

# Thoracic outlet syndrome: Its complexity is not disputed

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## Abstract

Thoracic Outlet Syndrome (TOS) has a complex history that has led to misunderstanding and confusion from physicians. We review the history of TOS to better understand where this confusion comes from. We explain the etiology of TOS to give improved recommendations for treatment of this unique patient population. We present the case of a patient with two types of TOS to further examine the workup and treatment of a TOS patient.

**Keywords:** Thoracic Outlet Syndrome, Neurogenic, Venous, Arterial

**Abbreviations List:** TOS: Thoracic Outlet Syndrome; nTOS: Neurogenic Thoracic Outlet Syndrome; vTOS: Venous Thoracic Outlet Syndrome; aTOS: Arterial Thoracic Outlet Syndrome

## Introduction

In 1818, Sir Astley Cooper described the first case of arterial thoracic outlet syndrome (aTOS) in a young woman with a prominent C7 transverse process [1]. In 1875, Sir James Paget and Leopold von Schroetter published the first cases of venous TOS (vTOS) [2,3]. In 1893, H. Lewis Jones published the first cases of neurogenic TOS (nTOS) [4]. Over the 200 years following Sir Astley Cooper's publication, numerous pre-eminent physicians and researchers published their research on thoracic outlet syndrome. Consider the contributions of, Paget, Halsted [5], DeBakey [6], Rontgen [7], Keen [8], Adson [9], Todd [10,11], Naffziger [12], Falconer [13], Wright [14], and Lord [15].

In the late 19<sup>th</sup> century following the introduction of diagnostic x-rays, the 'cervical rib syndrome' was born. Shortly thereafter, it became apparent that anomalous soft tissue structures in the thoracic outlet contributed to these entrapment syndromes [16]. In the mid-20<sup>th</sup> century physicians demonstrated multiple mechanisms of dynamic compression or entrapment caused by arm movement and positioning. By 1956, more than 25 distinct syndromes of upper extremity pain, paresthesia and weakness had been described when Peet et al. published their now-famous paper unifying all of these diverse mechanisms and patients under the umbrella of 'thoracic-outlet syndrome' [17].

Peet wrote, "The etiologic aspects of this complex of neurovascular symptoms remain confusing because so many anatomic deviations in the structures of the neck and shoulder have been described." However, clinicians seemed to favor a single term to encompass a broadly diverse group of patients with this confusing upper extremity pain syndrome.

To complicate matters further, Wilbourn published an opinion article regarding TOS in 1988 (based on 8 cases of surgical complications from first rib resections), stating, "many of the entities grouped under it have little in common beyond their known or presumed lesion site" [18]. Wilbourn introduced an arbitrary classification of "true" neurogenic TOS and "disputed" neurogenic TOS, without presenting any evidence to support the validation of these two terms with diagnoses or outcomes. His classification relied on the presence of muscle wasting and positive electrophysiological studies in patients with "true" neurogenic TOS, even though there is little evidence that EMG studies are sensitive or specific in the broad range of patients with TOS. Additionally, we do know that muscle wasting represents a late and potentially irreversible endpoint of any nerve entrapment

syndrome. With nTOS, grip strength testing with arms up and arms down correlates with symptoms and the elevated arm stress test (EAST) test. However, many patients with nTOS symptoms have normal electromyographies (EMGs), even when tested with the arm in the position of dysfunction [19].

Many physicians dismiss neurogenic TOS, partly due to this arbitrary and pejorative classification, which leaves many patients suffering with neither diagnosis nor treatment. Even clinicians who consider the diagnosis of thoracic outlet syndrome need to exclude other common entities, including cervical radiculopathy or myelopathy, peripheral entrapment syndromes (cubital tunnel syndrome, carpal tunnel syndrome, etc.), or primary orthopedic problems [20].

Confounding the diagnosis is the fact that a significant percentage of these patients have workers compensation claims, for whom there may be circumspection about potential malingering and "secondary gain." TOS also has a high incidence in patients with collagen-vascular disorders like Ehlers-Danlos Syndrome, who have a high incidence of complications with any surgical procedure, making clinicians less likely to want to treat, and therefore little incentive to completely diagnose [21].

Symptoms of nTOS include weakness (and eventually atrophy), numbness, pain (frequently non-dermatomal or multi-dermatomal due to the complex crossing of nerve fibers within the affected parts of the brachial plexus) [20]. In addition, there is usually a positional worsening of symptoms with arms elevated at least 90 degrees from the body, such as working with arms elevated at or above the head, or arms extended forward, such as driving a vehicle. There may also be focal pain and tenderness in the area where the compression is occurring, which may produce or aggravate distal symptoms.

In arterial TOS, repeated compression of the subclavian artery between the clavicle and the rib causes arterial wall damage and either narrowing of the lumen or aneurysm formation. Superimposed thrombus causes either local or distal acute arterial occlusion. In venous TOS, repeated compression of the subclavian vein beneath the subclavius tendon causes similar damage, resulting in thrombus and severely impaired venous drainage of the upper extremity. Either of these forms of TOS shows a dramatic clinical presentation, and are usually clinically obvious, necessitating urgent diagnosis and treatment. Positional symptoms in both aTOS and vTOS are also more the rule than the exception, but not pathognomic nor exclusive [20].

Beginning at the proximal thoracic outlet and working distally, the causes of plexus or vascular compression or injury include:

- Cervical rib or enlarged C7 transverse process, sometimes with an associated fibrous band, impinging on the C7 and C8 roots.
- Isolated fibrous band compressing or distorting the brachial plexus by itself, or between the band and another structure like the first rib.
- Abnormal scalene triangle caused by hypertrophied middle and anterior scalene muscles, broad or aberrant scalene muscle attachments on the first rib, or supernumerary muscles.
- Dynamic compression of one or more elements of the neurovascular bundle between the clavicle and the first rib, due to arm position, trauma, congenital bone anomalies, and other forms of

costoclavicular instability.

- Dynamic tension on the brachial plexus can occur at the level of the coracoid process, with arm elevation [22].

We still have a limited understanding of the complex biomechanical interactions of the shoulder region. Each scapula moves through six degrees of freedom; seventeen individual muscles control this motion. Spasm or weakness of subsets of these muscles can lead to abnormal scapular dynamics, which we believe lead to the development and progression of TOS. As a result, therapy that may focus on one form of scapular instability and one subset of muscles may worsen a situation where the spasm or weakness arises from a different subset of those 17 muscles. We believe that this leads to a high incidence of therapy failure.

Developmental soft tissue anatomic anomalies often cause or contribute to TOS [20]. Anomalous scalene muscles, scalene minimus variants, and fibrous bands are well-known, but often not considered as a possible causative component of TOS. These soft tissue anomalies are invisible to x-ray, but may be visualized on dedicated MRI studies. In patients with clear clinical evidence of TOS, normal radiographs do not rule out the disease.

Congenital or hereditary conditions that predispose to any of the above causes need to be evaluated so that progeny can also be counseled, and potentially genetic markers identified (and someday perhaps even edited). This way, therapy (non-invasive as well as invasive) can be directed towards treatment only for those likely to develop TOS, prior to permanent disability.

TOS-specific physical therapy has evolved considerably, and remains a vital component of assessment and treatment. Postural instability, muscle imbalances, breathing dysfunction, decreased thoracic and rib mobility, poor aerobic conditioning, ergonomics, low back pain, poor core strength, personal beliefs and coping mechanisms may aggravate TOS, or prevent symptomatic improvement if not adequately addressed. Upper extremity strengthening and stretching exercises, while commonly prescribed, are usually not the best first step for a TOS patient. Assessing nerve irritability and tolerance for movement is crucial. Any exercises given must be added in a way that does not aggravate the patient's condition. A TOS-experienced physical therapist can recognize and treat both TOS and other conditions that frequently coexist, while empowering the patient to calm their own symptoms and gain a sense of control. Bracing and physiotaping may also have a role in management of symptoms to minimize progression.

Invasive but nonsurgical treatments may include injections of steroids, local anesthetic, or botulinum toxin (BoTox™), and have good short-term and moderate intermediate term outcomes. They can serve both a therapeutic as well as diagnostic role in management of the disease [23].

New and less invasive surgical treatments, as well as the use of intraoperative neurophysiological monitoring, have lowered the bar for intervention in TOS patients. Treating physicians may be more willing to recommend these treatments earlier or more frequently than in the past when surgical treatments had higher incidences of major complications like vascular injury, pneumothorax, or nerve injury. Minimally invasive tenotomy for pectoralis minor syndrome, minimally invasive first rib resections, or minimally invasive resections of cervical ribs now allow for short incisions, ambulatory

procedures, and minimal morbidity treatments of TOS patients, reducing pain and post-operative morbidity [24,25].

Future interventions will likely also focus on restoring normal scapular dynamics and stability, including therapies or surgeries that lead to elevation of the scapula during elevation of the arms (when gravity imposes caudal displacement, leading to TOS exacerbations). Well-designed studies of scapular mechanics may help facilitate the diagnosis and treatment of this challenging condition.

These advancements will require clinicians, (neurologists, orthopedic surgeons, neurosurgeons, pain management doctors, emergency room physicians, vascular surgeons, and radiologists) to develop and share a common clinical pathway for evaluating this complex entity, and to share data that allows solid statistical evaluation of available diagnostic and treatment modalities. This will then allow clinicians to treat every patient effectively. Educating clinicians about the role that systemic conditions or diseases play in the diagnosis, conservative treatment, and surgical treatment of TOS patients is another critical step.

The complexity of TOS is often daunting to physicians who are already overwhelmed with differential diagnoses of conditions that they frequently treat. The thought of doing a specialized or complicated physical exam and/or radiographic work up necessary to eliminate the possibility, or diagnose the presence, of thoracic outlet syndrome (which only affects about 1% [3%-8%, Huang Zager [26]] of the general population) may result in some of these clinicians excluding this condition from their differential diagnosis

prematurely. Some may do so to save time and/or money, or to complete their workup more expediently, especially in an era where most physicians are graded or incentivized by how many patients they can see in a day, not necessarily the quality of the workup provided. Unfortunately, many physicians do not feel comfortable with their understanding of TOS, creating another barrier to appropriate workup.

This degree of intensive workup is necessary to make the correct diagnosis and to avoid overlooking TOS as a component of the patient's "pain generator." Treatment of TOS is more effective when initiated early in the course of the disease, to avoid re-clotting or re-occlusion in vTOS patients, or muscle atrophy in nTOS [27].

### Case Presentation

We present a patient with headaches, bilateral arm pain, and bilateral weakness. She has a positive EAST test bilaterally at 30 seconds. Right arm pain is worse with arms up, and this pain is reproduced by compression of the right costo-clavicular ligament. Left arm pain is worse with arms up, with weaker grip on Grip-X testing in the arm-up position, with reproduction of left radiating arm pain on compression of the left scalene muscles. Imaging demonstrates a narrowed costoclavicular interval on the right (1.0 cm, less narrowed on the left) with the arms abducted but no compression when the arms are down (Figure 1, Figure 2). This patient has two different types of TOS, both NTOS and ATOS, and because her story doesn't fall classically into what we call "Occam's Razor", she was given the diagnosis of fibromyalgia by her neurologist.

## Figure 1

- Coronal T2 STIR sequence, proprietary sequence (Neovista®)
- Red arrows point to clavicle and first rib on each side
- Right costoclavicular interval is narrower than left

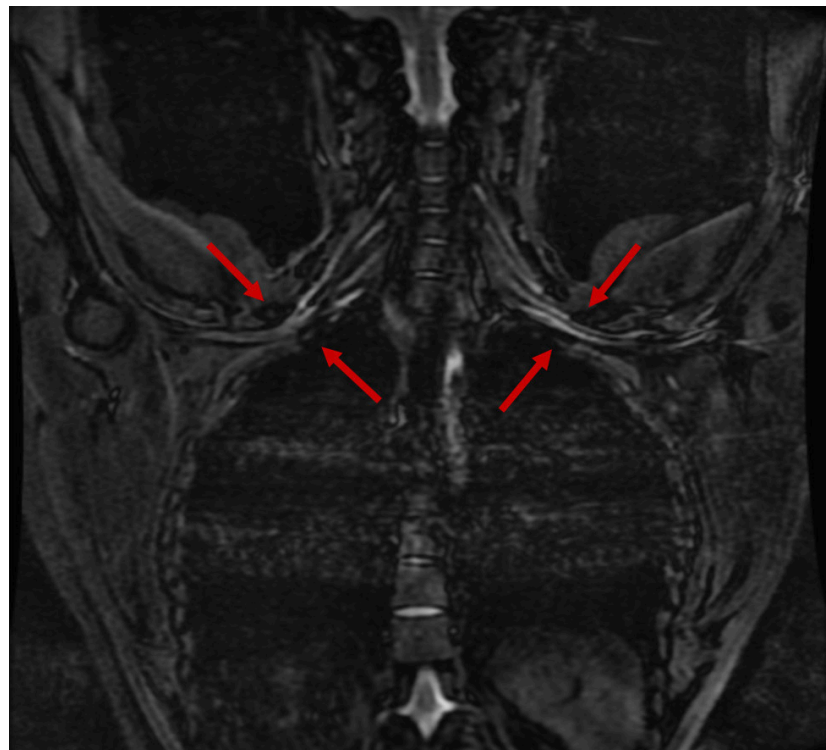
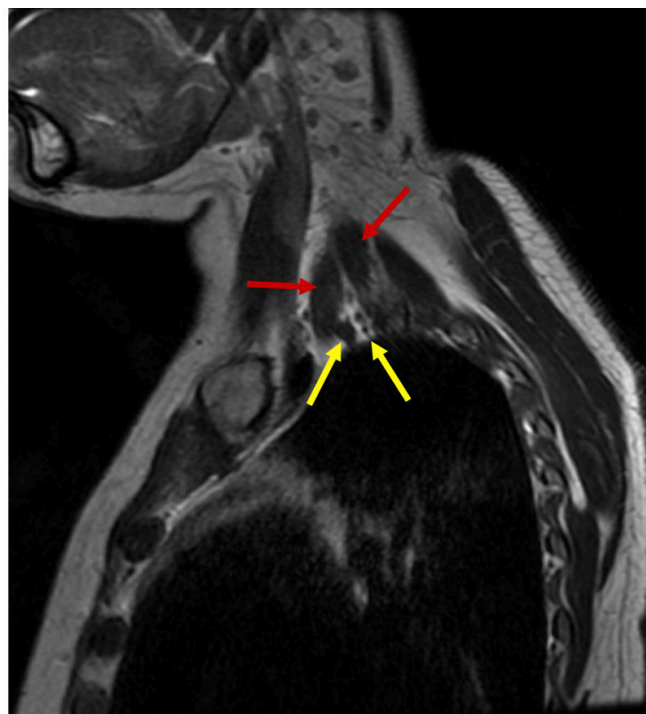


Figure 1: NeoVista imaging showing Coronal T2 STIR sequence.

## Figure 2

- T2 oblique sagittal sequence (proprietary of NeoVista®) showing the left scalene triangle
- Red arrows point to anterior and middle scalene muscles
- Yellow arrows point to subclavian artery (larger round structure on the left side of the scalene triangle) and the roots and trunks of the brachial plexus (smaller, vertically stacked structures on the right) within the scalene triangle



**Figure 2:** NeoVista imaging showing T2 oblique sagittal sequence.

### Conclusion

The concept of “disputed nTOS” should be discarded and referred to as “Early nTOS.” The adjective “disputed” is dismissive and does these patients an unnecessary disservice. Provocative questioning, insightful physical examinations, and diagnostic testing when indicated, need to be done routinely for patients with any symptoms that could be referable to spinal, shoulder problems, or peripheral nerve entrapments. Interventions need to be instituted early in the disease process to prevent progression of the condition, with a step-wise approach from least invasive to most invasive, keeping a focus on both local (outlet compression) and global (scapular mechanics) pathology. More research needs to be done to understand the biomechanics of the disease, improve and validate diagnostic tests and studies, and compare current and potential therapeutic interventions, to reduce the morbidity and increase the effectiveness of treatments.

### Conflicts of Interest

None to disclose.

### Statement of Non-duplication

I, Arthur L. Jenkins III, MD certify that this manuscript is a unique submission and is not being considered for publication, in part or in full, with any other source in any medium.

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