

## Who's Got a Pinched Nerve?

Edgar Romero, DC, DACNB

The ubiquitous pinched nerve - I must admit that I cringe every time I see an advertisement or a television commercial with an illustrious colleague preaching about how they will "relieve the pressure of the pinched nerve in your spine," or whatever other form their explanation may take. That's not to say we cannot achieve this result, but I cannot count how many patients have come to my office reporting unsuccessful treatment in other offices (medical and otherwise) following diagnosis of a pinched nerve, when in fact, they had nothing of the sort.

The "pinched nerve" can really be an excellent way to explain the power of a good chiropractic adjustment when properly delivered at the appropriate place and time. The image of a compressed nerve springing back to life after we have laid our hands upon the patient is potentially tremendous. The problem, of course, is that most of these nerves are not actually pinched.

We may be getting great results, but I believe understanding the true mechanism of what is occurring can serve not only the patient, but also the doctor, when we happen to come across a case that is not responding as expected. There is an excellent text by Jerome M. True, et al., titled *Myelopathy, Radiculopathy and Peripheral Entrapment Syndromes*. It covers, as True likes to put it, "everything from the neck down." It should be required reading for every health professional dealing with patients in pain, which would include all of us.

Peripheral nerves are composed of large-diameter "A" fibers, medium-sized "B" fibers, and "C" fibers, the smallest in diameter. Large-diameter nerves comprise motor neurons and fast pathways of sensation, such as two-point discrimination sensory pathways from muscles that maintain structural integrity, e.g., Ia and Ib motor afferents. The B fibers contain autonomic pathways primarily with some motor components, and the C pathways are mostly pain pathways and other functions. (For the most comprehensive explanation of nerve physiology, consult your nearest, friendliest neurology text.)

I've started with this minor primer because when compressing a nerve, it is most likely that we would compress or affect the larger things first, as opposed to the smaller ones. Any peripheral compression first reduces the firing rates and function of large-diameter afferents, eventually to be followed by degradation of the other pathways after an extended period of time. The reality of nerve compression is that we will lose sensory pathways related to large-diameter function first, like vibration and two-point discrimination. After a period of six days or so, we will see motor weakness and muscle atrophy.

Let's put this into clinical perspective. You have a patient present with loss of dorsiflexion of the foot and big toe on the left side, low back pain, and referral patterns down the back of the leg. I know you've seen a ton of these cases, just as I have. We do not even have to examine this baby, do we? Too easy; this is as classic as a blown L5-S1 disc. Depending on your technique, you may or may not be willing to adjust this patient. Perhaps flexion-distraction? Some people feel the [McKenzie technique](#) is the best approach. Let's put this suffering patient on the traction machine, perhaps; 20 visits minimum, of course. But, maybe, just maybe, we decide to check a little further, just to be diligent.

DTRs are normal, but let's ignore that because you can't be sure what they were before. Vibration is also normal bilaterally, but let's ignore that because sensory examination of the lower extremity is often inaccurate. Two-point discrimination is normal, but that can be a weird test, so forget that one, too. You happen to measure the size of each lower extremity, and they are even; in fact, the left leg is actually a little bigger (as mine is). Let's ignore that also, since we can't be sure what the sizes were before the "injury to the disc." Regardless of the "classiness" of the initial presentation, I would immediately be suspect of this "disc" if this patient came to my office with these signs and it had been going on more than six days.

With any true peripheral neuropathy, we will see degeneration of the nerve secondary to axonal changes, such that function distal to the site of compression is assessable. The most accurate test to evaluate true nerve compression is needle EMG, as a denervated muscle will present in a specific and classical way. If you are in doubt as to a true case of nerve compression, referral to one of your neurology-based colleagues who performs needle EMG is strongly warranted.

Distal to the site of the lesion, whether this is a herniated disc, a deviated first rib or a compartment syndrome, there will be atrophy of muscles and sensory loss, with vibration and two-point discrimination being the first things to go. In the previously mentioned case, without sensory loss, changes in the DTRs and any sign of muscle atrophy, I would initially be more likely to wonder if perhaps their upper extremity also showed some weakness. If this were the case, it would not be likely that a herniated L5-S1 disc would cause both upper and lower extremity weakness patterns, unless this was one world-breakingly massive disc.

The greater likelihood is that the patient is presenting with a higher level of lesion, such as a soft pyramidal weakness of the left side (if the upper extremity is also weak), or a disynaptic/post-synaptic inhibition of the extensors on the left due to some imbalance from the opposite side, or ... you get the idea.

As I have said many times before, you must treat what you see. Everything has a meaning and a reason for being there. If there is sensory loss, that will tell us something. If there is no sensory loss, that must also tell you something. Signs and symptoms are not to be ignored at our convenience so the diagnosis fits into a simple little box we can understand. Peripheral neuropathy can take many forms in many places in the body, but these general rules apply to all the sites.

Get *Myelopathy, Radiculopathy and Peripheral Entrapment Syndromes* and learn about these patients with peripheral neuropathy, because I can assure you they are coming in to your offices, and perhaps some of these "pinched nerves" are nothing of the sort.

As always, e-mail me with any questions or comments. Also, thanks to everyone for all the e-mails from the "[Weak Cyclist](#)" article (Nov. 18, 2008 issue). It was a lot of fun to hear from all of you.

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