Understanding and Diagnosis of Nerve-Related Pain

Chiropractors treat the nervous system. How many times have we heard that or said that to our patients? This is the first of at least two articles addressing techniques that attempt to diagnose and treat the mobility and sensitivity of nerves. This first article will try to give a deeper understanding of chronic pain and "nerve pain," and introduce David Butler's neurodynamic model. I'll start with a series of quotes from other DCs who are more elegant and well-read than me.

Geoffrey Bove, DC, PhD, is a leading nerve researcher at Harvard. The following quote was taken from the opening of an abstract of his most recent journal article: "Peripheral nerve inflammation can cause axons conducting through the inflamed site to become mechanically sensitive. Axonal mechanical sensitivity (AMS) of intact axons may explain symptoms in a diverse number of conditions characterized by radiating pain evoked by movement of the affected nerve."[1] *Mechanically sensitive* means these nerve axons had increased reactivity to both pressure and stretch. The study used chemical irritants directly on the nerves.

I'll quote Dr. David Seaman, in comments he made recently on an online discussion group:[2] "When it comes to radiculopathy what is often forgotten is that the nerves are basically connective tissue tubes that insulate axons. The epineurium can be likened to the loose CT of the facet joint capsule and annulus, and the perineurium is not dissimilar to the more dense CT in joint and disc. Interestingly, the epi- and perineurium have their own intrinsic nerve supply, most of which are the C-sensory fibers or group IV afferents – the classic nociceptors."

To help our patients with chronic pain and/or radicular pain, we need to understand the physiology of pain. This understanding has grown immensely over the past 20 years. The concept of nocioception vs. neuropathic pain is critical. I'll quote Dr. Craig Liebenson (also from an online post[2]): "Neuropathic pain is associated with central sensitization. This is typified by a) allodynia – pain to non-noxious stimuli and b) hyperalgeisa – exaggerated pain responses. Nociceptive pain is peripherally driven by chemical, thermal or mechanics insults to tissues housing nociceptors. It is not centrally maintained; however, it can be centrally influenced by descending modulation or inhibition via the enkephalin system."

Here's Ryan Van Matre summarizing neuropathic pain concepts:[2] "Neuropathic sensitivity is where the synapses which relay the nociception danger message (either in the peripheral or central nervous system) have become more sensitive to reaching threshold. Thus, they modify in their physiology to become:

1. More susceptible to incoming excitatory neurotransmitters (things hurt more than when they did before or things that didn't hurt before now hurt).
2. The gates which allow the positively charged ions to enter stay open longer (more bang for the buck).

3. There is an increase in the number of gate or "sensors" for the excitatory transmitters to activate (the Velcro team – more sticks).

"All of these changes can increase the sensitivity of the neuron, whereas previous nociception (danger message), which may not trigger threshold, may now sustain it. Other long-term processes can then come into effect, such as sensitivity-enhancing chemicals (e.g., cortisol) building in the tissue to also make the synapses more sensitive. It is essential to understand that this process is under constant modulation, increasing or decreasing as the environment dictates. This process can occur anywhere in the nervous system including the brain itself."

David Butler points out, "Enhanced sensitivity of the alarm system is nearly always a main feature in chronic pain. Remember that the pain is normal, but the processes behind it are altered."[3]

In summary, pain has both a peripheral and a central component. Pain is a process that goes up and down. The nervous system is constantly being modulated or changed toward more pain or less pain. Most chronic pain and most nerve pain, may have had its beginning in a nociceptive event or nociceptive process, but evolves toward central sensitization or toward a neuropathic process. Looking for the pathology, the hard anatomical cause of the pain is necessary, but you cannot stop there. Rule out the red flags, and then proceed to evaluate and treat whatever functional changes have occurred that reinforce the pain pattern. Chiropractors have been doing this for 100 years, mostly unconsciously – touching patients, reassuring them that we can help and changing the "container" to decrease the nerve irritation. In my own clinical evolution, I've found that I have come to really appreciate that decreasing the pain helps the healing. This is partially based on medical research showing better postsurgical healing when adequate pharmaceutical pain relief is provided. It's also based on my understanding of the plasticity of the nervous system. The pain experience reinforces and increases itself. It's a vicious cycle.

I recommend the book *Explain Pain* by David Butler. This brilliant work uses pictures and words to attempt to explain central and peripheral sensitivity, and how the nervous system gets fired up from pain. The understanding of nervous system sensitization outlined here is expanded upon in this book. It's aimed at patients, but is a good reinforcement for doctors as well. How many of you have seen patients who were improving until they saw their MRI with disc bulging and disc degeneration? I call this "installing a herniated disc in the patient's mind." Suddenly, the patient thinks they have a severe pathology instead of a normal aging phenomenon, and they start exhibiting fear-based behavior. How many fusions are performed because of ugly MRIs, rather than for good clinical reasons? Other good reading on this topic includes Don Hazen's Web site, **www.dhazen.com/neuropages/nerv_struct.html**, and Butler's 2001 book, *The Sensitive Nervous System.*

Diagnosing "Nerve Pain" and Understanding Neurodynamics

How can we assess the state of the peripheral nerves? I'll start with the lower extremity, as the tests for the sciatic nerves are much simpler and most of us are already familiar with them. We'll start by exploring Butler's work. Many evidence-oriented DCs are familiar with this work, although Butler's group, the Neuro Orthopedic Institute (NOI), currently refuses to teach to DCs. You can still buy the DVD and his books.

The main concept I get from studying neurodynamics is that you can objectively test for nerve tension. The straight-leg raise and its variations are classic examples with which all of us are somewhat familiar. Butler fine-tunes the straight-leg raise, adding variations that increase or decrease the tension on the dura and sciatic nerve via foot dorsiflexion or plantar flexion, femoral internal rotation and adduction, and passive cervical flexion. He also uses a sitting slump test to further assess the same dura/sciatic nerve axis. Butler has pioneered a set of similar tests for the upper body that isolate the median, radial and ulnar nerves.

Here's what you can do on any lower back (or buttock or leg) pain patient to determine whether the sciatic nerve is involved. Perform a straight-leg raise. Does it cause pain or tightness? Does the leg feel heavy compared to the other side? Where is the pain or tightness? In classic severe sciatica, you'll elicit pain shooting down the leg, but you also can get tightness or pain in the hamstrings, buttock or lower back. Then either increase the tension on the nerve by dorsiflexing the foot and/or flexing the head and neck. Does this same pain or tightness increase? Now decrease the tension by extending the head and/or by plantar flexing the foot. If the tightness or pain is just hamstring tightness, it won't change much with these variations. If the tightness or pain is mediated through the nerves, it will change. Butler has modified the straight-leg raise into a more sensitive test.

The sitting slump test is a similar variation on the sitting straight-leg raise. In this position, you can add lumbar flexion by increasing nerve tension or lumbar extension by decreasing nerve tension. You can have the patient lean forward at the waist (maintaining lumbar lordosis), thus increasing nerve tension; or lean back, decreasing nerve tension.

Butler's work is more sophisticated than my brief description. I'm just trying to give a quick synopsis. What these tests tell you is whether the sciatic nerve, its branches and/or the dura are somehow involved in this patient's problem. If the problem is limited to joint dysfunction, myofascial pain, ligament issues and/or core instability, these tests will be negative. When the neurodynamic tests are positive, it means the mobility of the nerve and its containers have been somehow compromised.

Butler goes on to teach a therapeutic model based on these tests. The doctor moves the patient through various motions that glide and/or stretch the involved nerves, and then teaches the patient to do these as home exercises. The idea is to free up the nerves through movement, helping the nerves glide through the spaces where they may be constricted.

I have come to appreciate Butler's diagnostic model. It gives us a functional test for the mobility and state of the nerves. Most of us already do this when we have a patient with

sciatica. We measure changes in the straight-leg raise, telling us if the patient is improving. We can do much more if we study and use Butler's model.

I use Butler's model for my diagnosis and as a "reality check." To improve the health of the nerves, I use several different models including the following:

- Release restrictions in the "container," the soft tissues and joints that surround the nerve. This is the classic chiropractic and soft-tissue model.
- The McKenzie model: Find directional bias or preference and teach the patient home exercises that reinforce this direction.
- Butler's neurodynamics, both as in-office therapy and as home exercises.
- Barral's approach of manual therapy (manipulation) directly to the nerve sheath.
- Craniosacral techniques and when indicated, coccyx manipulation. These aim directly at the dura and its connections.

My next article will go into further detail on treatment of the involved nerves contributing to the pain.

References

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2. SpineDocs Online: **www.spinedocsonline.com**.
3. Butler D. *Explain Pain.* Adelaide, Australia: Noigroup Publications, 2003.