

Persistent Nerve Pain After Spine Surgery

A patient guide

In brief: Persistent nerve pain after spine surgery describes leg or arm pain of a nerve, or neuropathic, character that continues after an operation that was technically adequate and that has done what it set out to do. It is a particular and often misunderstood problem, because the pain arises from the nerve itself, from the original injury to it, from chronic irritation, from scarring around it, or from changes in the way the nervous system itself processes pain, rather than from a fresh, surgically-correctable compression. This is precisely what sets it apart from its structural neighbours, such as recurrent disc herniation, residual or recurrent stenosis, and instability, each of which presents a clear target that an operation can address. When the scans show no new pressure on the nerves, the pain is best understood as neuropathic and as a chronic-pain condition, and the modern literature reflects this in a shift of name from failed back surgery syndrome to persistent spinal pain syndrome.

What persistent nerve pain after surgery is (and what it is not)

The starting point is the distinction between a structural problem and a neuropathic one. A great deal of spinal surgery is undertaken to take pressure off a nerve, whether by removing the fragment of a herniated disc, by widening a narrowed canal or exit hole, or by stabilising a segment that has become unstable. When such an operation succeeds in relieving the compression, the structural cause of the pain has been addressed. In most people, the nerve then settles and the pain that brought them to surgery eases over the weeks and months that follow.

In a smaller group, the pain does not settle in this way, or it settles and then returns, even though the original target has been dealt with and the scans show no new pressure on the nerves. This is the territory of persistent nerve pain after surgery. The pain is felt along the course of a nerve, in the leg after lumbar surgery or in the arm after neck surgery, and it carries the qualities that mark out nerve pain, such as burning, shooting, electric or pins-and-needles sensations, often with areas of numbness or heightened sensitivity. The defining feature is that this pain persists despite there being nothing fresh and mechanical left to decompress.

It is worth being clear about what this condition is not. It is not, in itself, a sign that the surgery was done badly or at the wrong level, although those possibilities always have to be excluded first. It is not the same as a recurrent disc herniation or a fresh narrowing, both of which are structural problems with a structural answer. And it is not simply pain that has been imagined or exaggerated; it is genuine pain that arises from real changes in the nerves and in the pain-processing pathways. Understanding it as a distinct entity, neuropathic rather than mechanical, is the single most important idea in this whole field, because it explains why the answer to it is so often not another operation.

Why the name has changed: from failed back surgery syndrome to persistent spinal pain syndrome

For many years, persistent pain after spinal surgery was grouped under the heading of failed back surgery syndrome. That term has fallen out of favour, and the reasons for the change are instructive in themselves.

The first difficulty is that the word failed misrepresents what has happened. There is no agreed definition of what counts as successful spinal surgery against which an operation could be judged to have failed, and an operation that achieves exactly what it set out to do, such as relieving a compressed nerve, can still be followed by persistent pain. To call that a failure is to confuse the technical result of the surgery with the experience of the person afterwards. The second difficulty is that the label is, in effect, pejorative. It implies fault, usually on the part of the

surgeon, where often there is none, and it focuses attention backwards, on the operation, rather than forwards, on the nature of the pain and how it might be helped.

For these reasons, a consensus proposal published in 2021 recommended replacing the term with persistent spinal pain syndrome, abbreviated to PSPS. This system divides the problem into two types. Type 1 describes persistent spinal pain where no spinal surgery has been performed, and Type 2 describes persistent spinal pain after spinal surgery, which is the situation discussed here and the direct replacement for the old label. A closely related term used by the International Association for the Study of Pain is chronic pain after spinal surgery. The point of all this renaming is not merely cosmetic. It reflects a genuine change in understanding, away from the idea of a botched operation and towards the idea of a chronic pain condition that needs to be described by its mechanism rather than by blame.

The crucial distinction: a structural target versus neuropathic pain without one

Everything in the assessment and understanding of this condition turns on a single division, which is worth setting out plainly. On one side lie the structural causes of persistent pain after surgery. On the other lies neuropathic pain that has no fresh structural target.

The structural causes are those in which something mechanical is once again pressing on or irritating a nerve, and in which imaging shows a lesion that an operation could, in principle, address. The recognised members of this group include a recurrent or residual disc herniation, in which disc material is once more in contact with the nerve; residual or recurrent stenosis, in which the central canal, the lateral recess or the exit foramen is still or again too narrow; instability or pseudarthrosis, in which a segment moves abnormally or a previous fusion has not united; adjacent segment degeneration, in which the levels next to a fusion wear and narrow; and, rarely, surgery at the wrong level. What unites these is that each presents a structural target, and for each, further surgery has a rational basis, because there is something concrete to correct.

The neuropathic causes are different in kind. Here the pain arises from the nerve itself and from the pain pathways, and the imaging shows no new compression that an operation could relieve. This group includes injury to the nerve root sustained at the time of the original problem or surgery; chronic irritation of a root that has been left sensitised; epidural fibrosis, which is scar tissue that forms around the nerves after any operation and can tether and irritate them without frankly compressing them; arachnoiditis, an inflammation and scarring of the lining around the nerve roots; and central sensitisation, a change in the spinal cord and brain that amplifies pain signals. The defining contrast is that in this group there is no structural target. The nerve is generating pain, but there is nothing fresh and mechanical to decompress.

The reason this distinction matters so much is entirely practical. Where a structural target exists, an operation has a logic to it. Where it does not, and the pain is neuropathic, the same operation has no target to address, and the literature consistently shows that further decompression in that setting rarely helps. The whole of careful re-assessment is therefore directed at working out, as reliably as possible, which side of this line a given person's pain sits on.

Why correctable causes must be excluded first

Because some causes of persistent pain after surgery are structural and potentially correctable, and others are neuropathic and not, the first task in assessment is a careful re-evaluation aimed at excluding the correctable causes before the pain is attributed to neuropathic mechanisms. This sequence is not a matter of preference; it is the only logical way to approach the problem.

The reason is that the two groups can feel similar to the person experiencing them, yet they call for completely different responses. A recurrent disc herniation pressing on a nerve and a sensitised nerve generating pain without any new compression can both produce leg pain of a nerve character. Only by re-examining the pattern of the symptoms, by testing the nerves, and above all by re-imaging the spine can the two be told apart. To assume

neuropathic pain without first looking for a structural cause would risk missing a problem for which an operation could genuinely help. To rush back to surgery without first excluding neuropathic pain would risk operating where there is no target to address.

Re-assessment therefore covers three strands. The clinical history and examination establish whether the pain has the features of fresh nerve compression or of established neuropathic pain, and whether there are signs that demand urgent attention in their own right. Imaging, principally magnetic resonance imaging, is used to look for any new or residual compression. And, where the distinction is difficult, contrast is used to help separate scar tissue from recurrent disc material, because the two behave differently when contrast is given. Only when this process has reasonably excluded a fresh, surgically-correctable cause is the pain understood as neuropathic and chronic in nature. None of this describes the path for any individual, whose assessment is a matter for direct evaluation; it is simply the reasoning that the literature applies to the problem as a whole.

How the nerve generates pain without compression: injury, fibrosis, arachnoiditis and sensitisation

To understand why pain can persist without any new pressure on the nerves, it helps to look at the several mechanisms the literature describes, because together they explain how a nerve can go on generating pain when there is nothing left to decompress.

The first mechanism is direct injury to the nerve root. The very problem that led to surgery, a compressing disc or a tight canal, may already have damaged the root, and a damaged nerve can continue to fire abnormally and to signal pain long after the pressure on it has been removed. Neuropathic pain is defined as pain arising as a direct consequence of a lesion or disease affecting the somatosensory system, the part of the nervous system that carries sensation, and an injured root is exactly such a lesion. This is why pain can be entirely genuine and neuropathic even when the scans look satisfactory.

The second mechanism is epidural fibrosis, the scar tissue that forms around the nerves after any spinal operation. Some scar is an inevitable part of healing. In some people it binds the sleeves of the nerve roots and the lining of the spinal canal to the surrounding structures, preventing the roots from stretching and gliding normally as the spine moves and the leg is raised, and tethering and irritating them in the process. The association between extensive scar and recurrent leg pain has been studied and remains debated, but fibrosis is recognised as a way in which a nerve can be irritated without being compressed in the ordinary sense.

The third mechanism is arachnoiditis, an inflammation and subsequent scarring of the arachnoid, one of the delicate membranes that surround the nerve roots. When it occurs, the roots may clump together and become persistently irritable, producing a neuropathic pain that has no single compressive point to relieve.

The fourth mechanism, and in many ways the most important for understanding chronic pain, is central sensitisation. This is a change within the spinal cord and brain in which the pain-processing pathways become amplified. After prolonged or intense input from an injured nerve, these central pathways develop increased background activity, enlarged areas from which they can be triggered, and heightened responses to ordinary signals. The result is that pain can persist, spread and become disproportionate to any remaining problem in the back or neck itself. Central sensitisation is one of the principal reasons that simply addressing the original site of trouble does not always abolish the pain, and it is a key part of why this condition behaves as a chronic-pain problem rather than a purely structural one.

How it tends to present

Persistent nerve pain after surgery usually announces itself as a continuation or a return of pain in a limb after an operation that was expected to relieve it. After lumbar surgery the pain is felt in the leg, following the course of a nerve, and after cervical surgery it is felt in the arm. The pain typically carries the hallmarks of nerve pain, described as burning, shooting, stabbing, electric or like pins and needles, and it is frequently accompanied by

areas of numbness, by tingling, or by patches of skin that are unusually sensitive, where a light touch feels unpleasant or painful.

The timing varies. In some people the limb pain never fully resolves after the operation, settling only partly before reaching a plateau at which a significant amount of nerve pain remains. In others the pain eases well at first and then returns over the following weeks or months. The pattern of the pain may also have changed from before surgery, becoming more diffuse, more constant, or less clearly tied to particular positions and movements than the original compressive pain had been. This change in character is itself one of the clues that the pain has become neuropathic rather than mechanical.

Alongside the pain itself, the wider effects of a chronic pain condition are commonly described in the literature. Persistent neuropathic pain disturbs sleep, limits activity, and is closely linked with low mood and anxiety, and these in turn can amplify the experience of pain through the same central pathways already described. This is not a sign of weakness or of pain being imagined; it is the recognised behaviour of chronic neuropathic pain, and it is one of the reasons the condition is approached as a whole-person problem rather than as a purely local one. The presence or absence of any of these features in a particular person is a matter for direct assessment rather than something that can be read from a description.

How it is assessed (re-examination and imaging)

The assessment of persistent nerve pain after surgery is built around the central question of whether a fresh, surgically-correctable cause is present, and it draws on clinical re-examination and imaging together.

The clinical re-assessment revisits the story of the pain and re-examines the nerves. The aim is to characterise the pain, to map its distribution against the nerves, and to look for features that point either towards renewed compression or towards established neuropathic pain. The examination tests power, sensation and reflexes, and it looks for the signs of nerve pain such as altered sensation and heightened sensitivity. Certain features, where present, are recognised as requiring attention in their own right, and these are assessed deliberately rather than assumed.

Magnetic resonance imaging, the MRI scan, is the principal investigation. It shows the soft tissues, the discs and the nerves, and it is the test best suited to revealing whether there is any new or residual compression that an operation could address. A particular challenge after surgery is telling scar tissue apart from recurrent disc material, because both can sit around a nerve and both can look similar on plain images. Here the use of a contrast agent, gadolinium, is valuable, because shortly after it is given, scar tissue takes up the contrast and appears bright, whereas recurrent disc material does not enhance in the same way. Contrast-enhanced MRI, particularly with techniques that suppress the signal from fat, is described in the literature as the single most useful way of making this distinction, which is exactly the distinction between a correctable and a non-correctable cause.

Where MRI cannot be performed or is unclear, computed tomography, sometimes combined with an injection of contrast into the spinal canal, has a role in showing the bony anatomy and any residual narrowing. The purpose of all this imaging is not to produce a label for its own sake, but to answer the practical question on which everything else depends: is there a fresh structural target, or is there not. Imaging and clinical examination together provide that answer, and neither alone is sufficient.

What the evidence shows about management (impersonal)

It is important to state at the outset that this section describes what the literature reports about approaches to persistent neuropathic pain after surgery, not guidance for any person. Decisions in practice are individualised and made through direct assessment. The consistent thread running through the evidence is that, once a fresh surgically-correctable cause has been excluded, the problem is managed as a chronic neuropathic-pain condition rather than with further decompression.

****The role of further surgery is limited and selective.**** Where re-assessment identifies a genuine structural target, such as a recurrent disc herniation or a residual narrowing, an operation directed at that target has a rational basis. Where it does not, the evidence is discouraging. A randomised trial comparing repeat lumbosacral surgery with spinal cord stimulation in people with persistent radicular pain found that those who underwent further surgery were more likely subsequently to cross over to stimulation than the reverse, and that stimulation was the more often successful of the two. The broader literature is consistent in reporting that further decompression where there is no clear target rarely helps, which is the central practical message of the whole subject.

****Neuropathic-pain medication**** is described as a mainstay of management, used in the same way as for neuropathic pain from other causes. National guidance and international reviews describe a small number of medication classes as first choices for neuropathic pain in general terms, including certain antidepressants that act on pain pathways and certain anticonvulsants. The literature is candid that the effects of these medicines are typically modest, that they help some people and not others, and that finding a tolerable and useful option often involves trying more than one. They are described as part of a wider approach rather than as a complete answer in themselves.

****Pain-rehabilitation and multidisciplinary programmes**** occupy a central place in the evidence for chronic spinal pain. A multidisciplinary biopsychosocial programme combines physical reconditioning, education about pain, psychological approaches such as cognitive behavioural therapy, and review of medication, delivered by a team drawn from several professions. A Cochrane systematic review found that such programmes were more effective than usual care, with moderate-quality evidence, in reducing pain and disability and improving the chances of returning to work. This approach reflects the understanding of persistent nerve pain as a chronic, whole-person condition influenced by central sensitisation and by the wider effects of living with pain.

****Spinal cord stimulation and neuromodulation**** are the best-evidenced device-based therapies for this specific problem. Spinal cord stimulation works by delivering small electrical impulses to the spinal cord through fine leads, altering the pain signals before they reach the brain, and it is typically assessed first through a trial period before any permanent system is considered. The pivotal randomised PROCESS trial compared stimulation alongside conventional management against conventional management alone in people with predominant neuropathic leg pain after back surgery, and found that markedly more people achieved meaningful leg-pain relief with stimulation at six months, with benefits in function and quality of life, and that the advantage was broadly sustained at two years. Later trials of newer stimulation patterns, including high-frequency stimulation, reported improved relief of back as well as leg pain. The literature is equally clear about the limits: across studies, roughly half of carefully selected people achieve clinically meaningful relief, the benefit can attenuate over time, and the implanted hardware carries a recognised burden of revision and, less commonly, infection. Stimulation is therefore described as an effective option for selected people with predominant neuropathic limb pain, not as a cure.

****Interventional procedures****, such as epidural injections and percutaneous adhesiolysis, in which scar tissue around the nerves is broken down through a fine catheter, are described in the literature as further options with a moderate evidence base, generally used when simpler measures have not given enough relief. As with everything in this field, their place is described as one element of an individualised plan rather than as a fixed step.

In summary, the evidence points away from further surgery once a structural target has been excluded, and towards a combination of neuropathic-pain medication, multidisciplinary pain rehabilitation, and, for selected people with predominant neuropathic limb pain, spinal cord stimulation, all within a framework of individualised, shared decision-making rather than fixed rules.

Outlook

The outlook in persistent nerve pain after surgery is best described honestly and in general terms, because it varies widely between individuals. This is a chronic-pain condition, and the realistic aim described in the literature is usually meaningful improvement in pain, function and quality of life rather than the complete abolition of pain.

Many people are helped substantially, but a complete return to a pain-free state is not the typical outcome, and approaches that promise it tend to disappoint.

What shapes the outlook most is the nature of the pain itself. Where careful re-assessment identifies a genuine structural target, addressing it can improve matters, which is one of the reasons that re-assessment comes first. Where the pain is neuropathic and without a target, the outlook is governed by how well the chronic pain can be managed across its several dimensions, by medication, by rehabilitation, and, in selected people, by neuromodulation. The evidence for spinal cord stimulation in particular shows that a substantial proportion of well-selected people gain lasting and meaningful relief, while a proportion do not, and that the benefit can lessen over time.

Across the whole field, the consistent themes are that early and accurate distinction between a structural and a neuropathic cause improves the chances of a good result, that persistent neuropathic pain is a genuine condition deserving of active management rather than dismissal, and that recovery is best understood as gradual improvement shaped by the individual problem. The variation is wide, and any individual outlook is a matter for assessment by the treating team rather than something that can be read from a category alone.