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

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### 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', 'pseudoradicular 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].

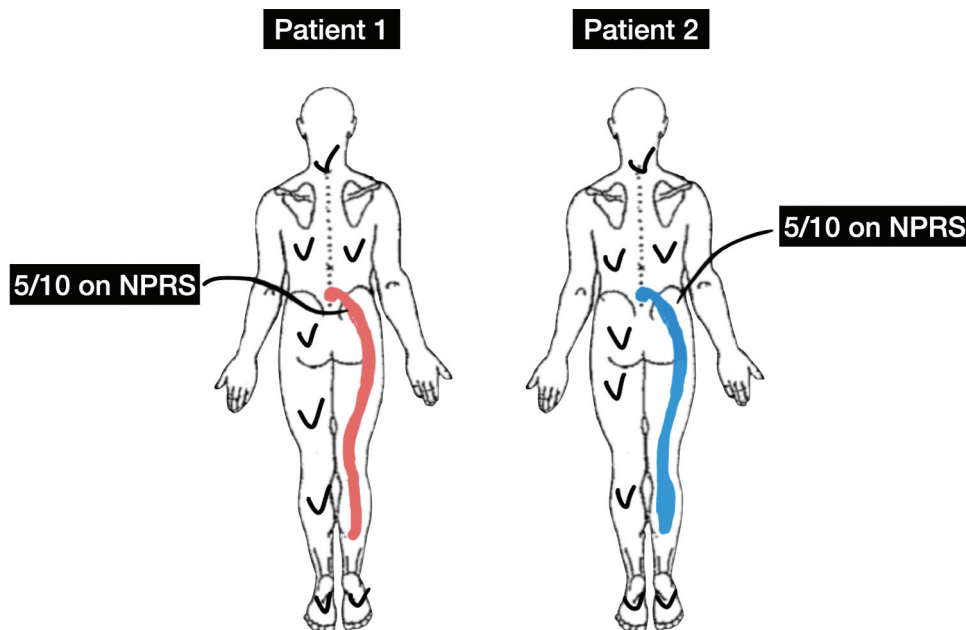
Nociceptive LBP stimulation of lumbar spine innervated structures (e.g the zygapophyseal joints, spinal ligaments or muscles, or the outer part of the lumbar disc) can induce the transduction of a noxious stimulus into an electric signal in the nervous system. This signal, often referred to as a warning signal, will be processed in the central nervous system with significant brain excitations [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

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**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

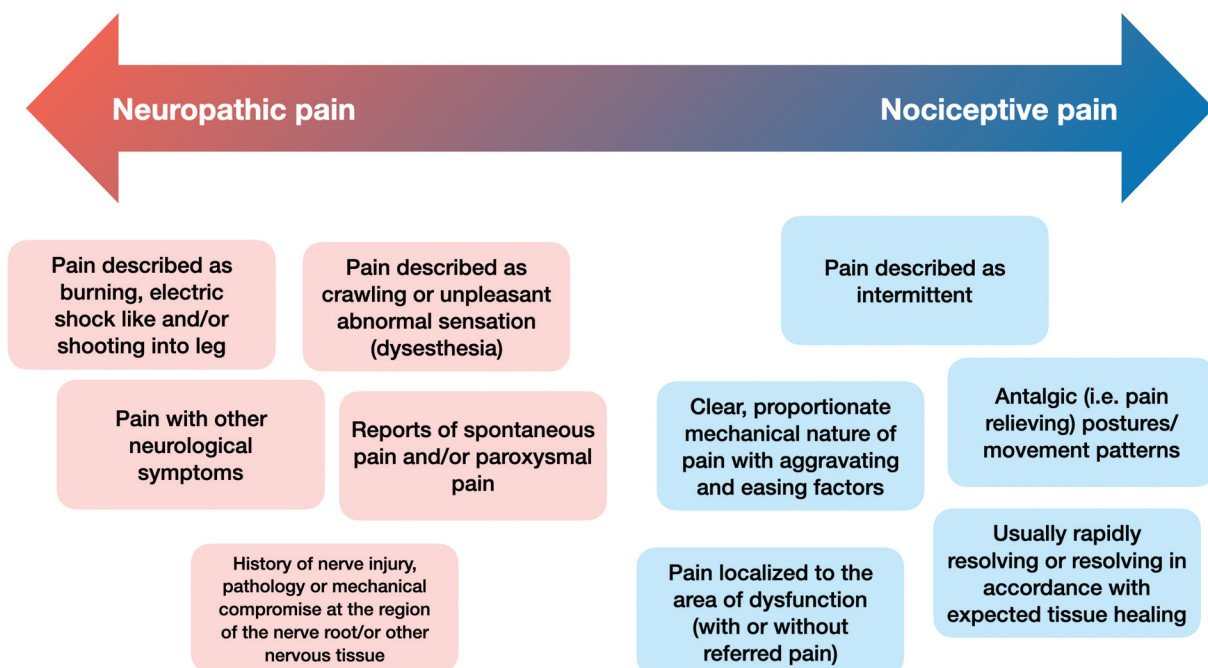


Figure 2. Consensus of clinical descriptors for neuropathic and nociceptive pain based on Mistry et al., 2020 [40] and Smart et al., 2011 [48].

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,

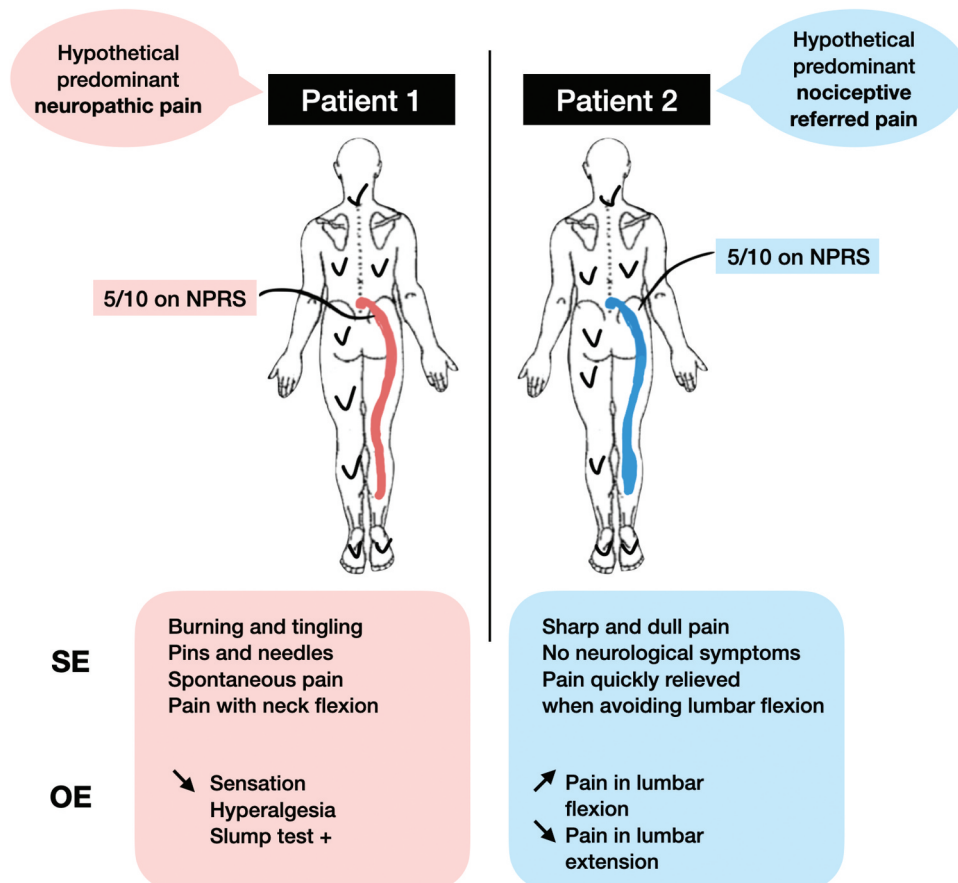


Figure 3. Body chart of patient 1 and patient 2 after a complete subjective and physical examination.

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.

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