JOURNAL OF MANUAL & MANIPULATIVE THERAPY
2023, VOL. 31, NO. 2, 57–63
https://doi.org/10.1080/10669817.2022.2092266

Low-back related leg pain: is the nerve guilty? How to differentiate the underlying pain mechanism

Antoine Fourré [a,b,c], Félix Monnier [a], Laurence Ris [a], Frédéric Telliez [d], Jef Michielsen [b,e], Nathalie Roussel [b] and Renaud Hage [c,f]

[a]Department of Neurosciences, Research Institute for Health Sciences and Technology, University of Mons, Mons, Belgium; [b]Faculty of Medicine and Health Sciences, Rehabilitation Sciences and Physiotherapy (MOVANT), University of Antwerp, Antwerp, Belgium; [c]Traitement Formation Thérapie Manuelle (TFTM), Physiotherapy Center, Brussels, Belgium; [d]Institut d'Ingénierie de la Santé-UFR de Médecine, Centre Universitaire de Recherche en Santé-Laboratoire Péritox, Université de Picardie Jules Verne, Amiens, France; [e]Orthopedic Department, University Hospital, Antwerp, Belgium; [f]Centre de Recherche et de Formation (CeREF), HELHa, Mons, Belgium

ABSTRACT

Low back pain (LBP) that radiates to the leg is not always related to a lesion or a disease of the nervous system (neuropathic pain): it might be nociceptive (referred) pain. Unfortunately, patients with low-back related leg pain are often given a variety of diagnoses (e.g. 'sciatica'; 'radicular pain'; pseudoradicular pain"). This terminology causes confusion and challenges clinical reasoning. It is essential for clinicians to understand and recognize predominant pain mechanisms. This paper describes pain mechanisms related to low back-related leg pain and helps differentiate these mechanisms in practice using clinical based scenarios. We illustrate this by using two clinical scenarios including patients with the same symptoms in terms of pain localization (i.e. low-back related leg pain) but with different underlying pain mechanisms (i.e. nociceptive versus neuropathic pain).

KEYWORDS

Neuropathic pain; clinical reasoning; nerve tissue; pain management

Introduction

Patients with low back pain (LBP) commonly experience pain that radiates to the leg. Approximately two-thirds of patients consulted for LBP in primary and secondary care have associated leg pain [1,2]. Healthcare Professionals (HCPs) often use terms such as 'sciatica', 'radiculopathy', 'radicular pain', 'pseudo-radicular pain' and 'referred pain' to refer to these symptoms [3–8]. However, a similar clinical picture may have different and overlapping underlying pain mechanisms (see Figure 1) [9]. This variety in terminology and overlap in pain mechanisms is not only confusing for patients and HCPs, but it also makes the process of clinical reasoning more challenging and complicated. The aims of this paper are therefore to (1) clarify and describe the underlying pain mechanisms as defined by the International Association of the Study of Pain (IASP) [1] and (2) help clinicians differentiate the predominant pain mechanism by using clinical scenarios.

Definition of nociceptive and neuropathic pain

While the pain experience is extremely common, there is no scientific consensus to define this pain experience. This is not surprising, given the complexity of pain and the multiple influencing factors. However, the authors used the terminology from the IASP to describe underlying pain mechanisms. This organization proposed distinct pain definitions to differentiate nociceptive and neuropathic pain [10]. Both types of pain mechanisms can explain low-back related leg pain.

Nociceptive pain

Nociceptive pain is defined by the IASP as '*pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors. Nociceptors are triggered by mechanical, chemical, or thermal stimuli arising from all innervated structures*' [11,12].

Nociceptive LBP stimulation of lumbar spine innervated structures (e.g the zygapophyseal joints, spinal ligaments or muscles, or the outer part of the lumbar disc) can induce the transduction of a noxious stimulus into an electric signal in the nervous system. This signal, often referred to as an warning signal, will be processed in the central nervous system with significant brain excitements [13] and can lead to pain [5,14,15]. In the case of nociceptive pain, the somatosensory nervous system functions normally [16].

In some patients, the pain is also felt in the leg, from a region that is topographically different from the source of nociception [7]. This phenomenon is considered as referred pain and might be explained by the convergence of nociceptive afferents on second-order neurons. Most often, the pain is perceived in regions

CONTACT Antoine Fourré info@antoinefourre.com Neuroscience Unit, Research Institute for Health Sciences and Technology, University of Mons, Mons, Belgium

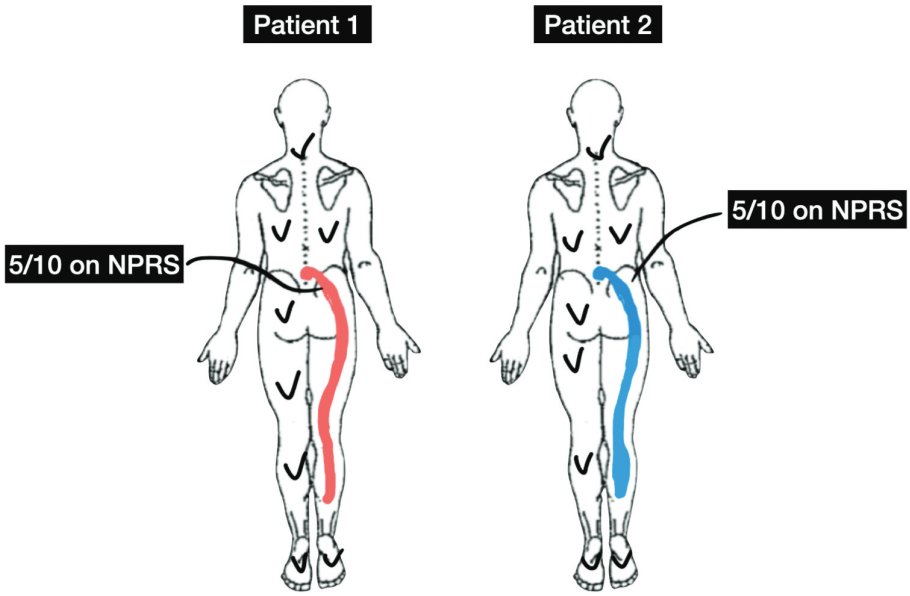

Figure 1. Description of two body-charts. **Patient 1 & patient 2**: These patients describe similar pain topography, localized in the lower back and irradiating in the buttock and the leg. The present pain intensity for both patients is 5/10 on the numeric pain rating scale (NPRS).

that have the same segmental innervation. Moreover nociceptive referred pain can also extend as far as the foot in some cases [5].

Neuropathic pain

The IASP defines neuropathic pain as '*pain caused by a lesion or a disease of the somatosensory nervous system*' [12,17].

Neuropathic pain is a syndrome caused by various diseases or lesions. The most common cause of neuropathic pain in patients with LBP is related to compression of neural structures (e.g. disc herniation) leading to inflammation or degeneration of nerve fibers [18–20]. The nervous system is affected by the generation of ectopic discharges that bypass transduction [9]. These disturbances may affect the nerve function resulting in sensory and motor deficits.

These patients often report that the pain in the leg is lancinating and radiates downwards in a specific root distribution [21]. The pain in the leg is usually worse than the pain in the back [21].

How to differentiate nociceptive referred pain and neuropathic pain

It is crucial for clinicians to determine which mechanism is predominant in the buttock and/or leg. Failure to differentiate mechanisms of radiating pain in the assessment of LBP patients leads to inappropriate investigations and treatment [18,22]. Pain in the leg does not always imply a lesion or disease of the nerve roots or the peripheral nervous system [23–25]. Predominant nociceptive or neuropathic pain management differs from patient to patient [26]. The next part will thus detail how to distinguish between these pain mechanisms.

Differentiating predominant neuropathic pain from nociceptive referred pain is a clinical challenge. While there is currently a paucity of standardization in the diagnosis, neuropathic pain is commonly identified on the basis of clinical criteria [27,28]. Quantitative sensory testing (QST) could be a useful tool, but it is mainly used in research studies [27]. It consists of several tests designed to quantify somatosensory function (gain or loss) in individuals, but is not sufficient alone for the diagnosis of neuropathic pain [29,30]. QST is not widely implemented by first-line practitioners, such as physiotherapists, for several reasons: it is time-consuming, extremely costly and there is a lack of standards in utilization and interpretation [30]. The diagnosis of neuropathic pain in first-line care is difficult to establish but some elements from the patient's history can suggest the presence or absence of neuropathic pain. None of them are pathognomonic, but clustering history elements from subjective and objective examination is the best way to reduce the risk of a wrong diagnosis [31]. Clinicians should primarily base their diagnostic strategy on predominant pain mechanism identification. The next part of this narrative review will cover the theoretical aspect of a subjective (SE) and objective (OE) examination, accompanied with a clinical assessment from the patients presented in Figure 1.

Subjective Examination (SE)

The symptoms described by the patient are the first step in theorizing the predominant pain mechanism. Listening to the patients is important in the differentiation process, as the words used by the patients to describe their pain will be variable between patients with neuropathic and nociceptive pain [27]. Figure 2 lists the most common clinical descriptions of neuropathic and nociceptive pain expressed by patients.

Neuropathic pain is generally referred in a dermatomal or cutaneous distribution [32]. The most common descriptors used by patients are burning, lancinating, and is accompanied by unusual tingling, crawling, or an electrical shock or shooting in the leg [9,26,27]. The description of a patient with neuropathic pain is often characterized by specific neurological symptoms, such as positive (hyperalgesia and/or allodynia) and negative (loss of function) sensory signs [16,33]. The patient may experience various sensations, such as paresthesia, mechanical or thermal hypersensitivity. Neuropathic pain is also characterized by spontaneous (arise without stimulation), evoked (abnormal responses to stimuli) or paroxysmal (sudden recurrences and intensification) pain [32,34].

These symptoms contrast with the description of patients suffering from nociceptive (referred) pain. Pain is usually localized to the area of injury/dysfunction (with or without referred pain) [32]. The symptoms are commonly described as intermittent and sharp with movement. The pain is proportional and in direct relation to pain and easing/aggravating factors [32].

To further refine the reasoning in the SE, a number of self-completion questionnaires with, or without, limited clinical examination (e.g. DN4, LANSS, PDQ) [34–37] have been developed to detect the presence of neuropathic pain, each with condition-specific discriminatory characteristics [38]. The '*Douleur Neuropathique en 4 questions*' (DN4) questionnaire (sensitivity 0,83; specificity 0,9) has been developed to differentiate neuropathic pain from nociceptive pain and seems to have specific discriminative features for low back pain [35]. The questionnaire is short, containing only 10 items, which gives a score, if greater than or equal to 4, indicates the probable presence of neuropathic pain. Seven items are used as a self-report questionnaire of sensory descriptors and 3 items are scored based on the OE. The speed and ease of administration of a questionnaire such as the DN4 make it a valuable complementary tool for clinicians. However, questionnaires should not replace a detailed subjective and objective examination. Although many screening tools have good sensitivity and specificity, they reportedly fail to diagnose 10–20% of patients diagnosed with neuropathic pain [38].

Interpretation of the clinical scenario

When asking patient 1 about the symptoms quality, the patient mentions pain in the buttock that radiates in the leg associated with painless sensations, such as burning, tingling and pins and needles in the calf. The patient further specifies that these sensations tend to increase when pain increases in the buttock. Concerning the aggravating or easing factors the patient explains that

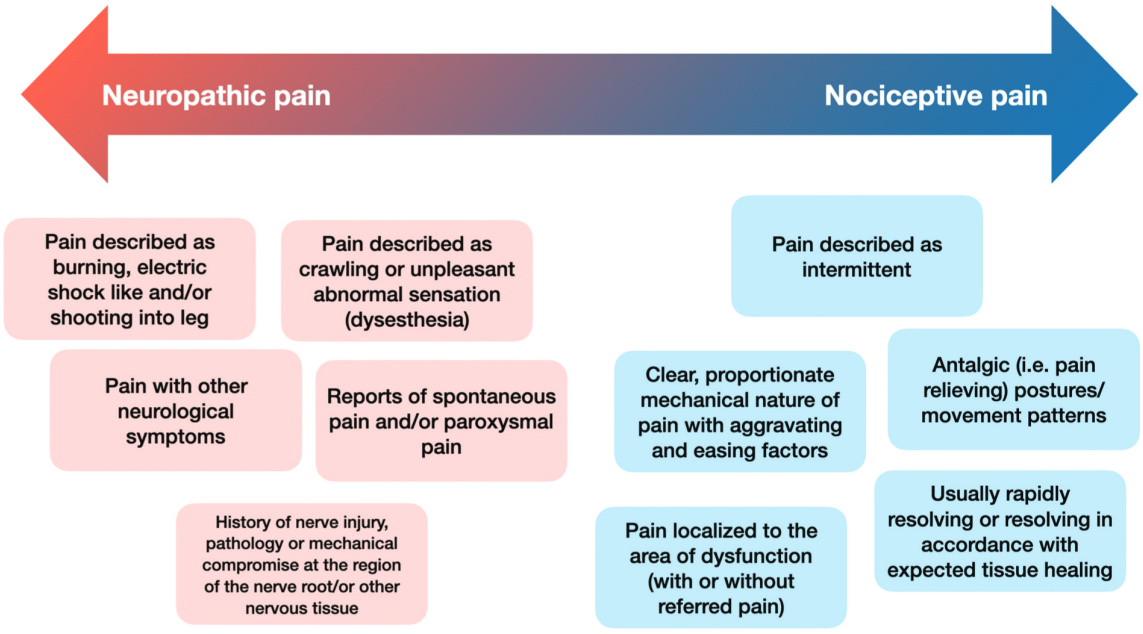

Figure 2. Consensus of clinical descriptors for neuropathic and nociceptive pain based on Mistry et al., 2020 [40] and Smart et al., 2011 [48].

pain is aggravated when getting out of the car with their neck bent. The patient also notices that the pain is easily provoked and takes longer to decrease. The pain is described as 'unpredictable' and may reappear spontaneously. The presence of positive neurological signs, the description of symptoms, and the fact that patient 1 describes the pain as unpredictable supports the hypothesis of predominant neuropathic pain [39].

When asking patient 2 about the quality of symptoms, the patient mentions pain mainly localized in the buttock but radiating in the leg to the calf. The pain is described as sharp and dull. Concerning the aggravating or easing factors, the pain worsens in all sitting positions with lumbar flexion. The pain increases in the transition from sitting to standing or standing to sitting. Patient 2 noticed that the intensity of pain quickly decreased in general and especially if lumbar flexion is avoided (e.g. when resting on the couch in a lying position). The presence of clear and proportionate symptoms, associated with aggravating and easing factors, and the fact that patient 2 does not describe any neurological positive or negative symptoms support the hypothesis of predominant nociceptive mechanism [32].

Although the subjective evaluations of patient 1 and patient 2 provide useful information to help define the underlying predominant pain mechanism, it is obvious that this is not sufficient to draw a definite conclusion regarding the pain mechanism. OE elements, such as neurological and neurodynamic testing, are necessary to further refine the hypothesis established during SE and lead clinicians to a differential diagnosis.

Objective Examination (OE)

A complete OE is carried out with observation, examination of active movements and examination of passive physiological and accessory movements. If a neuropathic pain mechanism is suspected, clinicians should conduct OE with caution and include a neurological evaluation of the patient's sensory, motor and autonomic functions to identify potential neurological dysfunction (including hypoesthesia and brushing testing from the DN4) [7]. In this instance, the neurological examination could highlight neuroanatomical pain distribution, positive and/or negative signs and symptoms (altered reflexes, sensation and muscle power) [40]. The presence of hyperalgesia/allodynia and/or other sensory abnormalities could indicate the presence of neuropathic pain.

Moreover, a neurodynamic examination should be integrated in the OE to assess the nervous system mechano-sensitivity [41,42]. A neurodynamic test is positive when at least reproducing the patient's symptoms and a change in these symptoms with a positive structural differentiation [43–45]. The most common lower limb test is the passive straight-leg raise test (SLR) [42]. The slump test is another neurodynamic test with a high sensitivity (0.9) to identify neuropathic pain in the lower limb [44]. It should be noted that, although most neurodynamic tests have good sensitivity, they generally have low specificity and should not be used independently [46]. However, it is possible to increase diagnostic accuracy by combining several neurodynamic tests [46,47]. More research is needed to determine the most relevant combination of neurodynamic tests for detecting neuropathic pain in LBP.

If there is no evidence during neurological and neurodynamic examinations to suggest the presence of neuropathic pain and in presence of consistent and proportional symptoms, then the predominant mechanism is probably nociceptive (referred) pain [48].

Interpretation of the clinical scenario

Since the SE of patient 1 suggests a predominant neuropathic pain mechanism, the OE should include a thorough neurological and neurodynamic examination. During the neurological examination, the patient describes a loss of sensation to light touch in the right calf and foot, as well as hyperalgesia on pinprick in comparison with the left leg. Concerning the neurodynamic evaluation, given the pain experienced when the neck is bent, the slump test is well suited to assessing mechano-sensitivity. The test is positive in patient 1 with symptoms reproduction and a positive structural differentiation. Information from SE and OE of patient 1 suggests the presence of a predominant neuropathic pain mechanism.

Elements gathered from the SE of patient 2 suggest the presence of a predominant nociceptive pain mechanism. Giving the elements from the SE, an OE including active movements and passive physiological and accessory movements should be performed. During active movements, the pain of patient 2 increased when bending forward and the range of motion is limited. The pain decreased quickly when returning in the starting position. This pattern is similar during the physiological movements. The pain is reproduced in a precise location (L4-L5) with unilateral posterior-anterior mobilization and decreased quickly after. Information from SE and OE of patient 2 suggests the presence of a predominant nociceptive pain mechanism. Even if a predominant neuropathic pain is not suspected, a neurological examination should be performed when a patient presents pain to confirm a normal function of the nervous system. Clinicians should remain attentive to any changes in symptomatology and perform further examinations if neuropathic components appear during the patient's follow-up appointment.

The OE in clinical practice is not described extensively in this paper as the main objective is to help clinicians differentiate between two predominant pain descriptors. The management of the patient should rely on a dynamic and patient-centered biopsychosocial (BPS) framework, including the different aspects of the International Classification of Functioning (ICF) model [49,50].

Predominant pain mechanism

The elements of SE and the OE of patients 1 and 2 suggest the presence of a predominant neuropathic pain mechanism and nociceptive referred pain mechanism, respectively (see Figure 3). The clinical examples presented are rather clear and easy to differentiate. However, in clinical practice the differentiation between the two is not so easy and the clinical reasoning is sometimes very complex for patients with low back related leg pain. Clinical descriptors, signs and symptoms could be confusing and might overlap. Although distinction is essential for effective management, neuropathic and nociceptive pain have several features in common. Pure nociceptive pain and pure neuropathic pain may in fact be very rare in practice [3,51,52]. Both share the same neurotransmitters, ascending spinal pathways, supraspinal signal processing regions and descending modulatory pathways [9]. The traditional view that these two mechanisms are completely separate entities is questioned by some experts and may be due to our propensity to classify items [9]. In most instances, it is probably a combination of the two mechanisms with, depending on the case, a neuropathic or nociceptive predominance. Clinicians should be aware that this predominance can change over time (see Figure 2) and assess the patient repeatedly over consecutive sessions.

Discussion and conclusion

The aim of this paper was to discuss pain mechanisms underlying low back-related leg pain and help clinicians to differentiate these mechanisms in clinical practice. The distinction between neuropathic and nociceptive pain is essential in establishing adapted and patient-centered management. If the clinician simply assesses the topography of the pain (e.g. irradiation in the buttock) these two pain mechanisms are easily confused and can lead to inappropriate investigations and management [5,22]. While the symptoms topography could be similar, description and behavior of pain completed by a thorough objective examination should be used to differentiate the predominant pain mechanisms. However, the terminology used in the literature seems difficult to translate from research to clinical practice. Moreover,

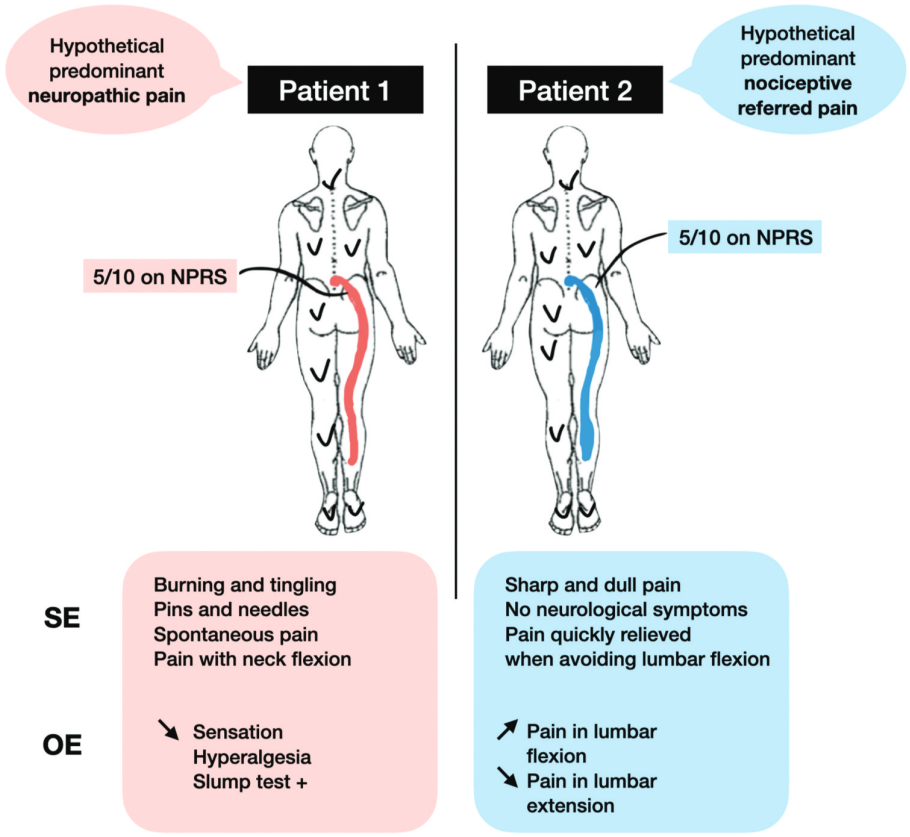

Figure 3. Body chart of patient 1 and patient 2 after a complete subjective and physical examination.

clinical practice is complex, and sometimes this differentiation seems too simplistic and insufficient for the patient's condition. These pain mechanisms should not be considered as distinct entities [9]. Overlap exists between these two pain descriptors and could explain the difficulties in implementing treatment based on mechanism [9]. Some patients display predominantly nociceptive components but can simultaneously display symptoms suggesting a neuropathic component. A patient's condition is not fixed in time and should be assessed repeatedly in the follow-up with a combination of subjective and objective examination to decide the predominant underlying pain mechanism. This is clinically important because the prognosis of a patient with neuropathic pain is worse than a patient with nociceptive pain and should lead the clinician to an adapted management [2]. Hence the management of a patient with predominant neuropathic pain includes specific medication [53], adapted passive treatments [41,43,54], an appropriate dosage of exercises [55] and education about the function of the nervous system [43]. Moreover, the clinician must be aware of the potential changes in the patient's symptomatology to prevent the aggravation of a potential serious condition [56]. Further research is necessary to better define diagnosis, prognosis and pathways of patients with low-back related leg pain.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

With the financial support of the European Regional Development Fund (Interreg FWVl NOMADe - N° 4.7.360).

ORCID

Antoine Fourré http://orcid.org/0000-0002-8494-4919
Félix Monnier http://orcid.org/0000-0001-8557-852X
Laurence Ris http://orcid.org/0000-0002-2140-8297
Frédéric Telliez http://orcid.org/0000-0001-6815-6050
Jef Michielsen http://orcid.org/0000-0002-4353-6601
Nathalie Roussel http://orcid.org/0000-0002-7409-878X
Renaud Hage http://orcid.org/0000-0002-6556-0899

References

[1] Hill JC, Konstantinou K, Egbewale BE, et al. Clinical outcomes among low back pain consulters with referred leg pain in primary care. Spine (Phila Pa 1976). 2011 Dec;36(25):2168–2175.
[2] Harrisson SA, Stynes S, Dunn KM, et al. Neuropathic pain in low back-related leg pain patients: what is the evidence of prevalence, characteristics, and prognosis in primary care? A systematic review of the literature. J Pain. 2017 Nov;18(11):1295–1312.
[3] Freynhagen R, Rolke R, Baron R, et al. Pseudoradicular and radicular low-back pain – a disease continuum rather than different entities? Answers from quantitative sensory testing. Pain. 2008 Mar;135(1):65–74.
[4] Van Boxem K, Van Zundert J, Patijn J, et al. Pseudoradicular and radicular low-back pain: how to diagnose clinically? Pain. 2008 Apr;135(3):311–312.
[5] Bogduk N. On the definitions and physiology of back pain, referred pain, and radicular pain. Pain. 2009 Dec;147(1):17–19.
[6] Valat J-P, Genevay S, Marty M, et al. Sciatica. Best Pract Res Clin Rheumatol. 2010 Apr;24(2):241–252.
[7] Baron R, Binder A, Attal N, et al. Neuropathic low back pain in clinical practice. Eur J Pain. 2016 Jul;20(6):861–873.
[8] Beynon R, Elwenspoek MMC, Sheppard A, et al. The utility of diagnostic selective nerve root blocks in the management of patients with lumbar radiculopathy: a systematic review. BMJ Open. 2019 Apr;9(4):e025790.
[9] Cohen SP, Mao J. Neuropathic pain: mechanisms and their clinical implications. BMJ. 2014 Feb 5;348(feb05 6):f7656–f7656.
[10] Scholz J, Finnerup NB, Attal N, et al. The IASP classification of chronic pain for ICD-11: chronic neuropathic pain. PAIN. 2019 Jan;160(1):53–59.
[11] Merskey H, Bogduk N. Part Ill: pain Terms: a current list with definitions and notes on usage. In: Classif chronic pain. Second ed. 2011; p. 209–214.
[12] Terminology | international association for the study of pain [Internet]. cited 2022 Mar 3]. Available from: https://www.iasp-pain.org/resources/terminology
[13] Marchand S. The physiology of pain mechanisms: from the periphery to the Brain. Rheumatol Dis Clin N Am. 2008 May;34(2):285–309.
[14] Tsao H, Tucker KJ, Coppieters MW, et al. Experimentally induced low back pain from hypertonic saline injections into lumbar interspinous ligament and erector spinae muscle. Pain. 2010 Jul;150(1):167–172.
[15] O'Neill S, Graven-Nielsen T, Manniche C, et al. Ultrasound guided, painful electrical stimulation of lumbar facet joint structures: an experimental model of acute low back pain. Pain. 2009 Jul;144(1):76–83.
[16] La Cesa S, Tamburin S, Tugnoli V, et al. How to diagnose neuropathic pain? The contribution from clinical examination, pain questionnaires and diagnostic tests. Neurol Sci. 2015 Dec;36(12):2169–2175.
[17] Jensen TS, Baron R, Haanpää M, et al. A new definition of neuropathic pain. Pain. 2011 Oct;152(10):2204–2205.
[18] Schäfer A, Hall T, Briffa K. Classification of low back-related leg pain—A proposed patho-mechanism-based approach. Man Ther. 2009 Apr;14(2):222–230.
[19] Yoshizawa H, Kobayashi S, Morita T. Chronic nerve root compression: pathophysiologic mechanism of nerve root dysfunction. Spine (Phila Pa 1976). 1995 Feb;20(supplement):397–407.
[20] Freynhagen R, Baron R, Tölle T, et al. Screening of neuropathic pain components in patients with chronic back pain associated with nerve root compression: a prospective observational pilot study (MIPORT). Curr Med Res Opin. 2006 Mar;22(3):529–537.
[21] Koes BW, van Tulder MW, Peul WC. Diagnosis and treatment of sciatica. BMJ. 2007 Jun 23;334(7607):1313–1317.
[22] Schäfer A, Hall T, Müller G, et al. Outcomes differ between subgroups of patients with low back and leg pain following neural manual therapy: a prospective cohort study. Eur Spine J. 2011 Mar;20(3):482–490.

[23] Konstantinou K, Dunn KM, Ogollah R, et al. Characteristics of patients with low back and leg pain seeking treatment in primary care: baseline results from the ATLAS cohort study. BMC Musculoskelet Disord. 2015 Dec;16(1):332.
[24] Saifuddin A, Emanuel R, White J, et al. An analysis of radiating pain at lumbar discography. Eur Spine J. 1998 Oct 19;7(5):358–362.
[25] O'Neill CW, Kurgansky ME, Derby R, et al. Disc stimulation and patterns of referred pain. Spine (Phila Pa 1976). 2002 Dec;27(24):2776–2781.
[26] Haanpää ML, Backonja -M-M, Bennett MI, et al. Assessment of neuropathic pain in primary care. Am J Med. 2009 Oct;122(10):S13–S21.
[27] Bouhassira D. Neuropathic pain: definition, assessment and epidemiology. Rev Neurol (Paris). 2019 Jan;175(1–2):16–25.
[28] Smith BH, Torrance N, Bennett MI, et al. Health and quality of life associated with chronic pain of predominantly neuropathic origin in the community. Clin J Pain. 2007 Feb;23(2):143–149.
[29] Rolke R, Magerl W, Campbell KA, et al. Quantitative sensory testing: a comprehensive protocol for clinical trials. Eur J Pain. 2006 Jan;10(1):77.
[30] Birklein F, Sommer C. Quantitative sensory testing—a tool for daily practice? Nat Rev Neurol. 2013 Sep;9(9):490–492.
[31] Callin S, Bennett MI. Assessment of neuropathic pain. Contin Educ Anaesth Crit Care Pain. 2008 Dec;8(6):210–213.
[32] Smart KM, Blake C, Staines A, et al. Mechanisms-based classifications of musculoskeletal pain: part 2 of 3: symptoms and signs of peripheral neuropathic pain in patients with low back (±leg) pain. Man Ther. 2012 Aug;17(4):345–351.
[33] Zilliox LA. Neuropathic Pain. Contin Lifelong Learn Neurol [Internet]. 2017;23(2). Available from: https://journals.lww.com/continuum/Fulltext/2017/04000/Neuropathic_Pain.13.aspx
[34] Bennett M. The LANSS pain scale: the Leeds assessment of neuropathic symptoms and signs. Pain. 2001 May;92(1):147–157.
[35] Bouhassira D, Attal N, Alchaar H, et al. Comparison of pain syndromes associated with nervous or somatic lesions and development of a new neuropathic pain diagnostic questionnaire (DN4). Pain. 2005 Mar;114(1):29–36.
[36] Nikaido T, Sumitani M, Sekiguchi M, et al. The Spine painDETECT questionnaire: development and validation of a screening tool for neuropathic pain caused by spinal disorders. Ikeda K, editor. PLOS ONE. 2018 Mar 21;13(3):e0193987.
[37] Haanpää M, Attal N, Backonja M, et al. NeuPSIG guidelines on neuropathic pain assessment. Pain. 2011 Jan;152(1):14–27.
[38] Cruccu G, Sommer C, Anand P, et al. EFNS guidelines on neuropathic pain assessment: revised 2009: neuropathic pain assessment. Eur J Neurol. 2010 Aug;17(8):1010–1018.
[39] Koury MJ, Scarpelli E. A manual therapy approach to evaluation and treatment of a patient with a chronic lumbar nerve root irritation. Phys Ther. 1994 Jun 1;74(6):548–560.
[40] Mistry J, Falla D, Noblet T, et al. Clinical indicators to identify neuropathic pain in low back related leg pain: a modified Delphi study. BMC Musculoskelet Disord. 2020 Dec;21(1):601.
[41] Shacklock M. Neurodynamics. Physiother Elsevier. 1995 Jan 1;81(1):9–16.
[42] Boyd BS, Wanek L, Gray AT, et al. Mechanosensitivity of the lower extremity nervous system during straight-leg raise neurodynamic testing in healthy individuals. J Orthop Sports Phys Ther. 2009 Nov;39(11):780–790.
[43] Nee RJ, Butler D. Management of peripheral neuropathic pain: integrating neurobiology, neurodynamics, and clinical evidence. Phys Ther Sport. 2006 Feb;7(1):36–49.
[44] Urban LM, MacNeil BJ. Diagnostic accuracy of the slump test for identifying neuropathic pain in the lower limb. J Orthop Sports Phys Ther. 2015 Aug;45(8):596–603.
[45] Nee RJ, Jull GA, Vicenzino B, et al. The validity of upper-limb neurodynamic tests for detecting peripheral neuropathic pain. J Orthop Sports Phys Ther. 2012 May;42(5):413–424.
[46] van der Windt DA, Simons E, Riphagen II, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. Cochrane back and neck group, editor. Cochrane Database Syst Rev [Internet]. 2010 Feb 17; [cited 2021 Nov 2]. 10.1002/14651858.CD007431.pub2
[47] González Espinosa de Los Monteros FJ, Gonzalez-Medina G, Ardila EMG, et al. Use of neurodynamic or orthopedic tension tests for the diagnosis of lumbar and lumbosacral radiculopathies: study of the diagnostic validity. Int J Environ Res Public Health. 2020 Oct;17(19):7046. PMID:32993094.
[48] Smart KM, Blake C, Staines A, et al. The discriminative validity of "Nociceptive," "Peripheral Neuropathic," and "Central Sensitization" as mechanisms-based classifications of musculoskeletal pain. Clin J Pain. 2011 Oct;27(8):655–663.
[49] Jull G. Biopsychosocial model of disease: 40 years on. Which way is the pendulum swinging? Br J Sports Med. 2017 Aug;51(16):1187–1188.
[50] World Health Organization. International classification of functioning, disability and health : ICF. Classif Int Fonct handicap santé CIF [Internet] Geneva: World Health Organization; 2001. [cited 2021 Sep]. Available from: https://apps.who.int/iris/handle/10665/42407
[51] Toda K. Pure nociceptive pain is very rare. Curr Med Res Opin. 2019 Nov 2;35(11):1991.
[52] Freynhagen R. on behalf of the author group. Response: letter: pure nociceptive pain is very rare. Curr Med Res Opin. 2019 Dec 2;35(12):2137.
[53] Attal N. Pharmacological treatments of neuropathic pain: the latest recommendations. Rev Neurol (Paris). 2019 Jan;175(1–2):46–50.
[54] Jones MA, Rivett D. Clinical reasoning in musculoskeletal practice - E-Book [Internet]. Elsevier Health Sciences; 2018. [cited 2021 Mar]. Available from: https://books.google.be/books?id=QFp0DwAAQBAJ
[55] Leitzelar BN, Koltyn KF. Exercise and Neuropathic Pain: a General Overview of Preclinical and Clinical Research. Sports Med - Open. 2021 Dec;7(1):21.
[56] Finucane LM, Downie A, Mercer C, et al. International framework for red flags for potential serious spinal pathologies. J Orthop Sports Phys Ther. 2020 May 21;1–23. 10.2519/jospt.2020.9971.