Thoracic outlet syndrome (TOS) is one of the most controversial subjects in entrapment neuropathies. Views differ regarding the pathogenesis, diagnosis, and management of TOS. Some authorities even question its existence. In spite of lack of understanding of the disease, clinicians often encounter patients whose pain or neurologic deficits in the upper limb cannot be explained after ruling out cervical spine diseases and other common causes of peripheral neuropathies. The goals of this article are to provide a comprehensive understanding of the syndrome by reviewing the anatomy of the region and the pathomechanism of the disease. Diagnosis can be established confidently in cases where the neurologic deficits are obvious and tests show positive findings. Issues surrounding cases where the objective findings are minimal are discussed. The duration of conservative treatment, indications for surgery, surgical approach, and patient selection for surgery are not standardized, and experiences vary across institutions. A number of studies in the past have shown conflicting results on the outcome of various surgical approaches, which has only added to the confusion. A systematic scientific approach aimed at understanding the pathogenesis of TOS, elucidating the criteria for diagnosis, and defining the treatment protocol has never been undertaken. Future endeavors should include multicentric, randomized, prospective clinical trials so as to answer these questions.

Until the first half of the nineteenth century, several terms were assigned to describe the symptom complex caused by neurovascular compression in the upper thoracic region based on the physiologic mechanism and structure that caused the compression (Table 1). Such rigid classification was the basis for explaining the pathogenesis, diagnosis, and treatment. In 1956, Peet et al, coined a common term, thoracic outlet syndrome, to encompass these various entities. It is now realized that strict classification is not practical. Patients tend to have multiple abnormalities, which may be responsible for the compression of the neurovascular bundle, and the clinical presentation is not unique to one syndrome.

ANATOMY

It has to be noted that TOS is a misnomer. Anatomically speaking, the true thoracic outlet is at the level of the diaphragm. Early
authors described the syndrome of neurovascular compression at the superior aperture of the thorax as TOS, and this incorrect term has since remained in the medical vocabulary. The anatomy of this region is complex, and understanding the relation between the neurovascular structures and the surrounding muscles and bony structures is critical in understanding the cause of compression.

The neurovascular complex traverses through three narrow straits within the thoracic outlet before entering the arm (Fig. 1). The first passage, the so-called “scalene triangle,” is mainly formed by muscles. The thoracic rib forms the base, and the anterior and middle scalene muscles form the anterior and posterior borders, respectively. The scalene muscles arise from the tubercles of the transverse processes of the cervical vertebrae and insert on the dorsal surface of the first rib. Although the hiatus transmits all the trunks of the brachial plexus, only the subclavian artery and lower trunk are closely related to the first rib. The subclavian vein passes in front of the anterior scalene muscle. After passage through the interscalene triangle, the neurovascular bundle passes through the costoclavicular triangle. Bordered by the first rib below, the clavicle and the subclavious muscle superiorly, and the upper border of the scapula and subscapularis muscle posteriorly, this hiatus transmits all the nerve trunks, the subclavian artery, and the vein. It has to be noted that the first rib is a structure common to both passages, and one of the surgical options is to resect the normal rib so as to pave the way to a wider passage. Finally, the neurovascular bundle traverses the subcoracoid space, a short tunnel just beneath the coracoid process of the scapula. The bundle is closely related to the fibrous clavipectoral fascia and the dense costocoracoid ligament anteriorly. As the flat tendon of pectoralis minor muscle ascends laterally to insert on the upper border of the coracoid process, it crosses the subcoracoid space. The ligament and tendon become taut during hyperabduction to significantly compress the space.

**PATHOMECHANISM**

Compression of the neurovascular structures as they course through the narrow passages of the upper thoracic aperture before entering the arm causes TOS. Any factor that compromises this narrow passage, which eventually leads to compression of neurovascular structures, can be included as a possible cause of TOS. These factors can be classified as:

- Anatomic or structural abnormalities: congenital (cervical ribs and fibromuscular bands) and acquired causes
- Physiologic or dynamic factors: developmental and postural factors
- Traumatic factors
- Idiopathic factors

A number of hypotheses have been proposed to explain the mode of compression in cases where structural abnormalities are not obvious.
thoracic outlet have been known to compromise the space through which the neurovascular structures course, the common anomalies are described here. Cervical ribs are supernumerary ribs and can be fully or partially developed. If a cervical rib is formed partially, it is associated with a ligamentous extension that inserts on the manubrium or the rib below. Incidence varies from 0.0004% to 1.5% according to various studies. Fifty percent of cervical ribs occur bilaterally, and, curiously, there is a female predominance. The mere presence of this anomaly does not confer the diagnosis of TOS because only 5% to 10% of individuals with cervical ribs actually develop the symptoms of TOS. The presence of this anomaly creates a barrier over which the neurovascular bundle has to acutely ascend to reach into the arm, which can result in microtrauma to the nerves from stretching and compression.

Fibrous bands are the most common congenital anomaly encountered that cause the symptoms of TOS. Various kinds of fibrous bands have been encountered, and attempts have been made to classify them according to their origin, insertion, and associated structures. As these anomalous structures traverse through the thoracic outlet, they tend to elevate, kink, and compress the neurovascular bundle against the surrounding anatomic borders of the thoracic outlet. Anomalous muscular insertion and muscle hypertrophy have been described to cause TOS, as they tend to trap the lower trunk of the plexus and the subclavian artery.
Acquired Anomalies

These anomalies are space-occupying lesions and can arise from the structures that form the borders of the thoracic outlet or from structures outside the outlet. Such lesions limit the neurovascular bundle into a narrow confinement. Fractures of the clavicle leading to malunion or formation of a large callus have been reported to cause TOS by reducing the costoclavicular space. Although uncommon, benign or malignant tumors of the first rib or the clavicle have been described as causing TOS. Among the lesions arising outside the outlet, a Pancoast tumor can compress the neurovascular structure. Rarely, malignant or inflammatory regional lymphadenopathy and tumors of the spinal cord or brachial plexus may present as TOS.

Dynamic Factors

Developmental Factors

It has been postulated that certain changes taking place during development can predispose to compression of the nerves and vessels. The position of the scapula dictates the inclination of the clavicle at the acromioclavicular joint, which, in turn, determines the width of the costoclavicular space. In infants, the scapula is high in the thorax, and the costoclavicular space is wider. As growth proceeds, the scapula descends as a result of the weight of the bearing limb. This, in turn, brings the clavicle closer to the first rib, thereby narrowing the costoclavicular space. Descent of the scapula is more pronounced in women, and this may explain the predominance of TOS in women.

Body Position and Habitat

Any factor that lengthens the descent of the neurovascular bundle so that the nerves and vessels become tensed as they traverse more obliquely against the first rib can cause TOS. Patients with cervicodorsal scoliosis often develop TOS symptoms on the side where the plexus has to travel a greater length. Poor physique of the shoulder girdle, debilitation, and age all tend to sag the scapula, which puts extra tension on the plexus.

Certain postures of the body tend to exacerbate or provoke the symptoms of TOS. Any position of the arm that stretches or compresses the plexus against the bones may consistently reproduce symptoms. Hyperabduction with external rotation of the arm is the most common position with which patients associate their symptoms. At arm abduction above 90°, the costoclavicular triangle closes, thereby compressing the cords between the bones. Patients usually complain that symptoms are aggravated at work when they use their arm overhead such as when a teacher writes on the blackboard or a painter paints the ceiling. Certain daily activities or household chores that involve using a repetitive motion of the upper extremity can worsen the symptoms.

Similarly, arm positions that depress the shoulder girdle (e.g., carrying heavy luggage, wearing a heavy bag over the shoulder) place pressure over the neurovascular structure by drawing the clavicle against the first rib. Hyperabduction and depression of the shoulder are maneuvers used by the clinician to provoke symptoms in patients with the diagnosis of TOS.

Traumatic Factors

The role of trauma is least understood of all, yet it has to be realized that it plays an important role. Most patients complain of the onset of TOS symptoms after an automobile accident or work-related injury that involves a strain of the ipsilateral neck or shoulder region. Not all patients who have trauma to the neck or shoulder region develop symptoms of TOS. Conversely, trauma in itself may be insufficient to cause TOS. It is most likely that trauma may be a precipitating factor in developing the symptoms in patients who have a predisposition because of congenital anomalies of the region. It has to be noted that there are few patients in whom the onset of symptoms occurs without any recognizable precipitating event. Whatever the cause, the final pathway in the pathogenesis of symptoms is the compression of the neurovascular bundle.
Clinical Presentation

Clinical features of the syndrome depend on the anatomic structures compressed. Accordingly, three distinct syndromes are recognized. These syndromes also help to delineate the presenting clinical signs and symptoms. They include (1) the arterial form caused by compression of the subclavian artery, (2) the venous form caused by compression of the subclavian vein, and (3) the neurogenic form caused by brachial plexus compression. The last form is by far the most common one encountered, and these syndromes rarely overlap.

Arterial Syndrome

The severity of symptoms depends on the extent of compression and injury to the subclavian artery. The symptoms and signs are secondary to the loss of blood supply to the arm. Mild ischemic symptoms may be the result of intermittent arterial compression at the thoracic outlet. Symptoms caused by compression may appear during certain postures of the arm (hyperabduction) or during exertion of the arm when the demand for blood supply increases. Patients are usually symptom-free between the attacks or during the summer season. Cold temperature always worsens the symptoms. During an acute episode, patients complain of pain followed by paresthesias; the pain is poorly localized and may involve the whole hand. Signs include a cool and pale extremity and the feeling of a stiff hand. Patients recover from the symptoms between the attacks. Such seasonal variation in the severity of symptoms is not seen with neurogenic or subclavian vein syndromes.

Sustained compression is a form of trauma to the arterial wall that can result in the formation of a mural thrombus leading to partial occlusion. Small emboli can dislodge from the thrombus and cause tissue necrosis, ulcers, and gangrene of the digits. These ischemic insults are more localized in the radial aspect of the hand, affecting mainly the index finger and the thumb.

Complete obstruction caused by a thrombus at the site of the injury secondary to compression or poststenotic dilatation resulting in turbulent blood flow is a rare complication. The severity of symptoms depends on the extent of collateral circulation in the arm. In acute cases, the presentation is typical of complete blood loss to the arm. Patients complain of severe pain and numbness. The limb is cool, pale, and pulseless and may become paralyzed and gangrenous. Formation of an aneurysm of the subclavian artery secondary to compression is rare.

Diagnosis is mainly based on presenting signs and symptoms. Several maneuvers have been described to aid in the diagnosis of arterial TOS. The following are the tests most commonly used:

- Adson test: with the patient in a sitting position, arms by the side, neck in extension, and facing either away or toward the affected side. The examiner feels the obliteration of the radial pulse of the affected arm when the patient is asked to inspire deeply.
- 90° abduction external rotation test: the examiner feels the radial pulse when the affected arm is abducted to 90° and externally rotated with the forearm in the 90° position. When the patient is asked to look in the opposite direction, the pulse may obliterate.
- Hyperabduction test: this simple test is positive when the radial pulse is diminished after elevating the arm above the head.
- Shoulder brace: many variants of this test have been described, but the test consists of bracing the shoulder backward and feeling for obliteration of the pulse.

Although these tests are employed to diagnose TOS, the specificity of these tests needs to be questioned. Studies have shown that positional obliteration of the radial pulse is seen in normal asymptomatic subjects. Positive results can be interpreted confidently only if they simulate the symptoms of the patient.

Arteriography of the subclavian system is the gold standard. This can aid in delineating the site of obstruction, demonstrate intramural lesions, and show dilatation. According to Sadler et al, positional arteriograms are of limited value, because positional arterial
compression occurs in normal asymptomatic subjects. Radiographs may reveal an associated cervical rib. There seems to be a general agreement about the indications for surgical intervention in cases of thromboembolic complication, poststenotic dilatation, and aneurysm formation. Transaxillary resection of the first rib is advocated along with resection of bony anomalies like a cervical rib or bony exostosis, if present, to decompress the passageway. Vascular repair may be necessary in certain cases.

**Venous Syndrome**

Venous compression at the thoracic outlet may be intermittent (positional), or venous obstruction may be associated with thrombosis. Venous thrombosis can develop at the site of compression. Acute symptoms develop after strenuous physical activity of the arm and are referred to as effort thrombosis or Paget-Schroetter syndrome. The affected limb feels heavy and becomes weak, tender, swollen, and cyanotic. Excruciating deep pain is felt in the chest, shoulder, and arm. Tingling paresthesias in the arm are common. Certain positions of the arm or strenuous physical activities can evoke intermittent compression. Symptoms are similar to the ones described previously.

In addition to compression, other causes of thrombosis have to be considered in the differential diagnosis, which includes iatrogenic (as a result of venous catheterization) or hypercoagulable states (malignancy, glomerulonephritis, cryoglobulinemia), vessel wall webs, and idiopathic factors.

Diagnosis depends on clinical suspicion. Venography is confirmatory and can show the site of obstruction or the presence of thrombus. Intermittent compression of the subclavian vein may be demonstrated during provocative maneuvers. The use and interpretation of Doppler flow measurement are difficult and of limited value. Venous pressure measurement has been used to support the diagnosis. Venous pressure higher than 10 mm Hg may be demonstrated in the affected limb compared with the contralateral limb.

The treatment in the intermittent type is to avoid provocative arm positions and to rest the arm. Anticoagulation is advocated to recanalize the thrombus. Arm elevation aids in reducing the edema. Venous thrombectomy has been suggested when a gangrenous or postphlebitic complication of thrombosis is an imminent danger. Along with thrombectomy, the first rib should be removed to relieve the obstruction of the venous system.

**Neurogenic Syndrome**

Compression of the brachial plexus is the most common form of TOS. Neurologic symptoms relate to the distribution of the lower trunk and the C8 through T1 roots, the elements of the brachial plexus that are closely related to the first rib and are prone to compression against it. Because the lower elements of the brachial plexus carry motor, sensory, and sympathetic fibers to the arm, symptoms relate to the fibers that are compressed.

**Sensory Symptoms**

Pain is the most common and earliest complaint. Pain is usually located in the supravclavicular region, axilla, back of the neck, and shoulder region going down into the arm. Some patients may have atypical pain in the retrosternal or parascapular region. If the retrosternal pain is felt on the left side, it can be confused with anginal pain. Pain is usually described as a dull constant ache that is difficult to localize. On most occasions, it does not respect the C8 through T1 dermatomes. Paresthesias are frequently more localized to the C8 through T1 dermatomes. Patients may complain of a “pins and needles” sensation and numbness on the medial side of the arm, forearm, hand, and fourth and fifth digits.

The sensory symptoms can be of a sudden or insidious onset. In most cases, patients are able to relate a major accident, fall, or any injury involving the shoulder region. In cases, where the onset is subtle, symptoms are recognized either during work (a barber may complain of pain and numbness in the hand after keeping the arm in a prolonged abducted position) or during routine household chores.

Although pain and paresthesias may be felt constantly, symptoms are aggravated by any
activities (e.g., carrying heavy loads on the shoulder or in the dependent arm) or position (e.g., combing hair, reaching toward a shelf) of the arm that put traction or pressure on the neural structures. These may be related to occupation, sports, or daily activities. Symptoms are usually worse at the end of a tiring day. Pain may be severe enough to disrupt a patient’s sleep during the night. In many instances, patients are forced to quit or change jobs to a less demanding one. In a few patients, cold weather seems to worsen symptoms.

Relief is bought about by discontinuing aggravating activities. Patients learn to relieve pressure over the brachial plexus by elevating the shoulder or resting the elbow on the table or on the arm of a chair.

Objective signs of sensory loss are usually a late finding. The percentage of patients with sensory symptoms who demonstrate sensory loss is unknown. Hypesthesia to pinprick and touch sensation is distributed in the C8 through T1 dermatomes. This hypesthesia eventually spreads to cover the whole hand. Vibratory sense, proprioception, and two-point discrimination may be affected variably.

Motor Symptoms

Complaints of subjective motor weakness may be elicited on questioning. Activities involved include hand grip, and fine hand movements are clumsy. Most notably, the intrinsic hand muscles innervated by the ulnar and median nerves are affected, which, in turn, have contributions from the C8 through T1 spinal nerves. Objective signs of muscle wasting are uncommon and seen in long-standing cases only. Muscle bulk and signs of paresis of the small intrinsic hand muscles should be assessed. There is a predilection for the first dorsal interosseous muscle (innervated by the ulnar nerve) and thenar muscles (abductor pollicis brevis and opponens pollicis innervated by the median nerve). The pattern of thenar muscle atrophy gives a characteristic guttering on the lateral aspect of the hand. There are several unsettled explanations as to the highly selective nature of muscle atrophy. The muscles of the medial forearm may also be affected, but this is usually not conspicuous.

Sympathetic Involvement

The C8 through T1 spinal roots carry sympathetic fibers, which supply most of the upper limb. Vasomotor disturbances accompany sensory and motor symptoms. Isolated cases of sympathetic involvement are rare but have been reported in the past. Symptoms of vasomotor instability include bluish red discoloration of the dependent arm and blanching of the hand affecting the whole hand. Intermittent attacks of cyanosis or pallor of the hand are usually precipitated by emotional disturbances or by exposure to cold, and the severity of symptoms is independent of arm position. Trophic changes of the hand have been described in long-standing cases, including thinning of skin, loss of hair, and thickening of nails.

Vasomotor involvement caused by TOS has to be differentiated from other causes of vasomotor instability. Disturbances like reflex sympathetic dystrophy, causalgia, Raynaud’s disease, secondary causes of Raynaud’s phenomenon (e.g., connective tissue disorder, obstructive arterial diseases, neurogenic lesions elsewhere, drug intoxication, dysproteinemias), acrocyanosis, and erythromelalgia should be kept in mind. Patients with Raynaud’s disease usually present with bilateral involvement for longer than 2 years without much progression of the disease. At times, a careful history and physical examination can elicit the secondary causes as a result of underlying disease. Acrocyanosis usually occurs in women and presents with bilateral cyanosis of the hands and, occasionally, the feet. Erythromelalgia is rare and involves sudden burning pain along with redness and increased temperature of the feet and hands.

DIFFERENTIAL DIAGNOSIS

The symptoms of neurogenic TOS overlap with the clinical presentation of commonly encountered entities. Clinicians are faced with the challenge of first excluding these causes before giving a diagnosis of TOS. Cervical degenerative diseases are common and important in the differential diagnosis. The
following signs are helpful:

- Patients with cervical disk disease have tenderness over the spinous processes and paraspinal muscles, whereas TOS patients tend to have tenderness over the supraclavicular fossa.
- Valsalva maneuvers and neck hyperextension usually provoke radiating pain going down the symptomatic arm in patients with degenerative disk disease.
- Downward pressure on the top of the head with the neck hyperextended and flexed toward the symptomatic side worsens the symptoms in patients with cervical herniated disks.
- Signs of upper motor neuron involvement might be elicited on examination in cases where the osteophytes compress the spinal cord.

Entrapment of peripheral nerves distal to thoracic outlet present with a confusing picture. The clinical picture caused by ulnar nerve neuropathy closely mimics the presentation caused by TOS. Examinations oriented specifically toward distinguishing ulnar entrapment from TOS are as follows:

- Tinel's sign at the medial epicondyle eliciting radiating paresthesias along the ulnar nerve distribution.
- Nerve conduction velocity studies show slowing of conduction of the ulnar nerve at the level of the elbow.
- Normal pinprick and light touch sensation are demonstrated on the radial aspect of the fourth finger in the case of ulnar neuropathy.

Certain non-neurologic diseases that can mimic the symptoms of TOS include musculoskeletal (arthritis, bursitis, tendinitis of the shoulder or elbow region) and visceral (angina pectoris, esophageal diseases, gastric ulcer, pulmonary diseases) causes.

It is important to recognize secondary causes of brachial plexus compression. Pancoast tumors can invade the thoracic outlet and compress the plexus. The diagnosis is made on eliciting a history of smoking, and a plain chest radiograph reveals an apical mass. The presence of Horner's syndrome is an indication for brachial plexus involvement.11

The supraclavicular and axillary regions are rich in lymph nodes. Enlargement of these nodes as a result of metastasis or inflammation can compress the brachial plexus.Palpating the supraclavicular and axillary regions can reveal adenopathy. Metastasis to the brachial plexus from breast carcinoma can result in TOS. Trauma to the upper thoracic region can lead to compression. Malunited clavicular fractures can result in callus overgrowth, which can impinge on the plexus.10,47 Atasoy3 reported a case of schwannoma of the C7 root causing compression of the thoracic outlet.

**DIAGNOSIS**

Suspicion for TOS should be raised in women with a long neck and sloping shoulders who present with a history of some trauma to the shoulder region and symptoms referred to the C8 through T1 dermatomes. TOS remains a diagnosis of exclusion; thus, a detailed history and physical examination should aim at ruling out other common entities that present with an overlapping or similar clinical picture.12,59,79

The authors like to distinguish two forms of neurogenic TOS. Most patients with brachial plexus compression present with the main complaint of pain. There are no objective signs of neurologic deficits present. Physical examination usually reveals no appreciable sensory loss or motor atrophy in the hand. Radiologic studies, nerve conduction tests, and electromyographic recordings show no abnormalities. Wilbourne and Porter have called the entity a disputed form of TOS. Much controversy exists as to its existence, because the diagnosis of this entity is based purely on clinical judgment. Physical signs specific to TOS that should help a clinician to support the diagnosis of TOS include (1) palpating the supraclavicular fossa to elicit tenderness and (2) tapping the supraclavicular fossa to elicit tingling paresthesias radiating down the arm. A positive Tinel's sign at the supraclavicular region can be considered specific for TOS.

Stress tests are maneuvers that provoke the symptoms of pain and paresthesias on the medial side of the hand and forearm. Such provocative tests further compress the neurovascular structures as they pass through
the narrowed outlet. Common tests are as follows:

- Hyperabduction test: the arm of the patient is raised above the level of the head.
- Costoclavicular test: pressure over the shoulder region may provoke symptoms.
- Military maneuver: eliciting the symptoms by elevation of the chin and pulling the shoulder joint behind in an extreme "attention" position.

Because of the lack of systematic clinical study, the reliability and specificity of these tests in diagnosing TOS are unknown.

In the true neurogenic type of TOS, there may be signs of intrinsic hand muscles atrophy and reduced pinprick and touch sensation in the C8 through T1 distribution. Radiologic and electrodiagnostic studies may indicate some abnormality. Unfortunately, there is no test that is pathognomonic of TOS. Positive findings in the ancillary tests are associated with the true form of TOS. The main purpose for ordering these tests is to exclude other diagnoses.

Radiologic studies are helpful in excluding the secondary causes of compression caused by structural anomalies. Plain radiographs of the cervical spine are helpful in showing a cervical rib, an elongated transverse process, or scoliosis of the region. Malunited fractures and malignant lesions arising from the bony wall of the thoracic outlet can be demonstrated. The cervicothoracic junction should be carefully inspected for malignant lesions such as a Pancoast tumor. Cervical degenerative diseases can be ruled out using plain radiographs. Discograms, myelograms, or MR imaging of the spine may be needed to further delineate the level of disk disease. CT is helpful in excluding intraspinal (e.g., tumor, syringomyelia) causes of sensory and motor symptoms. MR imaging of the cervicothoracic region has been shown to be useful by demonstrating distortion of the plexus. In addition to demonstrating bony anomalies of the region, MR imaging has the advantage of disclosing hypertrophy of muscles and radiologically invisible bands that can compress the neurovascular bundle.

Electrodiagnostic studies can help to support the diagnosis of TOS by excluding the common causes of neuropathies at sites other than the plexus such as ulnar neuropathy at the elbow and median neuropathy in the carpal tunnel.

Abnormal patterns of neuroelectric disturbances have been described in TOS patients. Electromyography may reveal denervation of intrinsic hand muscles innervated by the median and ulnar nerves. Prolongation of F-wave latency has been described in patients with atrophy of the hand muscles. Abnormalities in the somatosensory evoked potentials obtained after stimulating the ulnar nerve have been noted in a few patients. Urschel et al and Caldwell et al described the slowing of ulnar nerve motor conduction velocity across the thoracic outlet with enthusiastic results. They stimulated the plexus at the supraclavicular fossa and found abnormal conduction in the ulnar nerve in almost all their cases. This test is controversial, because many investigators have not been able to replicate the same results. The significance of electrodiagnosis in TOS is limited because most patients do not show any kind of abnormality. Numerous studies show a high variability in the outcome of these tests. Until a systematic clinical trial resolves the question, caution must be exercised when interpreting the data.

TREATMENT

Patients with TOS have often been misdiagnosed or passed off as histrionic. They are comforted to know that their symptoms are consistent with a diagnosis and that treatment can be instituted. As in most medical conditions, treatment can be divided into nonoperative and operative categories. The patient should avoid postures that exacerbate symptoms. A change in work habits, including attention to the work place can help those patients with repetitive stress disorder. Physical therapy likely plays a role, but the literature is lacking in outcome data. The general approach involves measures to strengthen the shoulder girdle and improve posture. Cervical traction can be added. Nonoperative care can help many patients to avoid surgery. The surgical approach to TOS varies depending on the philosophy and training of the surgeon. If the pathology is thought to be caused by
compression of the lower trunk from fibrous bands or a cervical rib, a supraclavicular approach is taken. If, however, the pathologic findings are thought to be more distal, resection of the first rib is performed. Within the vascular literature, treatment of vascular compression is approached most often with first rib resection.

Access to the first rib can be gained from a supra- or infraclavicular approach. Murphy described the supraclavicular approach to the first rib in 1910, but this approach was abandoned as being too dangerous. It was rediscovered in 1962, when Clagett introduced the posterior thoracotomy rib resection. Roos reported the transaxillary approach, which has become the most popular surgical approach for first rib removal. For some, a combined approach affords the best surgical outcome.

Supraclavicular Approach

For most neurosurgeons, this approach is the most familiar, providing direct visualization of the brachial plexus. The patient is positioned supine with the arms adducted at the side, and a general anesthetic is delivered. Muscle paralysis is avoided to allow electric stimulation. The incision is made just above and parallel to the clavicle. The platysma is sharply divided, and the lateral one third of the sternocleidomastoid muscle is divided. The omohyoid muscle can either be retracted or divided, and the fat pad of the posterior triangle is elevated. The phrenic nerve is located, often aided by electric stimulation, on the anterior scalene muscle and is protected for the duration of the case (Fig. 2). The anterior scalene muscle is then divided, and the bulk of the muscle is removed. The upper trunk of the brachial plexus is located along the lateral border of the anterior scalene muscle. The middle and lower trunk lies inferior and slightly posterior to the upper trunk. A branch of the thyrocervical trunk may have to be divided to allow exposure of the lower trunk.

A neurolysis is performed encompassing the upper, middle, and lower trunk of the brachial plexus. Moving proximal along the

![Figure 2. Structures encountered during supraclavicular approach to neurolysis of the brachial plexus. Often the anterior scalene muscle is removed before the neurolysis of the plexus is performed. Note the close relation of the phrenic nerve with the anterior scalene muscle. Ext = external; Int = internal; Trans = transverse; v = vein; m = muscle; ant = anterior; n = nerve; a = artery. (Adapted from Weinshel SS: Surgical exposure of the brachial plexus. In Benzel EC (ed): Practical Approaches to Peripheral Nerve Surgery. Illinois, AANS publications; with permission.)](image-url)
lower trunk, Sibson's fascia over the dome of the lung is released, and, separated by the first rib, the C8 and T1 roots are identified. In this area, several anomalous structures can be encountered, including (1) fibrous bands arising from the C7 transverse process, first rib, or apical lung pleura; (2) arterial vessels or a high arching subclavian artery; and (3) a cervical rib with fibrous bands arising from the end of the rib and inserting on the first rib. A cervical rib can be readily resected using this approach.

The first rib can be exposed by retracting the elements of the plexus. The rib can be removed through this exposure; however, this is a technically challenging procedure that places the nerves and vessels between the surgeon and the rib. As such, most surgeons prefer the transaxillary approach if the first rib is to be resected.

**Transaxillary Approach**

After delivery of general anesthesia, the patient is positioned prone, with the arm abducted, externally rotated, and suspended (3-lb weight). An incision is placed at the caudal extent of the axilla, where the skin breaks away from the chest wall (Fig. 3). Dissection is taken to the chest wall and up toward the axilla (see Fig. 3A). The intercostobrachial nerve, a branch...
of the T2 intercostal nerve, traverses the area
on its way to the arm and needs to be identi-
ified and protected. The first rib is palpated as
a broad flat structure. A localizing radiograph
can be obtained if there is any question as to
its identification. Deaver retractors are used to
elevate the soft tissues of the axilla, including
the vessels and nerves. The intercostal muscles
are stripped from the rib using a combination
of blunt dissection and cautery. The pleura is
located and divided. Palpation is used to iden-
tify any anomalous bands inserting on the rib
(see Fig. 3B).

Removal of the first rib can be accomplished
using a variety of techniques. The authors pre-
er a rib cutter in combination with rongeurs
used to remove the rib piecemeal. Resection
proceeds to within 2 cm of the transverse pro-
cess. An intraoperative radiograph can be ob-
tained to confirm the extent of the proximal re-
section. The lower trunk courses over the rib
and needs to be protected as does the T1 root as
it emerges from the foramen just below the in-
sertion of the first rib on the transverse process.
The anterior extent of rib resection terminates
distal to the subclavian vein. A cervical rib can
be resected from the transaxillary approach af-
ter completion of removal of the first rib; how-
ever, the authors prefer to remove cervical ribs
from a supraclavicular approach (see Fig. 3C).

If the pleura has been violated, it does not
have to be closed; however, the patient is main-
tained on positive-pressure ventilation until
the wound is closed. If the lung has been vi-
olated, a chest tube is placed. If there is a ques-
tion of lung injury, a postoperative chest radi-
ograph is obtained to look for a pneumothorax
while the patient is in the recovery room.

Outcome

As is the case with most surgical treatments,
patient selection remains the most important
criterion for a good outcome. With some ex-
ceptions, studies have suggested a high suc-
cess rate for surgical intervention using either
a supraclavicular or transaxillary approach.29,72
Patients with profound intrinsic muscle atro-
phy do not normally regain function; rather,
the surgery is performed to arrest the progres-
sive deterioration.

In one study, an orthopedic surgeon and a
vascular surgeon jointly conducted 30 op-
erations for TOS in 27 patients. Anterior
scalenectomy was performed by the supra-
clavicular route, except in one case, where the
infraclavicular route was used. The further
surgical procedure was tailored to the abnor-
malities identified (i.e., resection of a cervical
rib or band, medial scalenectomy). The first
rib was spared. At a median follow-up of 37
months, results were judged excellent or good
on 26 of 30 sides (87%); on the three occasions
when scalenectomy alone was performed, the
results were only fair or poor. There were no
major complications. The long-term outcome
in this series suggests that good results can be
obtained by the supraclavicular route without
resection of the first rib.53

Wenz and Husfeldt78 treated 67 patients with
a total of 80 TOSs between 1980 and 1994 (av-
erge follow-up of 6.6 years). Each of these pa-
tients was treated with transaxillary resection
of the first rib, excision of fibrous bands, and,
if necessary, excision of cervical ribs.

A total of 84.5% of the patients had complete
resolution or improvement of symptoms.78
Edwards et al31 studied 52 rib resections per-
formed in 46 patients with a median follow-up
interval of 33 months. Postoperative symp-
tomatic improvement was accepted as a confir-
mation of correct preoperative diagnosis. The
indications for surgery were neurologic symp-
toms (n = 42 excisions), arterial compromise
(n = 5), and venous compromise (n = 5). A to-
al of 42 patients (48 resections) showed imme-
diate improvement in symptoms after surgery,
although symptoms recurred in three patients
(four resections) between 6 and 8 months af-
after surgery. In the final 2 years of this study, 20
resections resulting in symptomatic improve-
ment were performed, suggesting a prevalence
for TOS of at least 10 per 100,000 population
per year. Thoracic outlet decompression was
performed more frequently in this series than
in many previous studies, suggesting that TOS
may be underdiagnosed.21
Donaghy et al performed 49 operations in 40 patients (33 women, seven men; age range, 22-62 years) with follow-up at 3 months to 20 years after surgery. Cervical ribs were removed in 23 patients together with fibrous band excision in nine. In the 17 patients without a cervical rib, the thoracic outlet was decompressed by resection of the first thoracic rib in nine and by supraclavicular operations in eight. After surgery, patients reported reduced pain (33 of 36 patients), and improved muscle strength (14 of 27 patients), and hand function (23 of 24 patients). After surgery, TOS recurred in two patients, and symptoms continued to progress in three patients in whom other diagnoses eventually emerged. Surgical complications were recorded in 10 patients but were transient and did not result in permanent symptomatic sequelae. These investigators concluded that surgical treatment of suspected neurogenic TOS relieves pain and sensory disturbance (90%) but is less effective for muscle weakness (50%). Surgery relieved sensory and motor abnormalities to a similar degree in patients with and without a cervical rib.

Franklin et al studied all injured workers in the Washington State Workers' Compensation system who received TOS surgery from 1986 to 1991 (n = 158). The main outcome measure was work disability status 1 year after surgery. Additional functional status and quality of life outcomes were determined by telephone survey an average of 4.8 years after surgery. A sample of workers with a TOS diagnosis who did not receive surgery during the period from 1987 to 1989 were identified as a comparison group (n = 95). It cannot be stressed enough that this was not a randomized study, and as such, comparison of the surgical group with the nonsurgical group is of limited value. Sixty percent of workers were still work-disabled 1 year after surgery. The strongest predictors of remaining disability were the amount of work disability before surgery, longer time between injury and TOS diagnosis, and older age at injury. There was no relation between type of surgery, presence of any provocative tests, experience of the surgeon, and work disability outcome. In follow-up surveys, 72.5% of workers still reported that they were "limited a lot" in vigorous activities. Compared with a nonsurgical sample of TOS patients, those receiving surgery had 50% greater medical costs and were three to four times more likely to be work-disabled. The nonspecific neurogenic TOS diagnosis, complexity of workers' compensation cases, and adverse event profiles are likely substantial contributors to the worse outcomes reported here. Goff et al compared surgery for neurogenic TOS between laborers and nonlaborers. Laborers with TOS were less likely to have a good result from surgical intervention and were unlikely to return to their original occupation.

**SUMMARY**

TOS represents a spectrum of disorders encompassing four related syndromes: arterial compression, venous compression, neurogenic compression, and a poorly defined pain syndrome. Patients can present with signs of arterial insufficiency, venous obstruction, painless wasting of intrinsic hand muscles, and pain. History and physical examination are the most important diagnostic studies, and radiographs of the chest and cervical spine and electromyography/nerve conduction studies are useful to identify other causes of pain and disability. Surgical intervention is indicated for patients failing nonoperative maneuvers and can usually yield satisfactory results. TOS may also be the most underrated, overlooked, and misdiagnosed, and the most important and difficult to manage peripheral nerve compression in the upper extremity.

**References**

DIAGNOSIS AND TREATMENT OF THORACIC OUTLET SYNDROME


Address reprint requests to
Allan J. Belzberg, MD, FRCSC
Johns Hopkins Hospital
600 North Wolfe Street, Meyer 5-109
Baltimore, MD 21287-7509