Dynamic Chiropractic

BACK PAIN

Neuropathic Low Back Pain: Where Does It Hurt?

ACUTE VS. CHRONIC VS. NEUROPATHIC LBP

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Chiropractors can easily recognize the pain that arrives from biological components of the back, but as anyone who has practiced more than six months realizes all pain has a psychological component as well.[1] In contrast to the acute low back pain that we treat on a daily basis, patients with chronic pain may involve pathological processes affecting the nervous system that potentially can be a disease all its own.

Because the processes involved are different, patients with acute and chronic pain should not be treated in the same way. Moreover, chronic pain is difficult to treat as the contribution from biological processes and psychosocial and environmental factors are often difficult to flesh out. Unfortunately, in the chiropractic practice, the aim of treatment in chronic pain patients is often to merely reduce suffering. These people then become chronic chiropractic patients. This may be because the neuropathic component of low back pain makes an accurate diagnosis and treatment difficult.

The Nature of Chronic Low Back Pain

Pain can be broadly described as nociceptive or neuropathic pain. These two types of pain are caused by different neurophysiological processes and may require different treatment modalities.[2-3] For our typical chiropractic patients, nociceptive pain results from the activity in neural pathways caused by actual or potential tissue-damaging stimuli (e.g. ,chemical, thermal or mechanical) and is mediated by polymodal pain receptors (e.g., A-delta and C fibres), which are located in the skin, bone, connective tissue, discs, muscles and viscera.[4]

Neuropathic pain, in contrast, is produced by damage to, or pathological changes in, the peripheral and/or central nervous system. The Special Interest Group on Neuropathic Pain of the International Association for the Study of Pain (NeuPSIG) has recently redefined *neuropathic pain* as "pain arising as a direct consequence of a lesion or disease affecting the somatosensory system."[5] To be more specific, the conventional definition of neuropathic pain is pain resulting from injury to or dysfunction of the nervous system in the absence of direct nociceptive input. The injury or dysfunction may involve peripheral or central nervous system structures.[6]

Nociceptive Pain Components

Chiropractors do not need a primer on structural components of low back pain associated with degeneration of bones, muscles, ligaments, joints and intravertebral discs. We clearly understand that the facet (zygapophyseal) joints have the ability to become nociceptive pain generators and that chiropractic care works appropriately for these mechanisms. Arthritis in these joints gives rise to mechanical pain or local nociceptive pain resulting in chronic activation of C fibres. Also, the annulus of a healthy vertebral disc is innervated by C fibres to a depth of about 3 mm. When the

disc cracks or is damaged, a neo-innervation (or a sprouting of C fibres) can occur, which may also result in chronic pain.[4]

Neuropathic Pain Components

The mechanisms involved in neuropathic pain are complex and involve both peripheral and central phenomenon, but one underlying dysfunction involves deafferentation within the peripheral nervous system. That old disc injury may very well have injured the nerve root directly and following a peripheral nerve injury (e.g., crush, stretch or axotomy), sensitization occurs, characterized by spontaneous activity by the neuron, a lowered threshold for activation and increased response to a given stimulus. Additionally, the connective tissue sheath around peripheral nerves is innervated by the nervi nervorum. Injury, compression and inflammation of the sheath may also cause pain.[7]

Neuropathic low back pain can also be caused by lesions of nociceptive sprouts within the degenerated disc (local neuropathic), mechanical compression of the nerve root (mechanical neuropathic root pain), or by action of inflammatory mediators (inflammatory neuropathic root pain) originating from the degenerative disc - even without any mechanical compression.[8] A local neuropathic low back pain can arise from the old disc injury as a result of direct damage to the nerve root by mechanical compression and/or through the action of inflammatory mediators,[9] long after our care has healed the disc.

Looking across the pain syndromes that enter our office, there is also the "in-between" group of nociceptive and neuropathic pain. Chronic low back pain patients, for example, often have pain components from both the nociceptive (tissue) and neuropathic (nerve) processes. These complex patients have back pain from both pain-generating mechanisms; what can be described as "mixed pain syndromes." The etiologies that can give rise to mixed pain syndromes of the lower back include: disc herniation, failed back-surgery syndrome (due to scarring), foraminal stenosis, and osteoporotic fractures of the vertebra.[8]

It's a *Big* Pain Problem

According to recent research, approximately 4 percent of the general adult population experiences back pain with a neuropathic component. The neuropathic low back pain patient has a much higher severity of pain and costs. A person suffering neuropathic back pain has as much as 67 percent higher back pain-related costs as an average patient with nociceptive back pain only.[10]

Large epidemiological studies show that 20 percent to 35 percent of patients with back pain suffer from a neuropathic pain component.8 Other studies have shown that 41 percent of chronic low back pain patients have neuropathic pain while 59 percent have nociceptive pain.[11]

Another problem with damage to nerves can occur if there is a loss of sensory input into the dorsal horn of the spinal cord, a process known as deafferentation.[12] This is the case also with chiropractic patients who have diabetes or other unrecognized forms of peripheral nerve damage in the lower extremity. This often results in pain signals being transmitted to the spinal cord in a way that makes up for the lack of sensory input into the dorsal horn.

In a sense, the spinal cord second order neurons make up their own response to the lack of normal background information.[12] This can result in severe neuropathic pain, sometimes delayed for years

after the injury, which arises suddenly without clear provocative incident. In fact, this type of neuropathic pain, which is particularly challenging to treat effectively, is found in 8 percent of patients affected by low back pain.[13]

Beyond How Much It Hurts

Diagnosing the source of the structural spinal pathology alone is not enough; neuropathic back pain evaluation goes beyond how much it hurts. Neuropathic low back pain may be highly prevalent in our chiropractic offices, but diagnosis and management remain difficult for most clinicians to pin down because there is no "gold standard." Most doctors rely on clinical experience, as the usefulness of current diagnostic tests (used to identify the source of low back pain) in clinical practice and for guiding treatment selection is unclear.[8] It is clear that we need to improve our understanding of factors that practicing chiropractors can employ so we can structure our services to better diagnose and treat the neuropathic low back patient, and perhaps stop its development in our chronic patients.

Since sensory symptoms likely translate into pain-generating mechanisms,[14] enrichment of the clinical exam and diagnostic skills that we employ to detect sensory losses in our patients' peripheral nervous system may show us potential treatments based on sensory profiles. Clearly, the task of treating chronic neuropathic low back pain is extremely complex and challenging. Methods to diagnose and treat the neuropathic component of our patients' pain and helping clinicians to better understand and identify these patients is essential. The ability to identify neuropathic pain mechanisms can lead us to innovative and individualized treatments, resulting in improved pain control in our patients with chronic and neuropathic low back pain.

References

1. Manchikanti L, Fellows B, Pampati V, et al. Comparison of psychological status of chronic pain patients and the general population. *Pain Physician,* 2002;5(1):40-8.
2. Finnerup NB, Otto M, McQuay HJ, et al. Algorithm for neuropathic pain treatment: an evidence based proposal. *Pain,* December 2005;118(3):289-305.
3. Helme RD. Drug treatment of neuropathic pain. *Aust Prescr,* 2006;29:72-5.
4. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine,* August 2006;31(18):2151-61.
5. Special Interest Group on Neuropathic Pain of the Int'l Association for the Study of Pain. www.neupsig.org
6. Wolfe GI, Barohn RJ. Painful peripheral neuropathy. *Current Treatment Options Neurol*, May 2002;4(3):177-88.
7. Asbury AK, Fields HL. Pain due to peripheral nerve damage; an hypothesis. *Neurology,* 1984;34:1587-90.
8. Freynhagen R, Baron R. The evaluation of neuropathic components in low back pain. *Curr Pain Headache Rep*, June 2009;13(3):185-90.
9. Mullemann D, et al. Pathophysiology of disk-related sciatica. Evidence supporting a chemical component. *Joint Bone Spine,* March 2006;73(2):151-8.
10. Schmidt CO, Schweikert B, Wenig CM, et al. Modelling the prevalence and cost of back pain with neuropathic components in the general population. *Eur J Pain*, Feb. 5, 2009. [Epub ahead of print]
11. Hassan AE, Saleh HA, Baroudy YM, et al. Prevalence of neuropathic pain among patients suffering from chronic low back pain in Saudi Arabia. *Saudi Med J*, December 2004;25(12):1986-90.
12. Fields HL, Hill RG. The Near and Far Horizon. In: *Neuropathic Pain: Pathophysiology and Treatment.* Seattle: IASP press, 2001:258-61.

13. Torrance N, Smith BH, Bennett MI, et al. The epidemiology of chronic pain of predominantly neuropathic origin. Results from a general population survey. *J Pain,* 2006;7(4):281-9.
14. Baron R, Tölle TR, Gockel U, et al. A cross-sectional cohort survey in 2,100 patients with painful diabetic neuropathy and postherpetic neuralgia. Differences in demographic data and sensory symptoms. *Pain*, July 8, 2009. [Epub ahead of print]

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