PAIN® 147 (2009) 17–19

PAIN®

www.elsevier.com/locate/pain

Topical review

On the definitions and physiology of back pain, referred pain, and radicular pain

Nikolai Bogduk *

University of Newcastle, Newcastle Bone and Joint Institute, Royal Newcastle Centre, PO Box 664J, Newcastle, NSW 2300, Australia

1. Introduction

Despite the efforts of the International Association for the Study of Pain [6,21], confusion still persists amongst clinicians about the definitions of back pain, referred pain, radicular pain, and radiculopathy. Basic scientists now stand to inherit this confusion as they develop animal models of back pain [7].

Pivotal to the definition of these entities are seminal studies undertaken 50, 60, and 70 years ago. The legacy of this pioneering work has not properly permeated medical education, publications or clinical practice.

2. Nociceptive back pain

By definition, nociceptive back pain must be pain that is evoked by noxious stimulation of structures in the lumbar spine. The quality of pain so evoked has been determined in studies of normal volunteers, in whom discrete noxious stimuli were delivered to selected lumbar structures. In the original studies, muscles of the back [17] or the interspinous ligaments [18] were stimulated, using injections of hypertonic saline. Others replicated these studies [2,13]. Later, the lumbar zygapophysial joints [22,23] and the sacroiliac joints [14] were stimulated with injections of contrast medium that evoked pain by distending these joints. The dura mater has been stimulated mechanically [27] and chemically [10]. Surgeons who have operated on patients under local anaesthesia have probed various structures mechanically, and showed that the posterior surface of the lumbar intervertebral discs are the most potent source of experimentally-induced back pain [12,20,29]. Uniformly, these experimental studies showed that noxious stimulation causes dull, aching pain in the back. Consequently, when it occurs clinically, this type of pain that should be inferred to be nociceptive back pain.

3. Somatic referred pain

Noxious stimulation of structures in the lumbar spine can produce referred pain in addition to back pain. The pain spreads into the lower limbs, and is perceived in regions innervated by nerves other than those that innervate the site of noxious stimulation – the core of the definition of referred pain [21]. Since the source of spinal referred pain lies in the somatic tissues of the lumbar spine it has been named somatic referred pain [3,5], in order to distinguish it both from visceral referred pain and radicular pain. Somatic referred pain does not involve stimulation of nerve roots. It is produced by noxious stimulation of nerve endings within spinal structures such as discs, zygapophysial joints, or sacroiliac joints. The proposed mechanism of referral is convergence of nociceptive afferents on second-order neurons in the spinal cord that happen also to subtend regions of the lower limb [21]. As a general rule, somatic referred pain is perceived in regions that share the same segmental innervation as the source. Since somatic referred pain is not caused by compression of nerve roots, there are no neurological signs.

Somatic referred pain is dull, aching and gnawing, and is sometimes described as an expanding pressure. It expands into wide areas that can be difficult to localize [2,13,18]. Once established, it tends to be fixed in location. Subjects often find it difficult to define the boundaries of the affected area, but can confidently identify its centre or core. The earliest studies depicted segmental maps of the referred pain patterns [13,18] (Fig. 1). However, although pain from different segments in the lumbar spine refers to different regions in the lower limb, patterns are not consistent amongst subjects or between studies. Most significantly, however, the pattern is not dermatomal. If anything, the pattern corresponds to the segmental innervation of deep tissues in the lower limb, such as muscles and joints. Moreover, although somatic referred pain tends most often to centre over the gluteal region and proximal thigh, it can also extend as far as the foot (Fig. 1). Such distributions have been evoked in normal volunteers and patients by stimulating the lumbar zygapophysial joints [22,23] or intervertebral discs [25], and relieved in patients by anaesthetizing their zygapophysial joints [11,23,26]. To be consistent with these experimental data, when dull aching pain that spreads into the lower limb and settles into a relatively fixed location occurs in patients, it should be recognized as somatic referred pain, when it occurs in patients.

4. Radicular pain

Radicular pain differs from somatic referred pain both in mechanism and clinical features. Physiologically, it is pain evoked by ectopic discharges emanating from a dorsal root or its ganglion [21]. Disc herniation is the most common cause, and inflammation of the affected nerve seems to be the critical pathophysiological process [3]. The clinical features of radicular pain were established in studies of patients who underwent surgery for disc herniation. In

* Tel.: +61 2 49223505; fax: +61 2 49 223559.
E-mail address: michelle.gillam@newcastle.edu.au.

0304-3959/$36.00 © 2009 International Association for the Study of Pain. Published by Elsevier B.V. All rights reserved.
doi:10.1016/j.pain.2009.08.020

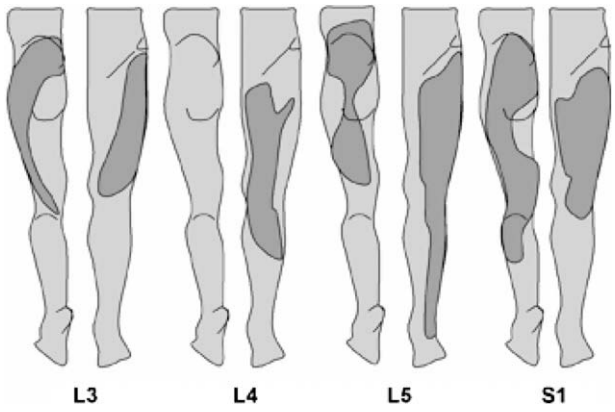


Fig. 1. Patterns of somatic referred pain evoked by noxious stimulation of the interspinous ligaments at the segments indicated. Based on Kellgren [18].

one study, the affected nerves and adjacent nerves were challenged by squeezing them with forceps in awake patients [24]. In another study, sutures were placed around the nerves, during surgery, and led out through the wound, so that they could be pulled on the following day [27]. The pain evoked was distinctive. It had a lancinating quality, and traveled along the length of the lower limb, in a band no more than 2–3 inches wide (see Fig. 2). This is the only type of pain that has been produced by stimulating nerve roots. So, reciprocally, it is only this type of pain that should be interpreted as radicular pain.

Significantly, squeezing or pulling normal nerve roots does not produce radicular pain. Only if nerve roots have previously been inflamed does mechanical stimulation evoke radicular pain [27]. For compression alone to be painful, it seems that it must involve the dorsal root ganglion. Although this has not been verified in experiments on human volunteers, it is borne out in animal studies.

Studies in laboratory animals have provided a neurophysiologic correlate of radicular pain. Squeezing normal nerve roots evokes only a momentary discharge, but squeezing a dorsal root ganglion, or squeezing an inflamed dorsal root, evokes discharges in Aβ as well as Aδ and C fibers [15,16]. Radicular pain, therefore, is not due to a discharge exclusively in nociceptive afferents; it is due to a heterospecific discharge in the affected nerve. The evoked sensation is very unpleasant but is not exactly pain, in a classical, nociceptive sense. The qualities of lancinating, shocking, or electric are consonant with more than nociceptive afferents discharging. Since the English language lacks a more precise word, this sensation is, nevertheless, by default, called pain.



Fig. 2. An illustration of the lancinating quality of radicular pain traveling into the lower limb along a narrow band.

The term – sciatica, is arcane. It stems from an era when the mechanisms of referred pain were not understood, and any referred pain was attributed to irritation of the peripheral nerve that passed through the region of pain. The taxonomy of the IASP recommends replacement by the term – radicular pain [21].

5. Radiculopathy

Radiculopathy is yet another, distinct entity [21]. It is a neurological state in which conduction is blocked along a spinal nerve or its roots. When sensory fibers are blocked, numbness is the symptom and sign. When motor fibers are blocked weakness ensues. Diminished reflexes occur as a result of either sensory or motor block. The numbness is dermatomal in distribution and the weakness is myotomal. However, radiculopathy is not defined by pain. It is defined by objective neurological signs. Although radiculopathy and radicular commonly occur together, radiculopathy can occur in the absence of pain, and radicular pain can occur in the absence of radiculopathy.

Careful clinical examination remains the best tool for diagnosing a radiculopathy. Electrophysiological testing should rarely be necessary. The indications and validity of electrophysiological testing are beyond the scope if this review, but they have been addressed elsewhere [1,4,9].

A common maxim is that the segmental origin of radicular pain can be determined from its distribution. This is not true. The patterns of L4, L5, and S1 radicular pain cannot be distinguished from one another [24,28]. Segments can be estimated only when radiculopathy occurs in combination with radicular pain. In that event, it is the dermatomal distribution of numbness – not the distribution of pain – that allows the segment of origin to be determined.

6. Discussion

Failure to distinguish radicular pain from somatic referred pain may lead to misdiagnosis and thereby mismanagement. Back pain and somatic referred pain are common, but radicular pain is not. When radicular pain has been strictly defined, its prevalence is only about 12% or less [8]. Mistaking somatic referred pain for radicular pain creates the erroneous impression that radicular pain is more common. Because of the strong possibility that somatic referred pain has been mistaken for radicular pain in the past, studies of the prevalence of radicular pain are not reliable [19].

With respect to clinical management, imaging is justified for the investigation of radicular pain and radiculopathy because imaging can often establish the causative lesion. The same does not apply for somatic referred pain. Plain radiographs, MRI scan, or CT scan are unable to reveal the cause of somatic pain, in the majority of cases. Moreover, they carry the risk of false-positive interpretations. Finding degenerative changes, disc bulges and possible nerve root compression is immaterial to the diagnosis if the patient has somatic referred pain, but can lead to unnecessary surgery if somatic referred pain is mistaken for radicular pain.

Since nociceptive back pain and somatic referred pain do not involve nerve injury, there are no grounds for expecting neurological symptoms or signs. In particular, allodynia should not be a feature; and, indeed, allodynia has never been recorded in cases of nociceptive back pain. In contrast, since radicular pain and radiculopathy do involve pathology of a nerve trunk, allodynia is a theoretical possibility, provided that the nerve suffers an appropriate injury. However, allodynia is not a typical feature of radicular pain or radiculopathy, unless there is true nerve damage and neuropathy rather than simply compression or inflammation.

Clinical practice is straightforward when presentations are unambiguous. A patient who is distressed by pain shooting down the lower limb, cannot lie comfortably, and on examination has numbness or weakness in the leg, clearly has radicular pain and radiculopathy. A patient with aching pain in the back, which spreads into the buttock and thigh, but no lancinating pain and no neurological symptoms, has nociceptive back pain and somatic referred pain.

Difficulties arise when patients have combinations. It is possible for a patient to have nociceptive back pain, due to internal disc disruption, for instance. This pain may be referred into the lower limb, in which case it is somatic referred pain. However, the disc may also herniate, or leak inflammatory chemicals onto the nearby nerve root. Chemical irritation of the nerve root will cause radicular pain. Radiculopathy may ensue as the nerve root becomes swollen and conduction block occurs. Each feature, then, has a separate cause and a separate mechanism; and invites separate investigation and treatment. Discectomy might remove the disc herniation and relieve the radicular pain; but it will not relieve the back pain and any somatic referred pain. Indeed, this is the common experience of surgeons: discectomy is highly successful for leg pain, but patients are left with their back pain.

Certain terms are misleading and unhelpful. There is no singular condition called "low back pain – sciatica". This term implies that the patient has a single condition that causes both symptoms, which is not correct. Patients can have back pain; they can also have sciatica; but the two symptoms have separate mechanisms and causes. Features, causes, and mechanisms of one entity cannot be attributed to the other. Thus, although disc herniation is the most common cause of radicular pain, it is not a common cause of back pain. The vast majority of patients with nociceptive back pain does not have radicular pain, and do not have a disc herniation. Nor is it necessary to invoke terms such as pseudo-sciatica or pseudo-radicular pain. There is nothing false about these symptoms. These terms are superfluous synonyms for somatic referred pain or, at times, peripheral nerve entrapment distal to the spine, neither of which has anything in common with radicular pain, other than being perceived in the lower limb. Indeed, because somatic referred pain is far more common than radicular pain, radicular pain should be regarded as the exception rather than somatic referred pain being relegated to some irregularity of radicular pain.

If clinicians ceased to confuse somatic referred pain and radicular pain, fewer patients would be mismanaged, and fewer would sustain iatrogenic problems. If basic scientist understand the distinction, animal models of nociceptive back pain would be developed that do not include neurological abnormalities.

Conflict of interest

The author has no conflicts of interest regarding this article.

References

[1] Andersson GBJ, Brown MD, Dvorak J, Herzog RJ, Kambin P, Malter A, McCulloch JA, Saal JA, Spratt KF, Weinstein JN. Consensus summary on the diagnosis and treatment of lumbar disc herniation. Spine 1996;21:75S–8S.
[2] Bogduk N. Lumbar dorsal ramus syndrome. Med J Aust 1980;2:537–41.
[3] Bogduk N. Clinical anatomy of the lumbar spine and sacrum. 4th ed. Amsterdam: Elsevier; 2005. p. 183–6.
[4] Bogduk N, McGuirk B. Medical management of acute and chronic low back pain. An evidence-based approach. Amsterdam: Elsevier; 2002. p. 65–7.
[5] Bogduk N, McGuirk B. Medical management of acute and chronic low back pain. An evidence-based approach. Amsterdam: Elsevier; 2002. p. 5–11.
[6] Charlton JE, editor. Core curriculum for professional education in pain. Seattle: IASP Press; 2005.
[7] Devor M, Tal M. What causes low back pain? Pain 2009;142:11–2.
[8] Deyo RA, Tsui-Wu YJ. Descriptive epidemiology of low-back pain and its related medical care in the United States. Spine 1987;12:264–8.
[9] Dvorak J. Neurophysiologic tests in diagnosis of nerve root compression caused by disc herniation. Spine 1996;21:39S–44S.
[10] El Mahdi MA, Latif FYA, Janko M. The spinal nerve root innervation, and a new concept of the clinicopathological interrelations in back pain and sciatica. Neurochirurgia 1981;24:137–41.
[11] Fairbank JCT, Park WM, McCall IW, O'Brien JP. Apophyseal injections of local anaesthetic as a diagnostic aid in primary low-back pain syndromes. Spine 1981;6:598–605.
[12] Falconer MA, McGeorge M, Begg AC. Observations on the cause and mechanism of symptom-production in sciatica and low-back pain. J Neurol Neurosurg Psychiat 1948;11:13–26.
[13] Feinstein B, Langton JNK, Jameson RM, Schiller F. Experiments on pain referred from deep somatic tissues. J Bone Joint Surg 1954;35A:981–7.
[14] Fortin JD, Dwyer AD, West S, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part I: Asymptomatic volunteers. Spine 1994;19:1475–82.
[15] Howe JF. A neurophysiological basis for the radicular pain of nerve root compression. In: Bonica JJ, Liebeskind JC, Albe-Fessard DG, editors. Advances in pain research and therapy, vol. 3. Berlin: Springer; 1979. p. 647–57.
[16] Howe JF, Loeser JD, Calvin WH. Mechanosensitivity of dorsal root ganglia and chronically injured axons: a physiological basis for the radicular pain of nerve root compression. Pain 1977;3:25–41.
[17] Kellgren JH. Observations on referred pain arising from muscle. Clin Sci 1938;3:175–90.
[18] Kellgren JH. On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. Clin Sci 1939;4:35–46.
[19] Konstantinou K, Dunn KM. Sciatica: review of epidemiological studies and prevalence estimates. Spine 2008;33:2464–72.
[20] Kuslich S, Ulstro C, Michael C. The tissue origin of low back pain and sciatica. Orth Clin North Am 1991;22:181–7.
[21] Merskey H, Bogduk N, editors. Classification of chronic pain. Descriptions of chronic pain syndromes and definition of pain terms. Seattle: IASP Press; 1994.
[22] McCall IW, Park WM, O'Brien JP. Induced pain referral from posterior lumbar elements in normal subjects. Spine 1979;4:441–6.
[23] Mooney V, Robertson J. The facet syndrome. Clin Orthop 1976;115:149–56.
[24] Norlen G. On the value of the neurological symptoms in sciatica for the localization of a lumbar disc herniation. Acta Chir Scandinav 1944;(Suppl. 95):1–96.
[25] O'Neill CW, Kurgansky ME, Derby R, Ryan DR. Disc stimulation and patterns of referred pain. Spine 2002;27:2776–81.
[26] Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. Clinical features of patients with pain stemming from the lumbar zygapophysial joints. Is the lumbar facet syndrome a clinical entity? Spine 1994;19:1132–7.
[27] Smyth MJ, Wright V. Sciatica and the intervertebral disc. An experimental study. J Bone Joint Surg 1959;40A:1401–18.
[28] van Akkerveeken PF. Lateral stenosis of the lumbar spine: a new diagnostic test and its influence on management of patients with pain only. Thesis, Rijksuniversitet te Utrecht; 1989.
[29] Wiberg G. Back pain in relation to the nerve supply of the intervertebral disc. Acta Orthop Scandinav 1947;19:211–21.