

Thoracic Outlet Syndrome

A patient guide

In brief: Thoracic outlet syndrome is the name given to a group of disorders caused by compression of the nerves or blood vessels as they pass from the neck and upper chest into the arm, through the narrow channels above and behind the collarbone. Most cases are neurogenic, meaning the lower part of the brachial plexus, the bundle of nerves supplying the arm and hand, is affected. Because those nerves arise from the lowest cervical and first thoracic levels, and because the symptoms of hand numbness, tingling and arm pain overlap so closely with those of a trapped nerve root in the neck, thoracic outlet syndrome sits at the very border between spinal and peripheral nerve practice. It is also one of the more contested diagnoses in medicine, ranging from a rare but clear-cut nerve injury caused by an extra rib in the neck, through to a common, hard-to-prove condition that is argued over to this day.

What thoracic outlet syndrome is (and why it borders on the spine)

The thoracic outlet is the passage through which the great nerves and vessels of the arm leave the trunk and enter the limb. It is not a single doorway but a series of narrow spaces, stacked one behind the other, that the neurovascular bundle must thread its way through on its journey from the neck to the armpit. When one of these spaces is too tight, or is narrowed by an extra structure, the contents can be compressed, and thoracic outlet syndrome is the result.

Three structures travel through this region and can be squeezed, and they give rise to the three recognised types of the condition. The brachial plexus, the network of nerves that supplies the whole arm, gives the neurogenic type. The subclavian vein gives the venous type. The subclavian artery gives the arterial type. The neurogenic form is overwhelmingly the most common, accounting for the great majority of cases, and it is the form that overlaps most closely with spinal disease.

The reason this condition borders on the spine is anatomical. The brachial plexus is built from the nerve roots that emerge from the lower cervical spine and the first thoracic level, namely C5 down to T1. The lowest part of the plexus, formed from the C8 and T1 roots, is the part most often caught in the thoracic outlet, because it sits lowest and must arch over the first rib to reach the arm. Those same C8 and T1 roots can also be compressed within the neck itself, by a worn disc or a narrowed exit canal, producing a cervical radiculopathy. The two conditions therefore draw on the same nerve fibres, produce overlapping symptoms in the same part of the hand, and are distinguished only by careful assessment. This is precisely why a spinal surgeon takes an interest in thoracic outlet syndrome: it is one of the principal conditions that must be separated from a trapped nerve in the neck.

A further reason for interest is that the commonest causes of true neurogenic thoracic outlet syndrome are anatomical features that arise from the spine itself, namely an extra rib growing from the lowest neck vertebra, or a fibrous band tethered to it. These are described in detail below.

The three types: neurogenic, venous and arterial

Although they share a name and a region, the three types of thoracic outlet syndrome behave quite differently and are worth setting out separately.

****Neurogenic thoracic outlet syndrome**** is by far the most common, making up around nine in ten cases in most large series. It arises from compression of the brachial plexus, and within it lies a crucial division, discussed at length below, between a rare but objectively demonstrable form and a common but contested one. Its symptoms are those of nerve irritation: pain, numbness, tingling and, in its severe form, weakness and wasting of the hand muscles, typically along the inner forearm and the little-finger side of the hand.

Venous thoracic outlet syndrome arises from compression, and sometimes clotting, of the subclavian vein where it passes between the collarbone and the first rib. When the vein clots after vigorous, repetitive use of the arm, the picture is known by the historical name of Paget–Schroetter syndrome, or effort thrombosis, and it classically affects young, active people after sustained overhead activity. The arm becomes swollen, heavy, bluish and uncomfortable. A related variant, in which the vein is squeezed intermittently without clotting, is sometimes called McCleery syndrome.

Arterial thoracic outlet syndrome is the rarest of the three. It arises from compression or damage of the subclavian artery, almost always in the presence of a bony anomaly such as a cervical rib, and it can lead to a narrowed, widened or clotted artery, with consequences for the blood supply of the arm and hand. Because it is nearly always linked to an extra rib, it shares an anatomical origin with the spine, even though its consequences are vascular.

This page concentrates on the neurogenic form, because it is the type that overlaps with cervical spine disease and that a spinal surgeon is most often asked to help distinguish.

The three compression spaces

The journey of the neurovascular bundle out of the trunk passes through three narrow compartments in succession, and compression can occur in any of them. Understanding the three spaces helps explain why symptoms vary and why surgery is directed where it is.

The first and most important is the **scalene triangle**, sometimes called the interscalene triangle. It is bounded by two muscles of the neck, the anterior and middle scalene, with the first rib forming its base. The trunks of the brachial plexus and the subclavian artery pass through this triangle, with the lowest trunk, formed from C8 and T1, resting on the first rib at the bottom. The subclavian vein, by contrast, passes in front of the anterior scalene and so lies outside the triangle, which is why pure venous problems tend to arise elsewhere. The scalene triangle is the commonest site of nerve compression, and it is here that an extra cervical rib or a fibrous band exerts its effect on the lowest part of the plexus.

The second space is the **costoclavicular space**, the gap between the collarbone above and the first rib below. The subclavian vein is most vulnerable here, which is why this is the usual site of effort thrombosis. The space narrows when the shoulder is drawn down and back, as in carrying a heavy load or standing at attention.

The third space lies beneath the **pectoralis minor muscle**, where it attaches to a bony prominence at the front of the shoulder blade. Raising the arm above the head tightens this muscle and narrows the space beneath it, which can compress the bundle. Compression at this level is sometimes called pectoralis minor syndrome and is regarded as a recognised contributor to neurogenic symptoms.

The cervical rib, the elongated C7 transverse process and fibrous bands

The structures that most clearly cause neurogenic thoracic outlet syndrome arise from the lowest vertebra of the neck, and they are the reason the condition is bound up with the spine.

A **cervical rib** is an extra rib that grows from the transverse process, the side projection, of the seventh cervical vertebra, and occasionally the sixth. It is a congenital feature, present in roughly half to one per cent of the population. Most cervical ribs cause no trouble at all and are discovered by chance, and only a minority, perhaps one in ten, ever produce symptoms. Even so, a cervical rib is found far more often in people with thoracic outlet syndrome than in the general population, with reported figures of around a third of cases, which makes it a powerful clue. Cervical ribs vary in form, and a long-standing description by Gruber divides them into four kinds, from a small stub that barely projects beyond the vertebra, through longer ribs whose tips approach the first rib, to ribs joined to the first rib by a band of fibrous tissue or cartilage, to ribs completely fused to it.

An **elongated C7 transverse process** is a milder version of the same theme, in which the side projection of the lowest neck vertebra is simply longer than usual without forming a separate rib. It is seen in around two per cent of people and can act in the same way as a small cervical rib.

The most important structures of all, however, are often invisible. **Fibrous bands**, sometimes called Roos bands after the surgeon who catalogued them, are tough cords of tissue that run from a rudimentary cervical rib or an elongated C7 process forwards to the first rib. They stretch and angulate the lowest part of the brachial plexus as it arches over the first rib, like a string drawn tight across the nerves. Because these bands are made of soft tissue, they do not show on ordinary X-rays, and they may be invisible on more detailed scans as well. They are therefore a recognised cause of genuine nerve compression in people whose plain films look entirely normal, and they are frequently found only at operation. The combination of a small cervical rib or elongated process with a taut fibrous band is the classic anatomical basis of the rare but definite form of the condition described next.

True neurogenic thoracic outlet syndrome and the Gilliatt–Sumner hand

Within the neurogenic group, one entity stands apart for being rare, definite and objectively demonstrable. It is known as true neurogenic thoracic outlet syndrome, and it is the form that most resembles a clear nerve injury.

It was described by Gilliatt and colleagues in 1970, in people who had a fibrous band running from a small cervical rib or an elongated C7 process to the first rib. The band stretches the lowest trunk of the brachial plexus, the part carrying the C8 and T1 fibres, and over time produces a slow, progressive loss of nerve function. It is genuinely rare, with estimates of around one case per million people, and it tends to occur in younger and middle-aged adults, more often women, usually on one side.

Its hallmark is a distinctive pattern of muscle wasting in the hand, known as the **Gilliatt–Sumner hand**. The striking feature is that the muscle bulk at the base of the thumb, the thenar eminence, wastes the most, even though this muscle is supplied by the median nerve, while the sensory loss is felt along the inner forearm and the little-finger side of the hand, in the territory of the ulnar nerve and the medial forearm. This curious split, profound wasting at the thumb base combined with numbness on the opposite, little-finger side, is the clinical signature of the condition. It reflects the fact that the lowest fibres of the plexus, derived from T1 and C8, are preferentially injured, and that the T1 fibres are damaged even more than the C8 fibres.

The diagnosis is anchored by electrical tests of the nerves, described below, which can demonstrate the lower-trunk injury objectively. It is this objective confirmation that separates true neurogenic thoracic outlet syndrome from the far more common contested form, and that makes it, when found, one of the clearest indications for surgery to remove the rib and divide the band.

Electrical tests of the nerves

The electrical study of the nerves, comprising nerve conduction studies and electromyography, is the single most valuable investigation in this field, both for confirming a true nerve injury and for separating the condition from its many imitators.

In true neurogenic thoracic outlet syndrome, the studies show a recognisable fingerprint. The sensory response recorded from the inner forearm, the medial antebrachial cutaneous nerve, is reduced or absent, and this is reported to be the single most sensitive finding, because that nerve samples the lowest part of the plexus directly. The motor response recorded from the thumb-base muscle is reduced out of proportion, reflecting the loss of T1 fibres. The sensory response from the little-finger side, the ulnar nerve, is reduced, while the sensory response from the thumb side, carried by the median nerve from higher levels, is preserved. Needle examination of the small hand muscles shows chronic changes of nerve loss.

This pattern matters most for what it allows a clinician to distinguish. In a trapped nerve root in the neck, a cervical radiculopathy, the injury lies very close to the spine, before the point at which the sensory nerve cell bodies sit, and as a result the sensory nerve responses are preserved even when the patient has marked numbness.

In a true plexus injury, by contrast, the same sensory responses are reduced. This single difference, preserved sensory responses pointing to the spine and reduced ones pointing to the plexus, is one of the most reliable separators in the whole assessment. In the common, contested form of thoracic outlet syndrome, the electrical studies are typically entirely normal, which is itself an important and much-debated finding.

The contested form and the long-running controversy

The great majority of people given a label of neurogenic thoracic outlet syndrome do not have the rare, objectively provable form. Instead they have pain, tingling, heaviness and a sense of the arm tiring easily, with normal electrical studies and no extra rib. This common form is the subject of one of the longest-running controversies in clinical medicine, and an honest account must set out both sides.

In the 1980s and 1990s, Asa Wilbourn and colleagues at the Cleveland Clinic argued that this group was being greatly over-diagnosed. They coined the term disputed neurogenic thoracic outlet syndrome for patients with the typical story but no objective abnormality, and they pointed to frequent confounding factors and to the difficulty of proving that the outlet was truly the source of the trouble. On the other side, many surgeons, particularly within vascular surgery, maintain that this is a real and treatable condition, a functional narrowing that simply does not leave a fixed electrical mark, and they rely instead on the history, on provocative positioning tests, on the response to an injection that relaxes the scalene muscle, and on dynamic imaging.

This dispute has not been resolved, and it is fair to say that the contested form remains a diagnosis reached by careful exclusion of other causes rather than by any single confirmatory test. For a spinal surgeon the practical consequence is clear and impersonal: the rare true form can be defined and confirmed, while the common contested form overlaps so heavily with cervical and peripheral nerve conditions that those must be considered and excluded with particular care. This page presents both perspectives without adjudicating between them.

Telling it apart from its neighbours

Because the symptoms of thoracic outlet syndrome are shared with several conditions that arise nearby, the heart of the assessment is differentiation, and a few recognised distinguishing features are worth describing factually.

The most important neighbour is ****cervical radiculopathy****, a trapped nerve root in the neck, especially at the C8 or T1 level, which produces ulnar-side hand symptoms that closely mimic lower-trunk thoracic outlet syndrome. Several features help separate them. Raising the arm above the head tends to ease the pain of a trapped neck nerve, because it takes tension off the root, whereas the same movement tends to worsen thoracic outlet symptoms. A test that loads the neck, such as the Spurling manoeuvre, points towards the spine, while the positioning tests of the outlet point away from it. And, as described above, the electrical studies separate the two: preserved sensory responses indicate the root, reduced ones indicate the plexus. A single trapped root also follows a tidy dermatomal pattern, whereas a lower-trunk plexus lesion crosses both C8 and T1 territories and includes the characteristic thumb-base wasting.

A second neighbour is ****cubital tunnel syndrome****, compression of the ulnar nerve at the elbow. It too causes numbness on the little-finger side and wasting of the small hand muscles. It is separated by the fact that it spares the thumb-base muscles, which are supplied by the median nerve, and spares the inner forearm sensation, since the nerve to that area branches off above the elbow. The electrical studies localise the slowing to the elbow.

A third is ****carpal tunnel syndrome****, compression of the median nerve at the wrist, which also wastes the thumb-base muscles but produces numbness on the thumb side of the hand, with the electrical abnormality localised to the wrist.

Finally there is the concept of the ****double crush****, first proposed by Upton and McComas in 1973, in which a nerve compressed at one point becomes more vulnerable to compression at a second point further along its course. It is invoked to explain how a person may have, for instance, both a trapped neck nerve and a trapped nerve at the wrist or elbow at the same time. The idea remains debated, but it carries a useful practical reminder, that relieving

one site of compression may not resolve symptoms if a second site coexists.

Provocative tests and their limits

A number of physical manoeuvres have long been used to try to reproduce the symptoms of thoracic outlet syndrome by placing the arm in positions that narrow the spaces. Their names recur throughout the literature, and they are useful as part of a wider assessment, but their limitations are equally well recognised.

The **Adson test** involves turning the head towards the affected side and breathing in deeply while the examiner feels for a change in the pulse at the wrist or for reproduction of symptoms. The **Roos test**, also called the elevated arm stress test, asks the person to hold both arms raised and bent at the elbow and to open and close the hands repeatedly for up to three minutes, watching for the symptoms to come on. The **Wright test** raises and rotates the arm to narrow the space beneath the collarbone and pectoralis muscle.

The difficulty with all of these is that they are not specific. A change in the pulse, in particular, occurs in a large proportion of people who have no symptoms at all, with reported figures of up to around sixty per cent of healthy individuals showing some vascular change on testing. The Roos test is sensitive, in that it usually turns positive in people who do have the condition, but it also turns positive in many who do not, including those with quite different nerve problems. The recognised conclusion is that no single provocative test confirms the diagnosis, and that a combination of positive tests, taken together with the history and the other investigations, is what builds the overall picture.

Imaging and other investigations

Imaging in thoracic outlet syndrome serves two purposes: to find the anatomical causes, such as a cervical rib, and to look at the nerves and vessels themselves.

A **plain X-ray** of the neck or chest is the natural first step for detecting a cervical rib or an elongated C7 transverse process, both of which are bony and therefore visible. It cannot, however, show the fibrous bands that are so often responsible, because these are made of soft tissue.

Computed tomography, particularly with contrast in the vessels, shows the bony anomalies clearly and can demonstrate compression of the artery or vein, especially when the scan is repeated with the arm in different positions. It is central to assessing the vascular forms.

Magnetic resonance imaging, including detailed imaging of the brachial plexus, can show the nerves being displaced or compressed, can reveal enlarged or extra muscles, and can sometimes demonstrate the fibrous bands. Imaging performed with the arm raised can show compression that only appears in certain positions. It is important to note that the sensitivity of these scans is only moderate, with one series reporting that around seven in ten cases were identified and the remainder missed, so a normal scan does not exclude the condition, and incidental findings of uncertain meaning are common.

An **injection of local anaesthetic into the scalene muscle**, which temporarily relaxes it, is used by some as a diagnostic aid, on the basis that marked relief after the injection supports the diagnosis and may help predict the response to surgery.

What the evidence shows about management — impersonal

It is important to be clear at the outset that the evidence base for treating neurogenic thoracic outlet syndrome is limited. There is no large, multi-centre randomised trial, and a Cochrane review concluded that the quality of evidence was low and that firm recommendations could not be drawn. Most of what is known rests on observational series, expert consensus and one small randomised trial. What follows describes what the literature reports, not guidance for any individual, and decisions in practice are made on a case-by-case basis.

****Conservative, non-surgical management**** is the recognised first approach for most neurogenic cases, and especially for the common contested form. The reported approach centres on physiotherapy aimed at posture and the position of the shoulder blade, stretching of the scalene and pectoralis minor muscles, gentle nerve-mobilising exercises, activity modification, and pain relief. Some series describe injections of botulinum toxin into the scalene muscle, with mixed results. Observational studies report that a meaningful proportion of people improve with physiotherapy, with figures spanning roughly a quarter to three-fifths, and that of those who do not improve, a majority go on to benefit from surgery.

****Surgical decompression**** is reported for true neurogenic cases with an objective deficit, for the vascular forms, and for contested cases that have not responded to good conservative care. The operations described include removal of the first rib, removal of a cervical rib, division of fibrous bands, removal of the scalene muscles, freeing of the nerves themselves, and release of the pectoralis minor tendon. These can be approached through the armpit, above the collarbone, or below it, including with robotic assistance. In true neurogenic disease the cervical rib and band are removed to relieve the stretch on the lower trunk.

The ****reported outcomes**** of surgery are generally favourable in selected patients, with series describing meaningful improvement in around seventy to ninety per cent. One report of a rib-sparing scalene operation described around nine in ten achieving at least half of their improvement within three to six months. Outcomes are consistently described as more predictable in the true and vascular forms than in the contested form. Whether the first rib must always be removed is itself debated, with some series reporting good results from scalene surgery alone, and others trialling less invasive alternatives.

****Trial evidence**** is dominated by a single randomised study, the STOPNTOS trial of 2022, which compared surgery with continued conservative therapy in people whose symptoms had not settled with physiotherapy, and which reported lower failure rates in the surgical group at one year. It was small and conducted at a single specialist centre, and it is not, on its own, a basis for general rules. The overall picture remains one of individualised, shared decision-making rather than fixed protocols.

Outlook

The outlook in thoracic outlet syndrome varies as much as the condition itself. For the common contested neurogenic form, many people improve with conservative measures over a period of weeks to months, and the literature regards a structured rehabilitation approach as the reasonable starting point, with surgery reserved for those who do not settle. The principal uncertainty in this group is that, because the diagnosis itself is contested, the response to any treatment is harder to predict and the results of surgery are more variable than in the clearly defined forms.

For true neurogenic thoracic outlet syndrome, the picture is different. Here a definite nerve injury is present, and the established expectation in the literature is that removing the cervical rib and dividing the band relieves the pressure on the nerve; pain and sensory symptoms often improve, although established muscle wasting may recover only partially, since nerve fibres that have been damaged for a long time recover slowly and incompletely. This is one of the reasons the condition, once recognised, is taken seriously.

For the vascular forms, the outlook depends on the state of the vessel and on prompt recognition, and these are managed within a vascular rather than a spinal framework. Across the whole field, the consistent themes are that accurate diagnosis, and in particular careful separation from cervical and peripheral nerve conditions, is the foundation of a good result, and that any individual outlook is a matter for assessment by the treating team rather than something that can be read from a category alone.