

Diagnosing thoracic outlet syndrome

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Thoracic outlet syndrome (TOS) is a common condition that is recognized uncommonly because the manifestations are varied and there is no single definitive test for it. Radiographs, MRIs, and even EMGs cannot make the diagnosis with certainty. Unfortunately many physicians doubt the diagnosis if the pathology cannot be seen radiographically or measured electrophysiologically.

Conceptually TOS is simple. It is a symptom complex caused by compression of the brachial plexus, or the axillary-subclavian artery or vein as they exit the chest. The clinical presentation is highly variable, depending on what parts of the brachial plexus are involved and to what extent the circulatory system is involved. The purely vascular forms of thoracic outlet syndrome are diagnosed easily but are uncommon. Neurologic compression is more difficult to diagnose. The physician needs to discern which symptoms relate to the brachial plexus, which relate to the major vessels, and which are totally unrelated. Because the presentation is so varied, each patient represents a challenge in physical diagnosis. Diagnosis is based on the total clinical picture, which is made up of a meticulous history, a complete physical examination, and a review of medical records.

As experience with this common though complicated disorder has increased, several basic concepts have emerged. Once these concepts are understood, it is possible to make an exact diagnosis and to plan specific, effective management.

Basic concepts of thoracic outlet syndrome

There is a mechanical predisposition

People who develop clinical manifestations of TOS have one or more congenital anomalies that predispose them to develop symptoms. Thoracic outlet syndrome is a mechanical space problem. Because of the invariable underlying congenital anomalies and often a superimposed injury causing chronic muscle spasm, the space for the major structures of the outlet becomes compromised. This restriction of space precipitates the symptom complex of TOS. Treatment is directed to correcting the space problems either by physical therapy, exercise, or surgical decompression. The longer the space problem exists the more damage is done to the structures, particularly to the nerves of the brachial plexus.

TOS symptoms are primarily neurologic

In 98% of cases the symptoms are neurologic. Fifteen percent may have some concomitant arterial symptoms, but arterial symptoms seldom exist alone. Symptoms caused by pure venous compression occur in 1.5% of patients and usually present as axillary subclavian vein thrombosis. Physicians who diagnose only the vascular forms of thoracic outlet syndrome are misdiagnosing the vast majority of patients they see who have this neurologic condition.

Structural anomalies cause the problem

These abnormalities have been well described [1]. Each has a specific effect and few of them compromise the circulation. For this reason, using circulatory tests as the basis for specific diagnosis

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commonly leads to error. Muscle spasm is important and aggravates the structural problems. Injuries lead to fibrosis, which further aggravates the structural problems.

Accurate diagnosis is based on the total clinical picture

There are no short cuts. A careful, even exhaustive history and physical examination are required, together with selective testing. Knowledge of other conditions that may belong in the domain of other specialists is required. When a patient presents with thoracic outlet syndrome symptoms, the goal should be to figure out an accurate diagnosis and not just to determine if thoracic outlet syndrome is present or absent. This may require a team of physicians and therapists.

Symptoms

TOS occurs in young and middle-aged adults and is three times as frequent in women as in men. Symptoms may develop spontaneously or following trauma of a type that causes chronic muscle spasm in the neck or shoulder region.

Arterial symptoms

Arterial symptoms arise from compression of the subclavian artery in the region of the first rib. These symptoms consist of coldness, pallor, and fatigue of the arm with exercise (coldness alone may be neurologic in origin). These patients often experience true claudication of the arm with exercise, particularly with the arm elevated. These symptoms are different from the whole arm numbness and heaviness associated with arm elevation in patients with neurologic thoracic outlet syndrome. A cervical rib or an elongated C7 transverse process is often present. Aneurysms, as described in 1916 by Halsted [2], may occur, and embolism may result, particularly in the presence of congenital thrombophilia. Fortunately aneurysms, ischemic rest pain, and digital gangrene are rare.

Venous symptoms

Venous obstruction at the thoracic outlet produces swelling, edema, cyanosis, and discomfort of the arm that is aggravated with exercise. Unfortunately the diagnosis often is missed until the subclavian vein thromboses without warning at the level of the first rib. When this occurs there is a sudden onset of edema, often with dusky

cyanosis, and limb discomfort. Thrombosis must be diagnosed early so that thrombolysis and subsequent surgical decompression can prevent a lifetime of postphlebotic arm symptoms, chronic heaviness, discomfort, and swelling that increases considerably with use of the arm [3]. Although some patients collateralize or resolve the obstruction, the prognosis without specific treatment is much worse than the more common catheter-induced thrombosis.

Neurologic symptoms

Trauma may precipitate the neurologic type of TOS in susceptible individuals. This may be a repetitive motion problem and is commonly occupationally related. Violinists, flautists, data entry personnel, and assembly line workers are particularly vulnerable. Any injury, especially a blow or jerking injury in the region of the neck or shoulder, that causes chronic muscle spasm, may precipitate this syndrome. Particularly problematic is the hyperextension–flexion whiplash injury of the neck. The initial injury may be minor and vehicle damage trivial. The onset of brachial plexus involvement with arm pain and paresthesias may be delayed by weeks or even months following trauma. Cervical and shoulder sprain symptoms often occur immediately, with persisting symptoms of neck and shoulder pain and stiffness. As the whiplash symptoms gradually improve with time and conservative treatment, the post-traumatic TOS involving the brachial plexus relentlessly worsens, adding confusion to the clinical picture. These patients need to be examined repeatedly during the course of therapy to track the evolution of symptoms and physical findings.

Thoracic outlet syndrome is predominately a brachial plexus injury. These patients may present with upper plexus TOS involving the C5, C6, or C7 nerves, or with lower plexus TOS involving principally the C8-T1 nerves or both. Upper plexus TOS is analogous to but not as severe as the Erb-Duchenne upper trunk injury, which involves avulsion of the upper roots of the brachial plexus; these patients present with a bad forearm and arm and a good hand. Lower plexus TOS is analogous to Klumpke injury, which presents with a good arm and a bad hand.

The usual upper plexus thoracic outlet syndrome [4] causes pain in the side of the neck that radiates upward to the ear and may even include the mandible, face, temple, and occipital regions, with hemicranial headaches. Some patients com-

plain of a “stuffy ear” on the affected side, although otolaryngologic examination is normal. The pain radiates posteriorly to the rhomboid area, anteriorly across the clavicle into the upper pectoral region, laterally through the trapezius and deltoid muscle areas, and down the outer arm. This pattern may simulate C5–C6 nerve root symptoms from a herniated disc affecting the radial nerve.

The usual lower plexus thoracic outlet compression patient presents with discomfort of variable intensity felt in the anterior or posterior shoulder region, radiating down the arm, in the medial brachial area, through the inner aspect of the forearm, and into the hand. Paresthesias affect principally the ring and small fingers in an ulnar nerve distribution. The ache often spreads up the side or back of the neck into the occipital or mastoid area of the skull, causing severe headaches and pain in the supra- and infraclavicular areas. Headaches may be severe and disabling, starting posteriorly at the base of the skull and radiating over the top of the head. They often are related to arm use rather than time of day. In some patients the pain may radiate to the anterior chest region, simulating cardiac angina.

The paresthesias reflect the neurologic origin of the symptoms and may accompany the aching pain or occur separately. They are felt as numbness, with or without tingling, and predominate in the fingers, sometimes in the entire hand. The small and ring fingers, or the index, long, and ring fingers may be specifically involved, depending on the nature of the compression. The whole arm may “go to sleep” at times, particularly when the patient is sleeping or working with the arm elevated. In the upper plexus type, numbness and tingling may be felt in the cheek, earlobe, back of the shoulder, or outer arm. Sleep deprivation is common. In the more advanced cases, weakness of the hand and loss of dexterity of the fingers frequently develops. In late cases there is muscle atrophy and impaired use of the arm without paralysis.

Arm exertion and elevation typically aggravates the symptoms. Heavy work during the day may result in misery at night, whereas quiet days may lead to more comfortable nights. Typically the symptoms occur after exercise instead of during exercise. This pattern separates the condition from most orthopedic shoulder problems that produce misery during exercise, and from herniated cervical disc, which generally causes constant rather than intermittent pain. Disk pain is typically more severe and sharp, radiating in a dermatomal

distribution, usually in the radial nerve area peripherally from a C4–C5 or a C5–C6 disc. TOS frequently involves the entire limb without dermatomal preference. If such preference is noted, it is most likely in the C8–T1 dermatomes. This is the typical location of the numbness, but the pain is often more generalized. Pain and numbness may radiate in the radial distribution. These symptoms suggest a disc or involvement of the upper plexus.

Median nerve compression at the wrist may be confused with TOS because it too causes numbness and pain in the arm. Symptoms are in the distribution of the median nerve rather than in the radial or ulnar nerve distribution. Simply by asking the patient which fingers are predominantly involved, the physician thus has a strong clue to the origin of the compression neuropathy. Carpal tunnel syndrome typically produces symptoms that originate in the hand and radiate up the arm in contrast to TOS, which produces pain in the neck and shoulder that moves down to the hand. Muscle cramping in the hand and forearm are more suggestive of carpal tunnel syndrome than TOS. Shoulder symptoms are common in carpal tunnel syndrome, but neck symptoms are not. Compression or injury of the ulnar nerve at the elbow, as occurs with “sleeper’s palsy,” and cubital tunnel syndrome can be confused with TOS, but again, these symptoms originate peripherally in the medial elbow and spread distally.

The symptoms of thoracic outlet compression are induced by exercise and by positional changes, such as outward reach or elevated use of the hand, as in setting hair, working overhead, doing housework, or painting. The aggravating activities in carpal tunnel syndrome are a prolonged grasp of the hand, as in holding a steering wheel or hammer, or pinch with the thumb, index, and long fingers, as in writing or sewing. The aggravating activities in cervical disc syndrome are turning of the head to one or both sides and lying on the side with the head unsupported. Thoracic outlet syndrome and cervical disc disease may have neck stiffness resulting from muscle spasm.

Any of the neurologic syndromes of the upper extremities may cause subjective coldness of the hand, even pallor, which may be interpreted mistakenly as arterial insufficiency. This phenomenon has long been recognized in neuropathies of the lower extremities, the classic case being the withered limb of poliomyelitis that is cold, weak, and pale bluish-white despite bounding pedal pulses. The hands may be sensitive to cold exposure, suggestive of Raynaud phenomenon,

again as a result of the neuropathy, not of arterial insufficiency.

As can be seen by the previous discussion, simple categorization of symptoms is helpful. The cervical disc syndrome generally presents with problems in the radial nerve distribution. Thoracic outlet symptoms are either ulnar from the lower part of the plexus or entire limb from compression of the plexus. Carpal tunnel syndrome is a pure median neuropathy; median nerve symptoms are never seen by themselves in TOS. Ulnar compression at the elbow produces distal symptoms in the ulnar distribution but few symptoms in the shoulder and fewer in the neck.

With both types of TOS the symptoms may be mild and intermittent at first and are generally ignored by the patient. Typically they increase gradually in frequency and severity to the point that the patient no longer can ignore them or relieve them by positional change or simple medication. As progression begins to affect job, sleep, or general activities, the patient finally seeks medical attention. This is an ideal time for the physician to make the proper diagnosis, reassure the patient of the simple mechanical nature of the problem, and institute measures to relieve brachial plexus compression in an attempt to halt progression of symptoms. Careful documentation of the symptoms and findings at this point is important so that progress of therapy can be monitored.

After determining the important points in the history, a detailed and appropriate physical examination must be undertaken. See [Table 1](#) for a summary of the important points in the history.

Physical examination

The examination should be focused on diagnosing all of the patient's maladies and should not be limited merely to testing for thoracic outlet entrapment. The typical TOS patient has seen several physicians before seeing a surgeon. Often these physicians have made the wrong diagnosis or told the patient that their problem is psychosomatic. Insurance and workers' compensation physicians commonly minimize the patient's symptoms. Most of these patients have some psychological damage from these experiences. A diagnosis of psychosomatic illness must be just as specific on physical examination as is the diagnosis of TOS.

Although the technique for separating the somatic from the psychosomatic by physical examination is beyond the scope of this article,

the key is to repeat tests of the same function and look for variations of response and to look for disparity between symptoms and physical findings. Frost described the techniques well and the reader is referred to his article [5].

After a general examination, attention is turned to the neck, shoulder, and upper extremities. A blood pressure difference between two arms is significant if it is more than 20 mm Hg and suggests compromise of the subclavian artery. The upper extremities are examined side by side for color, temperature, moisture, muscle atrophy, edema, abnormal finger nails, and hair growth.

Muscle strength of all muscle groups of the arm is tested. This includes evaluating the interosseous muscles using the interdigital card test and spreading the fingers apart against resistance. The patient extends the thumb against resistance and dorsiflexes the wrist against resistance to test radial innervated muscles. The muscles innervated by the median nerve are tested by flexing the thenar muscle against resistance, with the thumb kept straight. Thumb extension is tested. Biceps and triceps strength is evaluated against the examiner's resistance. In TOS, the biceps is strong bilaterally, but the triceps is noticeably weak on the affected side. In upper plexus TOS, weakness of the deltoid (C5), the biceps (C6), the triceps (C7), and the rotator muscles of the head may occur caused by C5, C6, and C7 root compression, but the weakness of the head rotators may be from "muscle splinting" because of increased pain caused by compression of the brachial plexus with head motion. Rotator cuff problems may lead to inability to abduct the arm normally.

Carpal tunnel syndrome is evaluated by attempting to elicit Tinel sign and by performing Phalen's test. A Tinel sign is also sought over the ulnar nerve at the elbow.

Muscle spasm is evaluated in the neck, pectoral region, and interscapular region. Range of motion of the neck is tested. With the patient's neck flexed, the examiner then percusses the lumbar, thoracic, and cervical spine up the midline over the spinous processes. Normally the spine is not tender, but it may be so in the upper thoracic and cervical regions after hyperflexion trauma. This is not diagnostic of TOS but of the sprain itself. Cervical disc syndrome patients complain of localized tenderness at the level of the disc involved, usually C5–C6, and an attempt should be made to elicit this anteriorly and posteriorly. Tilting the head away from the affected side increases tension on neck muscles and the brachial plexus. In upper

Table 1
Differentiating features of the clinical syndromes that mimic thoracic outlet syndrome

	TOS	Carpal tunnel	Cervical disc	Cubital tunnel	Shoulder pathology
Symptoms					
Pain	Neck, shoulder, arm (intermittent)	Wrist, volar forearm, fingers 1–3 (intermittent)	Neck and shoulder (constant), radial distribution	Elbow, last two fingers	Shoulder, radiation into upper arm if causes muscle spasm
Numbness	Ulnar nerve or whole arm	Median nerve, first three digits. May have ulnar symptoms if Guyon canal is involved	Radial nerve-web between fingers 1 and 2	Ulnar distribution	Numbness not predominant but may occur with muscle spasm
Awkwardness Aggravation	All fingers or fingers 4 and 5 Arm elevation	Fingers 1–3 Sustained grasp or pinch	Thumb Neck position, arm stretch	Fingers 4 and 5 Pressure on elbow	Caused by pain Shoulder use
Color	Normal, cyanotic, pale, depending on what is compressed	Normal or splotchy	Normal or splotchy	Normal	Normal
Edema	May or may not be present	Absent	Absent	Absent	Absent
Tests					
Percussion	+ Brachial plexus	+ Tinel volar wrist	+ Neck at disk level	+ Tinel sign at elbow	Shoulder joint
Compression	+ Brachial plexus, band spot	+ Phelan test	+ Neck at disc level and brachial plexus	+ Ulnar nerve at elbow	+ Joint, bursas, Acromioclavicular joint
Symptoms reproduced	Arm elevation, brachial plexus compression	Wrist flexion	Head turn, tilt, cranial compression	Pressure on ulnar nerve	Pressure on joint structures
Electrophysiologic tests	Useful only to rule out other conditions	Positive (unreliable if negative)	Positive (unreliable if negative)	Positive (unreliable if negative)	Negative
Radiographs	Look for cervical ribs, old fractures, long transverse process	Normal, look for arthritis or old trauma	Arthritis of spine, consider MRI or other specialized work-up	Negative	Order tests based on presumptive diagnosis
Treatment					
Conservative	Avoid aggravating activities, physical therapy	Wrist splint, steroid injection	Cervical traction	Protect elbow	Varies by specific condition
Indications for operation	Treatment failure with intolerable symptoms, atrophy, arterial or venous insufficiency	Failure of treatment, loss of function, thenar atrophy	Intractable pain, atrophy and loss of function	Treatment failure	Varies according to condition
Operation	Resection of first rib and/or anterior scalene with anomalies	Resection of transverse carpal ligament	Discectomy and fusion	Decompression	Varies according to specific diagnosis

plexus TOS, pain gradually spreads, reproducing the patient's principal symptoms. Finger percussion indicates the point of maximum neurologic involvement. The head is rotated away from the side to be examined and the same percussion test is performed over the anterior scalene muscle. Radiation of pain and paresthesias into the face and ear suggest upper brachial plexus involvement. These maneuvers produce no symptoms in patients with carpal tunnel syndrome or orthopedic shoulder problems unless they aggravate the underlying muscle spasm.

The examiner next should exert slight to moderate thumb pressure over the patient's brachial plexus in the supraclavicular fossa for a few seconds on each side. Normally this causes no symptoms, but in TOS it may reproduce symptoms, causing paresthesias down to the fingers. Although the plexus usually is tender in cervical disc syndrome, thumb pressure held over the plexus for a few seconds ordinarily does not reproduce the patient's neck, shoulder, and radicular pain down the arm as it does in thoracic outlet compression. Pressure should then be applied to the "band spot," a point in the base of the neck just anterior to the trapezius muscle at the junction of the neck and shoulder. If a patient has a congenital cervical band contributing to or causing outlet compression, there often is extreme point tenderness without pain or radiation, or with deep percussion or pressure in this location. In other conditions, pressure in this area is painless.

One of the best tests for cervical disc syndrome is to have the patient turn the head to one side and tilt back. If this position is held a short while, the patient with a ruptured disc often complains of pain in the side or base of the neck, and if a nerve is irritated, the usual radicular pain and paresthesias may develop gradually. After this position is held for several seconds, the examiner carefully presses the forehead downward to compress the cervical spine. Cranial compression is painless in normal patients but may be very painful in those with cervical disc deterioration, reproducing the radicular pain across the top of the shoulder or down the arm (Spurling test).

The neurologic examination is completed by testing pinprick sensation in the three peripheral nerve regions of each hand and evaluating the reflexes in the biceps, triceps, and brachioradialis muscles of the arms. Reflexes are normal in carpal tunnel and TOS.

After the neurologic examination has been completed the vascular system is evaluated. The

radial and ulnar pulses are palpated at each wrist with the hands in the lap, and then the radial pulse is monitored with the arm in the 90° abduction–external rotation position, with the head neutral or turned to the opposite direction (Wright test). This position may or may not be positive for pulse change in any of the syndromes mentioned, because it is positive, with noticeable damping or obliteration of the pulse, in approximately 7% of the normal population. For this reason pulse changes should be considered only corroborating findings in patients with arterial symptoms.

The Adson test, described in 1947 [6], is performed with the patient seated with the arms resting on the knees. The patient takes a long breath, elevates the chin and turns the head to the affected side. An alteration or obliteration of the radial pulse was believed to be a pathognomonic sign of the scalenus anticus syndrome. Because this test is positive as often in normal patients as it is in patients with TOS, it must be one of the most famous yet most unreliable tests in all clinical medicine. It is of historic interest only.

The most reliable test for TOS is the "elevated arm stress test," (EAST) described by Roos. It is performed by having the patient put both arms in the 90° abduction–external rotation position, with the shoulders and elbows in the frontal plane of the chest. The patient is then instructed to open and close the hands slowly over a 3-minute period. Normally the patient can perform this stress test for 3 minutes with only forearm muscle fatigue and minimal distress. In those with an outlet syndrome, the test reproduces the usual symptoms: gradual increase of pain in the neck and shoulder, aching progressing down the arm, and paresthesias developing in the forearm and fingers. Those with arterial compression develop arm pallor with the arm elevated and reactive hyperemia when the limb is lowered. Those with venous compression may develop cyanosis and swelling. Many patients with TOS are unable to complete this test and drop the arms to the lap in marked distress, which they recognize as reproduction of their usual symptoms. An occasional patient with carpal tunnel syndrome may get some numbness in the fingers, but this is from compression of the median nerve during squeezing of the hand and is confined to the first three fingers, a different response from TOS. A patient with cervical disc syndrome may get pain in the neck and shoulder from holding the arms elevated but feels minimal distress in the arm or hand. A patient with orthopedic shoulder problems

may experience intolerable symptoms but they are confined to the shoulder area. The important characteristics of a positive test include reproduction of usual symptoms that involve the entire extremity. The duration of tolerance of this test may reflect the severity of the TOS.

Nerve tension tests of the upper extremity have been described that correspond to straight leg raising as a test for nerve impingement in the lower extremity [7,8]. Upper limb tension tests have been described that are median, ulnar, or radial nerve dominant. Although they may be well accepted by physiotherapists and do provide objective measurements, their role in separating TOS from other upper extremity nerve problems is not well documented.

Ancillary testing

Attempts to substitute technologic approaches to expedite diagnosis for the more time consuming clinical evaluation have been generally unsuccessful. Testing should be done selectively based on the complete history and physical examination.

Anterior scalene block

Some physicians advocate anterior scalene block as an appropriate diagnostic test. Such a block temporarily paralyzes the anterior scalene muscle, and if spasm of this muscle is the proximate cause of the symptoms, this should be diagnostic. The test cannot be expected to diagnose other anatomic problems in which the anterior scalene is not involved. In addition, this test has been rather nonspecific, as there is overlap with somatic nerves and the brachial plexus. Performance of the block guided by electrophysiologic tests has also been proposed to overcome the overlap problem [9], but even this precision does not address the basic limitations of the test.

Cervical spine films

Cervical spine films should be evaluated in every case to see if there are bony abnormalities contributing to the problem. Old fractures of ribs or clavicles, exostoses of the first rib or clavicle, or tumors in the outlet, obviously increase susceptibility to neurovascular compression in this region. Spine films are checked specifically for degenerative cervical spine disease, disc space narrowing, osteophytes in the neural foramina on oblique films and congenital anomalies. The surgeon should always look at the films, as radiologists

not familiar with TOS may not attach significance to findings such as a C7 transverse process longer than T1 process and other structural anomalies of the first ribs and clavicles. Cervical ribs are important even if small; the anlage is usually complete to the first rib by a congenital fibrous band not visualized on radiograph. It has the same effect as a long cervical rib, as it elevates the lower part of the brachial plexus, making it more susceptible to compression.

Patients with a low-lying shoulder girdle have a narrow costoclavicular space that over a period of years or suddenly after trauma may cause costoclavicular scissoring compression of the neurovascular structures. This type of susceptibility is readily determined on the lateral view of a cervical spine series. Normally there are seven vertebrae seen above the top of the clavicle on the lateral view in males and seven or eight in females. It is not unusual, however, in women with the TOS to find that they have 9 or even 10 vertebrae (1 or 2 thoracic vertebrae) above the clavicular line, resulting in narrower costoclavicular spaces, again making them more susceptible to the development of symptoms of thoracic outlet compression.

Arteriography

Arteriography is rarely indicated. Positional arterial obstruction can generally be diagnosed by physical examination more accurately than it can by angiography because of positioning restraints in the angiography suite. Arteriography is indicated in the case of embolization, bruit with arm in neutral position, suspicion of aneurysm, and blood pressure difference between arms greater than 20 mm Hg. In TOS, these indications are important but are unusual.

Venography

Positional venography is indicated when venous thoracic outlet syndrome is suspected. The physiologic importance of a positional obstruction of the venous system can be estimated by looking for collateralization. With increasing use of MR angiography, patients have been identified who have compression of the subclavian vein in the AP plane, even though it cannot be seen on standard venography (Figs. 1 and 2). When venograms show no collateralization the importance of this finding is uncertain. Patients with venous TOS should have both arms studied because of the high incidence of bilateral abnormal compression.

Acute occlusion of the axillary subclavian system documented on venography requires special attention. Serious consideration should be given to catheter-directed thrombolysis followed by early surgical decompression to lessen the risk for subsequent re-thrombosis.

Computed tomography (CT) and magnetic resonance imaging (MRI)

CT and MRI studies have generally replaced myelography in the diagnosis of cervical disk disease. Although disc disease can occur in the presence of TOS, routine use to eliminate the possibility of cervical disc disease in patients who have TOS on history and physical examination is not indicated.

MRI or CT of the brachial plexus traditionally has been useful only in patients with the possibility of Pancoast tumors or metastases involving the brachial plexus. Using special sequences, Collins and others have performed detailed studies of the thoracic outlet; they claim to have the ability to diagnose TOS with a high degree of accuracy [10]. Many of these investigators have a different

understanding of TOS than do the surgeons, however, and focus primarily on vascular anomalies. They speculate that neurologic symptoms are caused by arterial or venous insufficiency of the nerves of the brachial plexus. The validity of this approach needs further evaluation. Another promising technique is high-speed multidetector CT studies with contrast. Spatial resolution is much better than with MRI and the study is faster. Individual muscles can be visualized and peeled back using computer techniques. Whether this technique allows visualization of the specific congenital anomalies and bands is under investigation at the authors' institution.

Neurophysiologic studies

A variety of electrophysiologic tests have been proposed for the diagnosis of TOS and none have received general acceptance. Most of these tests measure nerve conduction between two points and anatomic constraints are often problematic. Use of C8 nerve root stimulation in addition to other electromyographic studies has been proposed to potentially overcome the anatomic



Fig. 1. Normal positional venogram in a patient with arm swelling and thoracic outlet syndrome symptoms.

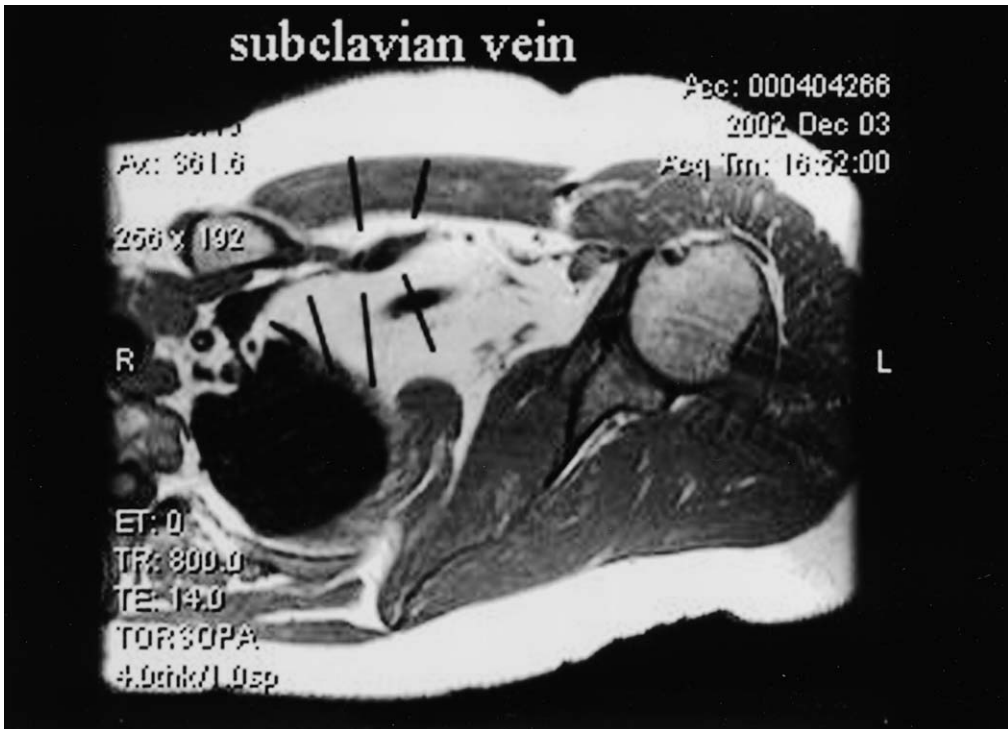


Fig. 2. Magnetic resonance image of the subclavian vein in the same patient shown in Fig. 1. Note anterior–posterior compression of subclavian vein not appreciated on conventional venography.

constraints, but there is wide variability with this test [11]. In addition, compression is positional and often intermittent. These tests are useful in diagnosing other causes of symptoms. Electrophysiologic studies may be positive in late cases. It is the hope of most surgeons that the diagnosis can be made before permanent damage takes place and wasting occurs.

Coagulation Studies

Hypercoagulability studies should be performed in all patients with embolization or venous occlusion. Patients developing thrombosis often have two things wrong (the two-hit theory). The first is the mechanical problem at the thoracic outlet and the second is hypercoagulability. Particular attention should be paid to hyperhomocysteinemia in arterial patients, and the Factor V Leiden or Factor 2 20210A abnormality, the most common causes of thrombophilia in humans. If the patient is heterozygous for Factor V Leiden, tests of Factor 5 polymorphism should be done also. In the case of a positive test, anticoagulation is indicated together with genetic counseling. Other tests for hypercoagulability may

be confounded in the presence of anticoagulation or coagulation and should be interpreted with caution.

Noninvasive vascular laboratory

Positional plethysmographic studies are effective in documenting positional arterial obstruction but add no additional information to the physical examination. Ultrasound is notoriously unreliable in diagnosing arterial or venous obstruction, as the problem is located between the first rib and the clavicle where ultrasound visualization is obstructed by bones, the exception being if venous thrombosis is identified lateral to the clavicle then the clinical diagnosis is confirmed. If no thrombus or obstruction is seen, further evaluation with venography, contrast, or MRI may be necessary.

Summary

Diagnosing and treating thoracic outlet syndrome can be challenging and frustrating. It must be emphasized that the diagnosis of TOS is a clinical one based on a detailed history and physical examination. This takes time and effort

and is often confounded by the patient's research on the internet and emotional problems usually resulting from the symptoms and lack of appropriate treatment. Years of inappropriate diagnosis and ineffective therapy take a heavy toll on these patients. Some have psychologic problems to the point that no treatment, no matter how well indicated, will make them well. Some have had symptoms so long that there is permanent neurologic damage. Each patient presents his or her own diagnostic challenge. Solving the problem and providing effective therapy can be rewarding for doctor and patient.

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