UNDERSTANDING SCIATICA

TOM JESSON

with ANNINA SCHMID

Illustrated by PETER JESSON

And TOM JESSON

PRAISE FOR UNDERSTANDING SCIATICA

'Absolute gold... There's not many anatomy/physiology books that one can't put down' - June, Specialist Physiotherapist in Spinal Triage.

'I was expecting a good read, but what you get is even better! Absolutely brilliant stuff and a MUST read for any clinician who works with sciatica' - Michael, Physiotherapist.

'Simply a good book as well as a good medical book. So much contemporary content, blended with a personal, readable format like the classic medical texts of the past.' - Greig, Orthopod & GP

'The book has made a massive difference to my practice and in explaining things to my patients.' - Philip, Spinal Advanced Practice Practitioner.

'Highly recommend this book. In addition to being informative and engaging it provides sensible, well-reasoned insight, useful for patients and clinicians alike' - Michael, US military Physical Therapist.

'The sciatica Bible' - Luke, Physiotherapist.

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FOREWORD BY ANNINA SCHMID

Sciatica is a complex condition. This becomes immediately apparent as I listen to the stories of people with sciatica in my research and clinical practice. Some report tingling, burning pain and electric shocks in their leg, while others experience a numb foot. Some find sitting impossible while for others this position is the only one that relieves symptoms. Some people have muscle weakness and sensory loss, while others experience cramps or strange feelings such as water trickling down their leg. Some people are highly functioning whereas others can hardly get out of bed. Some people recover spontaneously while some develop persistent pain with repeated flare ups. Some people benefit from physiotherapy or anti-inflammatory medication, while others' pain remains resistant to such treatment. Yet we summarise all this heterogeneity under the same label of 'sciatica'. It is therefore not surprising that it remains a challenge to make sense of 'sciatica' not only for people who experience this condition, but also for clinicians.

In research, we have made substantial advances in our understanding of sciatica and nerve injuries in the past years. However, as researchers we are sometimes so immersed in our complex theories, hypotheses

and data, that we struggle or lack time to communicate our findings in an easily digestible way. After all though, science is useless if our discoveries and their clinical relevance are not translated and communicated to clinical settings.

In the 'sciatica world', we are truly lucky to have Tom Jesson. This book is a stellar example of how understanding the basic scientific principles of neuroanatomy, biomechanics, neurophysiology, biology and pain neurosciences can help us understand 'sciatica' and its clinical nuances and distinct presentations. This book thereby elegantly juxtaposes complex scientific material and clinical pearls. It is a joy to see complicated research translated into clinically relevant and digestible concepts. Importantly, Tom manages this without compromising on accuracy. His enquiring, critical mind and outstanding ability to extract the essence from scientific texts are deeply reflected in this book.

It was an absolute pleasure to have contributed a small part to the refinement of this book. I am convinced it will lead to many 'aha' moments for its readers-as it has for me. Ultimately, this book and the knowledge it consolidates will help us be better clinicians, something we are all striving towards. I hope you will enjoy reading it and discovering that cleverly marrying science with clinics is highly exciting!

'The job of a clinician is to shut up, listen, care and know something.'

— ANON

(This book will help you with that last one!)

PART I

PART I: INTRODUCTION

1

REFERRED PAIN, RADICULAR PAIN, RADICULOPATHY… AND SCIATICA

'This is the history of medicine: giving a thing a name and then everyone thinks they know all about it...'

— PETER NATHAN, 1977

In 1994, the radiologist Pierre Milette wrote to the journal Radiology on the issue of referred pain, radicular pain and radiculopathy to ask simply, 'What are we really talking about?' (1).

According to Milette not only were clinicians confused about the terminology, but so were academics. 'If we seek to improve our understanding,' Milette wrote, 'it is mandatory to address this fundamental issue.'

Let's do that now.

Referred pain

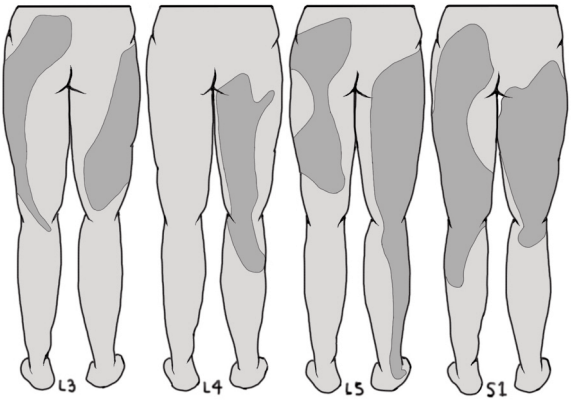
Referred pain is felt in a part of the body remote to that of the original injury. The original definition from the International Association for the Study of Pain is 'pain perceived as arising or occurring in

a region of the body innervated by nerves or branches of nerves other than those that innervate the actual source of pain' (2).

Why does this happen? The standard theory is that when danger messages (nociceptive signals) from an injury arrive in the spinal cord, they are mixed up with normal messages from other parts of the body. Those danger messages and normal messages are passed up the spinal cord to the brain together. The brain is unable to tell the two apart and creates a pain experience for both.

That's the standard theory, and it's perhaps a little over-simplified, but it gets at the basic idea of referred pain: the brain is 'confused' about the exact location of the problem.

There are two types of referred pain. First, visceral referred pain is caused by danger messages from internal organs like lungs, intestines, and kidneys. For example, danger messages from the spleen can be felt in the shoulder, and danger messages from the heart can be felt in the left arm.



Patterns of somatic referred pain. Adapted from a 193941 experiment, in this picture you can see some examples of patterns of referred pain originating from noxious stimulation of the lumbar interspinal ligaments at different segmental levels (3).

Second, somatic referred pain is caused by danger messages from somatic tissues like bones, cartilage and muscles. For example, danger messages from an intervertebral disc or a facet joint can be felt in the buttock and down the leg.

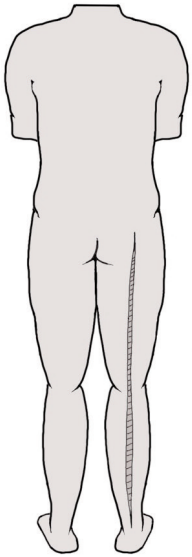
Referred pain is dull, aching, gnawing, often deep and difficult to localise.

Radicular pain

Radicular pain is a kind of nerve pain. It's caused by action potentials that emanate from the nerve root and/or the dorsal root ganglion (4). This, of course, is not where action potentials are supposed to come from. Ordinarily, they should start in nerve endings in target tissues such as skin, bone, muscle and so on.

Action potentials that emanate from within a nerve or its dorsal root ganglion are called 'ectopic impulses' (ectopic means 'in the wrong place').

In addition to these ectopic impulses, part of the clinical picture of radicular pain is likely also caused by a more generalised neuronal hyperexcitability. In response to injury at the nerve root, a neuron can amp up its defence strategy so that even normal, non-ectopic impulses are sparked more readily in response to stimuli.

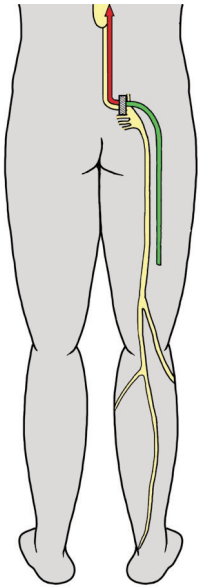


One way of representing the 'classic' radicular pain pattern. Other representations are more dermatomal. Adapted from Bogduk (4).

How does radicular pain feel? Classic radicular pain *roughly* tracks the territory of the affected nerve root. The pain is sharp, shooting, stabbing and usually severe. Often, the pain is accompanied by a dull

or burning background ache, as well as 'nervey' sensations like pins and needles and tingling.

Radiculopathy



Painful radiculopathy. Green line = normal impulses from the nerve tips: they are blocked or slowed at the injured nerve root, and will not (all) get to the brain. That's a radiculopathy. Red line = aberrant impulses emanating from the injured nerve root, and these *do* get to the brain, causing pain. That's radicular pain

Radiculopathy is another nerve problem. However, it is *not* a pain condition. Instead, the term describes *loss* of nerve function. 'Loss of nerve function' means that fewer action potentials are conducted up and down the injured nerve because of an injury to the nerve root or dorsal root ganglion. The nerve isn't doing its job.

A loss of nerve function is a pretty common everyday experience. If you sit too long and your leg goes numb, that's a loss of nerve function. A radiculopathy is not too different, although of course it involves the nerve root in the spine rather than the nerve trunk in the periphery, and often involves more lasting damage to the nerve too.

Radiculopathy manifests as a dulled or absent reflex response, a loss of sensation to different sensory stimuli (e.g., touch, sharp prick, warm/cold), and/or a loss of muscle strength.

The clinical picture is less clear cut

Of course, referred pain, radicular pain and radiculopathy can all occur together; all three can overap

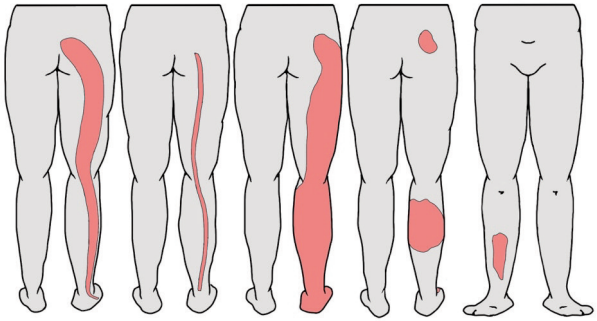
First, although radicular pain and radiculopathy can occur separately, they often co-exist as a 'painful radiculopathy'. This makes sense, of course - they both involve a problem with a nerve root, so it's not surprising that such a problem can cause both pain and loss of function. Sometimes the radicular pain is serious and the radiculopathy is mild, and sometimes it's the other way round - and everything in the middle.

And referred pain can also co-exist with radicular pain and radiculopathy. If you think about it, this makes sense. Imagine a big disc herniation that injures a nerve root and causes radicular pain. That disc herniation and all the associated inflammation might easily also trigger nociceptive signals from the disc and the surrounding tissues. That nociception might cause referred pain in the buttock or down the leg.

So, where there is radicular pain there can also be some somatic referred pain.

These mixed pain presentations partly explain why so many cases of radicular pain do not look like they are 'supposed' to look: it's often not only radicular pain, but radicular *and referred* pain.

Even aside from the presence of referred pain however, radicular pain itself can deviate from the 'classic' picture of a band of pain that tracks the territory of the affected nerve root, i.e. a dermatome of pain. In fact, numerous studies show that it's near-impossible to tell from the pain pattern alone what nerve root is causing pain (5–7); radicular pain doesn't obey the textbooks' dermatomes. On top of that, radicular pain can also expand beyond the expected band of pain to a wider territory (8).



Varieties of nerve root pain. From left to right: Classic band-like, dermatomal pain, often shown in textbooks; classic thinner band of pain, as described by Bogduk (4); extra-territorial spread; 'jumping' pain; occult patches of pain.

Sometimes, nerve root pain doesn't form a continuous line at all, but leaps from patch to patch, for example from the buttock to the shin to the big toe; or even just show up in one patch - *just* the buttock, or *just* the shin, or *just* the big toe (9). We don't want to overstate this variety; the classic picture is the classic picture for a reason. But it's important to know that radicular pain, like most pain, can present atypically.

Sciatica

> *'Amongst painful diseases, sciatica occupies a foremost place by reason of its prevalence, its production by a great variety of conditions, the great disablement it may produce, and its tendency to relapse; all of which have long ago led to its recognition as one of the great scourges of humanity'*
>
> — - VITTORIO PUTTI, 1927 (10)

The word ‘sciatica' is less a diagnosis and more of a vague gesture - 'there’s pain in the back of the leg... for some reason'. It doesn't really have any official definition and different people mean different things by it. It's a throwback to a time when we had much less medical knowledge. The eminent spinal surgeon Jeremy Fairbank even called sciatica 'an archaic term' (11).

One problem with ‘sciatica’ is that it means different things to different people. Of course, the same could be said for ‘referred pain’, ‘radicular pain’ and ‘radiculopathy’, which, as Milette complained, are often used carelessly. But for those words there is at least an official definition to refer to. Not so for sciatica. To some people, it means radicular pain, to others it means any pain that comes from the spine but is felt in the leg, including referred pain, and to still others it means any pain in the back of the leg that seems to have something to do with a nerve (12).

That said, sciatica is a recognisable word for laypeople, which is important. And it's a useful word for clinicians who want to refer to everything we’ve looked at above without having to say 'referred pain or radicular pain or radiculopathy or some mixture of the three'! It's kind of a catch-all term, in that respect. We use it all the time - in the title of this book, for example! So, whereas the scientific community discourages the use of the term ‘sciatica’ (13), its widespread use by laypeople and its usefulness as a colloquial catch-all means it is likely to stick around.

That’s enough place-setting! Let’s get started.

Key points on referred pain, radicular pain and radiculopathy:

- Referred pain is when pain from tissues like muscles, joints and discs is felt in the wrong place. It is usually a diffuse ache.

- Radicular pain is when pain from the nerve root in the spine is felt roughly in the territory of that root. In the case of lumbar radicular pain, that's down the leg. Radicular pain is usually sharp and severe.
- Radiculopathy is when an injury to the nerve root stops it from conducting impulses to and from the brain. This makes muscles weaker and sensation duller.
- Because all of these things can exist together, in different amounts, the clinical picture is often far from clear! Additionally, radicular pain itself has a varied presentation, not always appearing in the expected dermatome.
- Sciatica is an old-fashioned term without any specific meaning. Despite this, it is an easy way to refer to pain down the back of the leg that seems to be related to a nerve.

2

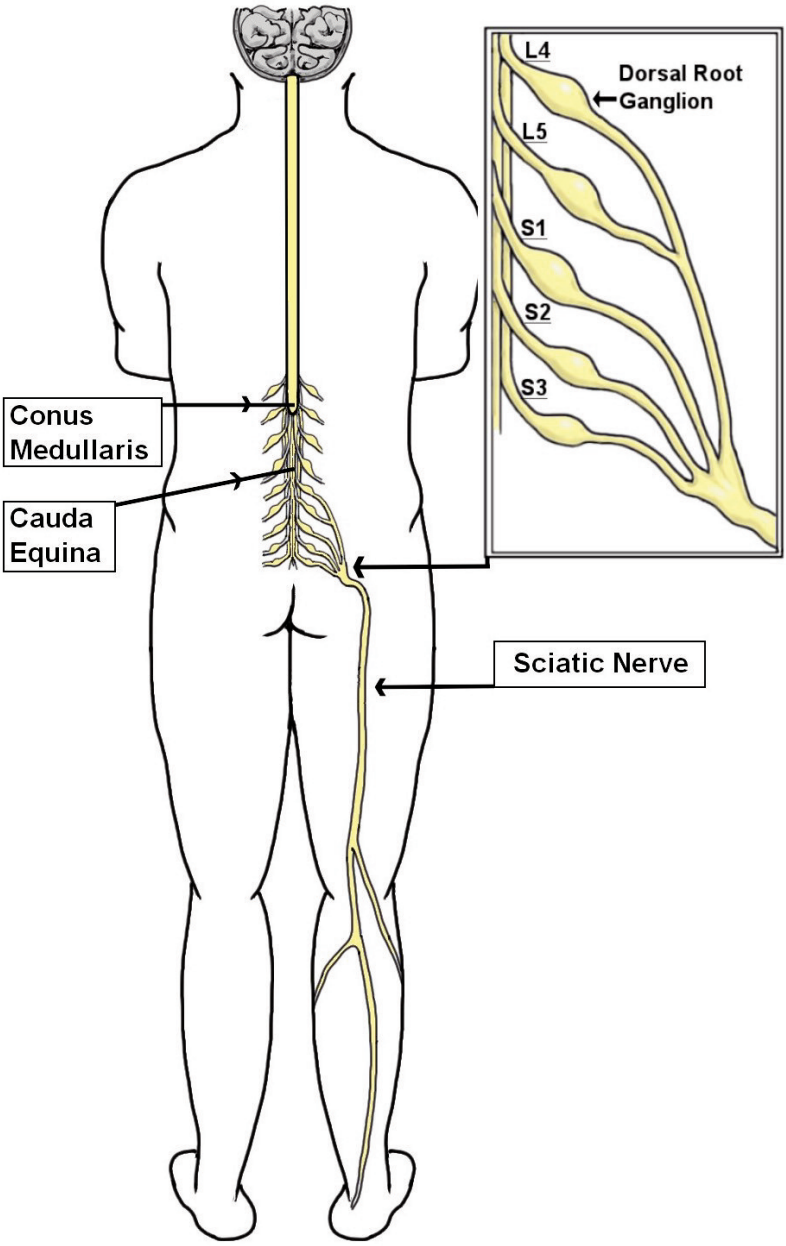
AN ANATOMY TOUR

Let's start with a tour.

On this tour, we will follow the primary sensory neurons. We will start at the top, where each primary sensory neuron synapses in the spinal cord in the upper lumbar spine, and many of them are bundled together as rootlets. Then, we'll follow these neurons downward through the spine as they bundle together again into nerve roots, then jumble together into the spinal nerve, and finally exit the spine through the intervertebral foramen. Finally, we'll continue to follow them down as, now bundled together as peripheral nerves, they continue their journey to the tips of the toes and other tissues.

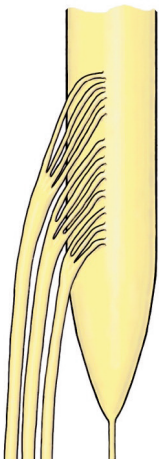
Rootlets become roots; many roots make up the cauda equina.

In infants, the end of the spinal cord is at about the L3 vertebral level. But as we age, it is outgrown by the rest of the body and by the time we are in our teens, the end of the spinal cord is further up the spine, at L1 or L2. The cord tapers to terminate at the conus medullaris.


L4
Dorsal Root
Ganglion
L5
S1
S2
S3
Conus
Medullaris
Cauda
Equina
Sciatic Nerve

The nerve roots enter and exit the spinal cord not as fully formed roots, but as rootlets. There are dorsal (posterior) rootlets, which are made up of sensory neurons carrying impulses from the body and its environment up to the spinal cord. And there are ventral (anterior) rootlets, which are made up of motor neurons carrying impulses from the spinal cord down to the muscles.

After the rootlets bud off from the spinal cord, they form sub-bundles, and then form bundles once more to become the nerve roots that make up the hanging tail of the cauda equina. When they bundle, dorsal rootlets stick together and ventral rootlets stick together, making separate dorsal sensory roots and ventral motor roots. Dorsal roots are thicker than ventral roots because there are more sensory than motor neurons in peripheral nerves.

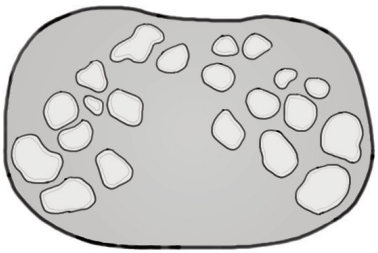


Schematic diagram showing how the cord tapers into the conus medullaris. The L4, L5 and S1 dorsal rootlets bud off the spinal cord and bundle to become nerve roots.

The lumbar spinal cord is small, about the size of a little finger, and the roots are smaller still: between two and four millimetres in diameter in the lumbar spine. When an anatomist is laying the roots out or holding them, they seem like tangled spaghetti, hanging off a fork. In the body, where they are guided and held by ligaments and connective tissue, they have a more orderly and linear appearance, like uncooked spaghetti still in the packet (although of course they are not hard but soft and pliable, with the consistency of rubber).

The cauda equina is inside the dural sac.

Everything we have seen so far is taking place inside the protective dural sac (sometimes called the thecal sac). The dural sac envelops the spinal cord in the cervical and thoracic spine and, after the spinal cord terminates in the upper lumbar spine, it descends into the lumbar spine to protect the nerve roots as they make up the cauda equina.



Cross section of the lumbar and sacral nerve roots in the dural sac, adapted from Cohen and colleagues (14)

The dural sac has two layers. The outer layer is made up of the tough dura mater ('dura' comes from the same root word as 'durable'). The inner layer is the thin, transparent arachnoid membrane.

Beneath the arachnoid membrane, pumping back and forth on its slow course around the brain and spine and bathing and nourishing the cauda equina, is the cerebrospinal fluid (CSF). The pressure of the CSF helps to balloon up the arachnoid membrane and dura mater into a plump sac shape (after it's been dissected from the body and it's lost its CSF, the dural sac is more of a sad, flat sleeve). As is evident from pictures of the dural sac in cross-section, the roots have quite a lot of space and shift around as we move.

Between the CSF and the nerve roots is the innermost layer of protection, the delicate pia mater. The pia mater, which covers the brain in the skull and the spinal cord in the cervical and thoracic spines, surrounds the individual nerve roots in the lumbar spine. If you were

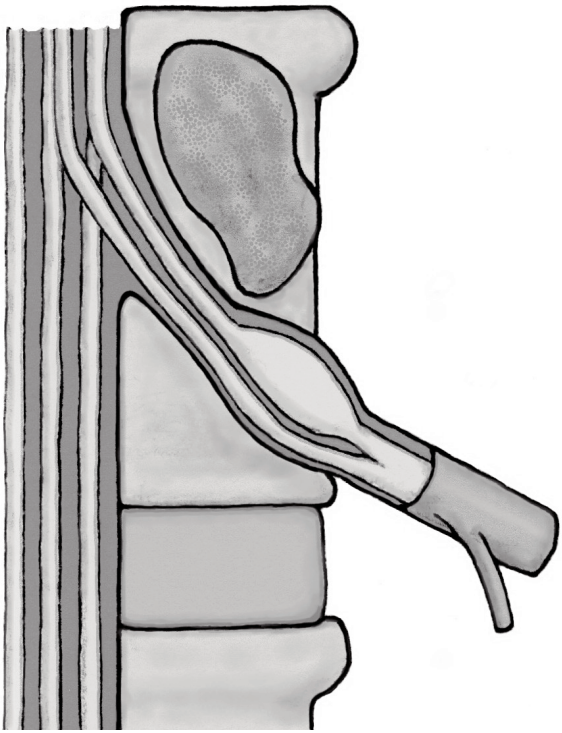
to remove the pia mater from a nerve root and roll the root in your fingers, it would de-bundle like loose twine into the separate rootlets that we originally saw budding off from the spinal cord.

If it was not already clear, these three layers - the dura mater, the arachnoid membrane and the pia mater - are the same contiguous membranes that cover the brain, too.

At each spinal level, roots change direction and head for the intervertebral foramen.

At each spinal level on the cauda equina's downward course through the dural sac, one dorsal and one ventral root per side will pair up and branch off together. They will go forth and innervate their particular patches of the low back and leg. As they branch off, the pair of roots take with them a portion of the dural sac, which will now form the nerve root sleeve. The sleeve binds the two roots more tightly than did the dural sac, holding them on course. As the space inside the sleeve is continuous with the space inside the dural sac, there is CSF in here too, nourishing the nerve roots.

In the short part of their course after they leave/enter the dural sac and before they leave/enter the spinal column completely, the nerve roots are at their most vulnerable. Although these 'extra-dural, intra-spinal' roots still have the protection of the dura mater and the arachnoid membrane (now in the form of the nerve root sleeve), they do not have the extra space and freedom of movement they had inside the dural sac. This makes them vulnerable to anything that would compress, stretch, pin or twist them - like a disc herniation. Not only that, but although the root sleeve is tough, it is far less tough than the layers of connective tissue that will protect peripheral nerves proper, once they are out of the spinal column and coursing down the leg.



This picture shows the dorsal and ventral nerve roots deviating from their downward course to leave the cauda equina and the dural sac. They take with them part of the dural sac which is now called the nerve root sleeve, shown in dark grey. (Somewhat confusingly, when a sensory and a motor nerve root are bound together like this, we still colloquially refer to them as 'the nerve root', i.e. as a singular when in fact they are distinct entities.) The dorsal root is continuous with the dorsal root ganglion. Shortly after, the roots blend to become the mixed spinal nerve. Before too long, they branch into the ventral and dorsal rami.

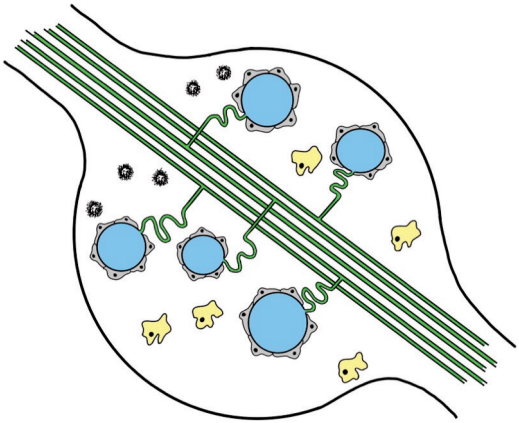
This short interval in which the dorsal and ventral roots are outside the dural sac but still inside the spinal column is called the radicular canal. It can be thought of as a passageway, with the door at the end being the opening of the intervertebral foramen. To pass through the canal, the roots, which have so far been descending in a straight line

downwards, have had to swoop laterally as if exiting on a sharp slip road (or off ramp, for American readers!). Emphasising this swooping motion, they hug the ceiling of the passageway within the foramen.

The dorsal root ganglion (usually) sits in the intervertebral foramen

Inside the radicular canal, the dorsal (sensory) roots exit from the dorsal root ganglia. (The ventral roots, which have their cell bodies within the ventral horn of the spinal cord, course closely by). The root sleeve, which has protected the two roots since they left the dural sac, ends here. The dura mater of the sleeve blends into the capsule of the ganglion, soon to become the tougher connective tissue layers that protect the peripheral nervous system. The arachnoid membrane below it pinches off just before it reaches the dorsal root ganglion. This pinching cuts off the CSF, which means this point marks the last outpost of the central nervous system and everything beyond it is the peripheral nervous system.

The dorsal root ganglion is usually located in the foramen, sometimes slightly distal or proximal to it. In the lumbar spine, it is about the size of the fingernail on your little finger. Inside it are the cell bodies of just over 10,000 sensory neurons, each one with a diameter of less than 100 micrometres, which is about the width of a strand of hair. These cell bodies manufacture all the parts that the primary sensory neuron needs in order to function, and ship them out to the rest of the cell. They sit off from the rest of the primary sensory neuron at the end of a T-junction.



Schematic diagram of the dorsal root ganglion. Axons, green, pass through. They send off T-junctions to cell bodies, blue. Cell bodies are surrounded by satellite cells, grey. Resident macrophages shown in yellow. T-cells cluster to the CNS-side of the ganglion.

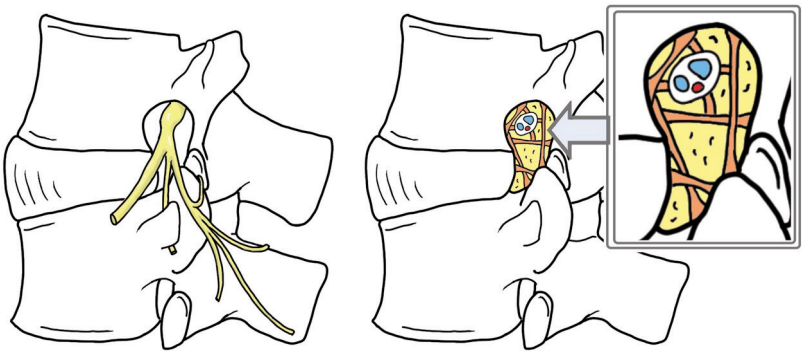
The cell bodies of sensory neurons are not the only cells inside the dorsal root ganglia. For instance, satellite glial cells surround each cell body. These glial cells have a protective and cushioning function akin to that of the Schwann cells in the peripheral nerve trunk. They also closely interact and communicate with neurons using, for instance, immune mediators. Additionally, the dorsal root ganglion also contains immune cells such as macrophages (15).

Although the ganglion is thicker than the nerve root, it still does not take up more than one third of the foramen. The rest of the space inside the foramen is taken up by blood vessels, ligaments, the sinuvertebral nerve (heading back into the canal) and cushioning fat. Below, you can see two drawings of the teardrop-shaped intervertebral foramen.

Distal to the dorsal root ganglion, the nerve roots become the mixed spinal nerve

Distal to the dorsal root ganglia, the pair of nerve roots, up until now held closely but separately, undergo a major change: they are finally woven together into the mixed spinal nerve. This means that the spinal nerve contains both the motor and sensory neurons of its spinal level.

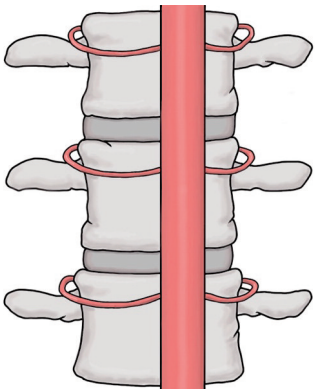
Compared to the length of the lumbar nerve roots, the lumbar spinal nerve is remarkably short: as soon as it has left the foramen, it branches off into two rami. The dorsal and ventral rami are mixed sensory and motor nerves that serve the structures of the spine and the lower limb, respectively. We have now left the nerve roots and the dorsal root ganglion behind.



Two views of the intervertebral foramen. The picture on the left shows the dorsal root ganglion blending into the mixed spinal nerve which soon splits into the dorsal and ventral rami. The picture on the right shows a cross section of the nerve root (blue). Note the dorsal and slightly smaller ventral root, accompanied by the distal radicular artery (red) in relation to the latticework of intra-foraminal ligaments (orange). Note also the fatty tissue (yellow) which takes up much of the rest of the space. The fact that the foramen houses these other tissues enables nerve root irritation without direct contact, as we will see later.

The nerve roots are supplied by the radicular arteries.

Let's pause here to look at how the nerve roots and the ganglion get their blood supply, because the blood supply is one of the most under-rated factors in radicular pain!



A view from the front of the vertebral bodies. At each spinal level, the descending aorta sends out a pair of arteries.

Making its way down from the heart, the descending aorta sits on the front of the vertebral bodies. At each spinal level, a pair of arteries branch off from the aorta and course backwards around each side of the vertebra. They look as if they are a pair of arms hugging the vertebral body. As they make their way backwards, these arteries send branches off to various parts of the vertebra, and one of these branches enters the intervertebral foramen. This is the distal radicular artery. As it enters the foramen, the distal radicular artery penetrates the spinal nerve and follows it in, in the opposite direction we took on our tour. When it reaches the nerve roots, it splits in two and continues to course up both. On its way past the dorsal root ganglion, it forms a plexus around it.

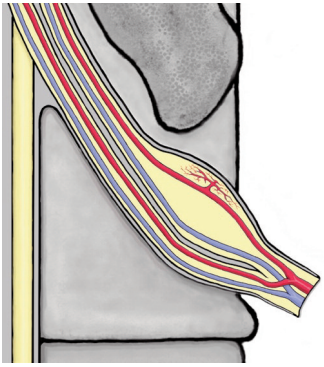
As it continues, it gives off more collateral branches which become tiny capillaries in each bundle of neurons inside the root. It ends about two thirds of the way up the roots. It is through this distal radicular artery that the heart pumps blood up the nerve roots, towards the spinal cord.

The proximal radicular artery begins in the conus medullaris and travels down the roots. At first it travels alone, because capillaries from the conus can supply the most proximal parts of the roots. But

after a few millimetres, the proximal radicular artery swerves and pierces the root. Like the distal radicular artery, it gives off collateral branches as it goes, which become tiny capillaries in each bundle of neurons. It is through the proximal radicular artery and its branches that the heart pumps blood down the nerves, away from the spinal cord.

The proximal radicular artery meets the distal radicular artery about one third of the way down the root, and the two blend into one another, their currents mingling.

Blood is drained from the root by radicular veins. Inside the nerve root, the veins run in spirals. Outside, they form large, thick plexuses throughout the radicular canal. The radicular veins drain de-oxygenated blood posteriorly, towards the spinous processes; the opposite direction from which it came. From there, it will continue to the lungs to be re-oxygenated.



Blood supply to the nerve root complex. The distal radicular arteries (red) enter the nerve roots through the spinal nerve, on the right. The proximal radicular arteries arrive from the left, having come from the conus medullaris, out of shot. Blood flow from each converges at a watershed about two thirds of the way up the root. Veins shown in blue.

The nerve root complex.

We are about to leave the spine, but before we do, let's pause one more time, look back and introduce a new term to describe much of what we have just seen: the nerve root complex. This term is used by Butler in his 2000 book The Sensitive Nervous System (16), and he writes that he took it from Rauschning (17), who was writing a couple of decades prior. You can also read Sunderland talking about the 'radicular complex' in 1974 (18). We will use ‘nerve root complex’ to mean the dorsal and ventral nerve roots, the dorsal root ganglion,

the nerve root sleeve and the radicular veins and arteries. It's a useful term when you want to refer to this collection of anatomically and physiologically different structures which bundle into a coherent whole, and all have their role to play in radiculopathy and radicular pain.

The plexuses become the femoral and sciatic nerves.

After the spinal nerve branches off into rami, the neurons in the ventral ramus travel off to the buttock and leg. Before they do, however, they intermingle neurons from other spinal levels in a structure called the lumbosacral plexus. Specifically, neurons from the L4, L5, S1, S2 and S3 spinal level combine to form the sciatic nerve, and neurons from the L2, L3 and L4 spinal level combine to form the femoral nerve. In other words, by the time our sensory neurons are in the buttock and leg, they are thoroughly jumbled up, each nerve contains neurons from many different spinal levels. This is why a radiculopathy, i.e. an injury at the nerve root, rarely causes complete strength loss: by the time the neurons that have been injured at the nerve root connect with muscle in the leg, they are working alongside uninjured neurons from other spinal nerve root levels. Typically, those uninjured neurons maintain some strength without their injured colleague.

(By the way, the name for each nerve root and spinal nerve is taken from one of the vertebrae where it exits. In the lumbar spine, a nerve is named for the vertebra above it. So, if we look at the intervertebral foramen between L4 and L5 we will see the L4 nerve roots and L4 spinal nerve. This rule is simple, but there is a source of confusion: because of a mismatch between the number of nerve roots and the number of levels, in the cervical spine we name them the other way round, after the vertebra below.)

Beyond the rami and the plexuses, nerves containing sensory and motor neurons from, mostly, multiple spinal levels, proceed into the

buttock and leg. Motor neurons end in muscles. Sensory neurons originate from everything we can feel: muscles, bones, ligaments, cartilage, connective tissue, the skin, and the follicle of the smallest hair. If we look closely at the tips of these sensory neurons, we will see that they either form a receptor (e.g. a Meissner or Pacinian corpuscle) or have free nerve endings which are covered with channels.

Sensory receptors and ion channels start action potentials.

At the tips of sensory neurons, there are sensory receptors and ion channels, which work together to convert stimulations into electrical signals called action potentials.

Ion channels are proteins embedded in the membrane of sensory neurons that allow ions to pass through, into and out of the neuron. There are lots of different types of ion channels, and you don't need to know them individually to understand this book. But here are a few examples that you might have heard of. There's TRPV1 channels, which react to thermal stimuli, including sunburn. There's acid sensing ion channels, which are activated by a drop in tissue pH during ischemia. And there are voltage gated sodium channels, which react to a change in membrane potential to create and transmit action potentials. In addition to ion channels, there's receptors such as cytokine receptors, which are activated by tissue inflammation. But, as we say, you don't need to remember each one, and it's sufficient to think of the ion channels and receptors as a group - the cell machinery that allows a sensory neuron to 'feel' what's going on around it.

Ion channels and receptors are not born in the neuron tips (where they mostly reside) but, as we saw earlier, way up in the dorsal root ganglion. To get down to the neuron tips, they are actively carried along microtubules, a process called axonal transport. That means that the axon of a nerve is not an inert wire waiting for impulses; it is also a conveyor belt for the machinery that makes the cell work. This

will become important later as we watch how radicular pain develops: as we will see, ion channels' and receptors' journey along an axon can be interrupted by an injury or inflammation, which makes them bunch up into an abnormally sensitive spot, contributing to neuropathic pain. Not only that, but after an injury to a primary sensory neuron, ion channels and receptors are also produced and shipped out along the axon in large numbers, which can make the neuron hyperexcitable.

And with that, we have finished our tour. We are down in the tissues, watching action potentials spark off in a primary sensory neuron's tips. They will zip up the neuron's axon, through the nerve root complex and up to the spinal cord and brain, in the opposite direction to that we've just traveled.

Now, you've seen everything you need to see to start building your understanding of sciatica.

Key points from the anatomy tour:

- The lumbosacral primary sensory neurons synapse with the spinal cord in the upper lumbar spine, and have their nerve terminals in the tissues of the pelvis, buttocks, legs and feet. The nerve root is just a short length of a bundle of these very long neurons.
- The lumbosacral nerve roots bud off from their respective spinal cord segment and travel down the lumbar spine as the cauda equina, where they are protected by the dural sac.
- At each spinal level, two pairs of sensory and motor nerve roots exit the dural sac, one pair on the left and one pair on the right. As they go, they take some of the dural sac with them as the nerve root sleeve.
- The nerve roots travel a short distance into the foramen. Around here, the dorsal nerve root has its ganglion, which

houses the cell bodies of the sensory neurons in the nerve root.

- Distal to the dorsal root ganglion, the sensory and motor nerve roots blend together into the mixed spinal nerve. This is the point at which the nerve root ends.
- The spinal nerve is very short, and the neurons inside soon branch off to innervate the lower back and leg.
- The nerve roots are supplied by the radicular arteries.
- The extra-thecal part of the nerve root, the dorsal root ganglion, the mixed spinal nerve, the nerve root sleeve and the radicular arteries can usefully be described as 'the nerve root complex'.
- Sensory neurons innervate all tissues that we can feel. In their nerve endings, they have ion channels and specialised receptors. These channels and receptors convert stimuli like touch, heat, cold, and chemical changes into action potentials, which are sent up to the spinal cord and may eventually reach the brain.
- These ion channels and receptors are manufactured in the dorsal root ganglion and transported to the sensory nerve endings by axonal transport. As we will see, problems with the manufacturing and transport of these channels and receptors is a significant mechanism of radicular pain.

PART II

PART II: THE NERVE

3

NERVE ROOTS CAN BE INJURED BY 1) MECHANICAL PRESSURE AND 2) CHEMICAL IRRITATION

'After all, it might be said that sciatica is hardly completely explained by the knowledge of disc protrusions...'

— LENNART SÖDERBURG, 1956 (19)

How pressure can injure nerve roots.

Mechanical pressure can injure nerve roots. But pressure means lots of different things - not just compression! To help paint the picture, here's an unofficial but hopefully intuitive typology of the three kinds of pressure that can injure nerve roots.

The most dramatic kind, and what most people think of when they picture a disc herniation injuring a nerve root, is squashing. Here are the researchers Smyth and Wright describing such a case: 'At operation there was a large disc protrusion pressing on the [...] nerve root, pushing it laterally. The root was wedged between the disc and the lateral wall of the spinal canal. Compression was severe' (20).

But squashing is probably rare compared to the second type of mechanical pressure, which is bowstringing (21).

Bowstringing happens because the root is relatively fixed in place against the disc side of the spinal canal by a set of ligaments. And, as we saw earlier, the root is further restricted by the tight swaddling of the root sleeve. Describing the fixity of the root, the neurosurgeon Murray Falconer wrote in 1943 that 'attempts to displace the [...] nerves are resisted, and if displaced the structure springs back into its original position when released' (22).

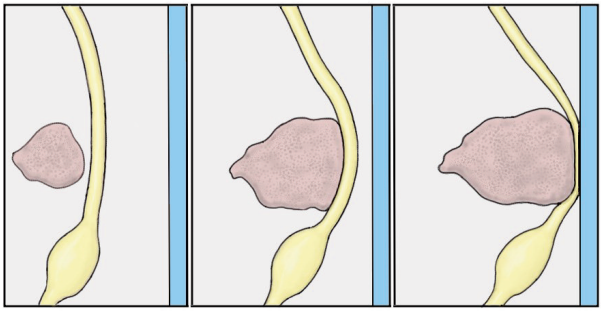
This fixity means that, in contrast to the roots inside the dural sac which can be compared to loosely-hanging ropes, once the roots are in the root sleeve they are more like guy ropes on a tent. And if you push your hand against the guy rope on a tent, it doesn't move much. Instead, it bowstrings. As it bowstrings, it distorts, stretching, thinning, and squashing in on itself. (Note that this is not simply 'compression'; the mechanical forces here are quite varied).

Nerve roots are not loose, but fixed at both ends like the guy rope on a tent

A disc herniation can have a similar effect on a nerve root. Observing this, Falconer described a 'compressing angulating as [the nerve root] was pulled taut and bent over the prolapse'. Here's Smyth and Wright describing the same thing: 'At operation there was a large dome-shaped herniation pressing on the [...] nerve root. The root was not compressed against the lamina.' This is bowstringing!

Finally, a nerve root can also be injured by mechanical pressure that is not as frank and direct as squashing or bowstringing. It might not even involve nerve root contact. This third type of pressure is 'crowding out'. As well as disc herniations, crowding-out can be caused by changes like osteophytes, ligament thickening, disc flattening, disc bulging or even oedema from inflammation or venous congestion (more on that later).

How can crowding-out injure a nerve if the nerve itself is not deformed and perhaps not even contacted? Well, the nerve root shares its space in the radicular canal with blood vessels, ligaments, fatty tissue and other nerves. So it can easily get tight in there. Think of standing in a crowded lift (or elevator!) and one more person gets on. As we will see soon, this little bit of added pressure on a root can be enough to affect its blood supply, causing a loss of nerve function, Schwann cell dysfunction and even inflammation.



Schematic diagrams showing the different types of mechanical pressure that a disc, or any other intra-spinal mass, can exert on a nerve: crowding out, bowstringing and squashing.

How chemical irritation can injure nerve roots.

Sometimes this chemical irritation is introduced by surrounding structures. Most commonly, the culprit is disc material. Disc material chemically irritates a nerve root for two reasons. First, it can contain pro-inflammatory chemicals that contact neural tissue when the disc ruptures. Second, the innermost material of a disc is foreign to the immune system, so it can cause an autoimmune-inflammatory reaction that catches neural tissue in the cross-fire. Smyth and Wright might have been seeing disc-material-induced chemical irritation when they described this painful nerve root: 'At operation there was a small nodular herniation touching the fifth lumbar-nerve root. The

root was quite free, there were no adhesions, nor did there appear to be any pressure' (20).

There are other surrounding structures that can introduce chemical irritation to a nerve root. For example, facet joint inflammatory chemicals can irritate the root (23). Malignant tumours release inflammatory chemicals, too (24).

Sometimes, chemical irritation is not introduced by a surrounding structure but arises as a response to mechanical pressure. Pressure, whether squashing, bowstringing or crowding-out, can damage a nerve (for example, through demyelination or axon degeneration). And damage can cause inflammation. Again, don't worry, we will look at all this in much more detail later, too!

You will have noticed by now that mechanical pressure and chemical irritation rarely work alone. Most often, they are accomplices. In various ways, each one can lead to the other. And the most common cause of radicular pain, disc herniations, can be a cause of both simultaneously.

Nevertheless, it helps to split them up into these two categories. It's a good way to make sense of things. For the rest of the first half of this book, we are going to use mechanical pressure and chemical irritation to understand how a nerve root can lose function, and how it can induce pain.

Key points for nerve root injuries

- Mechanical pressure can injure nerve roots.
- There are different types of mechanical pressure. Squashing is when a nerve root is compressed against something. Bowstringing is when a nerve root is displaced along its course. Crowding-out is when a nerve root is not necessarily

contacted physically by tissue, but is indirectly mechanically disturbed.

- Chemical irritation can injure nerve roots
- There are different kinds of chemical irritation. Disc material is a chemical irritant to nerve roots. Inflammation from nearby joints can irritate nerve roots. Pressure itself can cause an inflammatory reaction inside nerve roots. Ischaemia can also irritate a nerve.
- In reality, mechanical pressure and chemical irritation overlap, but looking at each one separately is a useful way to understand sciatica.