

Thoracic Spinal Stenosis

A patient guide

In brief: Thoracic spinal stenosis is a narrowing of the spinal canal in the mid-back, the long thoracic region that runs between the neck above and the lower back below. It is much less common than the narrowing that occurs in the lumbar spine or the neck, and it behaves quite differently from both. Because the thoracic canal contains the spinal cord itself, rather than the looser bundle of nerves found lower down, thoracic stenosis tends to declare itself as a disturbance of the cord, a myelopathy, with stiffness and unsteadiness in the legs, rather than as the walking-related leg pain of lumbar stenosis. The thoracic canal is also the narrowest, stiffest and most precariously supplied part of the spine, which gives this rare condition a character, and a set of difficulties, all of its own.

What thoracic spinal stenosis is (and why the mid-back is different)

Stenosis simply means narrowing. In the spine, it refers to a reduction in the space available within the spinal canal, the bony tunnel that protects the delicate neural structures running down the back. The same word is used at every level of the spine, but the consequences of narrowing depend entirely on what is being narrowed around, and this is where the thoracic spine stands apart from its neighbours.

In the lumbar spine, the lowest part, the spinal cord has already ended. What runs through the lumbar canal is the cauda equina, the loose bundle of nerve roots named after a horse's tail, which has a degree of slack and some room to move. In the neck, the cervical spine, the canal contains the cervical spinal cord, which also supplies the arms. The thoracic spine sits between the two, spanning the twelve vertebrae of the chest, each joined to a pair of ribs. Its canal contains the thoracic spinal cord, the long, relatively featureless stretch of cord that carries the messages travelling to and from the legs, the bladder and the bowel.

Three features make the thoracic canal unlike any other. First, it is the narrowest part of the whole spinal canal along much of its length, and the cord nearly fills it, so there is very little spare room to begin with. Second, the thoracic spine is braced by the rib cage and held in a gentle forward curve, the kyphosis, which makes it the stiffest and least mobile region of the spine. Third, the blood supply to the thoracic cord is sparse and comes through a vulnerable arrangement described later. Taken together, these features mean that even a modest encroachment on the thoracic canal can press on the cord, and that the cord here tolerates that pressure poorly.

This is the central idea behind thoracic stenosis. It is not simply lumbar stenosis higher up the back. It is a narrowing around the spinal cord itself, in the tightest and most unforgiving section of the spinal canal, and that single fact shapes how it presents, how it is investigated and how it is treated.

How it differs from lumbar and cervical stenosis

Because the word stenosis is shared across the spine, it is easy to assume that narrowing behaves the same wherever it occurs. It does not, and the differences are worth setting out plainly, because they explain much of what follows.

Lumbar spinal stenosis is by far the commonest of the three and is one of the most frequent reasons for spinal surgery in older adults. Its hallmark is neurogenic claudication, a heaviness, aching or tiredness that builds in the legs on standing and walking and eases on sitting or leaning forwards. This pattern arises because the cauda equina is being crowded; bending forwards opens the canal a little and relieves the symptoms. The problem is one of nerve roots, not of the cord.

Cervical spinal stenosis, in the neck, does involve the cord, and so it can cause a myelopathy, but because the cervical cord also serves the arms, it typically produces clumsiness of the hands and arm symptoms alongside any

disturbance in the legs.

Thoracic spinal stenosis is rarer than either, and it has a distinct signature. The thoracic cord does not supply the arms, so the arms are spared. What it governs is the legs and the pelvic organs, by way of the long tracts running through it. Narrowing here therefore tends to produce a slowly progressive stiffness and unsteadiness of the legs, often with a band-like sensation around the trunk, and later disturbance of bladder control, without any arm involvement at all. The presence of cord-type signs in the legs, with normal arms, points the experienced examiner towards the thoracic level. This contrast, claudication and root symptoms in the lumbar spine, hand and arm involvement in the neck, and isolated leg and trunk cord symptoms in the thoracic spine, is one of the most useful ways of understanding what makes thoracic stenosis different.

How it differs from thoracic disc herniation

A related but separate condition can cause confusion, and it is worth distinguishing clearly. A thoracic disc herniation is a focal problem: a single intervertebral disc, often hardened and calcified, bulges backwards at one level and may press on the cord from the front. It is dealt with on its own dedicated page. Thoracic stenosis, by contrast, describes a more general narrowing of the canal, which is usually multifactorial and may involve several levels.

The two can overlap, because a large central disc can contribute to canal narrowing, and both can produce a thoracic myelopathy. The distinction the literature draws is one of pattern and cause: a disc herniation is a focal, often calcified soft-tissue lesion arising from the disc itself, whereas stenosis is a broader narrowing produced by thickened ligaments, ossification, bony overgrowth or congenital narrowness, frequently in combination. Understanding thoracic stenosis as a problem of the canal, rather than of a single disc, helps keep the two ideas separate even where they meet.

What narrows the thoracic canal

In the lumbar spine, narrowing is overwhelmingly degenerative, driven by wear of the discs and facet joints and thickening of the ligaments. In the thoracic spine the picture is different, and a particular set of processes dominates, several of them involving ossification, the turning of soft ligament into bone.

****Ossification of the ligamentum flavum****, usually shortened to OLF, is the single commonest contributor in the published series. The ligamentum flavum is a strong elastic ligament that lines the back of the canal. When it ossifies, it forms hard, bony ridges that bulge inwards and press on the cord from behind. OLF has a strong preference for the lower thoracic spine, is often found on both sides at once, and may affect several levels. It is much more frequently reported in East Asian populations and in older men. Importantly, OLF is commonly seen on scans without causing any symptoms at all; the imaging finding is far more common than the clinical condition.

****Ossification of the posterior longitudinal ligament****, or OPLL, is the mirror image at the front of the canal. The posterior longitudinal ligament runs down the back of the vertebral bodies, and when it ossifies it presses on the cord from the front. In the thoracic spine it tends to favour the upper and middle levels and is often part of a wider tendency to form bone in spinal ligaments. Certain shapes of thoracic OPLL, particularly the so-called beak type, are recognised as especially difficult to treat.

****Degenerative change**** can also narrow the thoracic canal, through thickening of the ligaments, overgrowth of the facet joints and disc bulging, much as it does in the lumbar spine, though it is far less common here because the rib cage limits the wear and movement that drive it.

****Congenital and developmental narrowness**** describes a canal that was simply built small from the outset, which leaves less reserve for any later change to consume. Conditions such as achondroplasia, a form of restricted bone growth, can produce a constitutionally tight canal across several levels.

These processes often act together. A particularly difficult situation arises when the cord is squeezed from both front and back at once, for instance OPLL pressing forwards and OLF pressing backwards at the same level, leaving the cord pinched between them.

The narrow, rigid, rib-braced canal and the cord's blood supply

Two anatomical facts give thoracic stenosis much of its seriousness, and both deserve describing.

The first is the shape and stiffness of the region. The thoracic canal is the tightest section of the spinal canal, and the cord occupies a large proportion of the available space, so the margin for any narrowing is slim. The thoracic spine is also held in a forward curve and splinted by the rib cage, which makes it rigid. This rigidity has two effects. It limits the day-to-day wear that causes lumbar stenosis, which is part of why thoracic stenosis is rare, but it also means that when a lesion does encroach on the canal, the cord cannot easily shift away from it, and the gentle kyphosis tends to drape the cord against anything pressing from the front.

The second is the blood supply. The thoracic cord, particularly the middle section, lies in what anatomists call a watershed zone, a region at the far end of two arterial territories where the supply is thinnest. The lower two-thirds of the cord depends heavily on a single, variable artery, the artery of Adamkiewicz, which usually enters from the left side somewhere in the lower thoracic region. There is little in the way of backup. The practical consequence is that the thoracic cord is unusually sensitive to anything that reduces its blood flow, whether sustained pressure from a stenosis or the disturbances that can accompany surgery in this area. This precarious blood supply is one of the reasons the thoracic cord tolerates compression so poorly and why operating here carries particular risks.

How it tends to present (myelopathy rather than claudication)

The most important single point about how thoracic stenosis presents is that it usually does so as a myelopathy, a disturbance of the spinal cord, rather than as the walking-related leg pain of lumbar stenosis.

The onset is typically insidious. Rather than a clear event, there is a gradual change: the legs become stiff, heavy or unsteady; walking feels less secure; and balance, particularly on uneven ground or in the dark, may deteriorate. Many people describe a band-like or girdle sensation around the trunk or abdomen, a tightness that encircles the body at the level of the affected cord, which is quite characteristic and has no equivalent in lumbar stenosis. Numbness or altered sensation may spread up from the feet to a certain level on the trunk, a sensory level that marks the height of the problem. As the condition advances, disturbance of bladder control may appear.

When a doctor examines the legs, the findings are those of cord involvement: the reflexes are brisk rather than reduced, the muscles may be stiff and spastic, and certain reflexes that should be absent become present. Crucially, the arms are normal, because the thoracic cord lies below the level that supplies them. This combination, cord-type signs in the legs with entirely normal arms, points to the thoracic spine. Pain is often a minor feature or limited to non-specific back discomfort, which is one reason the condition can be slow to be recognised. None of this describes any particular individual; it is the general pattern reported in the literature, and the presence or absence of any feature in a given person is a matter for direct assessment.

Tandem stenosis and the diagnostic trap

One of the most important and best-recognised pitfalls in this field is the way thoracic stenosis can be overlooked because its symptoms are blamed on the much commoner lumbar disease. Understanding this trap is central to understanding the condition.

Stenosis can occur at more than one level of the spine in the same person. When narrowing is present at two or more of the cervical, thoracic and lumbar regions together, it is called tandem stenosis. The commonest combination is the neck and the lower back together; thoracic involvement, alongside either or both of the others, is rarer and is a particular source of difficulty.

The trap works like this. A person has symptoms in the legs. The lumbar spine is the usual culprit for leg symptoms, lumbar stenosis is common, and a lumbar scan often shows some narrowing because such changes are widespread with age. It is therefore tempting to attribute everything to the lumbar spine. But if the true driver of the leg symptoms is a thoracic cord compression higher up, then treating the lumbar spine alone will not help, and the cord problem continues to progress, with the risk that the damage becomes permanent.

The literature describes several clues that should make an assessor look higher than the lumbar spine. Cord-type signs in the legs, brisk reflexes, stiffness, certain abnormal reflexes, sit oddly with a purely lumbar problem, which usually produces the opposite, reduced reflexes. A band-like sensation around the trunk or a sensory level on the body points above the lumbar spine. Unsteadiness and spasticity that seem out of proportion to what the lumbar scan shows raise the same suspicion. A confusing mixture can occur when lumbar narrowing and thoracic cord compression coexist, producing both reduced reflexes at the ankles and brisk, abnormal reflexes elsewhere in the same legs, a mixed picture that itself prompts a look at the thoracic spine. The recognised lesson running through this literature is that leg symptoms should not be assumed to be lumbar in origin when signs of cord involvement are present.

How it is assessed (MRI, CT and whole-spine imaging)

The assessment of thoracic stenosis rests chiefly on cross-sectional imaging, because the cord and the soft tissues around it cannot be judged from ordinary X-rays.

****Magnetic resonance imaging, the MRI scan****, is the principal investigation. It shows the spinal cord directly, demonstrates where and how severely the canal is narrowed, and distinguishes whether the cord is being pressed from the front, from behind, or from both. It also reveals changes within the cord itself: a bright signal on certain sequences indicates that the cord has been injured or is swollen, a finding that carries weight when judging how advanced the problem is and how it may respond to treatment.

****Computed tomography, the CT scan****, is the better test for bone, and it is especially valuable here because so much thoracic narrowing is caused by ossification. CT shows the ossified ligaments clearly, defines the shape of OPLL, including whether it is the demanding beak type, and clarifies how far the bony change has progressed. MRI and CT are therefore complementary, one for the cord and soft tissues, the other for the bone.

Because of the tandem-stenosis trap described above, imaging of the whole spine, or at least imaging guided by the level the neurological findings point to rather than by the symptomatic limb alone, is often part of the assessment when cord-type signs are present. A recurring caution in the literature is that imaging findings must be interpreted alongside the clinical picture, because narrowing and ossification are frequently seen on scans in people who have no symptoms whatsoever.

What the evidence shows about management — impersonal

It is worth stating at the outset that the evidence base for thoracic stenosis is built largely on retrospective surgical series, many from East Asia where the condition is more common, together with reviews and a small number of guidelines. There are no large randomised trials directing treatment, and decisions in practice are individualised. What follows is a description of what the literature reports, not guidance for any person.

****Observation**** is the usual course where thoracic narrowing or ossification is found incidentally or causes only mild, non-progressive symptoms. The literature does not suggest that any conservative measure reverses established compression of the cord; the value of non-surgical management lies in monitoring and in managing symptoms, and the rationale for considering surgery is the prevention of further deterioration.

****Surgery**** is described in the literature for progressive myelopathy, and its central aim is decompression, relieving the pressure on the cord. The technique depends on where the compression is coming from. Narrowing from behind, as with ossification of the ligamentum flavum or degenerative thickening, is typically addressed by a posterior operation, removing the back wall of the canal and the offending bone to give the cord room. Because

removing this bone can affect the stability of the spine and allow the forward curve to worsen, fixation with implants is sometimes added. Compression from the front, as with thoracic OPLL or a calcified central disc, is more demanding and may call for more complex approaches, and the beak type of OPLL is singled out in the literature as particularly challenging.

The feature that most sets thoracic surgery apart from lumbar and cervical surgery is its higher neurological risk. The published series report rates of postoperative neurological deterioration considerably higher than those seen in lumbar decompression, with figures in some thoracic OPLL series in the region of one in eight to one in three, and overall complication rates, including leaks of spinal fluid, wound problems and deterioration, reported around forty to fifty per cent in some multicentre data. The reasons are exactly the anatomical ones described earlier: the cord has almost no reserve, its blood supply is precarious, and even small changes in alignment or handling can tip it into injury. For this reason, monitoring of the cord's electrical signals during the operation is commonly used to give early warning of any compromise.

As for results, the literature reports that many people improve after decompression, with recovery rates in OPLL series often around half, but recovery is frequently partial, and the goal is often to halt progression rather than to restore normal function. Outcomes are reported to be poorer where the deficit has been severe or long-standing, where the cord already shows signal change on the scan, and where the compression comes from the front. In short, the evidence points to observation for mild or incidental disease and decompressive surgery for progressive myelopathy, with the higher neurological risk weighed against the natural tendency of cord compression to worsen, all within individualised, shared decision-making rather than fixed rules.

Outlook

The outlook in thoracic stenosis depends heavily on how far the condition has progressed before it is addressed and on the nature of the narrowing. The general theme in the literature is that the thoracic cord tolerates sustained compression poorly and that an established myelopathy, left to its own devices, tends to deteriorate rather than to settle.

Where the condition is recognised while the deficit is still mild and the cord has not yet shown signal change, the reported chances of arresting and partly reversing the problem are better. Where the deficit has been severe or long-standing, or where the cord already shows injury on the scan, recovery is reported to be less complete, and the realistic aim may be to prevent further decline rather than to return to normal. The location of the compression matters too, with pressure from the front generally regarded as more troublesome than pressure from behind.

Across the literature the consistent messages are that thoracic stenosis is uncommon and easily mistaken for lumbar disease, that its cord-level nature gives it a seriousness out of proportion to its rarity, and that any individual outlook is shaped by the specific pattern of narrowing and the state of the cord, and is therefore a matter for assessment by the treating team rather than something that can be read from the diagnosis alone.