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Thoracic outlet syndrome: a controversial clinical condition. Part 1: anatomy, and clinical examination/diagnosis

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Thoracic outlet syndrome (TOS) is a frequently overlooked peripheral nerve compression or tension event that creates difficulties for the clinician regarding diagnosis and management. Investigators have categorized this condition as vascular versus neurogenic, where vascular TOS can be subcategorized as either arterial or venous and neurogenic TOS can subcategorized as either true or disputed. The thoracic outlet anatomical container presents with several key regional components, each capable of compromising the neurovascular structures coursing within. Bony and soft tissue abnormalities, along with mechanical dysfunctions, may contribute to neurovascular compromise. Diagnosing TOS can be challenging because the symptoms vary greatly amongst patients with the disorder, thus lending to other conditions including a double crush syndrome. A careful history and thorough clinical examination are the most important components in establishing the diagnosis of TOS. Specific clinical tests, whose accuracy has been documented, can be used to support a clinical diagnosis, especially when a cluster of positive tests are witnessed.

Keywords: Diagnosis, Examination, Literature review, Thoracic outlet syndrome

Thoracic outlet syndrome (TOS) is a frequently overlooked peripheral nerve compression that creates difficulties for the clinician regarding diagnosis and management.[1] The term 'thoracic outlet syndrome' was originally coined in 1956 by Peet to indicate compression of the neurovascular structures in the interscalene triangle corresponding to the possible etiology of the symptoms.[2–4] Since Peet provided this definition, the condition has emerged as one of the most controversial topics in musculoskeletal medicine and rehabilitation.[5] This controversy extends to almost every aspect of the pathology including the definition, the incidence, the pathoanatomical contributions, diagnosis and treatment.[6,7]

The controversy surrounding the definition exists because the term TOS only outlines the location of the problem without actually defining what comprises the problem. In response, investigators have categorized TOS as vascular versus neurogenic, where vascular TOS can be subcategorized as either arterial or venous and neurogenic TOS can subcategorized as either true or disputed. Furthermore, the term TOS fails to identify the compressing insult or mechanism, thus prompting individuals to propose an alternative nomenclature that identifies the anatomical factors involved.[6]

The disagreement regarding the definition of TOS makes the overall incidence of the condition difficult to track. Complicating matters, the recognized prevalence of the diagnosis varies between disciplines. For example, Campbell and Landau[8] estimated that surgeons diagnose TOS 100 times more frequently than neurologists. Cherington and Cherington[5] go further to imply that the diagnosis is made by surgeons according to potential reimbursement available for particular surgical procedures. Regardless of the overall incidence of TOS, it is estimated that over 90% of all TOS cases are of neurogenic origin, whereas less than 1% are arterial and approximately 3–5% are venous.[7] Neurogenic TOS has been further subcategorized as either true neurogenic or disputed neurogenic, with the former being defined as a condition with objective diagnostic findings and the latter being a condition without the same objective

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findings.[9] As a result, disparities in the definition have produced different opinions regarding diagnostic standards for TOS.[6,7,9,10] Furthermore, given the controversy surrounding the definition and diagnosis of TOS, conflict exists regarding the optimal treatment approach for this condition.[10–12]

The purpose of this paper is to provide the reader with clarity through: (1) a review of the relevant pathoanatomy; (2) a discussion regarding pathology and differential diagnosis; and (3) a presentation of the examination and special test measures, along with suggested diagnostic paradigms. A second manuscript (part 2 of this series) will examine both non-surgical and surgical management strategies appropriate for the treatment of TOS.

Review of pathoanatomy

The neural container described as the 'thoracic outlet' is comprised of several components. Proximally, the cervicoaxillary canal is divided by the first rib into two sections. The proximal portion of this canal is comprised of the interscalene triangle and the costoclavicular space, whereas the axilla comprises the distal aspect of the canal. The proximal portion is more clinically relevant, due to its role in neurovascular compression.[12] More specifically, the thoracic outlet includes three confined spaces extending from the cervical spine and mediastinum to the lower border of the pectoralis minor muscle (Fig. 1). The three compartments include the interscalene triangle, the costoclavicular space and the thoraco-coraco-pectoral space or retropectoralis minor space.[4,13] The interscalene triangle is bordered by the anterior scalene muscle anteriorly, the middle scalene muscle posteriorly, and the medial surface of the first rib inferiorly.

The trunks of the brachial plexus and subclavian artery are located in the interscalene triangle.[12] The subclavian vein does not cross the interscalene triangle but runs beneath the anterior scalene before joining the internal jugular vein to form the brachiocephalic vein. The costoclavicular space is bordered anteriorly by the middle third of the clavicle, posteromedially by the first rib, and posterolaterally by the upper border of the scapula.[4] The borders of the thoraco-coraco-pectoral space include the coracoid process superiorly, the pectoralis minor anteriorly and the ribs 2 through 4 posteriorly (Fig. 1).[14,15]

Several types of bony abnormalities exist that produce the compromising events related to TOS. Cervical ribs are supranumerary ribs originating from the seventh cervical vertebra and occur in less than 1.0% of the general population, with only 10% of those patients with the rib experiencing symptoms affiliated with its presence.[16] Samarasam *et al.*[3] found four main varieties of cervical ribs including: (1) type I: a complete cervical rib articulates with the first rib or manubrium of sternum; (2) type II: incomplete cervical ribs with a free end expanded to form a bulbous tip; (3) type III: an incomplete rib that is continued by a fibrous band; and (4) type IV: a rib that appears as a short bar of bone with a length of a few millimeters beyond the C7 transverse process. Additionally, an elongated C7 transverse process can produce neurovascular compression. Conversely, an abnormal first rib or clavicle can create compression through exostosis, tumor, callus or fracture of the first rib, subsequently irritating the brachial plexus.[13] When a clavicular fracture demonstrates malunion,[17] fragmentation,[18] or retrosternal dislocation,[19] the risk for TOS is enhanced.

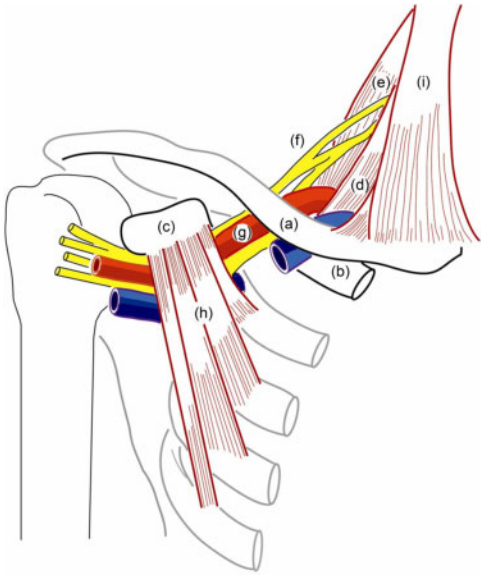

Figure 1 Diagram showing the thoracic outlet. (A) Clavicle; (B) first rib; (C) coracoid process; (D) middle scalene; (E) posterior scalene; (F) brachial plexus; (G) subclavian artery; (H) pectoralis minor; (I) sternocleidomastoid.

Soft tissue abnormalities may create compression or tension loading of the neurovascular structures found within the thoracic outlet container. For example, congenital abnormalities have been reported and include several anatomic variations of the scalene muscles. Demondion *et al.*[13] reported that scalene muscle variations include hypertrophy of the anterior scalene muscle, passage of the brachial plexus through the substance of the anterior scalene muscle, and a broad, excessively anterior middle scalene muscle insertion on the first rib. Further complicating the soft tissue compromised within the thoracic outlet, anomalous fibrous bands have been found within the thoracic outlet container, increasing the stiffness and decreased compliance of the container, resulting in an increased potential for neurovascular load.[20] These congenital abnormalities can be visualized by magnetic resonance imaging. In addition, an indirect sign of the presence of a tight fibrous band is elevation of the subclavian artery demonstrated on sagittal magnetic resonance imaging.[13] Roos classified 10 different types of

fibrous bands that tend to stiffen the already unforgiving boundaries of the thoracic outlet container. These anomalous structures traverse through the thoracic outlet and tend to reduce thoracic outlet container compliance, potentially elevating, kinking and compressing the neurovascular bundle against the surrounding anatomic borders of the thoracic outlet.[20,21]

Because the thoracic outlet is bordered by the clavicle, clavicular movement is essential for normal thoracic outlet container compliance. The normal clavicle is expected to elevate, retract and spin backwards during upper extremity elevation.[22] However, if this behavior is compromised, then the thoracic outlet container could be reduced and the brachial plexus placed at risk for increased load. This increased load could lead to direct neural compression, angulation, or tension loading.[23]

Clavicular movement is influenced by the structural and functional integrity of both the acromioclavicular joint (ACJ) and sternoclavicular joint (SCJ). The ACJ allows movement of the scapula on the clavicle in three planes about the coronal, sagittal, and vertical axes.[24,25] Ligamentous support for the ACJ (ACJ capsular ligaments) serves as a primary restraint for posterior axial rotation and posterior displacement of the clavicle on the acromion.[24,26] The integrity of the ACJ is indirectly controlled by the coracoclavicular ligament complex (trapezoid and conoid ligaments), which provides 75% of the constraint against axial compression of the clavicle toward the acromion.[24–26] Moreover, this complex tension loads during arm elevation, producing dorsal axial rotation of the clavicle about its longitudinal axis. Any compromise to this movement could contribute to TOS by compromising the container and loading the nerve tissue.

The SCJ is a diarthrodial, synovial, sellar joint. The various ligament systems (costoclavicular and sternoclavicular) reinforce the capsule and limit anterior-posterior movement of the medial end of the clavicle. The SCJ can be susceptible to anterior and posterior subluxations via direct and indirect trauma.[24,27] Any dysfunction of the clavicle associated with either a limit or subluxation of the SCJ can contribute to thoracic outlet compromise and subsequent symptom development.

Epidemiology

While the majority of TOS cases are diagnosed between the ages of 20 and 50 years,[4] TOS can occur in teenagers[28] or more rarely in pediatric patients.[29] Women are three to four times more likely to develop neurogenic TOS,[30] while the incidence of vascular TOS is more equal between non-athletic men and women,[31] but found to be even greater in competitive athletic men versus women.[32]

History and clinical examination

Diagnosing TOS can be challenging because the symptoms vary greatly amongst patients with the disorder, thus lending to other conditions including a double crush syndrome.[33,34] Diagnoses of the two vascular forms of TOS are generally accepted in all healthcare circles. On the other hand, neurogenic TOS, especially 'disputed' neurogenic TOS, is more difficult to diagnose because there is no standard objective test to confirm clinical impressions.[35]

A careful history and thorough clinical examination are the most important components in establishing the diagnosis of TOS,[36] which remains a diagnosis of exclusion. As such, other conditions that present with overlapping or similar clinical pictures must be considered during the examination process.[21] Further challenges are found in the fact that the diagnosis of entrapment neuropathies of the upper limb does not exclude TOS, constituting a double crush that is observed in approximately 50% of cases.[33,37,38]

Patient history

Vascular TOS can develop secondary to repetitive upper limb activities that lead to claudication, especially in young adults with arterial TOS. However, the same condition can develop spontaneously, unrelated to trauma. Conversely, neurogenic TOS more commonly develops following a macrotrauma to the neck or shoulder girdle area, such as a motor vehicle accident or work-related repetitive stressful activities.[7] Table 1 summarizes the clinical profile associated with vascular and neurogenic TOS.[7,31]

Signs and symptoms of TOS vary with every patient according to the location of the neurovascular tension and/or compression injury within the thoracic outlet. Symptoms of TOS can range from mild pain and sensory changes to limb- and/or life-threatening complications.[29,39] Patients can present with multiple unilateral or bilateral signs and symptoms associated with involvement of both neurogenic and vascular components.[40]

The quality, location and timing of symptoms all present valuable information to the clinician. Arterial TOS, while infrequent, can produce a series of profound symptoms. Patients suffering from this condition can present with pain, numbness in a non-radicular distribution, coolness to touch and pale discoloration, all of which worsen with cold ambient temperatures.[41] Conversely, venous TOS results in excruciating deep pain the chest, shoulder and entire upper extremity, accompanied by a feeling of heaviness that occurs especially after activity. The patient will present with cyanotic discoloration and distended collateral veins, potentially accompanied by edematous increases in the volume of the extremity.[42]

Table 1 Clinical profile and diagnosis of TOS

Arterial TOS	Venous TOS	True neurogenic TOS	Disputed neurogenic TOS
Often in young adults with a history of vigorous arm activity or spontaneous	Pain, often in younger men and often preceded by excessive activity in the arms or spontaneous	Common history of neck trauma preceding the symptoms, most commonly from car accidents and repetitive stress at work.	Common history of neck trauma preceding the symptoms, most commonly from car accidents and repetitive stress at work.
Pain in the hand but seldom in the shoulder or neck	Edema of the arm	Pain, paresthesia, numbness and/or weakness in the hand, arm (C8, T1 distribution) and shoulder, plus neck pain	Pain, paresthesia, and feeling of weakness in the hand, arm (C8, T1 distribution) and shoulder, plus neck pain
Pallor	Cyanosis	Occipital headaches	Occipital headaches
Claudication	Feeling of heaviness	Paresthesias, numbness during the day and also nocturnal, awakening the patient with pain or numbness	Paresthesias, often nocturnal, awakening the patient with pain or paresthesia
Coldness and cold intolerance	Paresthesias in the fingers and hand (secondary to edema)	Loss of dexterity	Loss of dexterity
Paresthesias		Cold intolerance	Cold intolerance
Symptoms usually stem spontaneously from arterial emboli.		Raynaud phenomenon, hand coldness, and color changes due to sympathetic overactivity as opposed to ischemia. 'Compressors': symptoms day>night Objectified weakness and/or sensory deficit='*true*' neurogenic	Raynaud phenomenon, hand coldness, and color changes due to sympathetic overactivity as opposed to ischemia. 'Releasers': symptoms night>day Subjective weakness and/or numbness='*disputed*' neurogenic
Diagnosis based on information above	Diagnosis based on information above	Diagnosis based on information above+cluster of at least two provocation tests positive and almost always elevated arm stress test (+)	Diagnosis based on information above+cluster of at least two provocation tests positive and often presence of cyriax release test (+)
Confirmed through doppler ultrasound and angiography in the seated position	Confirmed through venous ultrasound studies, venous scintillation scans, venography and plethysmography	Confirmed through (+) neurophysiological testing	No confirmation through objective testing neurophysiological testing are normal

Symptoms associated with neurogenic TOS include pain, paresthesia, numbness, and/or weakness. Here, the examiner should investigate if the symptoms are radicular or non-radicular in nature. Thoracic outlet symptoms will normally not produce symptoms that follow dermatomal and myotomal patterns, unless the thoracic outlet is accompanied by cervical or upper thoracic nerve root compression. Reports of paresthesia in the upper limb indicate mild perineural dysfunction,[43] while objectified numbness and/or weakness suggest true axonal compression, which is indicative of a more serious insult and a less favorable prognosis.[44] Symptoms' location most frequently reported in patients with TOS include paresthesia in the upper limb (98%), neck pain (88%), trapezius pain (92%), shoulder and/or arm pain (88%), supraclavicular pain (76%), chest pain (72%), occipital headache (76%), and parasthesias in all five fingers (58%), the fourth and fifth fingers only (26%) or the first–third fingers (14%).[7] Compression and irritation of the upper plexus (C5, C6, C7) can cause pain in the anterior aspect of the neck from the clavicle to the mandible, ear and mastoid area, occasionally radiating into the side of the face. These symptoms can spread into the upper chest anteriorly, the periscapular region posteriorly, and across the trapezius ridge down the outer arm and through the radial nerve distribution toward the dorsum of the thumb and index finger.[20] Patients with this distinct pattern of symptoms may have pathoanatomical anomalies that are typically multiple, most often located in the posterior scalenic triangle[45,46] between the anterior and middle scalene muscles[20] and occasionally compressed by a scalenus minimus muscle.[47] Patients with lower plexus (C8, T1) irritation will mainly complain of symptoms on the ulnar side of the arm and hand, potentially accompanied by symptoms found in the anterior shoulder and axillary regions.

Historical questioning should include the timing of symptoms, which may occur throughout the day during activity versus only present at night.[48] Many patients report awaking at night with paresthesia in the upper limb, a phenomenon coined in the literature as the 'release phenomenon'.[49] This phenomenon suggests release of tension and/or compression of the perineural blood supply to the brachial plexus and signals a return of normal sensation that is a prognostic indicator of favorable outcome. The clinician can refer to these patients as 'releasers'. On the other hand, other patients may experience their symptoms primarily throughout the day time while using prolonged postures (such as shoulder girdles protracted and depressed and the head forward) or activities (such as working over head with elevated arms) that would result in an increase in tension or compression of the neurovascular bundle of the brachial plexus. These patients can be referred to as 'compressors'.

Clinical examination

The examiner should record the position of the patient's head, shoulders, scapulae and arms in the seated and standing positions. The examiner should pay attention to the presence of rounded shoulders, forward head, increased thoracic kyphosis, as well as posterior tilt, downward rotation and/or depression of the scapulae. These postures tend to increase the tension loading of the brachial plexus.[50] Moreover, the patient may present with supraclavicular fullness, which could represent a first rib prominence versus soft tissue swelling.[51]

Visual inspection of the upper limbs includes observing for cyanosis and edema in case of venous compromise or paleness in case of vascular compromise (as previously described). Atrophy in the hand region should additionally be noted. If the patient has a cervical rib or an elevated first rib, the supraclavicular fossa may appear to be full.[51]

The supraclavicular fossa, including the brachial plexus found in the space, should be palpated for pain.[52] The brachial plexus is best palpated directly posterior to the pulsation of the subclavian artery with the head sidebent toward the contralateral side. The patient's hands are palpated for temperature changes and moistness for the sake of detecting sympathetically mediated symptoms.

The clinical examination begins by questioning the patient about the location and amplitude of present symptoms, such as pain. The patient is asked to report changes in the symptoms' amplitude associated with movements of the neck, shoulder girdle and upper limb.

In suspecting thoracic outlet problem, the cervical spine and shoulders should be examined. A description of cervical and shoulder clinical examination are beyond the scope of this discussion and are described elsewhere.[25,53] The decision to perform specific clinical testing for TOS is based on the examiner's clinical reasoning and is carried out mainly in the presence of neck–shoulder–arm symptoms that are non-radicular in nature and influenced by the position of the upper limb and/or neck. If the testing of the cervical spine, shoulder and TOS are negative, a peripheral compression neuropathy is suspected[54] and further testing should be performed in order to focus on those possibilities.

The diagnosis of arterial, venous and true neurogenic TOS has the advantage of implementing standard diagnostic tests. The validity of vascular diagnostic tests is improved when performed dynamically in the positions that produce the patient's symptoms.[55] The following diagnostic tests have been recommended: (1) venous ultrasound studies, venous scintillation scans, venography and plethysmography

for venous TOS;[31] (2) Doppler ultrasound and angiography in the seated position for arterial TOS;[55] and (3) nerve conduction velocities and electromyography of the medial antebrachial cutaneous nerve for the true neurogenic TOS.[30,56]

Conversely, no valid standard diagnostic test is available for disputed neurogenic TOS, resulting in controversies in the frequency of TOS diagnosis.[57,58] Commonly, nerve conduction velocities and electromyography are negative for disputed neurogenic TOS. Thus, the examiner must rely on a thorough history and a cluster of clinical TOS provocation test findings to solve the disputed neurogenic TOS diagnostic puzzle.

Provocative clinical testing for TOS has been reported to display high rates of false positive findings.[59] The reliability and validity of provocative clinical tests for TOS and one motion test for assessment of the presence of an elevated first rib are summarized in Table 2. The supraclavicular pressure test and the Adson's test more specifically address compromise to the plexus through the scalene triangles.[54] The costoclavicular maneuver evaluates provocation produced by costoclavicular space narrowing, while the Wright's test examines neural tissue compromise through the thoraco-coraco-pectoral gate.[54] The elevated arm stress test examines the result of loading the plexus throughout the TOS container, while the Cyriax release test examines the result of unloading the plexus in the same space. Finally, the upper limb neural tension test examines provocation to the neural tissue passing through the thoracic outlet container under a tension load.

The Adson's test and costoclavicular maneuver display a fairly large percentage of false positives when a change in the radial pulse is considered as a positive test.[60,61] Therefore, the clinician is encouraged to use the test position of those tests for symptom provocation and not as a test for radial pulse change. This is sensible, considering the low incidence of vascular involvement in TOS. Conversely, one can note from examining tabulated data that the Wright's test and the elevated arm stress test appear to display the greatest sensitivity for neurogenic and vascular TOS. The positive Cyriax release test[62] represents a 'release phenomenon', which is most relevant when correspondent to a history of nocturnal symptoms. These findings can be used as a treatment guide.

Upper limb tension testing is sensitive for irritation of the neural tissue including cervical roots,[63] brachial plexus and peripheral nerves[64] as well as for patients with arm pain syndrome.[65] It has been advocated for the diagnosis of neurogenic TOS with reported high sensitivity.[7] The test appears to be excellent for screening for sensitization of the neural tissue in the cervical spine, brachial plexus and upper limb but is not specific for one area. The test is recommended as part of the examination and for its usefulness in treatment that includes neural mobilization.[66,67]

Each of the previously discussed tests should be considered when establishing a diagnosis of TOS. From Table 2, one can see that the validity of any single test is troubled. In view of the lack of quality research reporting both sensitivity and specificity of TOS provocation testing, Gilliard *et al.*[61] showed that a cluster of two provocative tests displayed the highest sensitivity (90%), while a cluster of five positive provocative tests increased the specificity for TOS to 84%.

A comprehensive diagnosis of disputed neurogenic TOS is supported based on several levels of assessment. First, the diagnosis is supported based on a history that includes the presence of non-radicular symptoms in the neck–shoulder–arm worsened by movements and/or position of the neck, arms, and shoulder girdle, accompanied by the presence of a cluster of TOS provocation tests. Next, the diagnostic picture is completed with an assessment of postural dysfunctions and container mobility (thoracic outlet container testing). As stated earlier, the examiner should record the presence of rounded shoulders, forward head, and increased thoracic kyphosis, as well as posterior tilt, downward rotation and/or depression of the scapulae.[50] The mobility of the first rib can be assessed using the cervical rotation lateral flexion test (Table 2),[9] as an elevated first rib can potentially increase the tension on the neurovascular bundle of the brachial plexus. The mobility of the thoracic spine should be carefully assessed for lack of motion, especially in the direction of extension.[68] This assessment should be accompanied by an appraisal of the muscle length of the scalene muscles, especially in weight lifters, patients with severe chronic obstructive pulmonary disease and after whiplash. Shortening of these muscles can lead to non-compliance of the thoracic outlet container through its gates.[7,69]

To continue the assessment of the thoracic outlet container, the motion of the clavicle should be assessed during arm elevation, looking for decreased mobility. When hypomobile, the clavicle moves too quickly in a dorsal direction and produces a narrowing of the costoclavicular space.[54] In such a case the joint play tests of the ACJ and SCJ should be carried out. Hypomobility of these joints could lead to dysfunction in the movement of the clavicle and shoulder girdle, thus crowding the thoracic outlet container through which the brachial plexus courses.[54] Finally, glenohumeral end-range mobility testing is merited, where limits could force the clavicle to compromise the brachial plexus during end-range arm elevation.

Table 2 Description, reliability, sensitivity, specificity, and likelihood ratios of clinical tests for TOS

Test	Description	Reliability	Sensitivity	Specificity	LR+	LR−	References
Elevated arm stress	Patient seated with arms above 90 degrees of abduction and full external rotation with head in neutral position. Patient opens and closes hands into fists while holding the elevated position for 3 minutes. Positive test: pain and/or paresthesia and discontinuation with dropping of the arms for relief of pain.	1.0	52–84	30–100	1.2–5.2	0.4–0.53	33, 60, 61, 70–72
Supraclavicular pressure	Patient seated with arms at the side. Examiner places fingers on the upper trapezius and the thumbs contacting the anterior scalene muscle near the first ribs and squeezes the fingers and thumb together for 30 seconds. Positive test: reproduction of pain or paresthesia.	NT	NT	85–98	NA	NA	60, 72
Costoclavicular maneuver	Patient sits straight with arms at the side. Radial pulse is assessed. Patient retracts and depresses shoulders while protruding the chest. Position is held for up to 1 minute. Positive test: change in radial pulse and/or pain and paresthesia.	NT	NT	53–100	NA	NA	60, 70, 72
Adson's	Patient seated with arms at the side. The radial pulse is palpated. Patient inhales deeply and holds the breath, extends and rotates the neck toward the side being tested. Positive test: change in radial pulse and/or pain, paresthesia reproduction.	NT	79	74–100	3.29	0.28	60, 61, 70, 72
Wright's	Patient seated with arms at the side. The radial pulse is palpated. Examiner places the patient's shoulder into abduction above the head. The position is held for 1 to 2 minutes. Positive test: change in radial pulse and/or symptom reproduction.	NT	70–90	29–53	1.27–1.49	0.34–0.57	61
Cyriax release	Patient seated or standing. Examiner stands behind patient and grasps under the forearms, holding the elbows at 80 degrees of flexion with the forearms and wrists in neutral. Examiner leans the patient's trunk posteriorly and passively elevates the shoulder girdle. The position is held for up to 3 minutes. Positive test: Paresthesia and/or numbness (release phenomenon) or symptom reproduction.	0.92	NT	77–97	NA	NA	33, 62
Upper limb tension	Patient supine. Examiner standing on the side to be tested. Examiner depresses the shoulder girdle and abducts the shoulder to 110 degrees with slight extension and elbow flexion to 90 degrees. The forearm is then maximally supinated and the wrist and fingers extended. Finally, elbow extension is applied. The neck is sidebent to the contralateral side. Testing is stopped after any symptom reproduction. Positive test: reproduction of symptoms with the distal movement or neck movement and/or restricted elbow extension range of motion.	0.03	90	38	1.5	0.3	65, 73, 74
Cervical rotation lateral flexion	Patient seated. Examiner passively rotates the head away from the affected side and gently flexes the neck forward to end range moving the ear toward the ventral chest. Positive test: forward flexion part of the movement is notably decreased with a hard end feel.	0.42–1.0	100	NT	NA	NA	9, 75

Note: LR+: positive likelihood ratio; LR−: negative likelihood ratio.

The clinician must consider the presence of associated neural tissue irritations/entrapments along the entire course of the peripheral nerves associated with the brachial plexus. Rather than trying to differentiate TOS from a peripheral nerve entrapment as the sole cause of the patient's symptoms, the clinician is encouraged to consider the possibility of both conditions that result from a double crush event of the neural tissues along their course. In response, a clinician's suspicion for the relative contribution of TOS versus a peripheral nerve entrapment is influenced through the analysis of outcomes from the clinical testing ensemble.

A description of relevant testing for peripheral nerve structures and their resultant irritations/entrapments (including ulnar nerve at the cubital tunnel and tunnel of Guyon, dorsal interosseus nerve in the tunnels of the dorsal forearm, median nerve at both the pronator teres and carpal tunnel) is offered elsewhere[59] and is beyond the scope of this discussion. However, due to the potential for a double crush event, nerve irritation/compression/tension at the brachial plexus (TOS) can lend selected peripheral nerves to greater vulnerability and subsequent symptoms in the sites distal to the thoracic outlet. As a consequence, the patient suffering from entrapment in the thoracic outlet is at risk for developing a symptom profile that reflects both the TOS condition and the other irritation/entrapment(s). Thus, the clinician is encouraged to perform testing that is relevant both to TOS and the peripheral nerve irritation/entrapment.

The potential for double crush complicates the clinical presentation and makes a differential diagnostic process challenging for the clinician. Once the previously described testing (TOS provocation testing, Thoracic outlet container testing, and peripheral nerve irritation/entrapment testing) is completed,

Table 3 Double crush considerations for disputed neurogenic TOS

TOS provocation tests*	Thoracic outlet container testing†	Peripheral nerve provocation tests‡	Interpretation	Management decisions
(−)	(−)	(−)	Low suspicion for TOS and or peripheral nerve irritation/ entrapment condition. Symptoms are likely due to other condition, such as cervical spine or central nervous system.	Further testing merited before management is initiated
(+)	(−)	(−)	High suspicion for TOS without associated thoracic outlet container dysfunction; low suspicion for peripheral nerve irritation/entrapment	Treat TOS for symptom management
(+)	(+)	(−)	High suspicion for TOS with associated thoracic outlet container dysfunction; low suspicion for peripheral nerve irritation/entrapment	Treat TOS for symptom management and improvement of thoracic outlet container mobility
(+)	(−)	(+)	High suspicion for TOS without associated thoracic outlet container dysfunction; high suspicion for peripheral nerve irritation/entrapment; high suspicion for double crush	Treat TOS for symptom management; treat peripheral nerve irritation/entrapment
(+)	(+)	(+)	High suspicion for TOS with associated thoracic outlet container dysfunction; high suspicion for peripheral nerve irritation/entrapment; high suspicion for double crush	Treat TOS for symptom management and improvement of thoracic outlet container mobility; treat peripheral nerve irritation/entrapment
(−)	(+)	(+)	Low suspicion for TOS; high suspicion for thoracic outlet container dysfunction; high suspicion for peripheral nerve irritation/entrapment; high suspicion for double crush.	Treat for improvement of thoracic outlet container mobility; Treat peripheral nerve irritation/entrapment
(−)	(−)	(+)	Low suspicion for TOS; low suspicion for thoracic outlet container dysfunction; high suspicion for isolated peripheral nerve irritation/entrapment; low suspicion of double crush.	Treat peripheral nerve irritation/entrapment

*For any UE symptoms; A (+) cluster of tests increases the suspicion.
†For mobility loss/dysfunction in any joint system in the container, such as first rib, ACJ, or SCJ.
‡For specific peripheral nerve symptoms provoked through peripheral nerve clinical provocation testing.

then the clinician is left to interpret the clinical testing results. Table 3 presents a suggested approach to interpreting test results and clinical decision making for patients with potential complex presentations. The interpretation of said tests raises or lowers the clinician's suspicion for any combination of the following: TOS, thoracic outlet container dysfunction, peripheral nerve irritation/entrapment, and underlying double crush. Without consideration for related peripheral nerve irritations/entrapments and potential double crush, the management of complex patients may be incomplete.

Summary

The diagnosis and management of TOS has remained controversial, based on a potential lack of correspondence between neurophysiological testing and clinical examination outcomes. An appreciation for the complexity of the anatomical and mechanical features associated with the thoracic outlet container can serve as a foundation for understanding a patient's clinical presentation. A thorough historical account and clinical examination can guide the clinician towards a selection of specific tests that will support the diagnostic process. The accuracy of numerous tests has been established, and a clinician can implement these tests to strengthen a diagnostic suspicion. This is especially important when neurophysiological testing contradicts the clinical presentation. Rather than trying to differentiate TOS from a peripheral nerve entrapment as the cause of the patient's symptoms, the clinician is encouraged to consider the possibility of both conditions that result from a double crush irritation of the neural tissues along their course. In response, a clinician's suspicion for the relative contribution of TOS versus a peripheral nerve entrapment is influenced through the analysis of outcomes from the clinical testing ensemble.

Once a thorough history and clinical examination is completed, the clinician can decide upon a management strategy appropriate for the individual patient. Part 2 will discuss non-surgical, as well as surgical, treatment options. Non-surgical treatment focuses on symptom reduction and addressing the specific dysfunctions responsible for the patient's symptoms. Surgery is generally reserved for cases of vascular TOS or neurogenic TOS that does not respond to conservative measures.

References

1 Atasoy E. Thoracic outlet compression syndrome. Orthop Clin North Am. 1996;27:265–303.
2 Peet RM, Hendricksen JD, Anderson TP, Martin GM. Thoracic outlet syndrome: evaluation of a therapeutic exercise program. Prc Mayo Clin. 1956;31:281–7.
3 Samarasam I, Sadhu D, Agarwal S, Nayak S. Surgical management of thoracic outlet syndrome: a 10-year experience. ANZ J Surg. 2004;74:450–4.
4 Koknel Talu G. Thoracic outlet syndrome. Agri. 2005;17:5–9.
5 Cherington M, Cherington C. Thoracic outlet syndrome: Reimbursement patterns and patient profiles. Neurology. 1992;42:943–5.
6 Cuetter AC, Bartoszek DM. The thoracic outlet syndrome: controversies, overdiagnosis, overtreatment, and recommendations for management. Muscle Nerve. 1989;12:410–9.
7 Sanders RJ, Hammond SL, Rao NM. Diagnosis of thoracic outlet syndrome. J Vasc Surg. 2007;46:601–4.
8 Campbell WW, Landau ME. Controversial entrapment neuropathies. Neurosurg Clin N Am. 2008;19:597–608, vi–vii.
9 Lindgren KA, Leino E, Manninen H. Cervical rotation lateral flexion test in brachialgia. Arch Phys Med Rehabil. 1992;73:735–7.
10 Lindgren KA, Oksala I. Long-term outcome of surgery for thoracic outlet syndrome. Am J Surg. 1995;169:358–60.
11 Cherington M. Thoracic outlet syndrome: rise of the conservative viewpoint. Am Fam Physician. 1991;43:1998.
12 Urschel HC, Kourlis H. Thoracic outlet syndrome: a 50-year experience at baylor university medical center. Proc (Bayl Univ Med Cent). 2007;20:125–35.
13 Demondion X, Herbinet P, van Sint Jan S, Boutry N, Chantelot C, Cotten A. Imaging assessment of thoracic outlet syndrome. Radiographics. 2006;26:1735–50.
14 Richardson AB. Thoracic outlet syndrome in aquatic athletes. Clin Sports Med. 1999;18:361–78.
15 Remy-Jardin M, Doyen J, Remy J, Artaud D, Fribourg M, Duhamel A. Functional anatomy of the thoracic outlet: evaluation with spiral CT. Radiology. 1997;205:843–51.
16 Wood VE, Twito R, Verska JM. Thoracic outlet syndrome. The results of first rib resection in 100 patients. Orthop Clin North Am. 1988;19:131–46.
17 Fujita K, Matsuda K, Sakai Y, Sakai H, Mizuno K. Late thoracic outlet syndrome secondary to malunion of the fractured clavicle: case report and review of the literature. J Trauma. 2001;50:332–5.
18 Chen DJ, Chuang DC, Wei FC. Unusual thoracic outlet syndrome secondary to fractured clavicle. J Trauma. 2002;52:393–8; discussion 398–9.
19 Jain S, Monbaliu D, Thompson JF. Thoracic outlet syndrome caused by chronic retrosternal dislocation of the clavicle. Successful treatment by transaxillary resection of the first rib. J Bone Joint Surg Br. 2002;84:116–8.
20 Roos DB. Historical perspectives and anatomic considerations. thoracic outlet syndrome. Semin Thorac Cardiovasc Surg. 1996;8:183–9.
21 Sheth RN, Belzberg AJ. Diagnosis and treatment of thoracic outlet syndrome. Neurosurg Clin N Am. 2001;12:295–309.
22 Fung M, Kato S, Barrance PJ, Elias JJ, McFarland EG, Nobuhara K, *et al.* Scapular and clavicular kinematics during humeral elevation: a study with cadavers. J Shoulder Elbow Surg. 2001;10:278–85.
23 Fisher MA, Gorelick PB. Entrapment neuropathies. Differential diagnosis and management. Postgrad Med. 1985;77:160–74.
24 Culham E, Peat M. Functional anatomy of the shoulder complex. J Orthop Sports Phys Ther. 1993;18:342–50.
25 Sizer PS, Phelps V, Gilbert K. Diagnosis and management of the painful shoulder. Part 1: clinical anatomy and pathomechanics. Pain Practice. 2003;3:39–57.
26 Renfree KJ, Wright TW. Anatomy and biomechanics of the acromioclavicular and sternoclavicular joints. Clin Sports Med. 2003;22:219–37.
27 Wirth MA, Rockwood CA Jr. Acute and chronic traumatic injuries of the sternoclavicular joint. J Am Acad Orthop Surg. 1996;4:268–78.
28 Rigberg DA, Gelabert H. The management of thoracic outlet syndrome in teenaged patients. Ann Vasc Surg. 2009;23:335–340.
29 Arthur LG, Teich S, Hogan M, Caniano DA, Smead W. Pediatric thoracic outlet syndrome: a disorder with serious vascular complications. J Pediatr Surg. 2008;43:1089–94.
30 Machanic BI, Sanders RJ. Medial antebrachial cutaneous nerve measurements to diagnose neurogenic thoracic outlet syndrome. Ann Vasc Surg. 2008;22:248–54.
31 Urschel HC,Jr, Patel AN. Surgery remains the most effective treatment for paget-schroetter syndrome: 50 years' experience. Ann Thorac Surg. 2008;86:254–60; discussion 260.
32 Melby SJ, Vedantham S, Narra VR, Paletta GA Jr, Khoo-Summers L, Driskill M, *et al.* Comprehensive surgical management of the competitive athlete with effort thrombosis of the subclavian vein (Paget-Schroetter syndrome). J Vasc Surg. 2008;47:809–20; discussion 821.

33 Smith TM, Sawyer SF, Sizer PS, Brismee JM. The double crush syndrome: a common occurrence in cyclists with ulnar nerve neuropathy – a case-control study. Clin J Sport Med. 2008;18:55–61.
34 Abe M, Ichinohe K, Nishida J. Diagnosis, treatment, and complications of thoracic outlet syndrome. J Orthop Sci. 1999;4:66–9.
35 Sanders RJ, Hammond SL, Rao NM. Thoracic outlet syndrome: a review. Neurologist. 2008;14:365–73.
36 Rayan GM. Thoracic outlet syndrome. J Shoulder Elbow Surg. 1998;7:440–51.
37 Seror P. Symptoms of thoracic outlet syndrome in women with carpal tunnel syndrome. Clin Neurophysiol. 2005;116:2324–9.
38 Wood VE, Biondi J. Double-crush nerve compression in thoracic-outlet syndrome. J Bone Joint Surg Am. 1990;72:85–7.
39 Elman EE, Kahn SR. The post-thrombotic syndrome after upper extremity deep venous thrombosis in adults: a systematic review. Thromb Res. 2006;117:609–14.
40 Tilki HE, Stalberg E, Incesu L, Basoglu A. Bilateral neurogenic thoracic outlet syndrome. Muscle Nerve. 2004;29:147–50.
41 Pollack EW. Surgical anatomy of the thoracic outlet syndrome. Surg Gynecol Obstet. 1980;150:97–103.
42 Urschel HC Jr, Razzuk MA. Improved management of the Paget-Schroetter syndrome secondary to thoracic outlet compression. Ann Thorac Surg. 1991;52:1217–21.
43 Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. J Bone Joint Surg Am. 1999;81:1600–10.
44 Jordan SE, Ahn SS, Gelabert HA. Differentiation of thoracic outlet syndrome from treatment-resistant cervical brachial pain syndromes: development and utilization of a questionnaire, clinical examination and ultrasound evaluation. Pain Physician. 2007;10:441–52.
45 Rusnak-Smith S, Moffat M, Rosen E. Anatomical variations of the scalene triangle: dissection of 10 cadavers. J Orthop Sports Phys Ther. 2001;31:70–80.
46 Natsis K, Totlis T, Tsikaras P, Anastasopoulos N, Skandalakis P, Koebke J. Variations of the course of the upper trunk of the brachial plexus and their clinical significance for the thoracic outlet syndrome: a study on 93 cadavers. Am Surg. 2006;72:188–92.
47 Harry WG, Bennett JD, Guha SC. Scalene muscles and the brachial plexus: anatomical variations and their clinical significance. Clin Anat. 1997;10:250–2.
48 Murtagh J. The painful arm. Aust Fam Physician. 1990;19:1423–6.
49 Cyriax J. Textbook of orthopedic medicine: diagnosis of soft tissue lesions. 7th ed. Vol 1. London: Baliere Tindally; 1978.
50 Swift TR, Nichols FT. The droopy shoulder syndrome. Neurology. 1984;34:212–5.
51 Okereke CD, Mavor A, Naim M. Arterial thoracic outlet compression syndrome: a differential diagnosis of painful right supraclavicular swelling? Hosp Med. 2000;61:672–3.
52 Liu JE, Tahmoush AJ, Roos DB, Schwartzman RJ. Shoulder-arm pain from cervical bands and scalene muscle anomalies. J Neurol Sci. 1995;128:175–80.
53 Sizer PS, Phelps V, Brismée JM. Differential diagnosis of local cervical syndrome as compared to cervico-brachial syndrome. Pain Practice. 2001;1:21–35.
54 Winkel D, Matthijs O, Phelps V. Diagnosis and treatment of the upper extremities: nonoperative orthopaedic medicine and manual therapy. Gaithersburg (MD): Aspen Publishers; 1997.
55 Cornelis F, Zuazo I, Bonnefoy O, *et al.* Diagnosis of thoracic outlet syndrome. Value of angiography in the sitting position. J Radiol. 2008;89:47–52.
56 Seror P. Medial antebrachial cutaneous nerve conduction study, a new tool to demonstrate mild lower brachial plexus lesions. A report of 16 cases. Clin Neurophysiol. 2004;115:2316–22.
57 Roos DB. Thoracic outlet syndrome is underdiagnosed. Muscle Nerve. 1999;22:126–9; discussion 137–8.
58 Wilbourn AJ. Thoracic outlet syndrome is overdiagnosed. Muscle Nerve. 1999;22:130–6; discussion 136–7.
59 Cook C, Hegedus EJ. Orthopedic physical examination tests: an evidence-based approach. Upper Saddle River (NJ): PearsonPrentice Hall; 2008.
60 Plewa MC, Delinger M. The false-positive rate of thoracic outlet syndrome shoulder maneuvers in healthy subjects. Acad Emerg Med. 1998;5:337–42.
61 Gillard J, Perez-Cousin M, Hachulla E, Remy J, Hurtevent JF, Vinckier L, *et al.* Diagnosing thoracic outlet syndrome: contribution of provocative tests, ultrasonography, electrophysiology, and helical computed tomography in 48 patients. Joint Bone Spine. 2001;68:416–24.
62 Brismée JM, Gilbert K, Isom K, Hall R, Leathers B, Sheppard N *et al.* Rate of false positive using the cyriax release test for thoracic outlet syndrome in an asymptomatic population. J Man Manip Ther. 2004;12:73–81.
63 Wainner RS, Fritz JM, Irrgang JJ, Boninger ML, Delitto A, Allison S. Reliability and diagnostic accuracy of the clinical examination and patient self-report measures for cervical radiculopathy. Spine (Phila Pa 1976). 2003;28:52–62.
64 Mahmud MA, Merlo AR, Gomes I, Becker J, Nora DB. Relationship between adverse neural tension and nerve conduction studies in patients with symptoms of the carpal tunnel syndrome. Arq Neuropsiquiatr. 2006;64:277–82.
65 Quintner JL. A study of upper limb pain and paraesthesiae following neck injury in motor vehicle accidents: assessment of the brachial plexus tension test of elvey. Br J Rheumatol. 1989;28:528–33.
66 Orset G. Evaluation of the cervicothoracobrachial outlet and results of conservative treatment. Chir Main. 2000;19:212–7.
67 Berthe A. Considerations on rehabilitation of cervicothoracobrachial outlet syndrome. Chir Main. 2000;19:218–2.
68 Sobel JS, Kremer I, Winters JC, Arendzen JH, de Jong BM. The influence of the mobility in the cervicothoracic spine and the upper ribs (shoulder girdle) on the mobility of the scapulohumeral joint. J Manipulative Physiol Ther. 1996;19:469–74.
69 Kai Y, Oyama M, Kurose S, Inadome T, Oketani Y, Masuda Y. Neurogenic thoracic outlet syndrome in whiplash injury. J Spinal Disord. 2001;14:487–93.
70 Rayan GM, Jensen C. Thoracic outlet syndrome: provocative examination maneuvers in a typical population. J Shoulder Elbow Surg. 1995;4:113–7.
71 Howard M, Lee C, Dellon AL. Documentation of brachial plexus compression (in the thoracic inlet) utilizing provocative neurosensory and muscular testing. J Reconstr Microsurg. 2003;19:303–12.
72 Nord KM, Kapoor P, Fisher J, Thomas G, Sundaram A, Scott K, *et al.* False positive rate of thoracic outlet syndrome diagnostic maneuvers. Electromyogr Clin Neurophysiol. 2008;48:67–74.
73 Greening J, Dilley A, Lynn B. *In vivo* study of nerve movement and mechanosensitivity of the median nerve in whiplash and non-specific arm pain patients. Pain. 2005;115:248–53.
74 Bertilson BC, Grunnesjo M, Strender LE. Reliability of clinical tests in the assessment of patients with neck/shoulder problems-impact of history. Spine (Phila Pa 1976). 2003;28:2222–31.
75 Gilbert KK, Brismée JM, Dedrick G, Delgado B, Harle J, Miller J, *et al.* The interrater reliability of the cervical rotation lateral flexion test and correlation to first rib mobility testing. J Man Manip Ther. 2004;12:163–4.