Radiculopathy, radicular pain and referred pain: what are we really talking about?

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Pain that radiates from the neck down the arm, or from the back down the leg, is a common presentation in clinical practice. However, despite the importance of standardisation in physiotherapy practice, there exists an abundance of terms to describe such symptoms, that may not only be confusing to patients, but may worry them unduly if explanations imply that their body is vulnerable or damaged. Therefore, communicating a diagnosis to the patient in a way that is honest, reassuring and empowering is very important. This article also aims to clarify these terms, explain the pathophysiology behind them, consider their limitations in light of recent developments in pain science.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 To clarify the terminology of radicular pain, radiculopathy and referred pain.
2 To consider these terms in light of the complexity of pain science.
3 To address how to explain radicular pain and radiculopathy to our patients.

Introduction

In 1941, the rheumatologist Jonas Kellgren wrote a paper in *The Lancet* lamenting that back pain with leg pain was, in his experience, too often diagnosed as "sciatic neuritis" when in fact it was nothing of the sort (Kellgren 1941). He had performed a series of experiments showing that noxious stimulation of the ligaments and muscles of the spine produced segmental areas of referred pain running down the buttock, leg, shank and foot; a referred pain which he accused the "neurological school" of physicians of over-diagnosing as nerve pain. Assessing 90 x-rays of patients diagnosed with sciatica, Kellgren judged that 50 of them were in fact suffering referred pain.

Later, in 1994, the radiologist Pierre Milette wrote to the journal *Radiology* on the issue of radicular and referred pain to ask simply, "what are we really talking about?" (Milette 1994). Like Kellgren, Milette accused his colleagues of over-diagnosing sciatic nerve root pathology, assuming that any back / leg pain was neurogenic. He pointed to confusion surrounding the terminology for back / leg pain not only clinically but even at an academic level. "If we seek to improve our understanding", Milette wrote, "it is mandatory to address this fundamental issue".

Today, despite Kellgren and Milette's pleas, there is still some confusion about the terms *referred pain*, *radicular pain* and *radiculopathy*. This article aims to address this confusion on two levels. First, at the level of *communication between clinician and clinician*: in order to reduce unwarranted variation in practice it is important to, as Milette put it, be clear what we are really talking about. Second, at the level of *communication between clinician and patient*: as physiotherapists are becoming more aware of the impact of the words we use, and their power to both harm and heal, we need to improve on our common lexicon so we can explain nerve root pain in a way that is clear, reassuring and empowering.

Communication between clinician and clinician

BASIC TERMINOLOGY (TABLE 1)

Referred pain, or *somatic referred pain*, is

	MANIFESTATION	MECHANISM
SOMATIC REFERRED PAIN	Dull, aching, gnawing and difficult to localise pain, with an inconsistent and non-dermatomal pattern	"The convergence of nociceptive afferents on second order neurons in the spinal cord that also happen to subtend regions of the lower limb" (Bogduk, 2009)
RADICULAR PAIN	Lancinating, shocking, electric feeling in a thin band	Compression and / or inflammation leads to ectopic (i.e. spontaneous, abnormal) discharges emanating from the dorsal root or its ganglion
RADICULOPATHY	Objective neurological signs: loss of function in reflexes, power and sensation. (Not necessarily pain)	Compression and / or inflammation leads to demyelination and axonal damage, causing reduced impulse conduction along the nerve

TABLE 1: Basic terminology of the manifestation and mechanism of pain (Bogduk 2009; Baron *et al* 2016)

"AS PHYSIOTHERAPISTS, WE ARE BECOMING MORE AWARE OF THE IMPACT OF THE WORDS WE USE"

a result of nociception (danger messages) arising from the structures of the spine (Bogduk 2009; Baron *et al* 2016). The proposed mechanism is the "convergence-projection model". According to this model, afferents from the lumbar spine and from the lower limb converge on the same second order afferent in the spinal cord, and so the brain cannot discriminate between the two sources of input when they are projected up. As a result, the brain creates a pain experience for both, even though the lumbar spine is the only significant source of nociception. This is the same mechanism by which visceral referred pain, like the shoulder and arm pain of a heart attack, is assumed to work.

Somatic referred pain is dull, aching, gnawing and difficult to localise (Bogduk 2009). Figure 1 illustrates the results of Kellgren's (1941) aforementioned experiments to find the referred pain patterns for each spinal segment.

Radicular pain is a result of an action potential emanating from the axon of a nerve at the dorsal root, or at the nerve root ganglion, as opposed to resulting from stimulation of the nerve's peripheral terminals (Bogduk 2009; Baron *et al* 2016). This discharge is "heterospecific", a barrage of AB, Ad and C fibre discharges, and so technically it is not nociceptive.

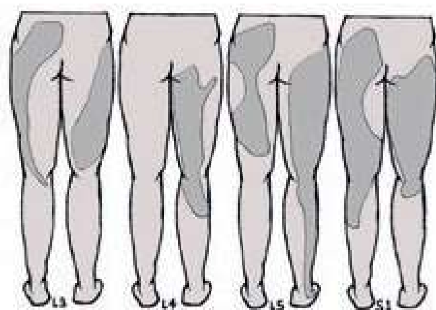

FIGURE 1: Patterns of somatic referred pain evoked by noxious stimulation of the interspinous ligaments at the segments indicated. Based on Kellgren (1941)

FIGURE 2: An illustration of the lancinating quality of radicular pain travelling into the lower limb along a narrow band.

Figure 2 illustrates the result of Kellgren's (1941) experiments to induce radicular pain. The subjects reported this pain as a lancinating, shocking, electric sensation.

The pathophysiological process is complex. Compression of the nerve, whether by a disc or its exudate, or by foraminal stenosis, reverses the downward pressure gradient necessary to maintain the nerve's blood supply causing it to become ischaemic. One way in which this leads to pain is by causing Schwann cell death and demyelination, which exposes a portion of axon from where spontaneous action potentials can emanate to be processed by the brain and experienced as pain (Schmid 2015). Another way in which compression and ischaemia lead to pain is by inciting intraneural inflammation, which alone is sufficient to sensitise a nerve to movement (Dilley *et al* 2005) and can spread to the spinal cord and the brain (Albrecht *et al* 2018). In addition, intraneural inflammation impairs axonal transport so that ion channel components being transported along the nerve bunch up in one place, creating a "hot spot" of excitability (Dilley *et al* 2013).

The mechanisms of radicular pain are not fully understood and more complex than is suggested by the neat description here; for a more comprehensive explanation see Schmid (2015).

As figure 1 demonstrates, experimentally induced radicular pain manifests in a clear line that approximates the territory innervated by the affected nerve root. Clinically, however, the boundaries of radicular pain are less clear. Bove *et al* (2005) found that, of 25 patients with lumbar radicular pain, all but one reported multi-dermatomal pain. Similarly, Furman & Johnson (2018) found that only radicular pain on the anterior, medial and lateral thigh had any correlation with its nerve root origin, whereas buttock, gluteal and calf pain had none (figure 3). The non-dermatomal presentation of radicular pain is likely because intra-neural inflammation has the potential to spread to the dorsal horn and beyond, which would sensitise neighbouring nerves (Schmid *et al* 2013a); and because nerve roots have multiple intrathecal anastomosis (cross linkages) (Furman & Johnson 2018).

Radiculopathy is the objective loss of function of a nerve resulting from damage to the nerve root (Bogduk 2009; Baron *et al* 2016). This manifests clinically as loss of muscle power, reflex response, and the sensation of light touch, hot / cold response and response to pinprick. As discussed, a compressive insult to the nerve can set off a chain events that cause it to become demyelinated and inflamed, in

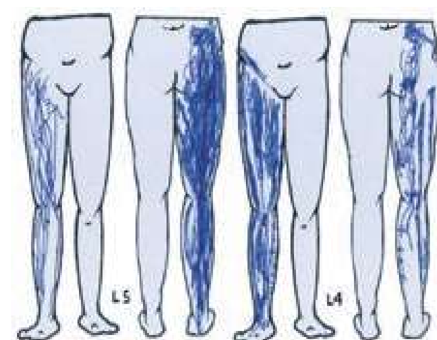

FIGURE 3: An illustration of the variation of induced radicular pain distribution in multiple patients. Based on Furman & Johnson (2018)

> "THE CLINICIAN SHOULD MAKE A DIAGNOSIS OF RADICULOPATHY BASED ON AN ACCUMULATION OF SIGNS RATHER THAN JUST ONE"

some cases there may also be axonal degeneration (Merskey & Bogduk 2012). A nerve in this poor state of health cannot readily conduct action potentials, leading to the loss of function we call radiculopathy. Radiculopathy is not a pain condition at all; it is a diagnosis that refers only to loss of nerve function and may or may not be painful (Merskey & Bogduk 2012).

NOTES ON MAKING A DIAGNOSIS

In order to diagnose a radiculopathy (as opposed to radicular pain), we need to see objective loss of nerve function, but it is important to remember that many of our tests for the loss of function of a nerve have imperfect specificity, i.e. will turn up false positives (Tawa *et al* 2017). This implies that the clinician should make a diagnosis of radiculopathy based on an accumulation of signs rather than just one (Tawa *et al* 2017).

When it comes to differentiating between radicular and referred pain there is no clear and validated method, although recent work suggests that a cluster of:

1. pain below the knee
2. leg pain worse than back pain
3. positive neurodynamic test
4. the report of pins and needles

is strongly associated with nerve root involvement (Stynes *et al* 2018).

As previously discussed, radicular pain is not necessarily a "line" of pain and rarely follows dermatomal patterns (Furman & Johnson 2018). In cases of doubt, the International Association for the Study of Pain (IASP) suggests simply documenting "lower limb pain" (Merskey & Bogduk 2012).

When documenting findings from an objective assessment, table 2 illustrates how it might be helpful to make the distinction between gain and loss of function (Schmid *et al* 2013a).

IMPORTANT CAVEATS

Having presented a classical way of categorising low back / leg pain and neck / arm pain it should be noted that, to make a pain diagnosis based on an underlying mechanism like "radicular" or "referred" is to reformulate something complex and emergent into something simple and linear, and as such is perilous.

The distinction between different kinds of axial / limb pain is not clear; for example, nerve entrapment pain is not necessarily neuropathic, as nervi nervorum (the nerves subserving the nerve trunk) can be a source of nociception (Teixeira *et al* 2016). Additionally, radicular and referred mechanisms can co-exist. Seemingly non-neuropathic pain can feature "occult" neuropathic symptoms (Spahr *et al* 2017), and, of course, pain itself is a higher construct. None of the above mechanisms "are" pain until this information reaches the brain (Brodal 2017).

Indeed, it is arguably not necessary to consider underlying mechanisms at all. They may in fact distract from variables like expectations, disability and pain severity, which appear to be more important for the prognosis and management of many musculoskeletal conditions (Croft *et al* 2015; Konstantinou *et al* 2017), but such considerations have to be held against the practical need for clinicians to communicate clearly and productively with each other, and between disciplines. For this reason, classification, though awkward, is arguably necessary.

Just as importantly, we must also consider how we communicate such labels as *radicular pain* and *radiculopathy* with patients.

Communication between clinician and patient

The clinical presentation of radicular pain has little association with findings on imaging (Karppinen *et al* 2001), but in a survey of people with a history of cervical radiculopathy, Weber *et al* (2017) found that 67% believed an MRI is more important than clinical findings in the decision-making process for their condition, and 47% said they would be willing to undergo surgery based on "abnormal" findings on an MRI alone, even if they had no or few symptoms.

It is no surprise that patients appear to have a biomedical understanding of radicular pain and radiculopathy, they share that perspective with much of the medical and allied health professions and with society at large. For our patients' sake, physiotherapists must promote a more evidence-based and less threatening understanding of these conditions (Moseley & Butler 2017). This is a delicate matter as there is a fine line between de-threatening a condition and trivialising it.

	DEFINITION	MANIFESTATION	TESTED BY	MECHANISM
GAIN	Abnormal excitability	Paraesthesia, spontaneous pain, hyperalgesia and allodynia, hyperreflexia, muscle spasm	Neurodynamic tests, Spurling's test, Tinel's test, pain on palpation	Demyelination, axonal degeneration and neuroinflammation
LOSS	Reduced impulse conduction	Hypoesthesia, anaesthesia, weaker / absent reflexes, muscle weakness, reduced sensation	"Hard neuro" deficits, pinprick, hot, cold testing	Demyelination, axonal degeneration but not neuroinflammation

TABLE 2: Distinguishing between gain and loss of function during objective assessment

There is no script for patient education, and where possible it should be tailored to the individual's prior knowledge and beliefs. That said, the following learning aims are likely to appear in most explanations of cervical radicular pain and radiculopathy. It is often possible to provide them in an "online commentary" to the objective assessment, to save the patient a lecture at the end.

CONCEPTS TO EMPHASISE (TABLE 3)

Since Victorian times, nerves have been thought of as a weak-point in our bodies, the slightest damage to which spells disaster (Bourke 2014). Patients may, therefore, benefit from knowing that nerves are big, tough structures with the dura mater and epineurium forming a layer of protection around them (Moseley & Butler 2017). Patients may also be pleased to hear that, like any other part of the body, nerves also like movement; a good stretch disperses inflammation and helps provide them with lots of oxygen.

The terms "trapped" and "pinched" nerves are probably nocebic, and in any event unlikely to be accurate given that the nerve root ganglion takes up just one third of the space in the outlet holes between the vertebrae, meaning that it potentially has room to move even after significant degenerative changes (Moseley & Butler 2017). Research by Schmid *et al* (2013b) has shown that mild compression, rather than tight constriction, is sufficient to induce nerve pain in rats, so "compressed" or "crowded" are more appropriate words to use. Indeed, it is neuroinflammation that is likely to be more associated with the patient's symptoms than nerve root compression (Albrecht *et al* 2018; El Barzouhi *et al* 2013), and an explanation in these terms would frame the issue accurately as a normal pathophysiological process, i.e. inflamed as opposed to an aberrant biomechanical event, i.e. trapped.

Where a patient has radicular pain with no objective loss of function, it is important to reassure them that there is no evidence that the nerve is damaged, but that it does appear to be irritated. Where there are signs of radiculopathy, i.e. loss of nerve function, it is important to be honest, i.e. advise that this implies demyelination and axonal degeneration rather than offer blind reassurance that "there is no damage", which is arguably unethical. However, the patient may benefit from hearing that this is common and normal, and that neural tissue, like any other soft tissue, is capable of healing and rebuilding. There is some evidence that loss of nerve function, as long as it is not progressive, is not particularly relevant for prognosis; indeed, it may be an indicator of a better than average outcome as it implicates a disc protrusion, which has a good chance of resolving spontaneously (Konstantinou *et al* 2017).

There is an opportunity to link nerve pain to the patient's wider health and wellbeing. Explain to the patient that nerves are connected to the brain and made of the same stuff so, if the brain is stressed or overworked, the nerve might not get a chance to calm down either. This means taking time to relax and sleep well is part of rehab. With regard to exercise, although central sensitisation is a theoretical concern when exercise is painful (Woolf 2004), there is no reason to think that even vigorous movement will cause structural damage to the nerve.

> "THE NERVES ARE CONNECTED TO THE BRAIN, SO IF THE BRAIN IS STRESSED, THE NERVE MIGHT NOT GET THE CHANCE TO CALM DOWN EITHER"

Nerve pain appears to cause patients more distress than somatic pain (Spahr *et al* 2017). Part of the reason for this may be that, for the patient, it is a strange sensation. By explaining that it is the nerve's job to send information about the body and its surroundings to the brain, and that when the nerve is irritated it can start firing off a lot of useless information that is similar to white noise, the patient can be reassured that, although they might feel all sorts of sensations from the nerve, the problem itself is likely to be minor and quite localised. Equally, the worrying symptom of night pain is easily explained; blood pressure drops at night, and when this is combined with the individual

	CONCEPTS TO EMPHASISE
NERVES	Big and tough, with a protective covering Move, bend and stretch with the rest of the body Enjoy movement. A workout gives them a good stretch, disperses any inflammation and brings them a fresh blood supply
"TRAPPED" NERVES	Even if you lose some space there is plenty spare Nerves are slidey and bendy – they are almost impossible to pinch. "It would be like picking up a lychee with chopsticks" More crowded out, or compressed, than trapped
PAIN AND DAMAGE	Zings and zaps do not mean that a nerve is injured – more likely it is sensitive Most painful nerves are sore, but safe If the nerve isn't doing its job as well as it usually does, then just like muscles and ligaments, it can repair and recover
UNUSUAL SYMPTOMS	Night pain is normal, it is because of a drop in blood pressure and often because you fall asleep in an awkward position Touchy nerves report all they possibly can to the brain – sometimes weird and wonderful things There is usually nothing wrong with the area you feel the pain

TABLE 3: Explanatory statements of nerve concepts (Moseley & Butler 2017)

"A DROP IN BLOOD PRESSURE, COMBINED WITH AN ODD SLEEPING POSITION, CAN RESULT IN A TEMPORARY ISCHAEMIA TO THE NERVE, AND EXPLAIN NIGHT PAIN"

sleeping in an odd position, it can result in a temporary ischaemia to the nerve (Moseley & Butler 2017).

With regard to prognosis, the assumption that radicular pain has a favourable natural history is not borne out by the literature. Although some reviews are optimistic (Casey 2011; Wong 2014), a recent high-quality trial of 609 patients with back / leg pain, in primary care, found that just 55% reported a 30% or more improvement in one year (Konstantinou *et al* 2017). This study also identified factors associated with improved disability: shorter pain duration, lower leg pain intensity, fewer other symptoms associated with back and leg pain (e.g. fatigue, stiff joints, poor sleep, loss of strength), and belief that the problem will be short lived (Konstantinou *et al* 2017). Clinicians are, however, not yet in a position to be confident forecasting an outcome for an individual. Patients can be counselled that there is a good chance their pain will fully resolve, but they need to know that there is also a good chance it may not.

Conclusion

This article began by asking the question: "what are we talking about really?" when we talk about axial / limb pain. As demonstrated, there are quite clear differences between radicular pain, radiculopathy and referred pain; although the clinician should maintain a healthy scepticism of the veracity and usefulness of any mechanism-based pain diagnosis. When it comes to explaining these conditions to our patients, there are many ways to give them hope for the future and confidence that they are safe to move; although we should not, in our enthusiasm, be tempted to give explanations that are false.

To reiterate Pierre Milette's statement of 24 years ago: "If we seek to improve our understanding, it is mandatory to address this fundamental issue." I hope this article has made a small contribution to that effort.

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