any postoperative blood collection into the chest cavity.[57]

In addition, we use an intraoperative nerve stimulator to demonstrate increased conduction as compared with before neurolysis. If a poor conduction through the trunks is appreciated at 0.5 mA and requires greater than 2 mA to achieve conduction, the surgeon proceeds with external neurolysis of the three trunks and roots (Fig. 9). If poor conditions are still present, then internal neurolysis with the use of microsurgical instruments is performed. Following internal neurolysis of the plexus, nerve stimulation is reassessed using the stimulator to reveal a normal motor conduction at 0.5 mA.

This technique advocates for the use of intraoperative neuromonitoring to identify the components of the plexus to help guide the surgeon in the performance of internal neurolysis in addition to external neurolysis, when appropriate. Even though not all compression-induced injuries to the plexus causing nTOS will be reversed by neurolysis, literature in external neurolysis has shown return of function to one or more muscle groups within 6–8 weeks postoperatively.[58,59] We believe that this will result in greater symptomatic improvement postoperatively.

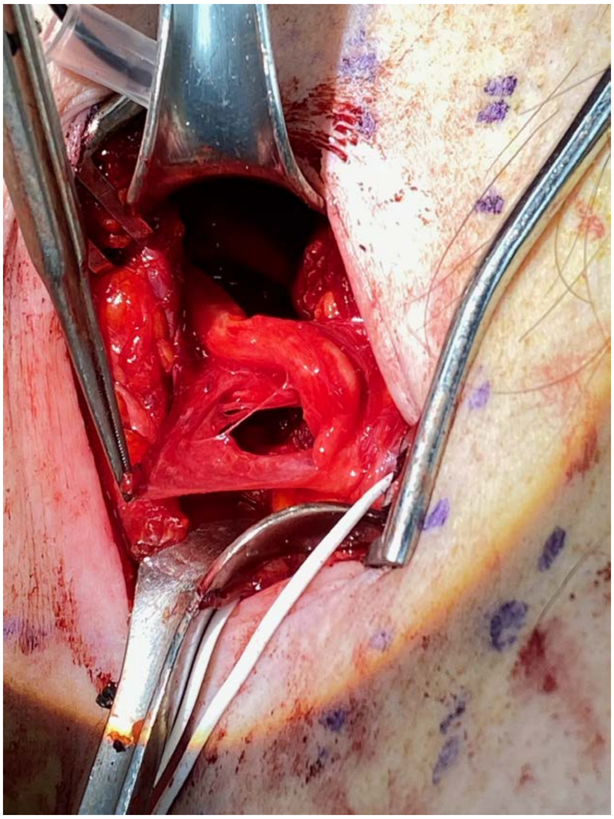

Fig. 9. External neurolysis of the trunks and roots of the brachial plexus.

FUTURE DEVELOPMENTS

The preliminary set of diagnostic criteria for nTOS produced by the Consortium for Outcomes Research and Education on TOS provides a useful tool to aid in the diagnosis of nTOS. Although this tool has been instrumental in assisting clinicians diagnose nTOS, it omits appropriate and available treatment options for nTOS, leaving the surgeon to determine the course of action based off the patient's underlying etiology and the surgeon's own preference. It also does not give weight to different aspects of the diagnostic criteria. A future study to establish levels of importance in the criteria, which can guide management, may be helpful.

CONCLUSIONS

TOS is a rare, yet serious condition with a myriad of clinical manifestations. A thorough understanding of anatomy and its variants, and the clinical signs and symptoms of TOS is needed to ensure accurate diagnosis. Although there are a variety of approaches to perform a decompression of the brachial plexus that may be causing TOS, we advocate for the supraclavicular approach.

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