BMJ Best Practice

Thoracic outlet syndrome

The right clinical information, right where it's needed

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Summary

- Compression of 1 or more of the neurovascular structures traversing the superior aperture of the chest. May affect neurological or vascular structures, or both, depending on the component of the neurovascular bundle predominantly compressed.

- Types include neurological, arterial, venous, and neurovascular/combined, and patients may present with signs and symptoms of nerve, vein, or artery compression or any combination of these. Neurological is the most common, while arterial, which is relatively rare, is arguably the most important to recognise owing to the risk of ischaemia.

- Neurological thoracic outlet syndrome primarily develops spontaneously in people in their late teens up to the age of 60 years, and is more common in women. It usually occurs in the setting of congenital abnormalities of the thoracic outlet, hyperextension injuries, repetitive stress injuries (e.g., work related), and external compressing factors (e.g., poor posture).

- Patients with venous or arterial occlusion require prompt evaluation for surgical intervention with thrombolysis and thoracic outlet decompression.

- For most cases of disputed neurological thoracic outlet syndrome, initial management is conservative and includes physiotherapy. Surgical management of nerve compression is indicated in individuals that show physical signs of nerve damage or who have failed conservative treatments.

Definition

Thoracic outlet syndrome (TOS) refers to the compression of 1 or more of the neurovascular structures traversing the superior aperture of the chest.[1] The thoracic outlet is the area between the neck and shoulder, over the top of the thorax, and under the clavicle to the axilla.

Knowledge of the thoracic outlet anatomy is cardinal for the physician to diagnose any type of TOS.[2] [3] The subclavian artery leaves the thorax by arching over the first rib behind the scalenus anticus muscle and in front of the scalenus medius muscle. It then passes under the clavicle and finally enters the axilla beneath the pectoralis minor muscle. The subclavian vein has an identical course, except that it passes anteriorly rather than posteriorly to the scalenus anticus muscle. The brachial plexus follows the route of the subclavian artery, but it lies a little more posteriorly and laterally. The axillary-subclavian vein traverses the tunnel formed by the clavicle and subclavius muscle anteriorly, the scalenus anticus muscle laterally, the first rib posterior-inferiorly, and the costoclavicular ligament medially.[4] The anatomical areas within the thoracic outlet that can typically impart neurovascular compromise include the interscalene triangle, the costoclavicular space, and the subcoracoid space, although it is possible that the sternal-costovertebral bony circle may also be involved.[2] [3] [5]

Epidemiology

Neurological TOS

- Primarily develops spontaneously in people in their late teens up to the age of 60 years. It is more common in women, and usually occurs in the setting of hyperextension injuries, repetitive stress injuries (e.g., work related), and external compressing factors (e.g., poor posture). Motor vehicle collisions and occupations involving prolonged time at a desk/computer or doing overhead tasks are commonly implicated. Overhead athletes (e.g., swimmers, tennis players) may also be affected. Paediatric and adolescent populations can also infrequently be afflicted, creating an entity termed early-onset TOS.[5] [8] [9] [10] [11] [20] [21] [22] [23]

Venous TOS

- Has been found to either occur in males and females equally or show a male predominance. It most commonly occurs at a younger age range (18-30 years) and rarely occurs in children or adolescents. Patients participating in frequent overhead exercise or activity are most commonly afflicted (e.g., swimmers, painters).[14] [16] [24]

Arterial TOS

- Epidemiological factors are less well defined due to its infrequent occurrence; however, like neurological TOS, it can occur in overhead athletes.[17] [18] [19] [25] [26] [27]

Aetiology

The anatomical relationships within the thoracic outlet provide potential areas of pressure leading to neurovascular-vascular compression. The anatomical areas within the thoracic outlet that typically impart neurovascular compromise include the interscalene triangle, the costoclavicular space, the subcoracoid space, and, more rarely, the sternal-costovertebral bony circle.[2] [3] [5]

Many factors can cause compression, elongation, or angulation of the neurovascular bundle at the thoracic outlet, but the basic aetiological factor is deranged anatomy.

Dynamic, static, congenital, traumatic, and occasionally atherosclerotic factors all contribute to deranged anatomy and therefore anatomical narrowing of the thoracic outlet.

Dynamic factors

- There is an unusually wide latitude of motion in the components of the shoulder joint. A moderate degree of motion takes place at the sternoclavicular articulation, this being one of the few universal joints in the body. The acromioclavicular articulation lets the inferior angle of the scapula move laterally, about 45°, during elevation of the arm. Finally, the articulation between the humerus and scapula allows the widest range of motion of any joint in the body. These movements, involving changes in relative position of regional structures, may result in compression or impingement on vessels and/or nerves. For example, when the arm is in full hyperabduction above the head, the axillary artery is bent 180° from its position when the arm is at the side. This motion pulls the vessel across the coracoid process and head of the humerus, as across a pulley, causing arterial compression.

Static factors

- Vigorous work or exercise may create relative muscle hypertrophy, thereby creating a relative narrowing of the spaces in the thoracic outlet through which neurovascular bundles pass (e.g., interscalene triangle, costoclavicular space, or subcoracoid space). Examples include factory workers with repetitive overhead stresses, people who work desk jobs with prolonged periods sitting at a keyboard, and repetitive overhead sport like tennis and swimming. On the other hand, a reduction in muscle mass and tone may be an important, though poorly understood, factor in explaining why TOS most commonly occurs in middle-age. Additionally, abnormal posture is a well known contributing factor in neurological TOS, creating compression of the brachial plexus in the interscalene triangle, costoclavicular space, and subcoracoid space.[6] [8] [14] [16] [17] [18] [19] [22] [23] [25] [26] [27] [28] [29]

Congenital factors

- Congenital factors can contribute to narrowing of all 4 spaces in the thoracic outlet. Congenital anomalies of the first rib can create stretching, angulation, or compression of the neurovascular bundle as it passes over the first rib in the sternal-costovertebral bony circle. Interscalene triangle narrowing can occur through the presence of a cervical rib, long cervical transverse processes, first rib abnormality, congenital fibrous bands, abnormal insertions of the scalene muscles onto the first rib, and supernumerary scalene muscles. Costoclavicular space narrowing can occur through the presence of a prominent costoclavicular membrane or through lower shoulder girdle positioning. The most common congenital defect in Paget-Schroetter syndrome is the congenital insertion of the costoclavicular ligament far laterally to the first rib.[30] Congenital factors to date do not contribute to most cases of TOS/pectoralis minor syndrome (PMS) from subcoracoid space narrowing.[5] [7] [8] [9] [11] [22] [29] [31] [32] [33] [34]

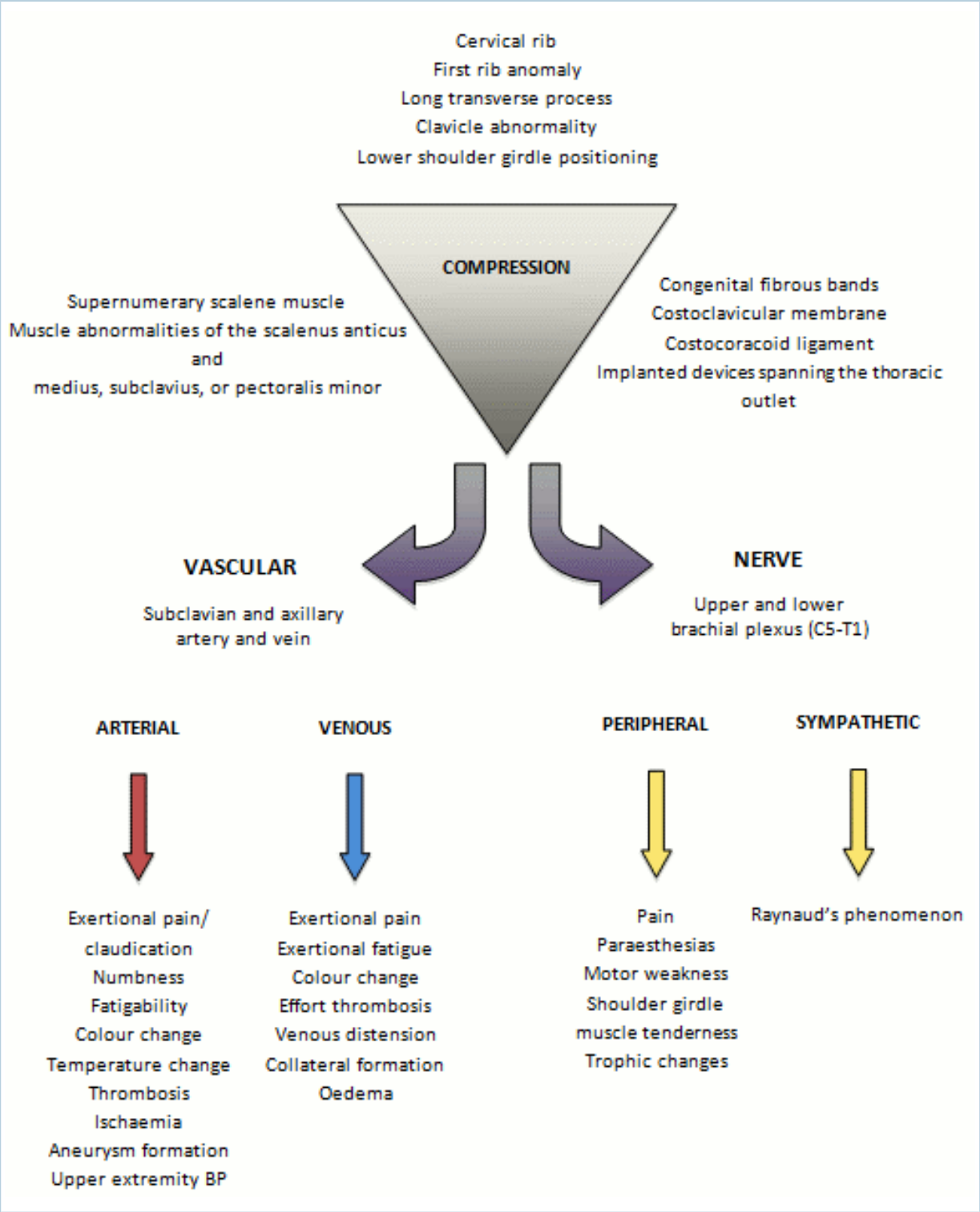
Traumatic factors

- Hyperextension injuries of the neck (e.g., whiplash or falls) are well known contributing factors in neurological TOS, creating compression of the brachial plexus in the interscalene triangle, costoclavicular space and/or subcoracoid space.[6] [8] [22] [23] [29] [35]
- Other traumatic causes include fractures of the clavicle [Fig-5]

  and subacromial dislocation of the humeral head. Occasionally, a crushing injury of the upper thorax may unduly stretch parts of the brachial plexus and/or thrombose the artery or vein.
- Acute injuries that do not result in the fracture of osseous structures surrounding the thoracic outlet can still create neurovascular bundle compression through the presence of pseudoaneurysm or haematoma formation. Callus formation from a previous fracture of the clavicle can also create compression of the neurovascular bundle in the costoclavicular space.[5] [8] [9] [10] [22] [35] [36] [37]
- Cases of arterial TOS have also been described as occurring technically outside of the thoracic outlet due to humeral head dislocations or humeral head compression from repetitive trauma.[17] [18] [38]

Atherosclerotic factors

- The degree of activity and effort that is well tolerated by a healthy, flexible artery may cause thrombosis in a vessel that is narrowed and sclerotic. This situation has been observed in several people in their 6th and 7th decades of life, whose shoulder girdles were anatomically normal for their age, but whose arteries were hardened and relatively inflexible.[39] This is a rare occurrence.



Thoracic outlet compression factors and resulting signs and symptoms

From the collection of Dr Chaney Stewman

Pathophysiology

Neurological TOS

- Repetitive stress injuries and hyperextension injuries of the neck have been found to cause acute haemorrhage and swelling of the scalene muscles and therefore narrowing of the interscalene

triangle.[6] [10] [22] [28] [35] Subsequently, fibrosis and therefore tightness of the scalene muscles ensues, which also creates narrowing of the interscalene triangle and potential angulation of the neurovascular bundle.[6] [8] [10] [22] [28]

- Relative hypertrophy of muscles within the thoracic outlet can cause narrowing and compression of the neurovascular bundle. Histological studies support this as they show scalenus anticus type 1 (slow twitch) fibre predominance and hypertrophy.[24]
- The congenital presence of cervical ribs, accessory muscles, fibrous bands, and prominent ligaments additionally contribute to some cases of TOS.[6] [10] [22] [28] [35]

Venous TOS

- The presence of a costoclavicular ligament contributes to many cases of venous TOS arising within the costoclavicular space, as it can create intermittent or positional venous obstruction.[4] [14]
- Idiopathic thromboses are possibly linked to hypercoagulable states (e.g., thrombophilia, malignancy). Relative muscle hypertrophy has also been described in both scalene muscles and the pectoralis minor muscle, which can contribute to positional venous obstruction in the interscalene triangle and subcoracoid space.[14] [16] [24]

Arterial TOS

- The same congenital factors and muscle hypertrophy that contribute to neurological TOS also occur with arterial TOS as the subclavian and axillary artery course intimately through the thoracic outlet with the brachial plexus.

Neurovascular/combined TOS

- A histological study has also shown that fibrosis within the scalene muscles is important in the pathology of traumatic TOS.[22] [40]

Classification

TOS can be classified into 4 main categories: neurological, vascular, neurovascular/combined, and other. Each category is then differentiated into further subtypes.

Neurological TOS (NTOS)

The most common form, and generally refers to an injury of the brachial plexus. NTOS is divided into 3 categories.

- True: a rare (<1% of NTOS), primarily unilateral disorder with specific diagnostic criteria. A cervical rib from which fibrous bands extend to the first thoracic rib is present, which causes stretching and compression of the proximal lower trunk of the brachial plexus and therefore ulnar nerve. Specific findings of ulnar neuropathy on electrodiagnostic studies are seen, as well as hand muscle atrophy.[5] [6]
- Disputed: the most common form of NTOS, comprising at least 95% of cases. Its name resulted from initial disagreements among clinicians regarding whether or not it is a true TOS versus a pain syndrome. In light of the ongoing debate, it has also been referred to in literature under other descriptors such as non-specific, postural, assumed, or symptomatic. This entity is also sometimes divided into 2 different subtypes based on whether the upper (20% of patients) or lower (80% of patients) rami of the brachial plexus are affected. The patient may or may not have an underlying

congenital abnormality of the thoracic outlet. Compression of the brachial plexus typically occurs at the interscalene triangle, costoclavicular space, subcoracoid space, and, more rarely, the sternal-costovertebral bony circle.[5] [6] [7] [8] [9] [10]

- Pectoralis minor syndrome (PMS): a clinical entity that is now re-emerging in TOS literature. It was initially described in 1945 within the spectrum of shoulder girdle syndromes as a hyperabduction syndrome, but was later excluded by area of anatomical compromise. It refers to brachial plexus injury within the subcoracoid space.[5] [6] [9] [11] [12] [13]

Vascular TOS

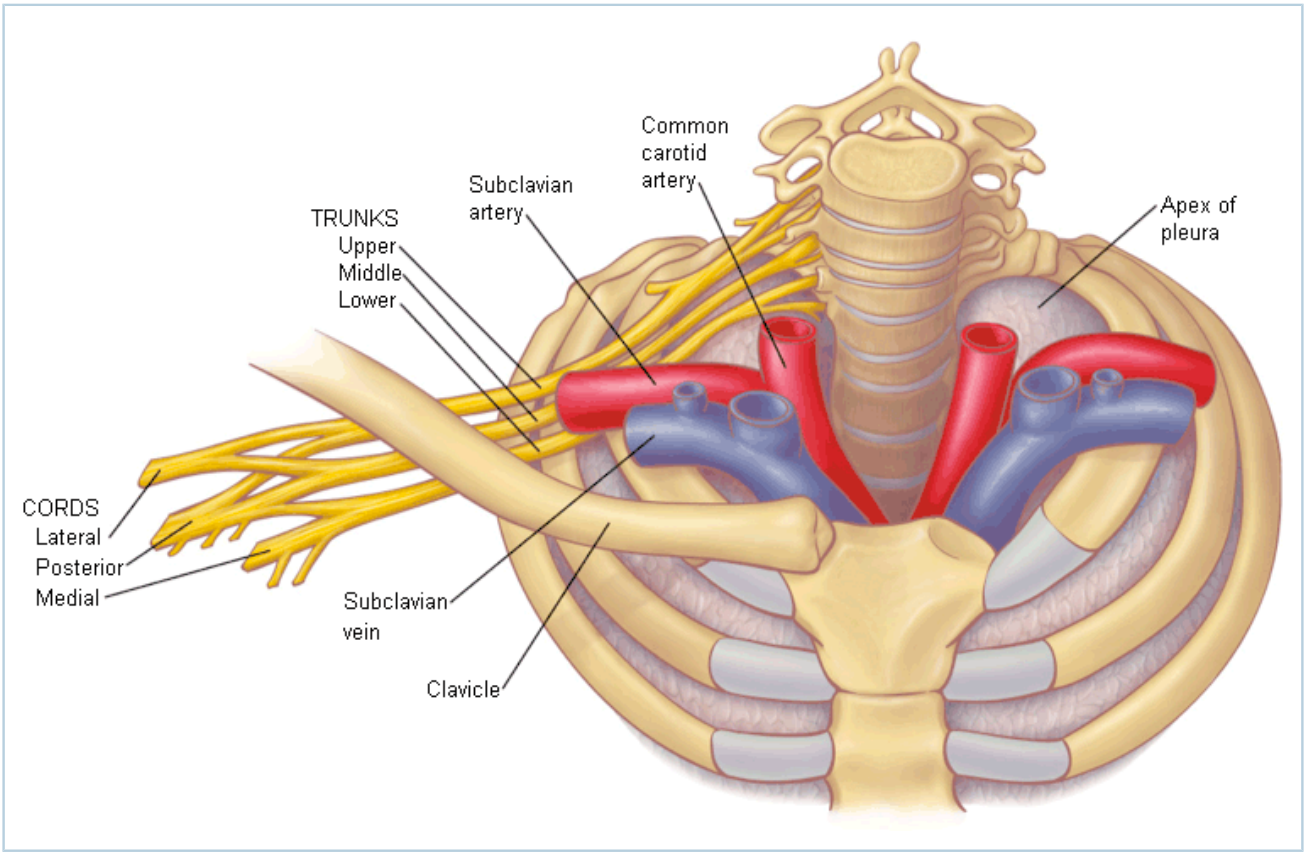
- Venous TOS (vTOS): refers to compression of the subclavian vein most commonly at the costoclavicular space, resulting in either obstruction or thrombosis. The costoclavicular ligament is commonly implicated in the concurrent setting of repetitive overhead activities, resulting in costoclavicular space narrowing.[14]
    - Paget-Schroetter syndrome is a subset of vTOS.[4] It is a thrombosis of the axillary-subclavian vein in the setting of strenuous and repetitive upper extremity activity, thereby often referred to as an effort thrombosis.[14] [15] [16]
- Arterial TOS (ATOS): refers to compression of the subclavian and/or axillary artery. Compression of these arteries typically occurs at the interscalene triangle, costoclavicular space, subcoracoid space, and, more rarely, the sternal-costovertebral bony circle. Though it is the least common type of TOS (accounting for approximately 1% of TOS cases), it remains clinically important due to the risk of ischaemia. Like vTOS, congenital abnormalities are often present, most commonly a cervical rib. Overhead activities have also been implicated.[17] [18] [19]

Neurovascular/combined TOS

- Given the close proximity of the brachial plexus to its surrounding vasculature, combined injuries can occur. The sympathetic nerves of the brachial plexus are intimately attached to the artery as well as being adjacent to the bone.
- Combined injuries commonly occur in traumatic TOS resulting in neurovascular injury most commonly from a traumatic midshaft displaced fracture of the clavicle.[5] [9]

Early-onset TOS

- A type of neurological or vascular TOS that may occur in children and adolescents.[10] [11] [20] [21] This type is not discussed any further in this topic as there are only a small number of studies evaluating this population.



Subclavian vein and artery pass over first rib and under clavicle. Brachial plexus traverses top of bony circle to join the artery. Apex of the pleura (cupula) shown on left side

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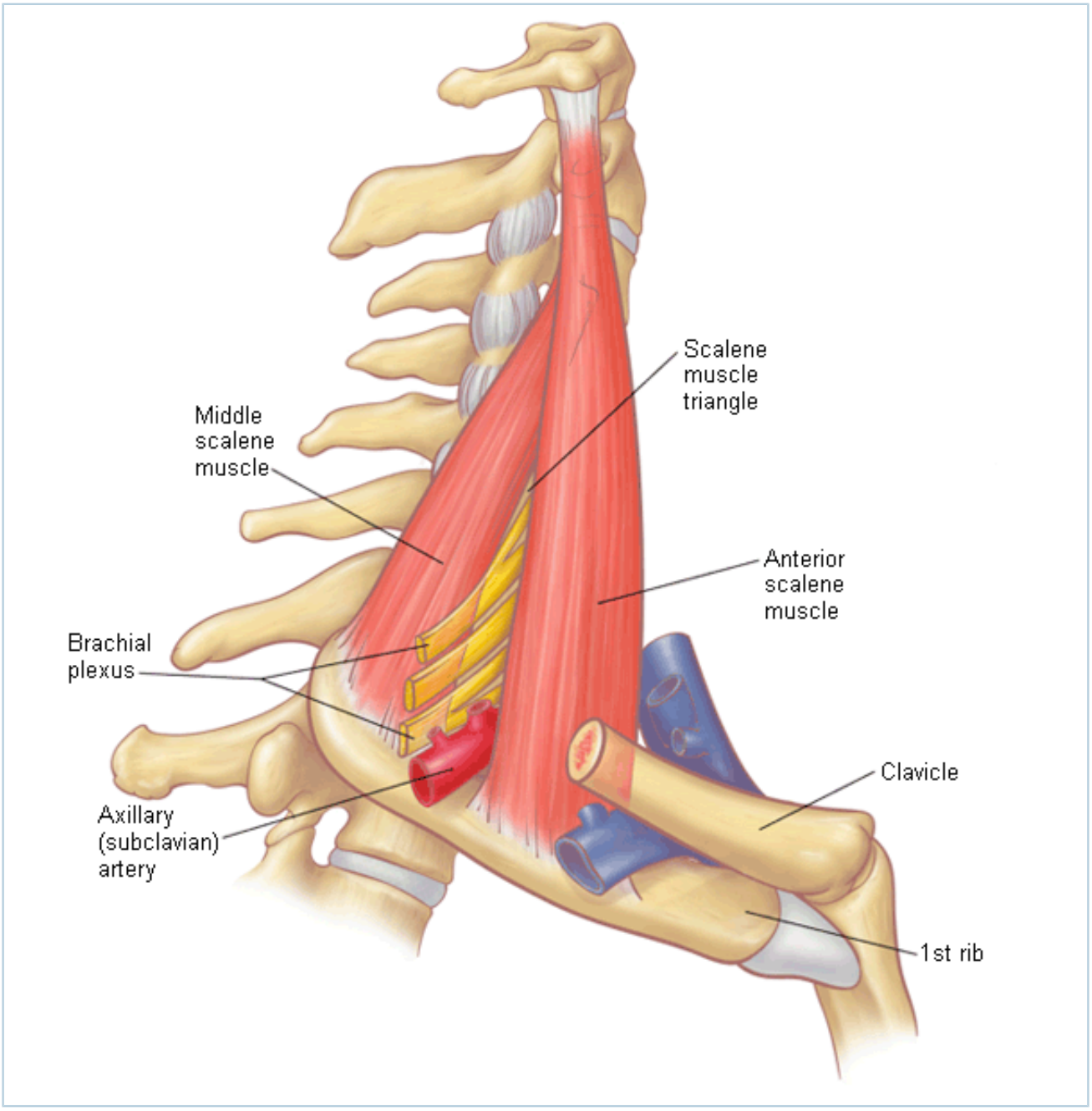

Scalene muscle triangle is the second major level of compression

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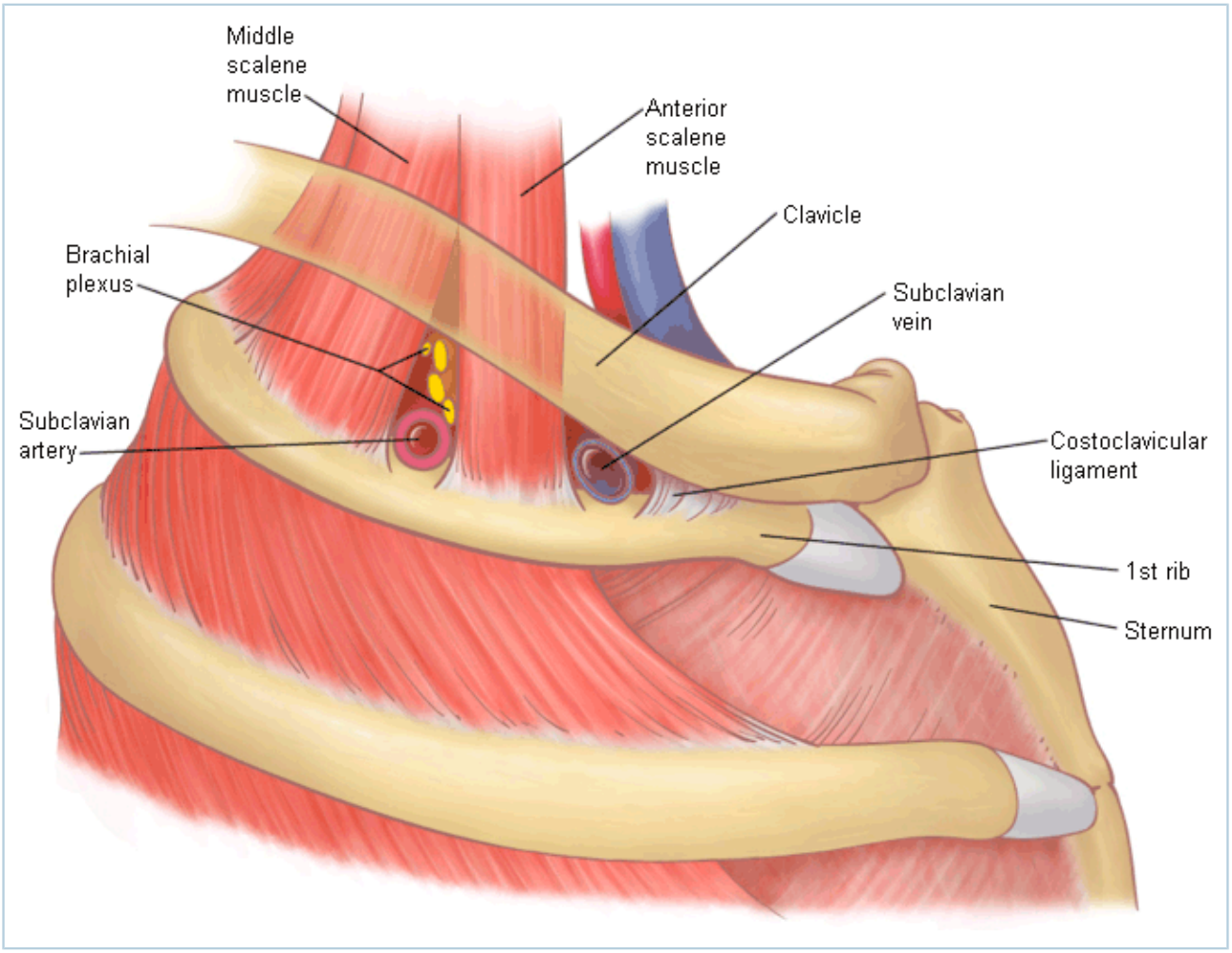

Cross-section of neurovascular structures traversing the thoracic outlet with clavicle above and first rib below

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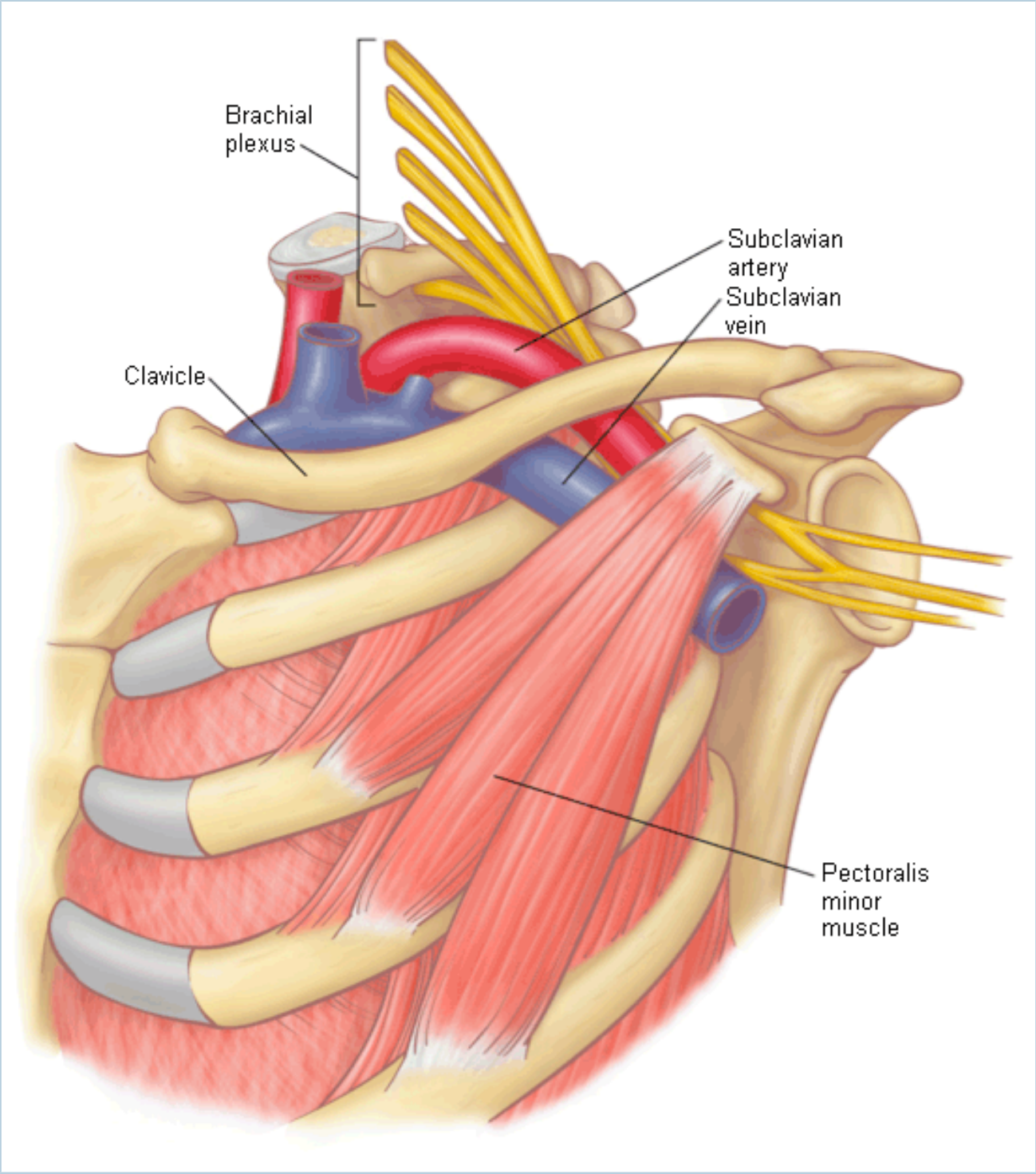

Neurovascular structures pass behind pectoralis minor muscle, another major area of compression. Pectoralis minor is a shoulder protractor, which can overpower the rhomboids. The shoulder retracts and alters the thoracic outlet, contributing to muscular imbalance and compression of the brachial plexus

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Primary prevention

Lifestyle should be modified to minimise compression and trauma to the thoracic outlet by the following methods:

- Maintaining good posture
- Optimising posture (including core stability)
- Optimising mechanics for overhead athletes (i.e., core and shoulder girdle stability)
- Avoiding repetitive jobs, or taking frequent breaks (rotating activity) during repetitive jobs
- Using ergonomic workstations.

Secondary prevention

In addition to optimising posture and work practices, patients can reduce recurrence by:

- Core strengthening
- Strengthening the shoulder girdle
- Keeping open the space between the clavicle and first rib using physical manoeuvres and stretches
- Loosening the neck and shoulder muscles (stretching the scalene muscles and pectoralis)
- Modifying behaviour (losing weight, correcting sleep positions).

Case history

Case history #1

A 30-year-old right-handed woman presents complaining of pain in the right side of her neck, shoulder, arm, hand, chest, and somewhat down her back. She describes her pain as dull and aching. She works as a computer operator and first noticed symptoms about 2 years ago. Along with the pain, she has developed severe numbness in her right arm and hand, which frequently wakes her at night. She notices that she drops things and has marked difficulty working over her head. Common household tasks have become very difficult for her (e.g., vacuuming, sweeping, mopping). Cold exacerbates her symptoms. She has previously had 2 courses of physiotherapy without improvement of her symptoms. Physical examination reveals 3+ supraclavicular tenderness on the right. She has a positive Adson's sign on the right and a positive Roos' test on the right in 5 seconds. Atrophy of the thenar eminence is noted in her right hand. Her grip is 2 out of 5 on the right, with a 1 out of 5 interossi on the right. Her ulnar conduction velocity on the right side is 40 m/second and on the left side is 55 m/second. Her median conduction velocity on the right side is 43 m/second and 58 m/second on the left side.

Case history #2

A 23-year-old man presents with swelling and pain in his left arm after strenuous exercising with upper extremity lever weights. Symptoms started 75 minutes after the exercises. The arm turned reddish, and he described it as 'feeling different than it ever had before'. He has Raynaud's phenomenon with marked cold sensitivity and writing increases his symptoms. No supraclavicular tenderness is present. He has a venous collateral over his left shoulder. He has a 4+ bilateral Adson's sign and a 4+ Roos' test on the left with mild anterior deltoid pain in 5 seconds. His grip is 4 out of 5 bilaterally and his interossi are 4 out of 5 bilaterally. Doppler ultrasonography of the left upper extremity demonstrated a clot in his left subclavian vein. The diagnosis of venous TOS (Paget-Schroetter syndrome) was confirmed.

Step-by-step diagnostic approach

Patients may present with signs and symptoms of nerve, venous, or arterial compression, or any combination thereof. Physicians should be guided by the patient's symptomatology as to which route(s) to investigate.

History and examination

Many historical factors can predispose patients to compression of the neurovascular components of the thoracic outlet: trauma (e.g., fracture of the clavicle), repetitive overhead activity, motor vehicle collisions, previous cardiovascular or thoracic surgery, cervical or bony abnormalities, poor posture, having large breasts or breast implants, obesity, and female sex.

There are many common examination findings and manoeuvres for diagnosing TOS; however, specific history and examination findings depend on the location of the compression.

Nerve (brachial plexus) compression

- Signs and symptoms include:[45]

- Paraesthesias in arms, hands, and/or fingers (unilateral or bilateral)
- Pain in head, neck, upper back, anterior chest, shoulder, arm, forearm, and/or hand
- Tenderness to palpation in the supraclavicular region, anterior chest wall, scalene muscles, trapezius, or pectoralis minor muscles
- Motor weakness in the shoulder girdle and hand
- Patchy sensory deficit along medial forearm and hand (if neurological [true] TOS)
- Hand muscle atrophy (most pronounced in thenar eminence muscles if neurological [true] TOS)
- Possible Raynaud's phenomenon
- Excessively sweaty hand(s)
- Possible 'multiple crush' (or 'double crush' if only two areas are involved) syndrome phenomenon: there may be multiple points of compression of the peripheral nerves between the cervical spine and hand, in addition to the thoracic outlet.[41] In these cases, less pressure is required at each site to produce symptoms. For example, a patient may have concomitant TOS, ulnar nerve compression at the elbow, and carpal tunnel syndrome[46]
- Possible sympathetic compression: symptoms include excessive cold or warm and sweaty upper extremities. Can sometimes mimic atypical chest pain (pseudoangina).

Venous (subclavian and axillary) compression

- Patient may have a history of implanted devices in the thoracic outlet (e.g., central venous catheters, pacemakers). Signs and symptoms include:[14] [16]

  - Intermittent episodes of upper extremity pain with vigorous upper extremity activity
  - Exercise fatigue of upper extremity
  - Effort thrombosis (i.e., sudden onset of upper extremity swelling and pain following sudden increase in causative activity)
  - Cyanosis, erythema, or dusky appearance of upper extremity
  - Subcutaneous venous collateral around shoulders (i.e., Urschel's sign)
  - Possible supraclavicular systolic bruit (may only be present with shoulder abduction)
  - Right upper extremity involvement (most common site).

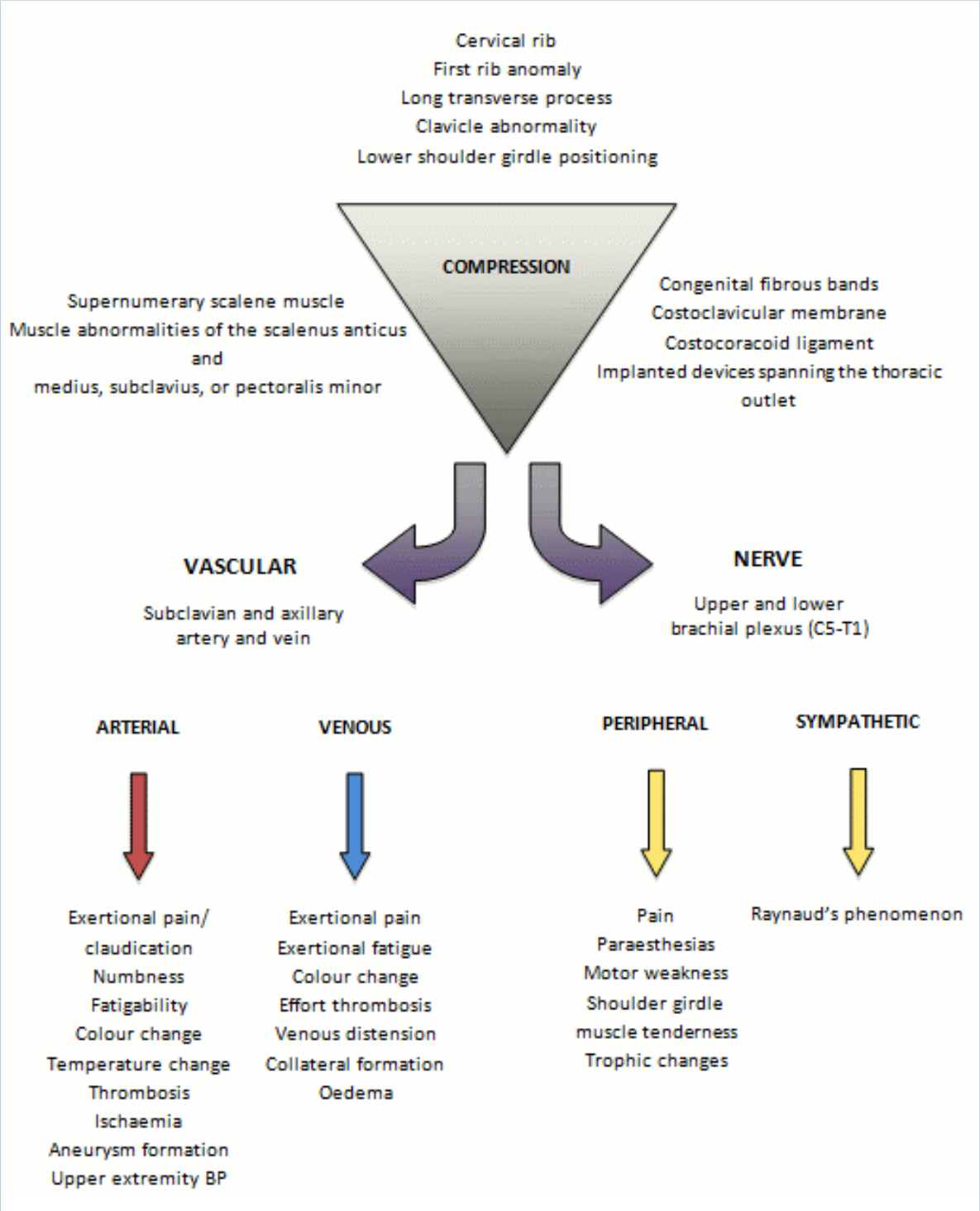
Arterial (subclavian and axillary) compression

- Signs and symptoms include:[17] [19] [27] [38]

  - Dull ache, numbness, or pain in upper extremity
  - Symptom improvement with rest and worsening with activity (e.g., exertional pain/claudication)
  - Early fatigability (e.g., dead arm sensation)
  - Cyanosis, pallor, or coolness of the upper extremity
  - Acute ischaemia and gangrene
  - Possible Raynaud's phenomenon
  - Excessively sweaty hand(s)
  - Blood pressure discrepancy (e.g., 20 mmHg difference) between extremities
  - Possible supraclavicular systolic bruit (may only be present with shoulder abduction).

Neurovascular/combined compression

- Patient may have a history of clavicular fracture.
- Possible sympathetic and arterial compression results in more severe symptoms of excessive upper extremity warmth or cold or sweating because of the additive or synergistic sympathetic stimulation. Trauma is frequently associated with sympathetic maintained pain syndrome or reflex sympathetic dystrophy. Sympathetic hyperactivity, also known as complex regional pain syndrome, may be a sympathetic response to pain rather than overt compression of the sympathetic chain.[45]



Thoracic outlet compression factors and resulting signs and symptoms

From the collection of Dr Chaney Stewman

Manoeuvres

Numerous provocative physical examination manoeuvres exist to reproduce brachial plexus impingement and frequently concurrent radial pulse obliteration. These manoeuvres produce subjective symptoms of pain and paraesthesias if positive or abnormal. These manoeuvres in isolation cannot be used for the diagnosis of TOS, and instead are used as confirmatory testing in conjunction with patient history and other diagnostic testing.[16] [17] [22] [47] [48] [49] [50] Manoeuvres include the following.

- Adson's (scalene) test: tightens the anterior and middle scalene muscles, thus decreasing the interspace and magnifying pre-existing compression of the subclavian artery and brachial plexus. The patient takes and holds a deep breath, extends the neck fully, and turns the head towards the side.
  [Fig-7]
- Costoclavicular test (also known as the Halsted test or military brace): shoulders are drawn downwards and backwards. This narrows the costoclavicular space by approximating the clavicle to the first rib and thus tends to compress the neurovascular bundle.
  [Fig-8]
- Hyperabduction test: hyperabducting the arm to 180° pulls the components of the neurovascular bundle around the pectoralis minor tendon, the coracoid process, and the head of the humerus.
  [Fig-9]
- Roos' test: both arms are placed at right angles to the shoulder, and the forearms are at right angles to the upper arms. Both hands are opened and closed as fast as possible to see if symptoms occur.
- Stretch test: the arm is abducted 90° with elbow extension, palm facing forwards, and thumb pointing up to the ceiling. The patient then laterally flexes the head to the opposite side. This stretches the plexus.
- Upper limb tension tests: a series of tests of all tissues in the upper limb with a preferential focus on the median nerve and its associated plexus and roots. They involve shoulder abduction, wrist suspension and extension, shoulder lateral rotation, elbow extension, and neck lateral flexion away from or towards test side.
- Wright's manoeuvre: simulating a lazy hand raise (externally rotate and abduct arm to 180° and flex at elbow).

Imaging

Bony abnormalities are present in up to 30% of patients as cervical rib, articulated or bifid first rib, fusion of first and second ribs, clavicular deformities, or previous thoracoplasties.[51] An initial cervical spine x-ray and/or CXR can reveal this and is the initial diagnostic test for all patients with TOS.

MRI of the neck, clavicle, and shoulder region should be considered in patients with suspected neurological TOS as it helps to provide anatomical detail of the soft tissue, and can identify abnormalities such as congenital fibrous bands.[5] [22] Non-contrast MRI is typically sufficient, unless a vascular component is suspected.[52] Images are obtained with the patient in both neutral and shoulder abduction positions to best localise the area of impingement.

If arterial TOS is suspected, CT angiography is the initial diagnostic imaging test performed. If venous TOS is suspected, Doppler ultrasonography is the initial diagnostic imaging test performed. CT angiography and Doppler ultrasonography are best performed with the patient both in neutral position and with shoulder abduction.[52] Magnetic resonance angiography or venography can also be considered

as alternatives. Conventional arteriography can also be performed, but has been replaced by alternative imaging for diagnosis and is largely used during cases of surgical intervention for arterial TOS. Contrast venography is the diagnostic standard for detecting venous TOS, but is performed in cases where alternative imaging is non-diagnostic or in cases where surgical intervention is planned.[16] [17]

Nerve conduction studies

Electrodiagnostic studies can be helpful indicators of brachial plexus impingement.[53] All patients with possible true neurological TOS should have confirmatory nerve conduction studies. EMGs are also used to help rule out alternative diagnoses (e.g., carpal or cubital tunnel syndrome or cervical radiculopathy).[22]

Pathognomonic findings on nerve conduction studies in the appropriate clinical picture for true neurological TOS include reduced motor amplitude of the median nerve, as well as reduced sensory amplitude of the medial antebrachial cutaneous and ulnar nerves.[5] Specifically, the median motor compound muscle action potential (recording thenar muscle, commonly the abductor pollicis brevis) is very low in amplitude, whereas the median sensory nerve action potential (recording the second digit) is normal. Additionally, the medial antebrachial cutaneous sensory nerve (supplying the medial forearm) action potential is either absent or very low in amplitude. The ulnar sensory nerve action potential (recording fifth digit) is slightly to moderately low in amplitude.

For examination of the ulnar nerve, the patient is placed on the examination table with the arm fully extended at the elbow and in approximately 20° of abduction at the shoulder to facilitate stimulation over the course of the ulnar nerve. The ulnar nerve is stimulated at the 4 points by a special stimulation unit that imparts an electrical stimulus at all points to obtain a maximum response.
[Fig-10]

When performed, the EMG should evaluate all muscles innervated by cervical nerve roots and the brachial plexus, including paraspinal muscles. Interpreting areas of abnormality can help identify the area(s) of compression.

Other tests

Targeted muscle blocks can also aid not only diagnosis but possibly response to surgical intervention in neurological TOS. Specifically, local anaesthetic is injected into the scalene anticus and pectoralis minor muscles. The muscle blocks can provide symptom relief if symptoms are due to brachial plexus compression at the interscalene triangle in cases of pectoralis minor syndrome.[5] [6] [10] [12]

FBC and coagulation studies can be considered in patients with venous TOS. They can help to evaluate for a hypercoagulable state as an aetiology for the venous thrombus if alternative aetiologies are not present (e.g., effort thrombosis, implanted devices, congenital abnormalities).

Risk factors

Strong

cervical rib or bony abnormalities

- Abnormalities in bone structure, or the presence of a cervical rib, can compress the neurovascular structures between the first rib and clavicle.

trauma

- Skeletal anatomy or upper extremity shifting (as a result of a traumatic incident or accident) can create a sudden onset of symptoms due to compression of the neurovascular structures between the first rib and clavicle.
- Neurovascular/combined (traumatic) TOS can be associated with (or can follow) fractures of the clavicle.

poor posture

- Compression can occur as a result of poor posture, or hunching of the upper shoulders and back, due to the anatomy of the thoracic outlet bending in such a way that the clavicle compresses the neurovascular structures on the first rib.

repetitive overhead activity

- Repetitive overhead activity, including work conditions, sport, or hobbies, cause the enlargement of the scalenus anticus (and other) muscles in the thoracic outlet, compressing the neurovascular structures.

motor vehicle collisions

- Motor vehicle collisions are a risk factor for both neurological (disputed) TOS and neurovascular (traumatic) TOS.
- Hyperextension injuries of the neck, as in the case of whiplash from motor vehicle collisions, have been commonly implicated in neurological (disputed) TOS.[5] [6] [9] [10] [28] [29] [35]
- High-velocity accidents resulting in significant trauma (e.g., fracture of the clavicle) have also been implicated in neurovascular (traumatic) TOS.[35]

large breasts or implants

- Compression can occur as a result of large breasts, due to the weight on the shoulders, which pulls the clavicle down on the neurovascular structures.

obesity

- Compression can occur as a result of obesity, which narrows the space in the thoracic outlet.

age (late teens to 60 years)

- Neurological (true) TOS afflicts a wide range of ages (i.e., late teens to 60 years). This is in contrast to neurological (disputed) TOS, which most commonly occurs in middle-aged adults. This is further contrasted by both venous and arterial TOS, which more commonly affect the younger population (approximate age range 18 to 30 years).[5] [9] [16]

female sex

- Neurological TOS is a female-predominant medical condition.[5] [9] [22]

Weak

hx of poliomyelitis

- Weakening of the shoulder muscle lets the clavicle down to compress the neurovascular structures in the thoracic outlet.[41]

pregnancy

- Weight gain narrows the thoracic outlet space.[41]

median sternotomy

- Operations that involve opening of the sternum can result in TOS due to stretching of the neurovascular structures between the first rib and clavicle.[41] [42] [43] [44]

History & examination factors

Key diagnostic factors

hx of repetitive jobs or overhead hobbies/activities (common)

- For example: computer operators, linotype operators, mail sorters, pump operators, athletes (particularly, football, baseball, tennis, and volleyball players, swimmers and divers, weightlifters, gymnasts).

pain in upper extremity and adjacent areas (common)

- Can occur in all types of TOS.
- Pain can occur in the head, neck, upper back, anterior chest, shoulder, arm, forearm, and/or hand.
- Tenderness to palpation may be present in the supraclavicular region, anterior chest wall, scalene muscles, trapezius, or pectoralis minor muscles.
- May be described as a dull ache in arterial TOS.
- Exacerbated by exertion in both arterial and venous TOS.

paraesthesias in arms, hands, and/or fingers (common)

- Common complaint in neurological TOS.
- Numbness may also be described in arterial TOS.
- May be bilateral or unilateral.

circulatory changes in upper extremity (common)

- Raynaud's phenomenon can be seen in neurological and arterial TOS.
- Cyanosis, pallor, or coolness of the upper extremity can be seen in arterial TOS. Acute ischaemia or gangrene is an emergent complication of arterial TOS.
- Cyanosis, erythema, or dusky appearance of the upper extremity can be seen in venous TOS.
- Sympathetic compression symptoms include excessive cold or warm and sweaty upper extremities. Can sometimes mimic atypical chest pain (pseudoangina).
- Sympathetic and arterial compression results in more severe symptoms of excessive upper extremity warmth or cold or sweating because of the additive or synergistic sympathetic stimulation.

upper extremity fatigue (common)

- Described in both arterial and venous TOS.
- Fatigue is often exacerbated by activity.
- Dead arm sensation has been reported in arterial TOS.

Other diagnostic factors

hx of clavicular fracture (uncommon)

- Neurovascular/combined (traumatic) TOS is often associated with a clavicle fracture, which can cause compression of the neurovascular structures in the costoclavicular space.

hx of cardiovascular or thoracic surgery (uncommon)

- Past surgical operations that could have caused compression on the neurovascular structures between the first rib and clavicle (e.g., median sternotomy).
- There may be history of implanted devices in the thoracic outlet (e.g., pacemakers, central venous catheters) in venous TOS.

palpation of cervical rib (uncommon)

- Presence of a cervical rib in the concurrent setting of repetitive stress can predispose people to TOS.
- Cervical ribs can sometimes be palpated on physical examination of the neck.

subcutaneous venous collateral around shoulders (Urschel's sign) (uncommon)

- Sometimes present in venous TOS.

motor weakness (uncommon)

- Present primarily in true neurological TOS.
- May occur in shoulder girdle and hand.

hyperhidrosis (uncommon)

- Present primarily in neurological or arterial TOS when there is sympathetic nerve stimulation.

thenar eminence muscle atrophy (uncommon)

- Hand muscle atrophy is seen in true neurological TOS.
- Consistent with median motor nerve conduction velocity abnormality.

exertional pain/claudication (uncommon)

- Feature of arterial and venous TOS.
- Symptoms of upper extremity pain can worsen with activity and improve with rest.

supraclavicular systolic bruit (uncommon)

- Can sometimes be auscultated on physical examination in arterial or venous TOS.
- May only be present with shoulder abduction.

blood pressure difference between extremities (uncommon)

- A blood pressure difference (e.g., of up to 20 mmHg) may be seen on physical examination between the thrombosed upper extremity and the contralateral normal upper extremity in arterial TOS.

positive Adson's (scalene) test (uncommon)

- Tightens the anterior and middle scalene muscles, thus decreasing the interspace and magnifying pre-existing compression of the subclavian artery and brachial plexus. The patient takes and holds a deep breath, extends the neck fully, and turns the head towards the side.[47]
  [Fig-7]
- Obliteration or decrease of the radial pulse in addition to production of symptoms and hand pallor suggests compression.

positive costoclavicular test (uncommon)

- Also known as the Halsted test or military brace.
- Shoulders are drawn downwards and backwards. This narrows the costoclavicular space by approximating the clavicle to the first rib and thus tends to compress the neurovascular bundle.[48] [Fig-8]
- Obliteration of the radial pulse with production of symptoms indicate compression.

positive hyperabduction test (uncommon)

- Hyperabducting the arm to 180° pulls the components of the neurovascular bundle around the pectoralis minor tendon, the coracoid process, and the head of the humerus.[48] [Fig-9]
- If the radial pulse is decreased, compression should be suspected.

positive Roos' test (uncommon)

- Both arms are placed at right angles to the shoulder, and the forearms are at right angles to the upper arms. Both hands are opened and closed as fast as possible to see if symptoms occur.[49]
- Test is given on physical examination to determine what symptoms the patient is experiencing with reference to their thoracic outlet (e.g., arms start to hurt, hands become numb, hands change colour).

positive stretch test (uncommon)

- The arm is abducted 90° with elbow extension, palm facing forwards, and thumb pointing up to the ceiling. The patient then laterally flexes the head to the opposite side. This stretches the plexus.
- A positive test is an uncomfortable pulling sensation of inner arm, sometimes into forearm or even into hand, on abduction. The patient may develop paraesthesias or heaviness of the extremity. A positive test of lateral flex is exacerbation of the symptoms.

positive upper limb tension tests (uncommon)

- A series of tests of all tissues in the upper limb with a preferential focus on the median nerve and its associated plexus and roots.[50] They involve shoulder abduction, wrist suspension and extension, shoulder lateral rotation, elbow extension, and neck lateral flexion away from or towards test side.
- Provides physical evidence of stretch of median, radial, and ulnar nerves. Allows side-to-side comparison and response compared with normal extremity movement.

positive Wright's manoeuvre (uncommon)

- The arm is externally rotated and abducted to 180° while the elbow is flexed (simulating a lazy hand raise).
- Can recreate patient’s paraesthesia symptoms in neurological TOS.

oedema/swelling of upper extremity (uncommon)

- Can be seen acutely in venous TOS.
- May be sudden onset of upper extremity swelling (and concurrent pain) following sudden increase in causative activity.

Diagnostic tests

1st test to order

Test	Result
cervical spine x-ray • Obtained in all suspected types of TOS. • Evaluates for bony abnormalities of the neck and shoulder girdle, which are present in about 30% of TOS patients.[51]	**bony abnormalities include: cervical rib, articulated or bifid first rib, fusion of first and second ribs, clavicular deformities, or previous thoracoplasties**
CXR • Obtained in all suspected types of TOS. • Evaluates for bony abnormalities of the neck and shoulder girdle, which are present in about 30% of TOS patients.[51]	**bony abnormalities include: cervical rib, articulated or bifid first rib, fusion of first and second ribs, clavicular deformities, or previous thoracoplasties**
EMG/nerve conduction velocity • Performed in cases of suspected true neurological TOS only to rule out alternative aetiologies (e.g., carpal tunnel syndrome). [Fig-10] • EMG measures muscle electrical activity at rest and during contraction. • Nerve conduction velocity measures the ability of nerves to transmit electrical impulses/signals.[5]	**reduced motor amplitude of the median nerve and reduced sensory amplitudes of the medial antebrachial cutaneous and ulnar nerves (true neurological TOS); normal in other types of TOS unless patient has concurrent condition**
CT angiography • Recommended if arterial TOS is suspected. • Intravenous contrast is injected into a contralateral vein and timed with concurrent spiral CT scan to obtain angiogram. • Evaluates for presence and location of arterial thrombus. • Provides anatomical detail regarding adjacent osseous structures that may be contributing to thrombus. Best performed both in neutral position and with shoulder abduction.[52] • Limitations include: ionising radiation, limited evaluation of soft tissue (i.e., brachial plexus) if concurrent neurological TOS is suspected, patient position (supine).[17]	**arterial thrombus, stenosis, or compression of the subclavian or axillary artery of the affected upper extremity; can also show aneurysm formation**
Doppler ultrasonography • Recommended if venous TOS is suspected. • Evaluates blood as it flows through vessels and can identify venous thromboses. Best performed both in neutral position and with shoulder abduction.[52] • Also used to confirm thrombosis resolution after treatment. • Limitations include: false-negative rate up to 30%, operator-dependent examination.	**venous thrombus, stenosis, or compression of the subclavian or axillary vein of the affected upper extremity; will show venous patency after treatment**

Other tests to consider

Test	Result
MRI neck/clavicle/shoulder • Considered in neurological TOS to help provide anatomical detail for diagnosing brachial plexus compression sites and preoperative surgical planning. • Non-contrast MRI is typically sufficient, unless a vascular component is suspected.[52] Images are obtained with the patient in both neutral and shoulder abduction positions to best localise the area of impingement.	**soft-tissue abnormalities: congenital bands, relative muscle hypertrophy (e.g., well-developed scalenus minimus muscle)**
muscle block • Recommended in patients with neurological TOS (disputed and pectorialis minor syndrome types). • Can help confirm diagnosis and determine those who may benefit from surgical intervention. • Scalenus anticus muscle block can be performed in disputed TOS by injecting local anaesthetic into the muscle belly.[6] • Pectoralis minor muscle block can be performed in pectoralis minor syndrome by also injecting local anaesthetic into the muscle.[12]	**transient subjective symptom relief; may also have negative provocative manoeuvres on repeat physical examination during symptom relief window**
conventional arteriography • Arterial catheter-directed injection of contrast under fluoroscopy. • Has largely been replaced by less invasive and costly modalities for diagnosis. • Used during catheter-directed thrombolysis procedure for treatment.	**arterial thrombus, stenosis, or compression of subclavian or axillary artery; may also show aneurysm formation, or arterial patency after thrombolysis procedure is successfully performed**
magnetic resonance angiography (MRA) • Lack of strong evidence currently in the use of MRA in diagnosing arterial TOS. • May be considered in cases of concurrent neurological TOS when soft-tissue anatomical detail is needed.	**arterial thrombus, stenosis, or compression of subclavian or axillary artery; may also show aneurysm formation; concurrent soft-tissue abnormalities may be visualised including congenital bands or relative muscle hypertrophy (e.g., well-developed scalenus minimus muscle)**
contrast venography • Diagnostic standard for detecting venous TOS. • Definitive test for diagnosing venous TOS if clinical suspicion remains high after negative Doppler ultrasonography testing. • Venous catheter-directed injection of contrast under fluoroscopy. • Limitations include: cost, invasive procedure, radiation exposure.	**venous thrombus, stenosis, or compression of the subclavian or axillary vein**

Test	Result
magnetic resonance venography (MRV) • Lack of strong evidence currently in the use of MRA in diagnosing venous TOS. • May be considered in cases of concurrent neurological TOS when soft-tissue anatomical detail is needed.	**venous thrombus, stenosis, or compression of the subclavian or axillary vein; concurrent soft-tissue abnormalities may be visualised including congenital bands, relative muscle hypertrophy (e.g., well-developed scalenus minimus muscle)**
FBC and coagulation studies • Consider in patients with venous TOS. • Can help to evaluate for a hypercoagulable state as an aetiology for the venous thrombus if alternative aetiologies are not present (e.g., effort thrombosis, implanted devices, congenital abnormalities).	**may suggest an underlying thrombophilia or occult malignancy**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Vasospasm, embolism, or insufficiency	• Severe chest pain.	• Angiogram showing narrowing or occlusion of affected vessel.
Coronary artery disease	• Chest pain/pressure.	• Coronary angiogram showing narrowing or occlusion of affected vessel.
Myocardial infarction	• Chest pain/pressure with possible radiation to jaw/shoulder/arm, dyspnoea, pallor, diaphoresis, and cardiogenic shock.	• ECG in ST-elevation MI: ST-segment elevation >1 mm in 2 or more anatomically contiguous leads or new left bundle branch block; ECG in non-ST-elevation MI: non-specific changes; ST-segment depression or T-wave inversion. • Troponins: elevated.
Angina pectoris	• Chest pain/pressure brought on by physical exertion and relieved by rest or at rest in a patient with a history of coronary artery disease/angina.	• ECG showing non-specific changes; ST-segment depression or T-wave inversion. • Troponins: normal.
Polymyalgia rheumatica	• Stiffness and pain in neck, shoulder, back, hips, thighs. • Affects adults >50 years.	• Elevated serum inflammatory markers (i.e., C-reactive protein, sedimentation rate).

Condition	Differentiating signs / symptoms	Differentiating tests
Buerger's disease	• Tobacco smoker. • Pain in the forearms/hands with activity. • Can have signs of ischaemia/gangrene of the fingers.	• Diagnosis is clinical.
Henoch-Schonlein purpura	• Joint pain with a rash and findings of kidney damage. • Typically occurs in children.	• Haematuria with or without kidney damage seen on basic metabolic panel.
Primary Raynaud's phenomenon	• Numbness/pain and coolness of fingers when exposed to cold temperatures/stress with associated colour changes of the fingers (pale/white or blue/purple during episode; red when episode is resolving).	• Diagnosis is clinical.
Carpal tunnel syndrome	• Pain and numbness primarily in the wrists; night-time worsening of symptoms. • Can reproduce pain with physical examination manoeuvres (i.e., positive Tinel's sign and Phalen's sign).	• Electrodiagnostic testing showing focal slowing of conduction velocity in the median sensory nerves across the carpal tunnel.
Cubital tunnel syndrome	• Pain and numbness primarily in the elbow through the ring and little fingers.	• Electrodiagnostic testing showing motor conduction velocity across the elbow is <50 m/second.
Brachial plexitis	• Also known as Parsonage-Turner syndrome, brachial plexopathy, and brachial neuropathy/neuritis. • Self-resolving, sudden onset, primarily unilateral piercing/sharp pain in shoulder and/or upper extremity lasting less than 7 to 10 days, followed by usually self-resolving upper extremity weakness. • May have a history of previous infection, injury, or surgery.	• Diagnosis is often clinical. • Electrodiagnostic testing shows abnormalities of the affected nerves.
Herniated or ruptured cervical intervertebral disc	• Pain radiating down the upper extremity.	• MRI cervical spine showing herniation and/or rupture of cervical intervertebral disc.

Condition	Differentiating signs / symptoms	Differentiating tests
Cervical stenosis and/or degenerative joint disease	• Stiffness and pain in upper neck. Possible radicular symptoms.	• Cervical spine x-ray showing cervical degenerative joint disease (i.e., intervertebral disc space narrowing, vertebral osteophytes, and facet hypertrophy).
Multiple sclerosis	• Most often relapsing-remitting variable symptoms that can include: numbness or weakness in extremities, radicular symptoms with neck flexion, fatigue, vision changes, tremor, unsteady gait, and/or bowel/bladder dysfunction.	• MRI brain and/or spine shows characteristic lesions.
Rotator cuff injury	• Pain in shoulder with possible arm weakness. • Provocative physical examination manoeuvres.	• MRI shoulder showing partial or complete tearing of one or more tendons of the rotator cuff (most commonly supraspinatus).
Adhesive capsulitis	• Pain in shoulder with decreased range of motion in shoulder joint.	• Diagnosis is clinical.
Shoulder impingement	• Pain in shoulder with certain movements and provocative physical examination manoeuvres (i.e., Neer's test and Hawkins' test).	• Diagnosis is clinical.
Glenohumeral/acromonial clavicular degenerative joint disease	• Pain in shoulder with movement and tenderness to palpation over joint spaces.	• X-rays show evidence of degenerative joint disease. • Evidence of degenerative joint disease on musculoskeletal ultrasonography of the acromioclavicular joint.
Superior/pulmonary sulcus tumour (Pancoast's tumour)	• Severe scapular, shoulder, and/or upper extremity pain.[54] • May also develop hand muscle weakness and/or atrophy. • Horner's syndrome can develop if invasion into the sympathetic ganglion occurs (e.g., ptosis, miosis, anhidrosis).	• CT chest or MRI neck/shoulder (if images include pulmonary sulcus) demonstrate a tumour.[55]

Condition	Differentiating signs / symptoms	Differentiating tests
Trigger points	• Pain in neck, shoulder, or upper extremity. • Pain reproduced with palpation of specific trigger points.	• Diagnosis is clinical.
Fibromyalgia	• Pain in neck, shoulder, upper extremity. Will also have pain in other locations of the body.	• Diagnosis is clinical.
Complex regional pain syndrome (CRPS)	• Chronic burning or throbbing pain in the upper (or lower) extremity out of proportion to what is expected following an injury or illness (type 1) or surgery (type 2; causalgia). • Can also have allodynia, oedema over the area of pain, changes in skin colour (e.g., whitening, mottling, blueness or redness), decreased range of motion, and/or weakness.	• 3-phase nuclear bone scan may show increased uptake/ activity on delayed phase/ pooling images (diagnostic utility is debated). • Radiographs may show evidence of previous injury, surgery, degenerative changes, and/or osteopenia.

Step-by-step treatment approach

For patients with neurological TOS, treatment is primarily conservative and focuses on physiotherapy; surgical intervention is reserved for patients who display motor deficits and muscle atrophy. For patients with venous or arterial TOS, prompt thrombolysis and surgical intervention is often required.

Neurological TOS (NTOS)

Treatment is primarily conservative and focuses on physiotherapy; surgical intervention is reserved for patients who display motor deficits and muscle atrophy (i.e., true NTOS). The preferred surgical approach depends on the type of NTOS and the pathological process leading to nerve compression.

True NTOS

- Patients present with objective signs of nerve compression: specifically, motor deficits such as weakness and atrophy of the involved muscle groups. These patients will also present with sensory complaints similar to patients with other types of TOS.
- The cause of compression in the vast majority of these cases will involve a bony structure, such as a cervical rib.
- Given the advanced nature of the neurological deficit, surgical intervention is the preferred approach, as many of these patients will not respond to conservative management. A supraclavicular or transaxillary approach can be used in these patients.

Disputed NTOS

- These patients will present with sensory symptoms and no objective evidence of nerve compression on examination or diagnostic studies such as electrodiagnostic testing.
- Most patients are initially treated conservatively with physiotherapy.[56] [57] [58] Core strengthening therapies help to improve posture and realign musculoskeletal structures. Physiotherapy is used to open up the space between the clavicle and first rib, improve posture, strengthen the shoulder girdle, and loosen the neck muscles.[1] This is accomplished by pectoralis stretching, strengthening the muscles between the shoulder blades, good posture advice, and active neck exercises (including chin tuck, flexion, rotation, lateral bending, circumduction).[58] Ergonomics are also a key factor in rehabilitation and the ability of the patient to return to work.
- Other conservative therapies include rest, appropriate work restrictions, and pharmacotherapy for pain relief. Pharmacotherapy should be limited to oral analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants. Local anaesthetic injections (e.g., lidocaine) are largely used for aiding diagnosis, but do provide temporary pain control. Botulinum toxin injections into the suspected abnormal muscle (e.g., scalene) have been used; however, this treatment has not shown long-term efficacy.[22]
- The usual indications for surgery are ineffective appropriate conservative therapy in a patient with a significantly reduced nerve conduction velocity (<50 m/second) and eliminating other possible aetiologies for the symptoms.[1] [59] Surgery may also be suitable in patients with a positive response to anterior scalene muscle anaesthetic injection and relief of symptoms by injecting into the pectoralis minor.

Surgery

- The commonly performed surgical procedures for the treatment of NTOS typically include at least one of the following components:

- Removal or release of anomalous structures (e.g., cervical rib)
- Neurolysis of the supraclavicular brachial plexus
- First rib resection (if it is found to be a compressive structure on the plexus)
- Removal or sectioning of the anterior and middle scalene muscles
- Possible release of the pectoralis minor muscle.

- The most commonly used surgical approaches are the supraclavicular and transaxillary approaches. The approach used is generally based on the surgeon's preference.
- The advantage of the supraclavicular approach is the relative ease of access to the scalene muscles and clear visualisation of the neural and vascular anatomy. Disadvantages include the need to manipulate the plexus and vessels, as well as potential injury to the phrenic nerve. [Fig-11]
- The main advantage of the transaxillary approach rests on the fact that the brachial plexus and major vessels do not need to be retracted. A disadvantage is that the intercostobrachial nerve is at risk for injury. [Fig-12]
- Pectoralis minor tenotomy can be considered in cases of NTOS where the area of nerve compression is felt to be in the subcoracoid space. It is also felt by some to be especially important to consider in those patients experiencing recurrence of symptoms after initial surgical intervention. This procedure can be performed by making a small vertical incision in the deltopectoral groove, dissecting the pectoralis major muscle to expose the pectoralis minor muscle and coracoid process, with subsequent separation of the pectoralis minor tendon from its adjacent short head of the biceps tendon and sectioning of the pectoralis minor tendon.[60] [61]
- Surgical complications in general include injury to the neural structures (i.e., plexus, intercostal nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.

Postoperative rehabilitation and pain control

- Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
- Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics.

Arterial TOS (ATOS)

Immediate surgical intervention is required if there is concern about ischaemia. A transaxillary approach is most often used in these cases. A supraclavicular or infraclavicular approach can sometimes be employed if bypass is necessary. A bypass is recommended if embolectomy cannot be achieved or an aneurysm is present.[17] If thrombosis is present, catheter-directed thrombolysis is recommended.

Surgical decompression of the thoracic outlet determined by area of compression (e.g., arterial repair if possible, first and/or cervical rib resection, congenital band lysis, accessory muscle resection, and pectoralis minor muscle resection) is also recommended. A patient with cervical or first rib arterial compression producing poststenotic dilation of the axillary-subclavian artery should undergo rib resection, preferably using the transaxillary approach, removing the ribs, both first and cervical, without resecting the artery. The dilation usually returns towards normal after removal of compression. Patients with compression from the first or cervical rib, producing aneurysm with or without thrombus, should undergo rib resection and aneurysm excision with graft using the supraclavicular and infraclavicular combined

approach. Thrombosis of the axillary-subclavian artery, or distal emboli secondary to TOS compression, should be treated with first rib resection, thrombectomy, embolectomy, arterial repair or replacement, and dorsal sympathectomy.[41]

Postoperative evaluation of arterial patency is essential. Perfusion of the limb and pulses should be routinely tested clinically. In cases where the clinical assessment is in question, assessment of arterial patency with CT angiography or arteriography may be necessary.

Postoperative rehabilitation and pain control is important. Selection of the specific analgesic is according to the treating physician's preference. Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. Postoperative anticoagulants are necessary if residual ischaemia is seen on postoperative evaluation or if the patient is deemed to have a hypercoagulable state. Anticoagulation options include heparin, enoxaparin, and/or warfarin, as novel oral anticoagulants (NOACs) have not been studied in this patient population to date.

Venous TOS (vTOS)

Immediate intervention is often required. If thrombosis is present, catheter-directed thrombolysis is recommended prior to surgical intervention. The subsequent approach to surgical intervention depends on the surgeon's preference. A transaxillary or supraclavicular approach is common; however, a transaxillary approach is often preferred, and an infraclavicular approach is also an option in these cases.[62] Surgical decompression of the thoracic outlet determined by the area of compression (e.g., first rib resection, anterior scalenectomy, and venoplasty or open venous reconstruction) is also recommended.

The ideal management of thrombosis of the axillary-subclavian vein (Paget-Schroetter syndrome) is thrombolysis; after clot lysis, the first rib is promptly resected with removal of compressive elements.[30] Through an antecubital indwelling venous catheter, venography is performed and thrombolytic therapy is initiated. The thrombolytic agent chosen and prescribed is determined by the treating physician. After clot lysis, the first rib is promptly resected with resection of the scalenus anticus muscle and removal of any other compressive element in the thoracic outlet, such as the costoclavicular ligament, cervical rib, or abnormal bands.[63] [64]

Intermittent or partial obstruction should be treated by first rib removal through the transaxillary approach, with resection of the costoclavicular ligament medially, the first rib inferiorly, and the scalenus anticus muscle laterally. The clavicle is left in place. The vein is decompressed, and all the bands and adhesions are removed. The transaxillary approach provides excellent visualisation of the vein and costoclavicular ligament. Also, the neurovascular structures are away from the dissection and do not have to be retracted, thus minimising their injury. In addition, first rib resection shortens the postoperative disability.[65]

The availability of thrombolytic agents, combined with prompt surgical decompression of the neurovascular compressive elements in the thoracic outlet, has reduced morbidity and the need for thrombectomy. It has also substantially improved clinical results, including the ability to return to work.[66] [67] [68] [69] Attempts to open the occluded vein mechanically with the use of laser or bypass grafts have uniformly been unsatisfactory.[4]

Postoperative rehabilitation and pain control is important. Selection of the specific analgesic is according to the treating physician's preference. Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. Postoperative anticoagulants are only necessary if residual thrombus is seen on postoperative evaluation or if the patient is deemed to have a

hypercoagulable state. Anticoagulation options include heparin, enoxaparin, and/or warfarin, as novel oral anticoagulants (NOACs) have not been studied in this patient population to date.

Often, patients who are not deemed to be surgical candidates (i.e., patients with significant medical comorbidities that would not tolerate extensive surgical intervention or prolonged anaesthesia) can still undergo catheter-directed thrombolysis to remove the venous obstruction. However, thrombolysis should not be considered in patients with ongoing contraindications to thrombolytic therapy (e.g., active bleeding). Additionally, cases in which thrombosis has been present for more than two weeks are typically associated with poorer outcomes. Post-thrombolysis, patients are treated with anticoagulation and arm elevation to help promote venous return to the heart and prevent venous pooling.[16]

Neurovascular (combined) TOS

Immediate surgical intervention is required for both the vascular and neurological components of combined TOS. Techniques include catheter-directed thrombolysis and surgical decompression of the thoracic outlet. The method selected depends on the pathology and type of thoracic outlet syndrome as detailed above, as well as surgeon preference.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Acute **(summary)**

Patient group	Tx line	Treatment
true NTOS	1st	surgery
true NTOS	plus	postoperative rehabilitation and pain control
disputed NTOS	1st	conservative management
disputed NTOS	adjunct	pharmacotherapy
disputed NTOS	2nd	surgery
disputed NTOS	plus	postoperative rehabilitation and pain control
arterial TOS (ATOS)	1st	surgery
	adjunct	catheter-directed thrombolysis
	plus	postoperative rehabilitation
	adjunct	anticoagulation
surgical candidate	1st	catheter-directed thrombolysis
surgical candidate	plus	surgery

Acute			(summary)
▪	surgical candidate	plus	postoperative rehabilitation
▪	surgical candidate	adjunct	anticoagulation
▪	non-surgical candidate	1st	catheter-directed thrombolysis
▪	non-surgical candidate	plus	postoperative rehabilitation
▪	non-surgical candidate	plus	arm elevation
▪	non-surgical candidate	adjunct	anticoagulation
neurovascular (combined)		1st	surgery
		plus	postoperative rehabilitation
		adjunct	anticoagulation

Treatment options

Acute

Patient group	Tx line	Treatment
true NTOS	**1st**	**surgery**

» Presents with objective signs of nerve compression: specifically, motor deficits such as weakness and atrophy of the involved muscle groups.

» Surgery is the preferred approach as many patients will not respond to conservative management.

» Surgical decompression of the thoracic outlet (e.g., removal of cervical rib, neurolysis of supraclavicular brachial plexus, first rib resection, removal or sectioning of the anterior and middle scalene muscles, release of the pectoralis minor muscle) is recommended.

» A supraclavicular or transaxillary approach can be used and choice depends on surgeon preference, although the supraclavicular approach is the most commonly used.

» The advantage of the supraclavicular approach is the relative ease of access to the scalene muscles and clear visualisation of the neural and vascular anatomy. Disadvantages include the need to manipulate the plexus and vessels, as well as potential injury to the phrenic nerve.

» The main advantage of the transaxillary approach rests on the fact that the brachial plexus and major vessels do not need to be retracted. A disadvantage is that the intercostobrachial nerve is at risk for injury.

» Pectoralis minor tenotomy can be considered in cases of NTOS where the area of nerve compression is felt to be in the subcoracoid space. This procedure can be performed by making a small vertical incision in the deltopectoral groove, dissecting the pectoralis major muscle to expose the pectoralis minor muscle and coracoid process, with subsequent separation of the pectoralis minor tendon from its adjacent short head of the biceps tendon and sectioning of the pectoralis minor tendon.[60] [61]

» Complications in general include injury to the neural structures (i.e., plexus, intercostal

Acute

Patient group	Tx line	Treatment
		nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.
true NTOS	plus	**postoperative rehabilitation and pain control** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
disputed NTOS	1st	**conservative management** » Presents with sensory symptoms and no objective evidence of nerve compression on examination or diagnostic studies such as electrodiagnostic testing. » Initially treated conservatively with rest, work restrictions, and physiotherapy.[56] [57] [58] » Core strengthening therapies help to improve posture and realign musculoskeletal structures. » Physiotherapy is used to open up the space between the clavicle and first rib, improve posture, strengthen the shoulder girdle, and loosen the neck muscles.[1] This is accomplished by pectoralis stretching, strengthening the muscles between the shoulder blades, good posture advice, and active neck exercises (including chin tuck, flexion, rotation, lateral bending, circumduction).[58] » Ergonomics are also a key factor in rehabilitation and the ability of the patient to return to work.
disputed NTOS	adjunct	**pharmacotherapy** » Pharmacotherapy should be limited to oral analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants (e.g., cyclobenzaprine). » Local anaesthetic injections (e.g., lidocaine) are largely used for aiding diagnosis, but do provide temporary pain control.

Acute

Patient group	Tx line	Treatment
		» Botulinum toxin injections into the suspected abnormal muscle (e.g., scalene) have been used; however, this treatment has not shown long-term efficacy.[22]
		Primary options
		» ibuprofen: 400 mg orally every 4-6 hours when required, maximum 3200 mg/day
		OR
		Primary options
		» cyclobenzaprine: 5-10 mg orally (immediate-release) three times daily when required
disputed NTOS	**2nd**	**surgery**
		» Usual indications for surgery are ineffective appropriate conservative therapy in a patient with a significantly reduced nerve conduction velocity (<50 m/second) and eliminating other possible aetiologies for the symptoms.[1] [59] Surgery may also be suitable in patients with a positive response to anterior scalene muscle anaesthetic injection and relief of symptoms by injecting into the pectoralis minor.
		» Surgical decompression of the thoracic outlet (e.g., removal of cervical rib, neurolysis of supraclavicular brachial plexus, first rib resection, removal or sectioning of the anterior and middle scalene muscles, release of the pectoralis minor muscle) is recommended.
		» The most commonly used surgical approaches are the supraclavicular and transaxillary approaches. The approach used is generally based on the surgeon's preference. The advantage of the supraclavicular approach is the relative ease of access to the scalene muscles and clear visualisation of the neural and vascular anatomy. Disadvantages include the need to manipulate the plexus and vessels, as well as potential injury to the phrenic nerve.
		» Pectoralis minor tenotomy can be considered in cases of NTOS where the area of nerve compression is felt to be in the subcoracoid space. This procedure can be performed by making a small vertical incision in the deltopectoral groove, dissecting the pectoralis major muscle to expose the pectoralis minor muscle and coracoid process, with subsequent separation of the pectoralis minor tendon from

Acute

Patient group	Tx line	Treatment
		its adjacent short head of the biceps tendon and sectioning of the pectoralis minor tendon.[60] [61] » Complications in general include injury to the neural structures (i.e., plexus, intercostal nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.
disputed NTOS	**plus**	**postoperative rehabilitation and pain control** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
arterial TOS (ATOS)	**1st**	**surgery** » Immediate surgical intervention is required if there is concern for ischaemia. » A transaxillary approach is often recommended. A supraclavicular or infraclavicular approach can sometimes be employed if bypass is necessary. A bypass is recommended if embolectomy cannot be achieved or an aneurysm is present.[17] » Surgical decompression of the thoracic outlet determined by area of compression (e.g., arterial repair if possible, first and/or cervical rib resection, congenital band lysis, accessory muscle resection, and pectoralis minor muscle resection) is recommended. A patient with cervical or first rib arterial compression producing poststenotic dilation of the axillary-subclavian artery should undergo rib resection, preferably using the transaxillary approach, removing the ribs, both first and cervical, without resecting the artery. The dilation usually returns towards normal after removal of compression. Patients with compression from the first or cervical rib, producing aneurysm with or without thrombus, should undergo rib resection and aneurysm excision with graft using the supraclavicular and infraclavicular

Acute

Patient group	Tx line	Treatment
		combined approach. Thrombosis of the axillary-subclavian artery, or distal emboli secondary to TOS compression, should be treated with first rib resection, thrombectomy, embolectomy, arterial repair or replacement, and dorsal sympathectomy.[41] » Postoperative evaluation of arterial patency is essential. Perfusion of the limb and pulses should be routinely tested clinically. In cases where the clinical assessment is in question, assessment of arterial patency with CT angiography or arteriography may be necessary. » Complications in general include injury to the neural structures (i.e., plexus, intercostal nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.
	adjunct	**catheter-directed thrombolysis** » If thrombosis is present, catheter-directed thrombolysis is recommended.
	plus	**postoperative rehabilitation** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
	adjunct	**anticoagulation** » Postoperative anticoagulants are necessary if residual ischaemia is seen on postoperative evaluation or if the patient is deemed to have a hypercoagulable state. » Anticoagulation options include heparin, enoxaparin, and/or warfarin. Novel oral anticoagulants (NOACs) have not been studied in this patient population to date. **Primary options** » heparin: consult specialist for guidance on dose **-or-**

Acute

Patient group	Tx line	Treatment
		» enoxaparin: consult specialist for guidance on dose **--AND/OR--** » warfarin: consult specialist for guidance on dose
surgical candidate	1st	**catheter-directed thrombolysis** » Immediate intervention is often required. If thrombosis is present, catheter-directed thrombolysis is recommended prior to surgical intervention. » Through an antecubital indwelling venous catheter, venography is performed and thrombolytic therapy is initiated. The thrombolytic agent chosen and prescribed is determined by the treating physician. » After clot lysis, the first rib is promptly resected with resection of the scalenus anticus muscle and removal of any other compressive element in the thoracic outlet, such as the costoclavicular ligament, cervical rib, or abnormal bands.[63] [64] » The ideal management of thrombosis of the axillary-subclavian vein (Paget-Schroetter syndrome) is thrombolysis; after clot lysis, the first rib is promptly resected with removal of compressive elements.[30]
surgical candidate	plus	**surgery** » A transaxillary or supraclavicular approach is common; however, a transaxillary approach is often preferred, and an infraclavicular approach is also an option in these cases.[62] » Surgical decompression of the thoracic outlet determined by the area of compression (e.g., first rib resection, anterior scalenectomy, and venoplasty or open venous reconstruction) is recommended. » Intermittent or partial obstruction should be treated by first rib removal through the transaxillary approach, with resection of the costoclavicular ligament medially, the first rib inferiorly, and the scalenus anticus muscle laterally. The clavicle is left in place. The vein is decompressed, and all the bands and adhesions are removed.

Acute

Patient group	Tx line	Treatment
		» The transaxillary approach provides excellent visualisation of the vein and costoclavicular ligament. Also, the neurovascular structures are away from the dissection and do not have to be retracted, thus minimising their injury. In addition, first rib resection shortens the postoperative disability.[65] » Postoperative evaluation of arterial patency is essential. Perfusion of the limb and pulses should be routinely tested clinically. In cases where the clinical assessment is in question, assessment of arterial patency with CT angiography or arteriography may be necessary. » Complications in general include injury to the neural structures (i.e., plexus, intercostal nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.
surgical candidate	**plus**	**postoperative rehabilitation** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
surgical candidate	**adjunct**	**anticoagulation** » Postoperative anticoagulants are necessary if residual ischaemia is seen on postoperative evaluation or if the patient is deemed to have a hypercoagulable state. » Anticoagulation options include heparin, enoxaparin, and/or warfarin. Novel oral anticoagulants (NOACs) have not been studied in this patient population to date. **Primary options** » heparin: consult specialist for guidance on dose **-or-** » enoxaparin: consult specialist for guidance on dose **--AND/OR--**

Acute

Patient group	Tx line	Treatment
		» warfarin: consult specialist for guidance on dose
non-surgical candidate	1st	**catheter-directed thrombolysis** » Patients who are not deemed to be surgical candidates (i.e., patients with significant medical comorbidities that would not tolerate extensive surgical intervention or prolonged anaesthesia) can still undergo catheter-directed thrombolysis to remove the venous obstruction. However, thrombolysis should not be considered in patients with ongoing contraindications to thrombolytic therapy (e.g., active bleeding). Additionally, cases in which thrombosis has been present for more than two weeks are typically associated with poorer outcomes. » Through an antecubital indwelling venous catheter, venography is performed and thrombolytic therapy is initiated. The thrombolytic agent chosen and prescribed is determined by the treating physician.
non-surgical candidate	plus	**postoperative rehabilitation** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period.
non-surgical candidate	plus	**arm elevation** » Arm elevation is recommended to help promote venous return to the heart and help prevent venous pooling.
non-surgical candidate	adjunct	**anticoagulation** » Postoperative anticoagulants are necessary if residual ischaemia is seen on postoperative evaluation or if the patient is deemed to have a hypercoagulable state. » Anticoagulation options include weight-based dosing of heparin, enoxaparin, and/or warfarin. Novel oral anticoagulants (NOACs) have not been studied in this patient population to date.

Primary options

Acute

Patient group	Tx line	Treatment
		» heparin: consult specialist for guidance on dose **-or-** » enoxaparin: consult specialist for guidance on dose **--AND/OR--** » warfarin: consult specialist for guidance on dose
neurovascular (combined)	**1st**	**surgery** » Immediate surgical intervention is required for both the vascular and neurological components of combined TOS. » Techniques include catheter-directed thrombolysis and surgical decompression of the thoracic outlet. » The method selected depends on the pathology and type of thoracic outlet syndrome as detailed above, as well as surgeon preference.
	plus	**postoperative rehabilitation** » Physiotherapy is paramount, focusing on posture modifications, shoulder girdle strengthening, and ergonomics. » Pain control with oral and/or intravenous analgesics is appropriate in the immediate postoperative period, with the selection of the specific analgesic agent depending on physician preference. Oral analgesics often need to be continued after the immediate postoperative period. » Rest and work restrictions are also recommended.
	adjunct	**anticoagulation** » Postoperative anticoagulants are necessary if residual ischaemia is seen on postoperative evaluation or if the patient is deemed to have a hypercoagulable state. » Anticoagulation options include heparin, enoxaparin, and/or warfarin. Novel oral anticoagulants (NOACs) have not been studied in this patient population to date.

Primary options

Acute

Patient group	Tx line	Treatment
		» heparin: consult specialist for guidance on dose **-or-** » enoxaparin: consult specialist for guidance on dose **--AND/OR--** » warfarin: consult specialist for guidance on dose

Recommendations

Monitoring

Neurological TOS can recur after conservative treatment with physiotherapy and after operative treatment. Symptoms can recur from 1 month to 10 years after surgical intervention; however, in most instances, recurrence is within the first 3 months.[70] Patients should be advised to monitor for symptoms of recurrent compression of thoracic outlet neurovascular structures.

Patients who suffered from vascular TOS should be educated regarding signs and symptoms of recurrence, so that they seek immediate evaluation in the unexpected event of recurrence.

Patient instructions

Patients should be aware of the high risk of recurrence of neurological TOS and contact their physician if they have any symptoms: aching or burning pain often associated with paraesthesia, involving the neck, shoulder, parascapular area, anterior chest wall, arm, and hand.

Patients should also adhere to any exercises as directed by their physiotherapist to help prevent recurrence. Optimising and maintaining posture, avoiding repetitive working practices, losing weight, and correcting sleeping positions can reduce compression to the thoracic outlet and so also reduce the likelihood of recurrence.

Complications

Complications	Timeframe	Likelihood
post-sympathectomy neuralgia	**short term**	**low**
Pain typically appears in the shoulder and upper arm on the lateral aspect. Clinical history usually substantiates this if the symptoms occur within the first 3 months. Tests show increased sympathetic activity and suggest a rebound phenomenon from the non-sympathectomised adjacent dermatomes. Rebound may be a regeneration of nerve fibres or increased response of peripheral nerves to catecholamines. Symptoms can be resolved in 3 to 6 weeks with conservative management.[74]		
cardiovascular complications	**short term**	**low**
Delay in the diagnosis of arterial TOS can result in aneurysm formation, thrombosis, embolisation, ischaemia, and retrograde embolic CVA/stroke.[17] [18] [75] [76] Diagnosis is clinical (e.g., signs of local ischaemia on physical examination or signs of CVA/stroke). Management is through emergent revascularisation of the extremity. Delay in the diagnosis of venous TOS can also result in embolisation (incidence of pulmonary embolism can range between 10% to 20% of cases).[16] Diagnosis can be made clinically (e.g., shortness of breath, pleuritic chest pain), with abnormal vital signs (e.g., tachycardia, tachypnoea, hypoxaemia), possibly an abnormal ECG (e.g., SI QIII TIII pattern), and confirmatory imaging for pulmonary embolism (e.g., V/Q scan or CT angiography of the chest).		
permanent nerve damage	**variable**	**low**
Delay in the diagnosis of true NTOS can result in progressive and permanent nerve damage. Management is through surgical exploration of the thoracic outlet.		

FOLLOW UP

Complications	Timeframe	Likelihood
surgical complications	**variable**	**low**
Complications include injury to the neural structures (i.e., plexus, intercostal nerve, phrenic nerve), bleeding, infection, pneumothorax, and incomplete nerve release.		

Prognosis

Prognosis in neurological TOS is variable. Symptoms may persist despite aggressive physiotherapy and therefore require surgical intervention. Symptoms can recur from 1 month to 10 years after surgical intervention; however, in most instances, recurrence is within the first 3 months.[70] When symptoms persist after surgical intervention, it is important to consider pectoralis minor syndrome (PMS) as an undiagnosed entity that may require follow-up treatment (targeted physiotherapy and/or pectoralis minor tenotomy).[12]

Prognosis in vascular TOS is largely favourable after appropriate surgical interventions are performed.

Physiotherapy

All patients with symptoms of neurological compression should be involved in physiotherapy. This can be used in isolation as the sole treatment modality or preoperatively and/or postoperatively (depending on the urgency of surgical intervention). Many patients with neurological (disputed) TOS and PMS are successfully treated with physiotherapy alone, focusing on posture correction, stretching, and strengthening. For patients who require surgical intervention, postoperative physiotherapy is paramount, again focusing on posture correction, stretching, and strengthening. If postoperative symptoms persist despite physiotherapy, re-operation may be indicated.

Re-operation

Two distinct groups of patients require re-operation.

- Pseudorecurrences happen in patients who never had relief of symptoms after the initial operation. Cases can be separated aetiologically as follows: 1) the second rib was mistakenly resected instead of the first, 2) the first rib was resected leaving a cervical rib, 3) a cervical rib was resected leaving an abnormal first rib, or 4) a second rib was resected leaving a rudimentary first rib.[71]
- The second group includes patients whose symptoms were relieved after the initial operation but who developed recurrence because a significant piece of the first rib was left in place at the initial operation, and a small subgroup whose first rib was completely resected but developed excessive scar formation involving the brachial plexus.[72]

Re-operation for recurrent TOS is preferably performed through the posterior thoracoplasty approach to provide better exposure of the nerve roots and brachial plexus, thereby reducing the danger of injury to these structures, as well as providing adequate exposure of the subclavian artery and vein.[73] This approach also provides a wider field for easy resection of any bony abnormalities or fibrous bands, and allows extensive neurolysis of the nerve roots and brachial plexus, which are not always accessible through the limited exposure of the transaxillary approach. The anterior or supraclavicular approach is inadequate for re-operation.

Some physicians treating neurological TOS suspect that recurrence of symptoms or absence of resolution of symptoms after initial surgery may be due to an undiagnosed underlying PMS. In cases of neurological TOS, it is important to consider whether PMS may be contributing and determine whether pectoralis minor tenotomy would be an appropriate surgical intervention.[22] [60] [61]

Diagnostic guidelines

North America

ACR appropriateness criteria: imaging in the diagnosis of thoracic outlet syndrome

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Summary: Imaging serves to localise the compression site, the compressing structure, and the compressed organ or vessel.

Key articles

- Wilbourn AJ. Thoracic outlet syndromes. Neurol Clin. 1999;17:477-497. Abstract
- Ferrante MA. The thoracic outlet syndromes. Muscle Nerve. 2012;45:780-795. Abstract
- Sanders RJ, Rao NM. The forgotten pectoralis minor syndrome: 100 operations for pectoralis minor syndrome alone or accompanied by neurogenic thoracic outlet syndrome. Ann Vasc Surg. 2010;24:701-708. Abstract
- Illig KA, Doyle AJ. A comprehensive review of Paget-Schroetter syndrome. J Vasc Surg. 2010;51:1538-1547. Full text Abstract
- Durham JR, Yao JS, Pearce WH, et al. Arterial injuries in the thoracic outlet syndrome. J Vasc Surg. 1995;21:57-70. Full text Abstract
- Gil HY, Yun MJ, Kim JE, et al. Brachial plexus injury following median sternotomy. Korean J Anesthesiol. 2012;63:286-287. Full text Abstract

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Images

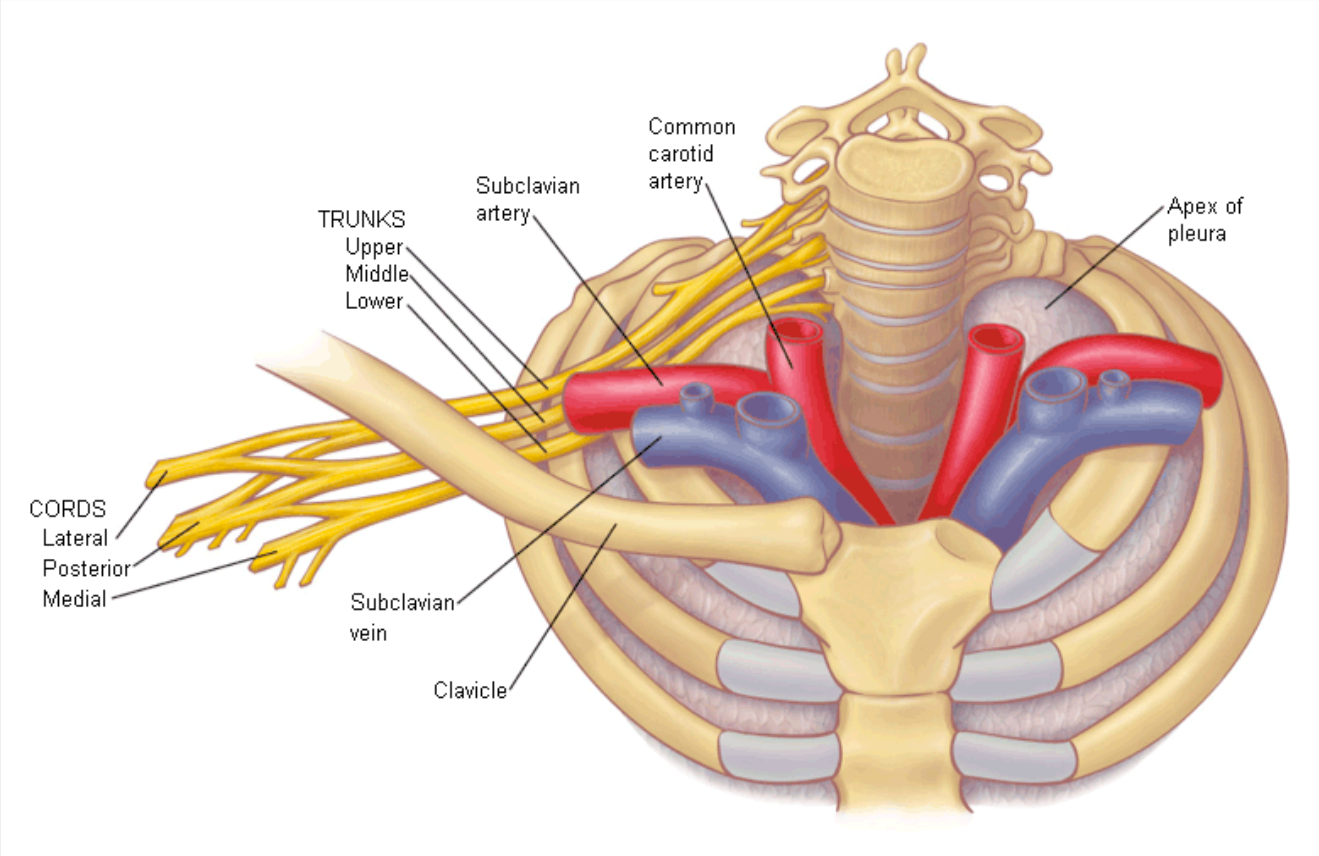

Figure 1: Subclavian vein and artery pass over first rib and under clavicle. Brachial plexus traverses top of bony circle to join the artery. Apex of the pleura (cupula) shown on left side

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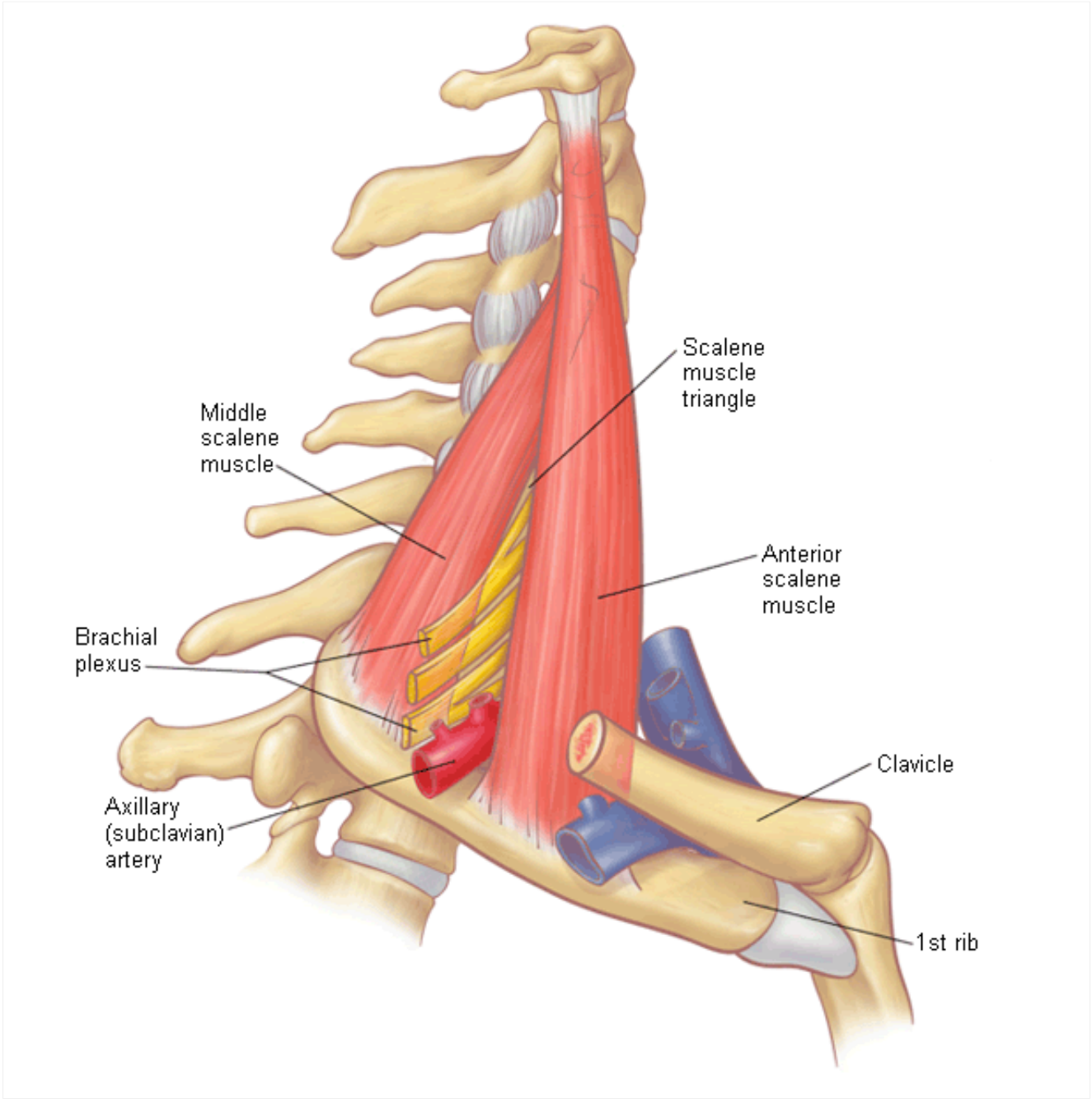

Figure 2: Scalene muscle triangle is the second major level of compression

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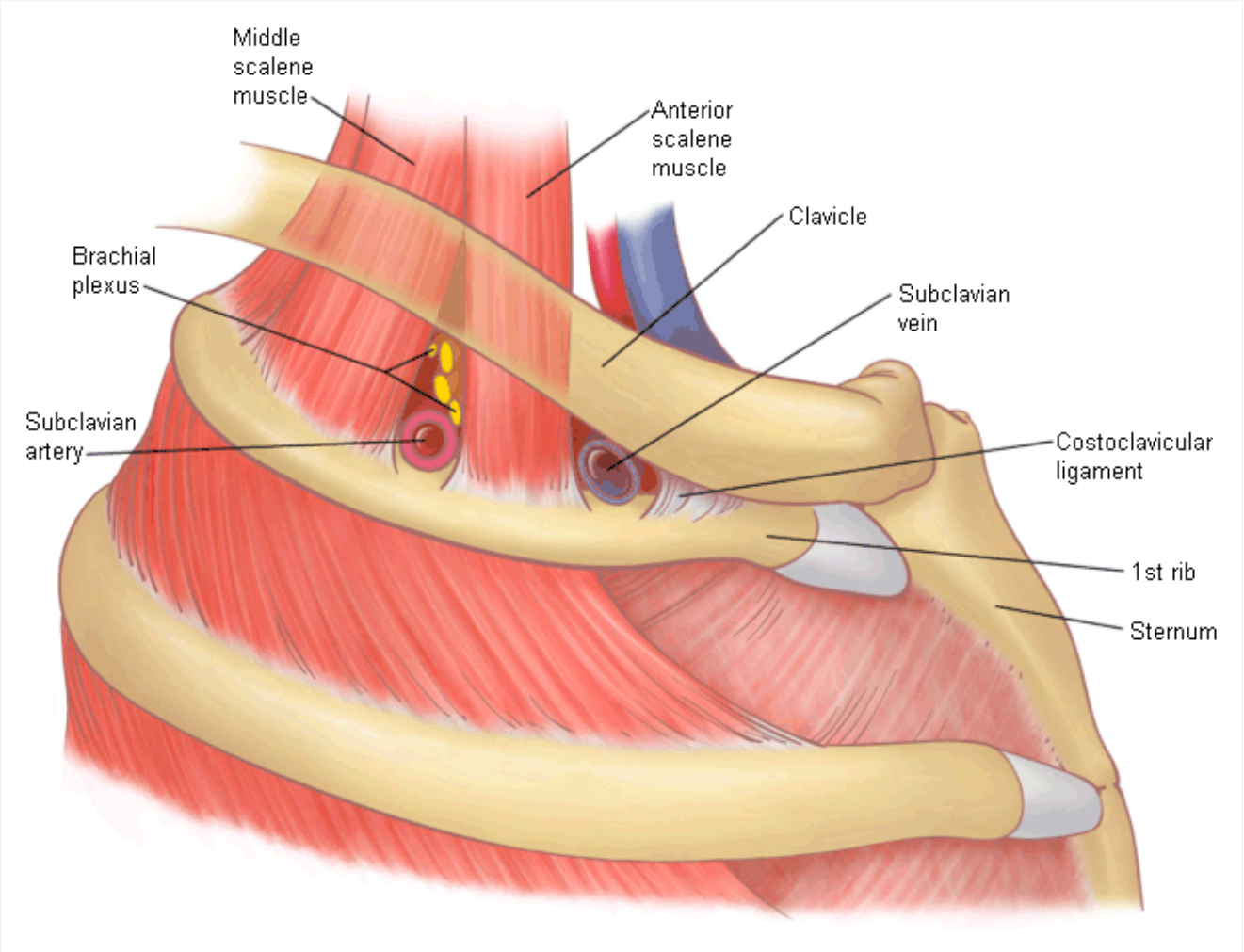

Figure 3: Cross-section of neurovascular structures traversing the thoracic outlet with clavicle above and first rib below

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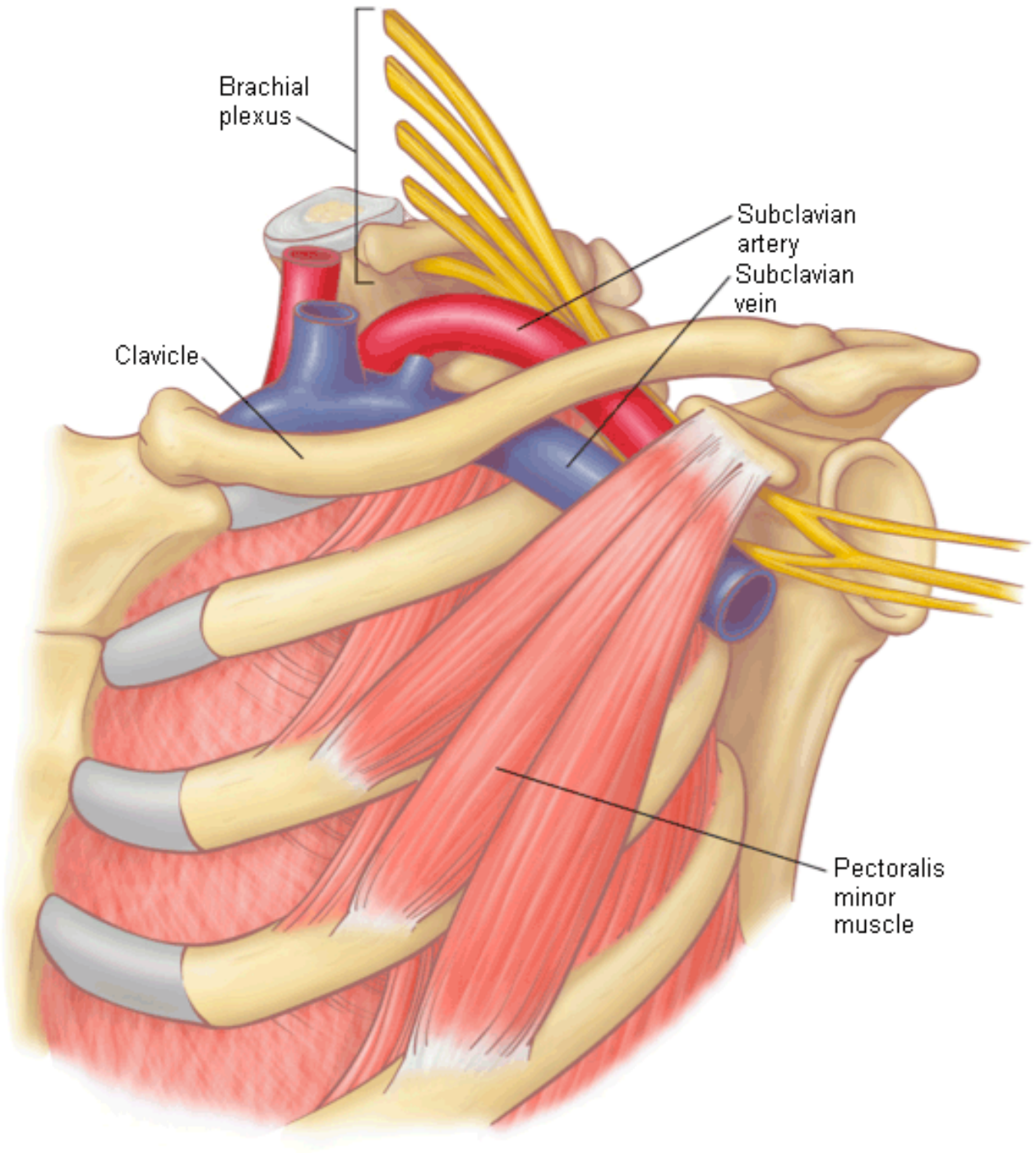

Figure 4: Neurovascular structures pass behind pectoralis minor muscle, another major area of compression. Pectoralis minor is a shoulder protractor, which can overpower the rhomboids. The shoulder retracts and alters the thoracic outlet, contributing to muscular imbalance and compression of the brachial plexus

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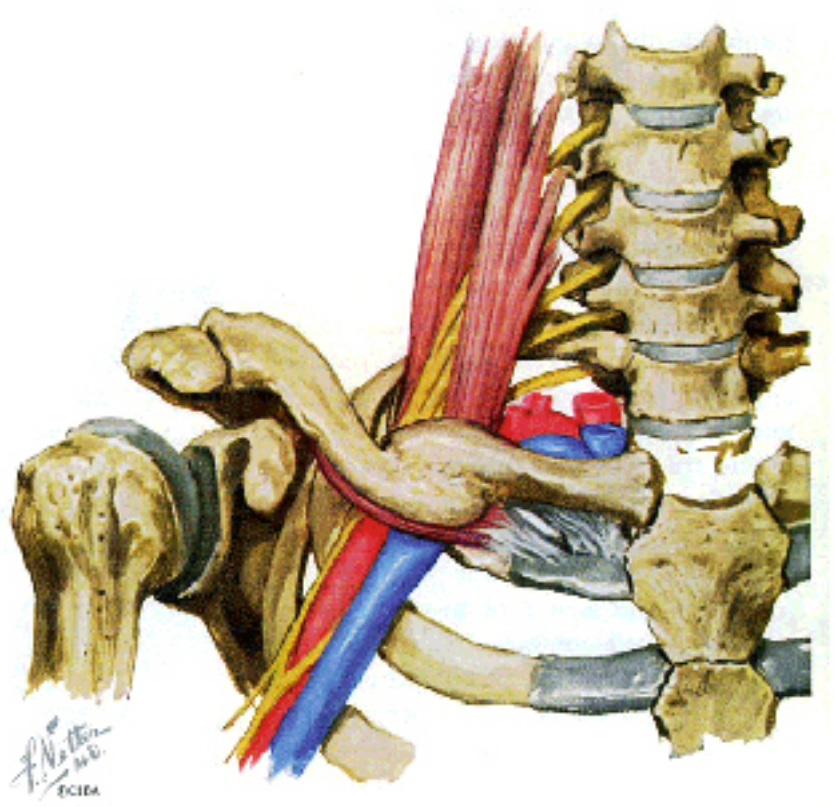

Figure 5: Fracture of clavicle with malunion compressing vessels and nerves

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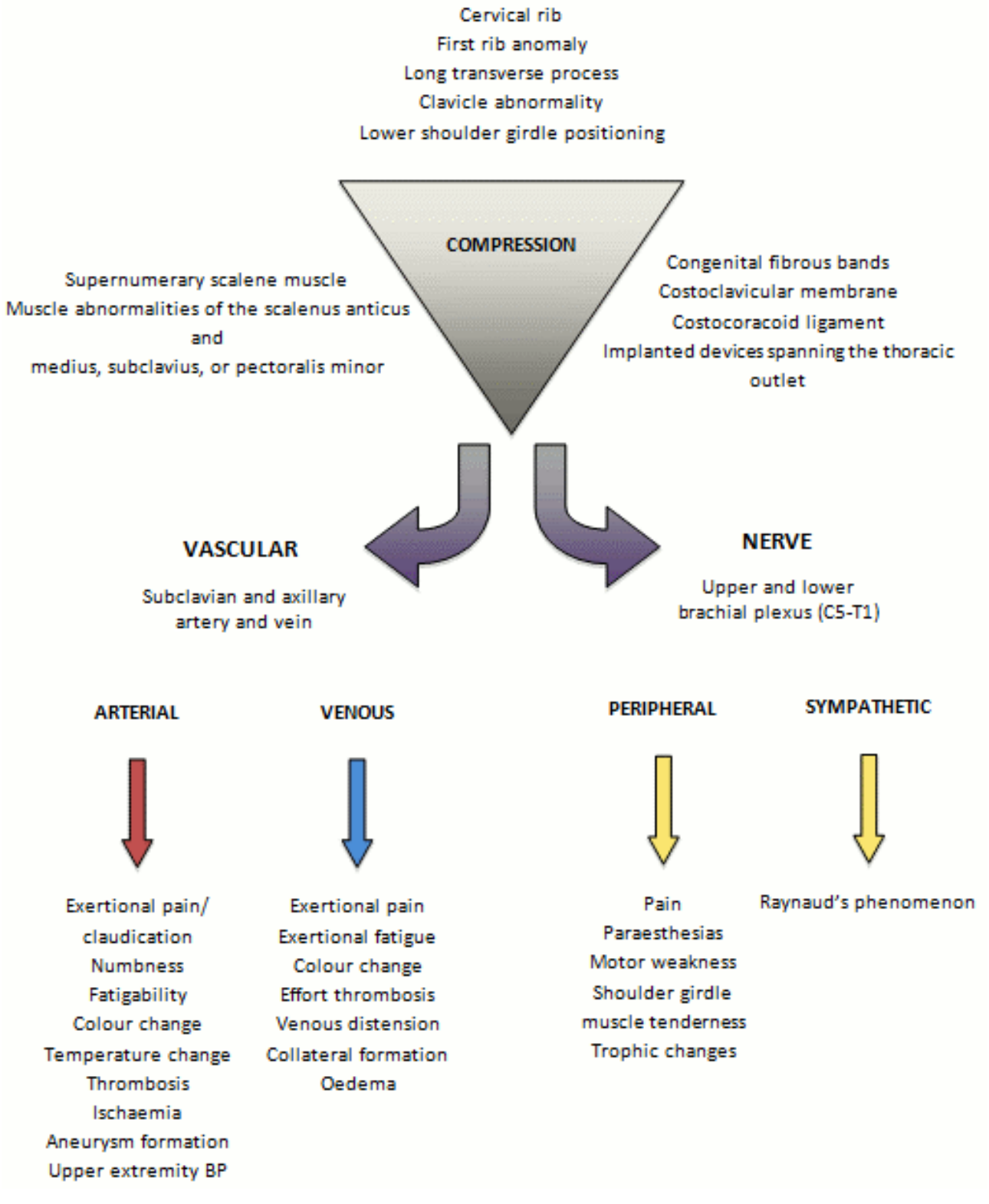

Figure 6: Thoracic outlet compression factors and resulting signs and symptoms

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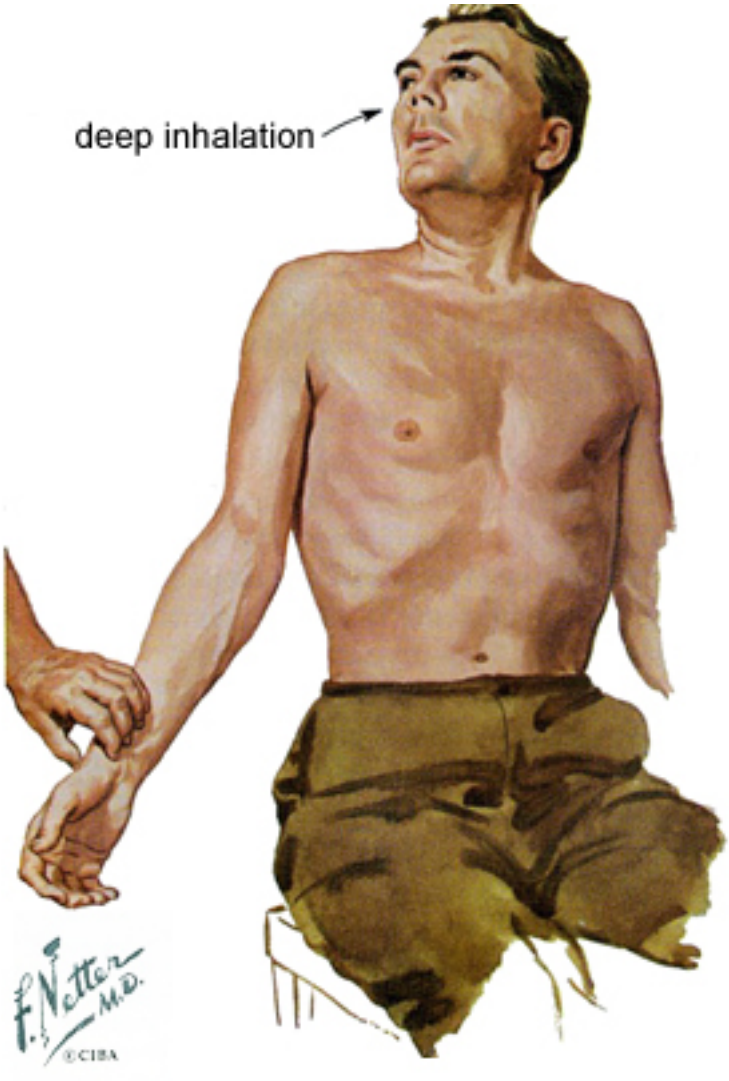

Figure 7: Adson's manoeuvre tightens anterior and middle scalene muscles, decreasing interspace and magnifying pre-existing compression of subclavian artery and brachial plexus. Patient takes and holds a deep breath, extends the neck fully, and turns head towards side. Obliteration or decrease of radial pulse suggests compression

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Figure 8: Costoclavicular test draws shoulders downwards and backwards, narrowing costoclavicular space by approximating clavicle to first rib, tending to compress neurovascular bundle. Changes in radial pulse with production of symptoms indicates compression

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Figure 9: Hyperabduction of right arm with anatomical structures. When arm is hyperabducted to 180°, neurovascular bundle components are pulled around the pectoralis minor tendon, coracoid process, and head of humerus. If radial pulse is decreased, compression should be suspected

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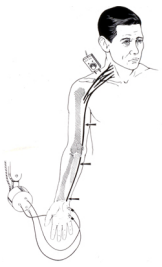

Figure 10: Nerve conduction velocity measurement technique. There is a 'blip' on the oscilloscope when the electric current hits the electrode

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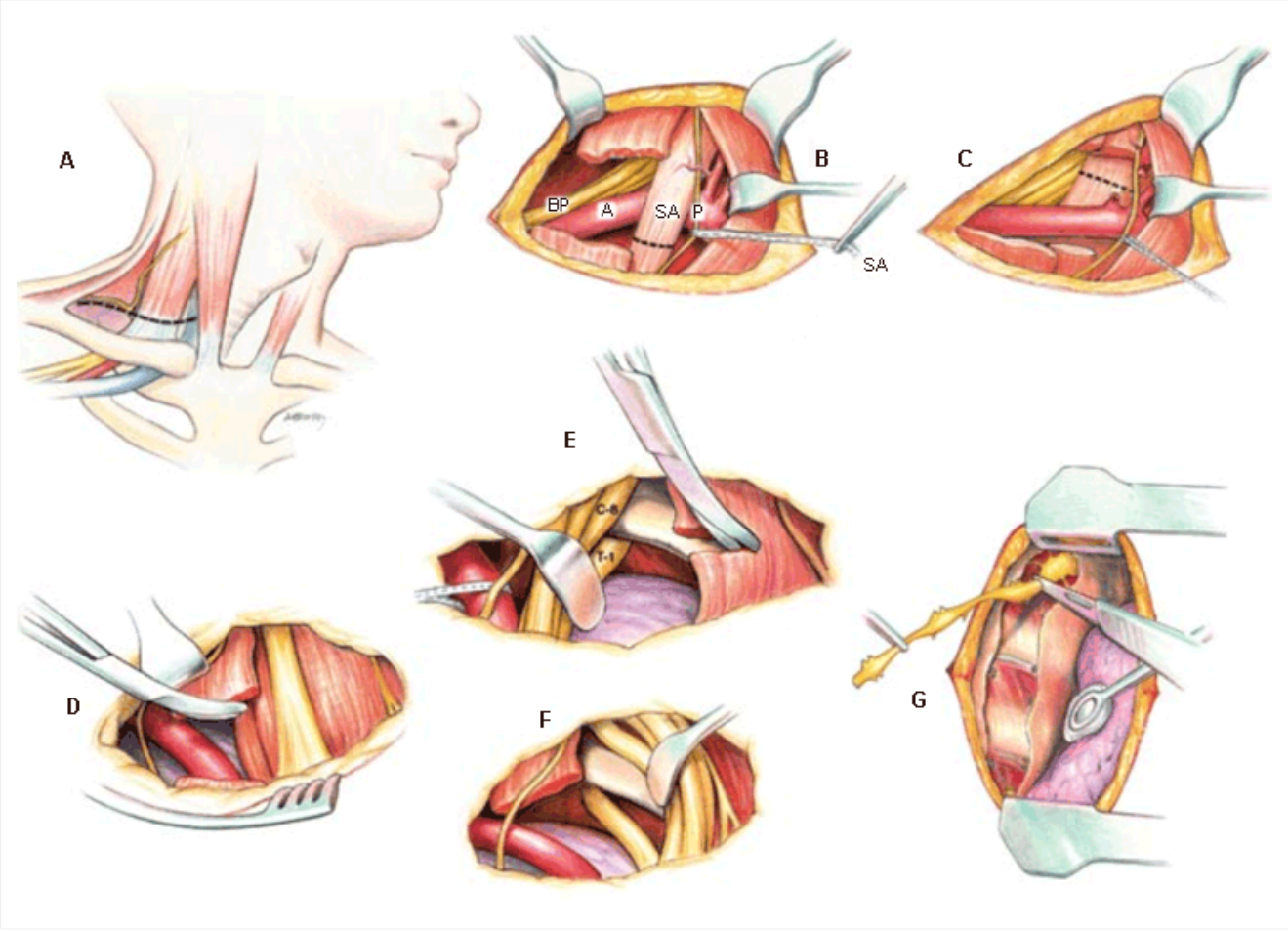

Figure 11: Supraclavicular approach. (A) Supraclavicular incision, (B) division of scalenus anticus muscle, (C) supraclavicular retraction of neurovascular structures, (D-F) resection of first rib, (G) supraclavicular dorsal sympathectomy. A: subclavian artery; BP: brachial plexus; P: phrenic nerve; SA: scalenus anticus muscle

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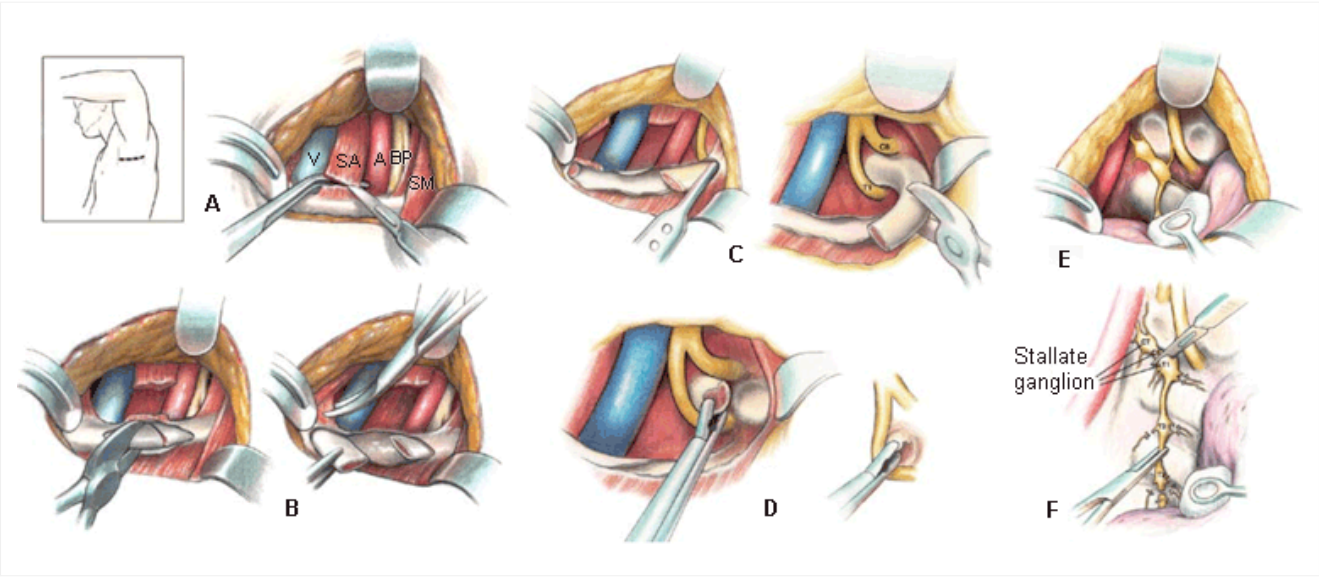

Figure 12: Transaxillary first rib resection. (A) Division of scalenus anticus muscle (SA), (B) division of first rib and anterior resection, (C) posterior resection of first rib, (D) resection of head and neck of rib, (E) identification of dorsal sympathetic chain, (F) division through lower stellate ganglion above T1 and below T3 ganglia. A: subclavian artery; BP: brachial plexus; SA: scalenus anticus muscle; SM: sternocleidomastoid muscle; V: subclavian vein

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