Diagnosis of chronic radiating lower back pain without overt focal neurologic deficits: what is the value of segmental nerve blocks?

The purpose of this paper is to discuss the value of spinal segmental nerve blocks in establishing diagnosis in chronic lower back pain patients with pain radiating to the leg without overt focal neurologic deficits. These patients represent a large population. Establishing diagnosis is often problematic and the effectiveness of presently available therapies is low. Indication, character of radiating pain, pain diagnosis, factors influencing segmental nerve blocks, reliability of segmental nerve blocks in diagnosing pain, recently performed studies on the reproducibility of segmental nerve block effects and considerations with respect to ‘segmental pain’ will be discussed herein.

Segmental nerve blocks (SNBs) are applied for diagnostic purposes in patients with radiating pain to determine the pain-conducting segmental spinal level [1–7]. SNBs have been applied in patients with chronic lower back pain radiating to the leg (CLBP-r) to select those eligible for a radiofrequency procedure of the dorsal root ganglion (RF-DRG) [7,8]. A positive SNB can further be used to establish the indication for spinal segmental nerve-injection therapy [9–14]. Huston and Slipman [14] and Gajraj [15] have reported that SNBs are valuable in the assessment of sciatica, but warn of the mainly retrospective nature and procedural limitations of the studies describing the predictive value. High sensitivity and specificity are attributed to SNBs when used to predict surgical outcomes in patients with specific radicular syndromes. These patients suffer from nerve compression with secondary neurologic deficits as a result of a lumbar herniated disc or spinal stenosis [3–6,14,16–19]. Hogan, in contrast, postulates that no role has been demonstrated for SNBs in evaluating patients for neuroablative procedures [20].

Radiating pain

CLBP-r patients may exhibit pain following a spinal segmental pattern or a nonsegmental pattern, whereby a segmental pattern is defined as being concordant to the innervation area of a spinal (segmental) nerve. Nonsegmental radiating pain may be referred – caused by local sources in the posture and motor apparatus of the back [21] – but may also be related to structures outside the back. Furthermore, radiating pain can be related to a neuropathy of a peripheral nerve.

When pain is felt in one neuraxial segment one needs to distinguish between pain with a specified, diagnosable, cause and that in which no certain cause can be established. When a pathoanatomic cause, related to a spinal segmental nerve, nerve root or dorsal root ganglion, is ascertained, and pain follows the innervation area of a spinal nerve (i.e., dermatome, myotome or sclerotome), it is defined as radicular pain. When radicular pain is accompanied by sensory changes in the corresponding dermatome, by a decrease in motor function in the corresponding myotome, positive spinal nerve stress tests and decreased tendon reflexes corresponding to the symptomatic level, it is defined as radiculopathy.

It should be emphasized that a substantial amount of CLBP-r pain does not conform to the diagnostic criteria for radiculopathy. In many of these patients, obvious causal pathology related to the spinal nerve suspected to be involved cannot be demonstrated with the presently available diagnostic tools. Nevertheless, patients may experience pain that follows a segmental or a segment-like pattern. To differentiate between radicular pain and radiculopathy, we propose to define this type of radiating pain, in which a specified cause cannot yet be found, as segmental pain. A classification with respect to different types of pain radiating into the leg is shown in Table 1.

Pain diagnosis

Radiologic examinations, such as plain radiography, myelography, discography, computed tomography (CT) and magnetic resonance imaging (MRI) have a low specificity with respect to establishing the cause or source of the pain. For example, a herniated disc may be the cause, but the compressed and excited dorsal root ganglion is
the source \([7,22–25]\). Potential pain-generating conditions, such as a herniated disc, spinal stenosis and epidural fibrosis, can be present in symptom-free patients, and \textit{vice versa}. Thus, the quest for a pathoanatomic cause for CLBP-r remains a challenge. In this search, spinal endoscopy is a promising diagnostic and potential therapeutic tool which can be performed in addition to (and in the future perhaps as replacement of) presently available radiologic and clinical neurologic examinations. This technique has received more attention recently and is of interest due to its ability to aid in diagnosing pathology in the epidural space that cannot as yet be demonstrated in another fashion, such as MRI or CT. Using spinal endoscopy, abnormalities such as spinal nerve inflammation, can be visualized that may compromise or threaten radicular nerves \([26–29]\).

Neuroinflammatory and neuroimmunologic processes in the spinal cord, spinal nerve root, dorsal root ganglion or spinal nerve \([30–33]\) may explain the presence of radicular pain that cannot at present be diagnosed by radiologic examination. These processes, as well as the presence of chronic pain itself, may result in altered nervous system function (i.e., neuroplasticity). Such neuroplastic mechanisms can lead to pain that persists after an initial nociceptive triggering process or event, which may no longer be present or detectable later on \([34–37]\). The interpretation of this type of pain can be difficult because other pain sources that can generate radiating pain may also be present concurrently, such as spondyloolisthesis, disorders of facet joints, intervertebral disc(s) and sacro–iliac joint or tendomyogenic structures. Olmarker demonstrated that the intervertebral disc is a potential source of biochemical substances that may directly and indirectly lead to excitation of dorsal root ganglion cells (phospholipase \(\text{PLA}_2\) and tumor necrosis factor \(\text{TNF}\)-\(\alpha\)) \([30]\). This complex picture is further complicated if pain originates from regions outside the back, the peripheral nervous system or the CNS. Finally, multisegmental innervation of the spine and dermatomal overlap \([38,39]\), presence of neuronal networks in the spine \([21,40]\), and influence of psychogenic and behavior-related factors can all coalesce to make the clinical diagnosis of CLBP-r extremely difficult. Thus, pain originating from the spine or related structures will be referred multisegmentally as a result of the multisegmental innervation \([21,40]\). This pain is defined as pseudoradicular and is generally felt in parts of more than one dermatome. Pseudoradicular pain can mimic segmental radiating pain, despite having no actual segmental origin.

In the case of nerve-root compression, the presence of radiating pain with a dermatomal pattern in the leg (radicular pain) seems to be one of the most significant diagnostic features. The sensitivity of this diagnostic symptom is reported as between 90 and 99\% \([41,42]\). However, not one single physical test or examination appears to have an equally high sensitivity and specificity for radiculopathy \([43]\). In summary, the diagnostic accuracy of history taking and physical examination still remains unclear in the diagnosis of lower back pain with radiation to the leg.

Factors influencing segmental nerve blocks

The diagnostic use of SNBs is based on the assumption that they can identify segmental pain and the spinal level by using significant pain reduction as an end point. However, there is no gold standard against which the SNB result can be measured to date. Therefore, there is a strong need to develop measures to confirm the effectiveness of SNBs. Changes in sensory and motor

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**Table 1. Radiating pain.**

<table>
<thead>
<tr>
<th>Pathoanatomical substrate</th>
<th>Neurologic deficits</th>
<th>Pain distribution in neuraxis segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Segmental</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radiculopathy</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Radicular pain</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Segmental pain</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Nonsegmental</td>
<td>−</td>
<td></td>
</tr>
<tr>
<td>Referred pain</td>
<td>−</td>
<td>+ (or ?)</td>
</tr>
</tbody>
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**Box 1. Techniques used to test human spinal nerve function.**

- **Nerve stimulation**
  - Mechanical
  - Electrical
- **Nerve block**
  - Conduction block with local anesthetic agent
function could be a useful tool to clinically document reliability. Despite the fact that SNBs have been used for many years in back-pain diagnostics, only a small number of studies have systematically described the clinical effects of SNBs. Box 1 shows methods which could be helpful in testing spinal segmental nerve function in humans. Table 3 shows the most commonly applied tests for quantifying alterations in spinal nerve function.

Furthermore, the technique used to identify the spinal nerve root, the dorsal root ganglion or the spinal nerve must be reliable and reproducible. The needle should be introduced and inserted into the upper, dorsal part of the intervertebral foramen (intra- or extraforaminally) and should be documented radiologically. Use of imaging guidance via fluoroscopy or CT is strongly recommended. The spinal nerve, spinal nerve root or dorsal root ganglion may be mechanically or electrically stimulated via the tip of the needle, evoking paresthesias in the corresponding dermatome and provoking muscle contractions in the corresponding myotome. A low volume of radio contrast dye (0.2–0.5 ml) should also be injected to visualize its spread around the target neural structure. In this way, the structure is made visible and it allows for the assessment of any unwanted spread of the injected solution. Spread should be limited to the target structure. It is important that neural structures lying at spinal segmental levels above or below the target level are unaffected. To prevent unintended nontargeted spread of the injected agent to adjacent neural tissues, a low volume should be injected extradurally (sometimes this is described as peridurally), either in- or outside the intervertebral foramen.

Theoretically, anesthetizing the mixed spinal nerve extraforaminally should block afferent signals coming from peripheral sites distal to the

<table>
<thead>
<tr>
<th>Function</th>
<th>Examination</th>
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<tbody>
<tr>
<td>Sensory/pain</td>
<td>Quantitative sensory testing</td>
</tr>
<tr>
<td></td>
<td>• Electrical: gradual and continuous</td>
</tr>
<tr>
<td></td>
<td>• Mechanical: gradual and continuous</td>
</tr>
<tr>
<td></td>
<td>• von Frey hairs: graded and discontinuous</td>
</tr>
<tr>
<td></td>
<td>• Temperature: gradual and continuous</td>
</tr>
<tr>
<td>Pinprick</td>
<td>(semiquantitative mapping)</td>
</tr>
<tr>
<td>Brush</td>
<td>(semiquantitative mapping)</td>
</tr>
<tr>
<td>Intramuscular injection with hypertonic NaCl</td>
<td></td>
</tr>
<tr>
<td>Motor</td>
<td>Muscle force in myotome</td>
</tr>
<tr>
<td>Spinal reflexes</td>
<td></td>
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<tr>
<td>Electromyography</td>
<td></td>
</tr>
<tr>
<td>Sympathetic</td>
<td>Skin infrared thermography</td>
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<tr>
<td></td>
<td>Skin impedance (galvanic reflexes)</td>
</tr>
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<td></td>
<td>Skin temperature</td>
</tr>
</tbody>
</table>
injection and thus prevent centripetal conduction (Figure 1). In an intraforaminal block, signals from the so-called sinuvertebral nerves should also be blocked. These nerves conduct afferent signals from the spine itself, such as from neighboring intervertebral discs, anterior and posterior ligaments of the spine and ventral dura [21, 40]. Although the dorsal ramus, originating just outside the intervertebral foramen, may remain out of reach of an intraforaminal block, it should be noted that the sensory fibers from the dorsal nerve pass through the dorsal root ganglion and consequently, are also blocked. This nerve branch innervates local muscles in the back and the neighboring facet joints. Furthermore, it has been reported that pain generated proximal to the nerve block may be relieved by a conduction block performed distal to the exciting locus. In this way, pain related to proximal spinal nerve root excitation and experienced in the leg and the back [44, 45] is affected by a distant block.

The concept of ‘controlled blocks’ for zygapophysial joint blocks [46] to increase reliability and improve interpretation may also be applied for SNBs – blocks are performed on two different occasions, with a short- and a long-acting local anesthetic agent in equipotent dosage. The duration of pain reduction should correspond with the duration of action of the local anesthetic agent used. It may be expected that not only duration of pain reduction, but also duration of other concomitant changes, for example, in sensory and motor function, should be concordant with the duration of effect of the local anesthetic agent used. However, in our experience, this is not always the case. To further increase reliability, the presence of multisegmental innervation should also be taken into account [21, 40]. Thus, we would suggest that SNBs should be performed on at least two or three spinal levels. These double controlled blocks with long- and short-acting local anesthetics have been advocated as gold standard [46]; however, in view of the discussion above with regard to single SNBs, this is hardly likely. Even controlled blocks do not distinguish between the source and cause of pain.

When the local anesthetic reaches the target neural structures it has to diffuse into these structures before it can exhibit its blocking property. Local effects may be affected by factors such as the physical and chemical properties of the local anesthetic agent. Furthermore, size and position of the dorsal root ganglia may vary in relation to the intervertebral foramen [47–53]. There is a large variation in anatomic positions of dorsal root ganglia. Three positions are possible:

- Outside the foramen
- Inside its aperture
- Actually within the spinal canal (Figure 2)

In this context, the use of radiocontrast dye and electrostimulation may be helpful to raise insight into the variability of the dorsal root ganglia topography. Furthermore, spinal nerve
roots and ganglia have an internal topographic organization regarding nervous and non-nervous cells [49]. So far, it is unknown whether there is a relationship between electrostimulation and the intraganglionic topographic organization. Therefore, the effects of the local anesthetic within the innervation area of a spinal nerve can be expected to vary, dependent on its penetration into the dorsal root ganglion and spinal nerve.

**Reliability of segmental nerve blocks**

Pain patterns, pain reduction and concomitant changes in sensation and muscle force by SNB should be clearly associated with the blocked spinal segmental nerve. To date, no data are available with respect to the reproducibility of sensory effects, pain reduction and motor effects by SNBs. The same holds true for elicited paresthesias.

SNBs should be sensitive and specific. North and colleagues reported a high sensitivity, but a low specificity for SNBs in the context of sciatica patients [45]. In these patients, blocks performed at spinal level L5 and S1 were compared with sciatic nerve blocks, consecutive blocks of the medial branch of the dorsal ramus (supplying the facet joints) and subcutaneous injections with local anesthetics. Surprisingly, they found that the two blocks performed far from the affected spinal nerves also resulted in significant pain reduction in a substantial number of patients compared with SNBs of the affected spinal nerve. Subcutaneous injections did not lead to pain reduction. The authors argued that negative blocks may have some predictive value, but that isolated, positive, pain-reducing blocks are to be considered nonspecific. All included patients had positive diagnostic imaging findings of ongoing nerve-root compression or a positive history of root compression, which had been identified surgically.

In CLBP-r patients who have no detectable and specific underlying diagnosis for their radicular pain, it is impossible to generate data on sensitivity and specificity with respect to SNBs. However, an alternative or better tool than SNB to identify segmental pain is not available at this moment.

The lack of a diagnostic golden standard in patients with chronic, nonspecifiable, radiating pain emphasizes the need to develop other methods by which to monitor the quality and reliability of SNBs, by systematically documenting sensory and motor [39,54,55]. Such methods could include:

- Paresthesias elicited by electrostimulation
- Changes in sensory function, and changes in muscle force

Findings resulting from monitoring these signs should correspond to the blocked spinal level. The method should be consistent and reproducible. Until recently, no studies had been performed to answer these questions. However, with respect to the large population of CLBP-r patients without overt focal neurologic deficits and with respect to the frequent need for diagnosis and treatment of these patients, this is extremely relevant.

**Studies on the reproducibility of SNB effects**

Interesting findings of a series of studies into the relationship between segmental pain in the leg, pain reduction and changes in sensory and
motor function after SNBs in CRLB-r patients without overt focal neurologic deficits \[39,54,55\] will be discussed here.

The first finding is that the incidence, location and extent of skin areas with hypesthesia for pin prick after SNB are very variable. These results can be seen in a so-called density map of hypesthetic effects for pin prick after SNB (Figure 3). The extent of the total skin area where hypesthesia is found in this series appears to be extremely large. Of note is the fact that in some patients, no hypesthesia develops at all, although all nerve blocks were technically adequate. It seems that patterns of pain radiation and hypesthesia, which mostly exceed the boundaries of the standard dermatomes, can be better understood if overlap by neighboring dermatomes is taken into account in the representation of dermatomes (Figure 4). The resulting 'adapted' dermatomes are then seen to be twice as large as in standard dermatomal maps. Using the map with 'adapted' dermatomes, sensory clinical SNB effects occur more often within the dermatomal boundaries. In contrast, the variability of paresthesias elicited by electrostimulation is much lower, being mainly experienced in the central part of the standard dermatome. The reproducibility of paresthesias elicited with electrostimulation via the tip of the needle appears to be high: 80% of experienced paresthesias are found to be present within the boundaries of the corresponding standard dermatomes and in 98%, paresthesias are found within the boundaries of the corresponding, 'adapted', dermatome. Nevertheless, the relation with pain remains poor: when pain is experienced in a specific 'adapted' dermatome, concurrent pain reduction, paresthesias and hypesthesia are present in only a third of this dermatome.

When the sensory function is tested with pin prick before SNB in CLBP-r patients without overt focal neurologic deficits, it is found that in the majority of cases, a variable pre-block hypesthesia is present \[55\]. This alteration in sensory function may change in time and location. Although a large variability in extent of post-block hypesthesia is found, the changes with
block are nonsignificant compared with the pre-block situation. The presence of pre-block alterations in sensory function may be the result of neuroplastic effects due to the chronicity of pain in these patients.

When the SNB effects on motor function are examined [54], it appears that average muscle force within the corresponding myotome decreases with block. However, the muscle force in the corresponding myotome increases if pain is reduced by the nerve block. This finding can be interpreted as follows: in patients with chronic pain in the leg, pain has an inhibitory effect on the muscle force (so-called diffuse noxious inhibitory control or DNIC, a phenomenon also attributed to neuroplasticity) [56]. After pain reduction, inhibition should cease, which normalizes muscle force.

These observational studies demonstrate that long-lasting back pain with segmental radiation to the leg, even when specific causes have not been found, induces neuroplastic changes in both the sensory and motor system. It is possible that the large variability in sensory effects with SNB is also related to these neuroplastic changes. However, the role of multisegmental innervation should not be forgotten in this context. It should be emphasized that the segmental changes related to sensory and motor function are poorly reproducible in CLBP-r patients. Only the elicitation of paresthesias with electrostimulation is reliably reproducible in a dermatomal fashion. At present this combination of clinical signs present after SNBs is not useful to assess the quality of SNBs.

**Does segmental pain exist?**

As discussed above, attempts to select only those patients who have segmental pain from the large population of CLBP-r patients have remained futile. Even assessing the effectiveness of SNB by measuring subsequent successful treatment outcome as end point did not resolve the problem. The diagnostic value of SNBs as selection tool for successive segmental invasive pain treatment could also not be confirmed in a recent study by Geurts and colleagues [8]. In a prospective, randomized and placebo controlled study they demonstrated that radiofrequency treatment of lumbosacral dorsal root ganglia in patients with radicular pain, selected with SNBs, were not effective. However, it should be added that in that study, controlled, double blocks were not used for patient selection. Besides the lack of treatment effectiveness itself, this could also be attributed to SNB-associated properties, such as low selectivity of SNBs or absence of applying double controlled SNBs, or to wrong concepts or hypotheses with respect to the phenomenon of segmental pain. Thus, ‘segmental pain’ as a clinical concept lacks experimental evidence for its existence when no specific cause can be demonstrated. It appears that, with respect to underlying mechanisms in chronic radiating pain, we will have to find other conceptual frameworks. A possible alternative framework would involve the aforementioned mechanism of neuroplasticity. With the presently available diagnostic tools we cannot clearly demonstrate, or exclude, processes such as (neuro-) inflammation or persistent neuroplasticity. It should be noted that neuroplasticity can be segmental, such as sensitization of dorsal root ganglion neurones and glial cells [35]. However, the lack of consistent segmental effects of SNB makes it unlikely that it will aid in the diagnosis of this type of problem, either.

In future, studies of pain-related neuroplastic changes in sensorimotor systems may provide us with more insights in underlying pain mechanisms. It is of eminent importance that we obtain a better understanding of the mechanism involved in the development of pain, of the processes facilitating the chronification of pain, and of its impact on systems involved. If segmental effects are related to pain, then diagnosis and treatment should take into account that the nerve cells involved are connected both to peripheral (sensory and motor function) and central neural structures higher in the neuraxis. Increasing numbers of studies performed in the last few years have demonstrated that pain, chronicity, neuroplasticity, cerebral functions, emotions, cognitions and behavior are all strongly related to each other. Longer-lasting back pain with nonspecifiable ‘segmental’ pain should be viewed as being a part of a more extended and complex system that demonstrates functional plasticity. It would seem that our conceptual frameworks with respect to the concept of ‘segmental pain’ and with regard to diagnostic ‘segmental’ nerve blocks as a main tool have to be reconsidered. Further studies should demonstrate whether or not this assumption is correct.

We have to bear in mind the fact that the diagnosis ‘segmental pain’ is a constantly evoking concept. Our diagnoses are dependent on our diagnostic tools, and these diagnostic tools should provide us with a better insight into underlying mechanisms. Therefore, an important future goal in the context of CLBP-r patients would be to develop more sophisticated diagnostic techniques that enable us to better identify pain-induced neuroplastic changes in the nervous system.
SPECIAL REPORT – Wolff, Groen & Wilder-Smith

Highlights

- Segmental nerve blocks (SNBs) should identify the spinal nerves conducting radiating pain in patients with chronic lower back pain (CLBP) without a specifiable cause or source.
- SNBs applied in CLBP patients with pain radiating to the leg without focal neurological deficits show a large variability with respect to segmental effects.
- Various factors such as neuroplasticity and multisegmental innervation complicate the interpretation of pain and SNB-related segmental effects.
- The conceptual framework with respect to segmental pain and SNBs as a diagnostic tool should be reconsidered.

Bibliography


Chronic radiating lower back pain – SPECIAL REPORT


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