Thoracic Outlet Syndromes:

Symptoms, Diagnosis, Anatomy and Surgical Treatment

David B. Roos, M.D., FACS

The thoracic outlet syndromes (TOS) are distinct symptom complexes affecting the neck, shoulder, arm, and hand that are caused by compression or irritation of the brachial plexus, subclavian artery, or vein as they pass over the apex of the lung through the thoracic outlet and costoclavicular space. Depending on the predominant symptoms, these syndromes may be divided into three categories: neurologic, arterial, or venous. The neurologic type may be further divided into three categories: the upper plexus type, the lower plexus type, and the combination of the two.[5] Each results from anatomic abnormalities formed either congenitally or resulting from trauma that affects one or more of the major neurovascular structures passing through the thoracic outlet.[4]

The neurologic type is by far the most common, comprising about 97% of all cases of the outlet syndromes. It affects principally young and middle-aged adults from the teens to the late 40s, but is practically unknown in children, and rare in adults over 50. Women are predominantly affected by this type by a ratio of 2 or 3 to 1. Although the neurologic symptoms may develop spontaneously, in approximately two-thirds of the cases they are precipitated by trauma that results in chronic muscle spasm in the neck and shoulder region.

Dr. Roos is Clinical Professor of Surgery at the University of Colorado Health Sciences Center, Denver, Colorado.

Address correspondence to Dr. Roos, 1721 E. 19th Avenue, Denver, CO 80218.

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Symptoms. The neurologic symptoms of TOS may be considered under categories of pain, paresthesias, and paresis. Because the compression or irritation of the nerves of the brachial plexus varies with arm activities, positions, and rest, the symptoms usually are intermittent, unlike the more constant but similar neurologic complaints occurring with cervical disc or carpal tunnel syndromes. The neurologic symptoms seem to fall into two distinct categories: those from compression or irritation of the upper nerves of the brachial plexus, C5, 6 and 7, as opposed to those from the effects of the lower nerves of the plexus, C8 and T1. Patients with upper plexus involvement report pain in the front and side of the neck, radiating from the clavicle upward just behind the sternocleidomastoid muscle to the ear, anteriorly into the upper chest, posteriorly into the scapular or rhomboid muscle area, laterally through the trapezius muscle over the cap of the shoulder, and down the *outer* aspect of the arm, sometimes as far as the back of the thumb and index finger in a typical C5-6 pattern commonly seen with herniated discs at that level.

The pain of the *lower* plexus involvement is quite different, originating low in the supraclavicular fossa, passing through the infraclavicular region into the axilla, then down from the back of the axilla through the inner aspect of the brachium to the inner elbow and forearm into the ring and small fingers, the typical ulnar nerve distribution. Patients may have referred pain in the rhomboid region medial to the scapula as well, and both types may cause posterior cervical muscle spasm and pain resulting in severe occipital or hemicranial headaches.

Most patients with TOS also have intermittent paresthesias of the involved arm and hand. Other dysesthesias of itching, burning, or coldness may be felt in the distribution of the pain of the upper or lower plexus involvement.

Paresis gradually develops as the symptoms increase, and includes heaviness, fatigue, and weakness of the upper limb, and loss of fine coordination of the fingers.

The venous symptoms of TOS consist of swelling, cyanosis, fatigue, heaviness, and aching of the upper extremity. If the subclavian vein is being compressed extrinsically by anomalous structures, the symptoms are intermittent and positional, worse with exertion or elevation of the arm, and clear with resting and dependency of the limb. If the symptoms persist and never clear, they may be caused by intrinsic thrombosis of the subclavian vein. In both instances, venous congestion is aggravated by limb use and exercise because activity quickly increases the arterial demand and flow into the extremity, which can no longer be balanced by simultaneously increasing venous egress due to obstruction of the main venous channel. A paradox may be seen in these instances which differs from venous occlusion of the lower extremities. When the limb is elevated, especially in abduction, venous engorgement may rapidly develop and the veins of the wrist and forearm may distend rather than empty, as one would expect with elevation above the heart. This results from constriction of the thoracic outlet itself from muscle contraction to elevate the limb, and rotation of the clavicle which closes the costoclavicular space. Venous insufficiency is exaggerated if the limb is exercised in this position because the arterial demand will increase but venous emptying is impaired by compression of the major subclavian vein as well as the collaterals passing through the outlet.[6]

The arterial symptoms of TOS consist of early muscle fatigue and cramp

with repetitive use; cold sensation, pallor, or dusky cyanosis of the fingertips from digital emboli; and, in an extreme case, severe ischemic pain, empty cutaneous veins, and even digital gangrene. These symptoms are the most serious and difficult to manage, but fortunately they are the least common. The predominantly neurologic symptoms constitute approximately 97% of the cases of TOS; the venous insufficiency symptoms comprise about 2%; and the arterial insufficiency only about 1% of the entire spectrum of patients with TOS.

It is evident that performing artists, especially instrumentalists, could be seriously affected, or even disabled, by the protean symptoms noted above. In my practice, I have seen 11 string instrument players who developed symptoms of TOS that seriously affected the stamina, speed, and dexterity of their left arms and hands. Four were music students studying under scholarship who became so disabled they were unable to play, thus threatening their scholarships, education, and future careers. Another was the first violinist and concertmaster of a major orchestra who felt his performance deteriorated so much that his job was in jeopardy. Each had typical symptoms of the neurologic type of thoracic outlet syndrome that failed to respond to various conservative measures, and ultimately required surgical decompression of the outlet by transaxillary first rib resection. Each had good to excellent response, returned to unrestricted practicing, and continued their careers. The four students maintained their scholarships and graduated with a degree in music.

String Instrumentalists Requiring Surgery for TOS

Instrument	*Patients*
Violin	6
Cello	3
Viola	1
Bass	1

Each was incapacitated or about to give up their instrument prior to operation, but each resumed playing without restrictions postoperatively.

Case Report. K.H., a 21-year old, right-handed college music student on scholarship began playing violin at the age of four. A few months prior to being referred to me, her left hand began to tire easily, developed a tremor, and her long, ring and small fingers cramped and felt numb and tingly. Previously she could practice several hours daily without distress, but her practice time gradually diminished to 2 hours, then 1 hour a day, and then only briefly. Avoiding practicing for 3 weeks at a time failed to improve her symptoms. She was awakening at night with aching, throbbing discomfort in the back of her shoulder and inner left forearm. She was threatened with loss of her scholarship if she were unable to continue playing in the school orchestra and chamber groups, which became impossible with her increasing symptoms.

From her history and physical examination, I believed she had typical left thoracic outlet syndrome of lower plexus type causing tremor, cramping, weakness, and loss of dexterity of her left hand and fingers. Because of the severity of this disability, and failure of her symptoms to respond to intervals of complete rest for 2–3 weeks at a time, mild exercise, heat, anti-inflammatory and muscle relaxant medications, early operation was advised.

She underwent left transaxillary first rib resection, had an uneventful postoperative course, and missed only a few days of classwork. I asked her to avoid the violin for several weeks, then gradually resume playing. Initially she felt symptoms in the left arm and hand when she played more than one-half hour, but these gradually faded and her practice time increased over the ensuing months. Six months postoperatively she was playing with no restrictions. She fulfilled all her course and performance requirements through her senior year to receive her bachelor's degree in music with a major in violin.

She then entered the master's program, and also taught young pupils on a regular basis the following two years. Again she performed without interruption, fulfilling all requirements for the master's degree in music. The program for her graduate recital, which she played almost flawlessly, is noted below. Those familiar with the violin repertoire will recognize this as an extremely difficult program at the master's level. She continued to teach and play in several orchestras. Finally, after national competition, she was awarded a faculty position teaching violin at a prominent school of music 4½ years after her operation for disabling thoracic outlet syndrome.

PROGRAM

Francesco Maria Veracini (1690–1750)	Sonata in e Minor Largo Allegro con fuoco Minuet and Gavotte Gigue
Wolfgang Amadeus Mozart (1756–1791)	Adagio in E major, K. 261
Wolfgang Amadeus Mozart	Rondo (Arr. by Fritz Kreisler)

Intermission

Johann Sebastian Bach (1685–1750)	Partita No. 1 in b Minor Sarabande Double Tempo di bourrée Double
Igor Stravinsky (1882–1971)	Suite Italienne Introduzione Serenata Tarantella Gavotta con due Variazioni Scherzioso Menuett e Finale
Fritz Kreisler (1875–1962)	Variations on a Theme of Corelli

Etiology. Patients in whom the thoracic outlet syndromes develop apparently have *anatomic abnormalities* in the outlet that make them susceptible to various neurovascular symptoms. These anomalies are of several types, the most notable being the skeletal anomaly of cervical rib. An elongated transverse process of the seventh cervical vertebra (longer than T1 on cervical spine x-rays) apparently is an abortive attempt at formation of a cervical rib. This is supported by the usual finding at operation in such patients of a congenital fibromuscular band arising from the tip of an incomplete cervical rib or an elongated C7 transverse process that passes under the T1 nerve and inferior trunk of the plexus, and attaches to the medial edge of the first rib just behind the scalene tubercle. These are the most striking anomalies as they are readily apparent on x-rays of the neck and chest, but far more common are the fibromuscular congenital bands of variable configurations in the same region that inevitably are found at surgical exploration in severe cases. Currently, I have identified 10 different types of anomalous

fibromuscular bands in the thoracic outlet that are in direct contact with the neurovascular structures and may cause compression or irritation, particularly of the nerves, though they may involve the subclavian artery and vein also. These anomalous bands may occur singly but more commonly are found in various combinations.[4]

Other anomalies may also contribute to neurovascular compression, such as exostosis or pseudarthrosis of the first rib, hypoplasia of the first rib, which may appear radiologically to be a cervical rib, and abnormalities of the clavicle such as fracture callous formation or pseudarthrosis. The bone anomalies are usually apparent radiologically, but the soft tissue anomalies, which are much more common, are not demonstrable by any radiographic technique. They are found and appreciated only by the surgeon during operative exploration of the outlet, and only then if they are carefully looked for!

Understanding the various anomalies in the outlet and their effect on the neurovascular structures is basic to the understanding of TOS, and in determining appropriate diagnostic tests and their effective treatment. Failure to appreciate these anomalies, their frequency, and their variations in the general population perpetuates the confusion, misdiagnosis, and mismanagement that has been associated with TOS for decades.

Diagnosis

The diagnosis of TOS depends on a high index of suspicion, familiarity with the various syndromes and anatomy involved, a detailed history and careful physical examination of the neck, shoulder, arm, and hand. Differentiation of TOS from other nerve compression syndromes, such as carpal tunnel, cubital tunnel, herniated cervical disc, and spondylosis must be made before effective treatment can be offered. The Adson test performed with the hand in the lap and head turned and extended to the ipsilateral side has been found to be useless for this diagnosis. Radial pulse variations with the arms elevated or shoulders braced also have little or no bearing on the diagnosis in that pulse changes in those positions are variations of normal found in 60–90% of the general population without symptoms.[8,9] Electromyograms and nerve conduction studies seldom give useful information and usually are not worth the considerable expense and patient discomfort they entail.[1]

Routine arteriograms and venograms are useless and merely confirm in a very expensive way the insignificant information that can be more easily obtained by feeling the pulse and listening for bruits with a stethoscope. Arteriograms are indicated only in unusual and specific cases in which one suspects, from the history and examination, that the patient may have an aneurysm or arterial occlusion, or is forming clots and emboli. The venograms, however, are required to clarify the diagnosis of subclavian vein compression or thrombosis, as various conditions may cause swelling and discomfort in the arm. When specifically indicated, venograms should be performed with the arm dependent and then elevated above shoulder level to demonstrate external compression mechanisms and collateral vein formation, which are required for the diagnosis of subclavian vein thrombosis.

In the upper plexus type of TOS, the upper trunk of the plexus is quite tender to palpation and percussion, especially when the patient's head is tilted to the opposite side, putting the sensitive upper trunk (C5 and 6) on the stretch.

The 3-minute elevated arm stress test (EAST) in the "stick-up position" will reproduce the symptoms of all three types of TOS in essentially all cases, and it is the simplest, cheapest, and most reliable test of all for these syndromes. Because the thoracic outlet is essentially closed in the 90° AER position, the compression mechanisms affecting the brachial plexus, subclavian artery or vein are immediately brought into play, and with mild exercise of the hand for 3 minutes, the usual symptoms will be reproduced in any of the three types of TOS or their various combinations. This test is simple, painless, quick, noninvasive, free, and is the most effective test available for TOS.[4]

Treatment

The conservative treatment of the neurologic or vascular symptoms of TOS may be soothing and temporarily beneficial but rarely offers permanent relief in the moderate to severe cases. Fortunately, surgical decompression of the neurovascular structures in the thoracic outlet or the side of the neck in upper plexus TOS may alleviate severe, even disabling symptoms in most cases when all attempts at conservative management have proven ineffective.[2,7]

Indications for surgical treatment of the neurologic type of TOS are straightforward: (1) intolerable pain; (2) progressive loss of function or strength or muscle atrophy of the hands; (3) threatened loss of job, impaired performance, or absenteeism resulting from the symptoms; (4) chronic loss of sleep; (5) unacceptable change of lifestyle caused by the symptoms; (6) significant personality changes resulting from chronic pain, lack of sleep, frustration, and depression resulting from restriction of activities imposed by the symptoms; (7) chronic use of strong medications, especially narcotics, in an effort to tolerate the symptoms.

Several operations have been devised over the past 76 years since the first report of removal of the first thoracic rib for TOS symptoms in 1910.[10] Currently, the surgical treatment usually performed for the lower plexus type of neurologic TOS is transaxillary first rib resection with shortening of the lower end of the anterior and middle scalene muscles, and resection of all anomalous fibromuscular tissues. For upper plexus, C5, 6 and 7 neurologic symptoms, supraclavicular total anterior scalenectomy offers the best chance of relief.[3,5] For intermittent nonthrombotic venous occlusion of the subclavian vein, transaxially first rib resection and removal of all anomalous soft tissue structures around the vein proves quite effective. This may also improve the chronic venous congestion following thrombosis of the subclavian vein if conservative treatment with anticoagulants and elevation proves ineffective.[6]

The surgical treatment of severe arterial compression or complications in the thoracic outlet is determined by arteriography. A subclavian plaque or source of emboli must be resected and replaced by graft; a fusiform aneurysm may be left intact if the compression mechanism, almost always a long cervical rib, is removed, but a sacular aneurysm, which may be the source of emboli from mural thrombi, must be resected.

Complications

Potential complications of these various operations may be minor, severe, or fatal. Minor complications may be slight postoperative pneumothorax or pleural effusion, or intercostobrachial nerve dysesthesias in the axilla and inner brachium, which may take a few months to clear; postoperative wound infections and hematomas are rare.

The major complications include phrenic nerve or brachial plexus injury, thoracic duct fistula after left-sided exploration for scalenectomy, or life-threatening hemorrhage from disruption of the subclavian artery or vein. In my own series of 1640 transaxillary first rib resections and 280 supraclavicular total anterior scalenectomies, six patients had temporary phrenic nerve palsy without significant complications and complete resolution; three had severe hemorrhage which was controlled, and the vessel repaired with complete recovery of the patient and extremity; and none has suffered a brachial plexus injury. Four patients had a lymphocele from operative injury to the thoracic duct, three of which required neck re-exploration to oversew the leaking lymphatics, and one required a chest tube for chylothorax. All recovered completely without residual symptoms. One patient developed Guillain-Barré syndrome with bilateral phrenic nerve palsy after first rib resection. She had complete recovery of diaphragmatic function in 3 weeks, but the weakness of her legs persisted for months.

Conclusions

With our present understanding of the etiology, anatomy, anomalies, and symptoms of the various thoracic outlet syndromes, accurate diagnosis can usually be made on clinical grounds if the physician has the proper index of suspicion and takes the time to perform a careful and thorough history and physical examination. If conservative measures fail to control or improve the symptoms to the extent the patient can lead a reasonably normal life comfortably without strong medications or severe compromise of family and job activities, properly selected surgical treatment performed with meticulous technique usually offers gratifying relief with substantial improvement in 90% of the cases that were uncontrollable by all conservative measures.

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