Imaging Assessment of Thoracic Outlet Syndrome

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The thoracic outlet includes three compartments (the interscalene triangle, costoclavicular space, and retropectoralis minor space), which extend from the cervical spine and mediastinum to the lower border of the pectoralis minor muscle. Dynamically induced compression of the neural, arterial, or venous structures crossing these compartments leads to thoracic outlet syndrome (TOS). The diagnosis is based on the results of clinical evaluation, particularly if symptoms can be reproduced when various dynamic maneuvers, including elevation of the arm, are undertaken. However, clinical diagnosis is often difficult; thus, the use of imaging is required to demonstrate neurovascular compression and to determine the nature and location of the structure undergoing compression and the structure producing the compression. Cervical plain radiography should be performed first to assess for bone abnormalities and to narrow the differential diagnosis. Computed tomographic (CT) angiography or magnetic resonance (MR) imaging performed in association with postural maneuvers is helpful in analyzing the dynamically induced compression. B-mode and color duplex ultrasonography (US) are good supplementary tools for assessment of vessel compression in association with postural maneuvers, especially in cases with positive clinical features of TOS but negative features of TOS at CT and MR imaging. US may also allow analysis of the brachial plexus. However, MR imaging remains the method of choice when searching for neurologic compression.

Abbreviation: TOS = thoracic outlet syndrome

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Introduction
The thoracic outlet includes three confined anatomic spaces, the congenital or acquired narrowing of which may lead to compression of blood vessels or nerves or both. Dynamically induced compression of the neural, arterial, or venous structures crossing one of these tunnels leads to thoracic outlet syndrome (TOS). The diagnosis is based on the results of clinical evaluation, particularly if the patient’s symptoms can be reproduced when various dynamic maneuvers, including elevation of the arm, are undertaken.

In this article, we review the anatomy of the thoracic outlet and discuss and illustrate the functional anatomy, clinical features, causes, imaging features, and treatment of TOS.

Anatomy of the Thoracic Outlet
The thoracic outlet, or cervicothoracobrachial junction, includes three confined spaces, extending from the cervical spine and the mediastinum to the lower border of the pectoralis minor muscle, which are potential sites of neurovascular compression. These three compartments are the interscalene triangle, the costoclavicular space, and the retropectoralis minor space (Figs 1, 2). (1, 2).
The interscalene triangle is the most medial of these compartments. It is limited anteriorly by the anterior scalene muscle, posteriorly by both the middle and posterior scalene muscles, and inferiorly by the first rib (Fig 3). The anterior scalene muscle originates from the anterior tubercle of C3–C6 and inserts inferiorly onto the scalene tubercle of the first rib. The middle scalene muscle originates from the posterior tubercle of C2–C7 and inserts inferiorly onto the first rib behind the scalene tubercle, from which it is separated by the subclavian groove. The posterior scalene muscle is the deepest of the three scalene muscles. It arises from the posterior tubercle of C4–C6 and inserts inferiorly onto the second rib. The interscalene triangle is crossed by the subclavian artery, which occupies the floor of the space, and by the three trunks of the brachial plexus. The upper (C5-C6) and middle (C7) trunks cross the upper part of the interscalene triangle above the subclavian artery. The lower trunk (C8-T1) is located in the inferior part of the interscalene triangle, behind the posterior part of the subclavian artery. The subclavian vein does not cross the interscalene triangle but runs beneath the anterior scalene muscle before joining the internal jugular vein to form the brachiocephalic vein (3).

The intermediate compartment of the thoracic outlet is the costoclavicular space. This compartment is limited superiorly by the clavicle, anteriorly by the subclavius muscle, and posteriorly by the clavicle and the anterior and middle scalene muscles.
both the first rib and the middle scalene muscle (Fig 4). The subclavius muscle originates from the junction of the first rib with its costal cartilage; its fibers run upward and laterally and insert into the inferior surface of the clavicle. This space contains the subclavian vein anteriorly, the subclavian artery immediately posterior to it, and the three cords of the brachial plexus, arranged in a triangular configuration. The lateral cord, formed by the anterior division of the upper and middle trunks, and the medial cord, formed by the anterior division of the lower trunk, are located above the subclavian vessels, the lateral cord being anterior to the medial cord. The posterior cord, formed by the posterior division of the upper, middle, and lower trunks, represents the posterior edge of the triangular configuration. It is located above the medial and lateral cords.

The retropectoralis minor space is the most lateral of the three compartments. It is limited by
the posterior border of the pectoralis minor muscle anteriorly, by the subscapularis muscle posteriorly and superiorly, and by the anterior chest wall posteriorly and inferiorly (Fig 5). The neurovascular arrangement in this space is quite similar to that seen in the costoclavicular space. Just lateral to the pectoralis minor muscle, the cords divide into five terminal branches (median nerve, ulnar nerve, musculocutaneous nerve, axillary nerve, and radial nerve).

**Functional Anatomy of TOS**

TOS is a dynamically induced compression syndrome that is produced especially by elevation of the arm. Several studies that used CT and MR imaging in healthy volunteers have demonstrated that upper limb elevation does not induce any
obvious change in the interscalene triangle (Fig 6) but does produce narrowing of both the costoclavicular space and retropectoralis minor space (4–9) (Figs 7, 8). The costoclavicular space is by far the most frequent site of arterial compression, while the interscalene triangle is the second most frequent site (9). Neurologic compression appears to be as frequent in the costoclavicular space as in the interscalene triangle (9). The retropectoralis minor space has rarely been reported as a potential site of compression.

Clinical Features of TOS
TOS is one of the most controversial subjects in medicine (10). Although TOS is conceptually simple, its diagnosis remains difficult and confusing. The term *thoracic outlet syndrome* was coined by Peet et al (11) in 1956 to indicate compression of one or several of the neurovascular structures crossing the thoracic outlet. The most common age range for this syndrome is 20–40 years, with a female-to-male ratio of 4:1 (10). The symptoms of TOS are typically reproduced or exacerbated by activity requiring elevation or sustained use of the arms, such as reaching for objects overhead or lifting. Compression of the neurovascular bundle
may occur in all three compartments of the tho-
racic outlet. Three distinct syndromes may be
encountered, individually or combined, depend-
ing on the injured component of the neurovascu-
lar bundle: neurogenic syndrome, arterial syn-
drome, and venous syndrome. Clinical signs and
symptoms include pain, numbness, tingling,
weakness, and other disorders of the upper ex-
tremity. According to the literature, neurogenic
TOS is observed in 90%–95% of the cases and
vascular TOS in 5%–10% (12,13). However,
these percentages may reflect a recruitment bias,
as other authors have reported mainly series of
vascular TOS (5,6,9).

Several provocative clinical tests are performed
as part of the physical examination. Four basic
maneuvers with several variations have been de-
scribed: the Roos test, Adson test, Wright test,
and costoclavicular test. The elevated arm stress
test maneuver introduced by Roos and Owens
(14) consists of asking the patient to place both
arms in 90° abduction and external rotation with
the shoulders braced posteriorly and then to open
and close the hands slowly for 3 minutes. Most
patients with neurogenic TOS are unable to com-
plete this test (12). The Adson test is performed
by holding the patient’s arm down and checking
the radial pulse while the patient inhales deeply
and keeps his or her head extended and turned
toward the involved extremity (15). However, this
test may also be positive in normal subjects and is
therefore not very reliable (2,10,16). The costo-
clavicular compression test is performed by hav-
ing the examiner depress the patient’s shoulder
and ask the patient for evidence of symptoms.
The Wright test (hyperabduction test) is per-
formed with the patient in a sitting or standing
position with the shoulder hyperabducted and
rotated externally (17). The patient is asked
whether he or she experiences any symptoms in
the extremity, and any change in pulse is noted.
As elevation of the upper limb has been reported
to be relevant in diagnosing TOS (18–21), this
maneuver has been chosen as a postural maneu-
ver, combined with imaging techniques such as
CT or MR (4–9).

In the case of neurogenic TOS, the symptoms
may be sensory or motor, although subjective sen-
sory symptoms of pain and paresthesia predomi-
nate (2). Patients may present with upper plexus
TOS involving the C5, C6, and C7 nerves. In
upper plexus TOS, pain is generally located in the
side of the neck and radiates upward to the ear
and occipital region. The pain may also radiate
posteriorly to the rhomboid area, anteriorly across
the clavicle into the upper pectoral region, later-
ally through the deltoid and trapezius muscle
areas, and down the outer aspect of the arm
(10,22). In most cases, however, patients present
with lower plexus TOS, corresponding to com-
pression of the C8 and T1 nerves. Pain is usually
distributed in the anterior or posterior shoulder
region and radiates down the arm, in the medial
brachial area and along the inner aspect of the
arm. Paresthesia affects mainly the ring and little
fingers, with an ulnar nerve distribution (22). The
autonomic innervation of the arm must also be
considered, as it can account for some autonomic
features, such as symptoms of vasomotor distur-
bance.

Figure 8. Effect of arm elevation on the retropectoralis minor space in an asymptomatic
subject. Sagittal T1-weighted MR images obtained with the arm alongside the body (a) and
after arm elevation (b) show narrowing of the retropectoralis minor space.
In arterial TOS, the symptoms are caused by arterial insufficiency. They include weakness, cold, and pain in the extremity, caused by ischemic neuritis of the brachial plexus. In the case of severe compression, subclavian artery thrombosis with peripheral embolization can be observed (23). Venous TOS consists of swelling and cyanosis of the extremity, with pain, a feeling of heaviness in the upper limb, and venous distention of the upper arm and shoulder region (10). Acute subclavian-axillary vein thrombosis refers to a Paget-Schroetter syndrome or effort thrombosis (2,10).

In many cases, classification as arterial or neurologic compression remains difficult. Moreover, arterial and neurologic compression can be observed together and some physiopathologic mechanisms are interrelated. For example, nerve compression involves closely related mechanical and ischemic factors, with disturbance of intraneural microcirculation (2). In many cases, however, symptoms are vague and nonspecific and clinical diagnosis is often difficult, requiring the use of imaging methods and electrophysiologic criteria, such as electromyography or somatosensory evoked potentials. This information is very important because treatment for thoracic outlet symptoms is aimed at alleviating or reducing the compression inside the narrowed space.

Causes of TOS

Anatomic abnormalities or acquired disease of the skeletal and soft-tissue structures forming or bordering on the three compartments may cause mechanical compression or direct irritation of the neurovascular structures. These abnormalities can be divided into two groups: bone abnormalities and soft-tissue abnormalities (12) (Table).

Bone Abnormalities

Skeletal and bone abnormalities include cervical ribs, elongated transverse process of C7, exostosis of the first rib or clavicle, and excessive callus of the clavicle or first rib.

Cervical Rib.—A cervical rib is a supernumerary rib originating from the seventh cervical vertebra (Fig 9). According to the literature, cervical ribs, which are present in less than 1% of the normal population, have been reported in 5%–9% of patients with TOS (24,25). Cervical ribs may be complete or incomplete, in association with fibrous bands (2,16). Complete cervical ribs are fused with a tubercle located on the upper aspect of the first thoracic rib. This fusion point is usually adjacent to the site of insertion of the anterior scalene muscle. As a result, the supraclavicular course of the subclavian artery is usually displaced anteriorly. Incomplete cervical ribs, although they do not articulate directly with a thoracic rib, usually have an associated fibrous band that can insert onto the first thoracic rib and compress adjacent neurovascular structures (Fig 10) (2,16).

Elongated Transverse Process of C7.—A C7 transverse process is considered elongated if it extends beyond the tip of the T1 process immediately below it, as seen on cervical radiographs (Fig 9). Like a cervical rib, an elongated C7 transverse process can lead either directly or indirectly to
neurovascular compression, in association with a fibrous band or an abnormal middle scalene muscle.

Abnormal First Rib or Clavicle. — The first rib and clavicle, which form the jaws of the costoclavicular “pliers,” are important anatomic structures. Abnormal development or orientation of the first rib may lead to undue neurovascular compression. Disease processes or acquired abnormalities, such as exostosis, tumor, callus, or fracture of the first rib or clavicle, may also irritate the neurovascular structures and in particular the brachial plexus (Fig 11).

In time, bone abnormalities can lead to severe arterial complications. Indeed, repetitive trauma at the site of compression by these abnormalities can damage the arterial intima and lead to atherosclerotic changes, aneurysm, and thrombosis or embolism (5,26). The thickening and fibrosis of the arterial wall, as well as the inflammatory changes in the adventitia, may explain the fact that the dynamically induced symptoms initially observed may later become permanent (fixed stenosis).

Soft-Tissue Abnormalities

In the soft-tissue group, congenital fibrous bands and ligaments as well as congenital or acquired muscular changes (hypertrophy, fibrosis, etc) and soft-tissue posttraumatic changes have been reported.

Congenital Soft-Tissue Abnormalities. — Several complex anatomic variations of the scalene muscles may be responsible for TOS (1,2,27,28). They include hypertrophy of the anterior scalene muscle, origin of the anterior and middle scalene muscles from a common belly that divides in two distally, passage of the brachial plexus through the substance of the anterior scalene muscle, a broad middle scalene muscle inserting more anteriorly on the first rib than is normal, interdigitation between the anterior and...
middle scalene muscles, and supernumerary muscles (including a possible scalenus minimus muscle) extending from the transverse processes of C6 and C7 to the first rib behind the scalene tubercle or to the cupola of the lung (Fig 12) (16,22,28). Anomalous fibrous bands, extensively described by Roos (16), have also been reported in patients with TOS. They may arise from a cervical rib, the first thoracic rib, an elongated C7 transverse process, or the anterior and middle scalene muscles. They insert onto the first thoracic rib or the cupola of the lung. Some of these bands are fibromuscular in nature and similar to variations of the scalene muscles.

**Acquired Soft-Tissue Abnormalities.**—These abnormalities include posttraumatic and postoperative fibrous scarring. As regards posttraumatic causes, two groups of patients have been differentiated (12). Patients who have suffered direct trauma to the brachial plexus or the muscles make up the first group. The trauma habitually results from a whiplash flexion-extension injury to the neck. The second group consists of patients suffering from work-related repetitive microtrauma (activities requiring repeated elevation of the upper limb or heavy lifting). It has been hypothesized that such trauma may induce fibrosis and spasm of the scalene muscles, leading to elevation of the first rib and consequently to its impingement on the neurovascular structures. Repeated microtrauma may also induce local perineural inflammation of the soft tissues (12). However, the exact mechanism whereby trauma precipitates TOS is still unclear.

**Posture and Predisposing Morphotype**
Thin women with poor posture and weak muscular support of the shoulder girdle have been reported to be predisposed to developing TOS (12). Indeed, drooping and sagging of the shoulder increase acromioclavicular descent and compression of neurovascular structures in the costoclavicular space.

**Imaging Features of TOS**

**Plain Radiography**
Radiographs of both the cervical spine and chest should be systematically obtained in order to search for bone abnormalities that may contribute to the problem (eg, cervical ribs, elongated C7 transverse process, degenerative spine disease, or bone destruction related to a primary or secondary neoplasm) (Fig 9) (2,16).

**Arteriography and Venography**
Conventional arteriography and venography may demonstrate the presence of extrinsic compression. Unfortunately, they do not allow a clear depiction of the impinging anatomic structure, and they tend to be replaced by less invasive procedures (CT, MR imaging, sonography).
CT Angiography

Spiral CT examination of TOS is performed first with the arms alongside the body and then with the arms elevated in an attempt to reproduce the neurovascular compression (a position intermediate between those of the Roos and Wright maneuvers). Intravenous injection of contrast medium is often considered in order to obtain CT angiograms (5,6). To opacify the subclavian and axillary arteries only without producing any venous artifacts, the contrast medium must be injected into a vein on the side opposite that being examined. The recommended method consists of beginning to scan 15–20 seconds after the start of a monophasic injection of 90 mL of iodinated contrast medium at a rate of 4 mL/sec (6). By comparing the images obtained with the arms alongside the body and after elevation, it is possible to assess the narrowing of the various compartments, as well as any dynamic compression of the neurovascular structures.

Arterial compression is well assessed with CT by using arterial cross sections produced by sagittal reformation of data obtained both in the neutral position and after postural maneuvers (Fig 13). Whereas sagittal reformation allows assessment of the location and severity of the arterial compression, volume-rendered images of the thoracic outlet before and after postural maneuvers allow simultaneous analysis of bones and vascular bundles, which are well visualized (6). Arterial stenosis has been expressed as the percentage of reduction of the cross-sectional area or the diameter of the artery (6,9). Venous compression is very difficult to incriminate because such compression is frequently observed in asymptomatic individuals in all the compartments of the thoracic outlet after arm elevation (5,9,29). Venous thrombosis and collateral circulation are well demonstrated by means of iodinated contrast media and constitute objective signs of venous TOS (Fig 14). Unfortunately, they are late-stage consequences of venous compression (9).

The limitations of CT include the difficulty of carrying out a fine analysis of the brachial plexus...
due to the limited CT contrast resolution, to which must be added two limitations that CT shares with MR imaging. First, abduction of the shoulder is limited by the size of the CT tunnel itself (upper limb elevation of >130° is impossible); second, the CT study is carried out with the patient in the supine position. In fact, the position of the patient undoubtedly has an effect on dynamically induced compression, as demonstrated by Scherrer et al (30) in 1979. In angiographic studies performed in 115 patients, these authors showed that 32% of false-negative results were related to use of the supine position during the examination. Finally, CT is an ionizing radiation technique, and the administration of iodinated contrast medium may be contraindicated or result in adverse side effects. In our clinical practice, this imaging method has been found to be especially helpful in analyzing the relationship between the vessels and the surrounding bony structures.

**MR Imaging**

MR imaging has the advantages of being a noninvasive and nonionizing technique offering excellent soft-tissue contrast. MR imaging of TOS is classically performed by using a phased-array body coil. Accurate observation of all the anatomic components of the thoracic outlet is possible, especially with use of sagittal T1-weighted sequences (7,9,31,32). The sagittal plane has been reported to be especially helpful for the depiction of vascular and nervous compressions, as they typically have an anteroposterior and craniocaudal direction. Coronal sequences can also supplement the examination, as they may provide a good view of the brachial plexus and also demonstrate fibrous bands (9). Whatever the plane, the sequences must be performed with the arm in a neutral position and, most of all, after hyperabduction of the arm. Interestingly, a study reported that, except for venous thrombosis, all the forms of neurovascular compression were demonstrated only with the arm elevated, which highlights the usefulness of this postural maneuver when TOS is suspected.

Arterial and venous compressions may be assessed by comparing the arterial cross-sectional area at the considered location with the arm in a neutral position (alongside the body) and after arm elevation (Figs 15, 16). Arterial compression may also be detected by analyzing the arterial caliper along the course of the vessel (Figs 15–17). MR angiography appears to be complementary to analysis of the arterial cross-sectional area on sagittal MR images (33–35) (Fig 15). This sequence may be especially helpful in detecting unobtrusive poststenotic aneurysmal dilatation. When venous TOC is suspected, venous thrombosis and collateral circulation must be looked for, but the limitations involved are the same as with CT.

MR imaging also allows a good analysis of the brachial plexus thanks to the excellent soft-tissue contrast it provides. The criteria used for determining the presence of neurologic compression are disappearance of the fat surrounding the brachial plexus and close contact with the adjacent bony structures (9) (Fig 18). Neurologic compressions seem to occur with the same frequency in the interscalene triangle and in the costoclavicular space (9).

Finally, MR imaging is also an efficient technique when searching for muscle hypertrophy

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**Figure 15.** Arterial compression in a 24-year-old woman. (a, b) Sagittal T1-weighted MR images, obtained after arm hyperabduction, show compression of the subclavian artery (arrow) in the costoclavicular space. Compare the cross-sectional area of the artery inside the costoclavicular space (a) with the cross-sectional area at the exit from the costoclavicular space (b). (c) MR angiogram shows the arterial stenosis (arrow).
**Figure 16.** Arterial compression in a 47-year-old man. Contiguous sagittal T1-weighted MR images, obtained after arm elevation, show compression of the subclavian artery (arrow) by a cervical rib (arrowhead) in the costoclavicular space.

**Figure 17.** Neurovascular TOS in a 20-year-old woman. Consecutive sagittal T1-weighted MR images of the costoclavicular space, displayed from medial (a) to lateral (c), show compression of the brachial plexus (arrowhead) in a and b and compression of the subclavian artery (arrow) in a. Compare the caliber of the artery in a and in b and c and the morphology of the brachial plexus in a and b and in c.

**Figure 18.** Neurogenic TOS in a 44-year-old woman. Sagittal T1-weighted MR images, obtained with the arms alongside the body (a) and after arm elevation (b), show narrowing of the costoclavicular space after hyperabduction and compression of the brachial plexus (straight arrow) between the clavicle (curved arrow) and first rib (arrowhead).
(scalenes, subclavius, or pectoralis minor muscles), abnormal muscles (eg, scalenus minimus) (Fig 12), and fibrous bands (9,32) (Fig 10). The visibility of such abnormal structures represents a fundamental advantage of MR imaging over other imaging techniques and may influence treatment. An indirect sign of the presence of a tight fibrous band is elevation of the subclavian artery, which is well demonstrated on sagittal images (9).

MR imaging has the same practical limitations as CT, that is, the restriction of arm elevation due to the size of the tunnel and the supine position of the patient. In our experience, another limitation is experienced in very thin individuals with little adipose tissue, in whom the delineation and individualization of the anatomic structures may be difficult.

**Ultrasonography**
Continuous-wave Doppler ultrasonography (US) has the advantage of enabling assessment of blood flow during performance of the clinical tests used in clinical practice. This technique is frequently used to confirm the clinical suspicion of arterial TOS. However, it is based on indirect signs of more proximal arterial stenosis and does not demonstrate the exact site of the arterial compression. The usefulness of power Doppler US in association with B-mode scanning has recently been reported in the assessment of subclavian and axillary arterial cross-sectional areas in patients with clinical suspicion of arterial TOS (36). B-mode scanning may allow detection of anatomic abnormalities such as aneurysmal dilatation and deviation of vessels. Color duplex sonographic examination associated with postural maneuvers (arm in neutral position, 90°, 120°, and 180° of abduction) may demonstrate alterations of the blood flow, such as complete cessation of blood flow or increase of blood flow velocity (29,37,38).

The main advantage of this technique is the direct comparison between dynamically induced symptoms and concomitant visualization of the vessels. The other fundamental advantage of sonography is the possibility of performing the examination with the patient in an upright or seated position, as in clinical examination, in contrast to CT and MR imaging, which are performed with the patient in a supine position. Unfortunately, this technique does not allow an accurate overview of the thoracic outlet region nor in particular an analysis of the region of the pulmonary apex. It must always be kept in mind that thoracic outlet symptoms may reveal locoregional disease such as a superior sulcus lung tumor (Pancoast-Tobias). For this reason, sonography should not be used solely to assess TOS; rather, it appears to be a valuable supplementary technique to CT or MR imaging in the event of clinical discordance, especially in patients with positive clinical features of TOS but negative features of TOS at CT and MR imaging (36).

Sonography also allows delineation of the brachial plexus structures (39), but its accuracy depends on the experience of the operator, especially as regards the assessment of this complex anatomic region. Moreover, to the best of our knowledge, the assessment of neurologic compression by means of this technique has not yet been reported.

**Treatment of TOS**

**Initial Management**
A conservative approach is the rule in the initial treatment of neurogenic TOS. Therapeutic efforts are focused on relaxing the scalene muscles and strengthening the postural muscles through physical therapy, combined with hydrotherapy and massage. Pain medication, nonsteroidal anti-inflammatory agents, and muscle relaxants are often useful adjuncts in treatment.

The initial treatment of arterial TOS is focused on revascularization in order to remedy acute ischemia if necessary. This step is typically performed via brachial artery thromboembolectomy. Demonstration of a fixed arterial lesion, either occlusive or aneurysmal in nature, is an indication for surgical reconstruction (40). Since these lesions are secondary to extrinsic compression, there is no significant role for endovascular approaches to their management (41).

The initial treatment of effort thrombosis generally involves contrast venography and catheter-directed thrombolytic therapy. This frequently results in restoration of venous drainage and resolution of the acute symptoms. Patients then receive maintenance treatment with intravenous heparin, which is converted to oral anticoagulation with warfarin. Most patients with venous TOS are candidates for surgical decompression.

**Surgical Strategies**
A first-rib resection via a supraclavicular approach represents the predominant treatment strategy, but some teams have recently adopted a highly selective approach in which supraclavicular scalenectionomy is the principal surgical strategy and first-rib resection is reserved solely for vascular forms of TOS.
First-rib resection and subclavian artery reconstruction are required when any degree of aneurysmal degeneration is present, particularly if the patient has had preoperative symptoms of digital thromboembolism, as well as for persistent occlusive lesions of the arterial wall that are still present after scalenectomy (41).

Patients with disabling neurogenic or arterial TOS may present with symptoms characteristic of sympathetic overactivity. In these situations, cervical sympathectomy may be a useful adjunct to thoracic outlet decompression.

Thoracic outlet decompression for venous TOS includes scalenectomy, brachial plexus neurectomy, resection of the first rib, and circumferential venolysis. In situations where external venolysis and paracervical first-rib resection have not succeeded in relieving the venous obstruction, additional venous reconstruction may be required.

Conclusions
The diagnosis of TOS is clinically based. Imaging may be helpful in informing the clinician as to the anatomic structures undergoing compression, the location of that compression, and the anatomic structures responsible for it (whether normal or abnormal). Indeed, all these features may influence the treatment. Radiographs may demonstrate predisposing bone abnormalities (elongated C7 transverse process, cervical rib). To the best of our knowledge, the respective advantages of the complementary imaging techniques have not been assessed in comparative studies. In the case of neurogenic or neurovascular symptoms, MR imaging in association with postural maneuvers has proved useful, especially in demonstrating brachial plexus compression and the existence of fibrous bands. CT with contrast medium injection (if not contraindicated) and postural maneuvers appears effective in demonstrating vascular compression by means of volume-rendered images, which allow analysis of the relations with bony structures. Color duplex sonographic examination and B-mode scanning, in association with postural maneuvers, is a valuable supplementary tool to CT and MR imaging when the results of the latter prove negative.

References


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Page 1744
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Page 1792
Venous compression is very difficult to incriminate because such compression is frequently observed in asymptomatic individuals in all the compartments of the thoracic outlet after arm elevation (5,9,29).

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Accurate observation of all the anatomic components of the thoracic outlet is possible, especially with use of sagittal T1-weighted sequences (7,9,31,32).