Neuropathy A. Kirkner

Neuropathy, a condition where the nerves running from the brain or spinal column become damaged, is a very troublesome problem for clinicians and patients alike. Patients view the condition as incurable and see the only treatment as a lifetime's worth of cascading medications. Often, they take one family of medications to offset the effects of another. Despite the drugs, however, many patients continue to experience burning, tingling, a loss of sensation and balance, the development of wounds from trauma or infection, and even the threat of amputation. Clinicians dislike having to share this diagnosis with patients, particularly since they feel there is not much else to offer. They also know too well the downward spiral that their patients will likely be forced to endure. The question persists: what can be done for these patients to help them lead more comfortable lives? Before we [attempt] to answer that, let's first discuss the etiology of neuropathy.

There are three major types of neuropathy: post-chemo, diabetic related, and idiopathic. There are other ways to get neuropathy: mono neural neuropathies that are a result of trauma, and disease-related neuropathies that are a result of other illnesses such as Guillain Barre, **Charcot Marie Tooth,** and multiple sclerosis. Here, I am only going to address the more common neuropathies associated with diabetes, post-chemo, and idiopathic because of their similarity in pathology, and because they are so prevalent in our society.

Peripheral neuropathy has a singular pathology that, in my opinion, is mostly overlooked. In each and every case there has been vascular embarrassment that has caused the loss of C-fibers to the tissue. The microcirculation that has been necrotized by diabetes, post-chemo or from a cause that is unknown (idiopathic), must be restored before the C-fibers can regenerate. The other nettlesome problem that exists with this diagnosis is there is no way to measure C-fiber function. Currently, no reliable neurological test exists to measure these unmyelinated fibers, and therefore any improvement or further deterioration is reliant solely on patient feedback. There is the flash response from laser Doppler that claims to measure change in the vascular flow when the C-fibers fire. Unfortunately, success of this test has been spotty at best and, at present, is expensive and has no established criteria1. So, we are left with a problem that cannot be measured and that has a very difficult treatment protocol.

The question then becomes: how can we improve the microcirculation in a specific area? In order to do this, we must induce angiogenesis in a specific area of the body in order to promote growth of the C-fibers. How does the body accomplish angiogenesis? The major factor of angiogenesis is nitric oxide (NO). Through activation of guanalate cyclase (GC), NO leads to cGMP formation, which then stimulates growth of the microcirculatory bed 2. By following this

chain reaction back to the beginning, we can see that it would be beneficial to activate endothelium-based NO at the site of ischemia, the area damaged by the reduced blood flow.

What are the best activators of NO? There are several agents that activate NO: nitroglycerin, L-argenine, and Sildenafil, to name a few. But ingesting or injecting medications is not the answer, since the delivery is generalized. In the case of neuropathy, we need a specifically-focused NO release. The best choice is near-infrared light delivered to the area of neuropathy. Near-infrared light—either collimated (laser) or non-collimated (by light-emitting diodes)—has been demonstrated to locally increase angiogenesis. The efficacy of using a laser is limited by its small area of delivery and by the need of the clinician to hold the laser-head device at the site for the entire treatment. Non-collimated near-infrared light, using LEDs, can be delivered to a large area and can be left, unattended, with no limitations such as bony prominences, etc. Whichever method is chosen, however, the development of angiogenesis and subsequent regrowth of the C-fibers will result in your patients proclaiming that they have restored sensation, diminished pain, and improved balance.
Understanding how to restore microcirculation to areas of the body damaged by restricted blood flow is a huge breakthrough for patients suffering from neuropathy. [As a result], we should no longer view neuropathy as a hopeless condition. Our ability to stimulate angiogenesis and to regrow C-fibers, along with critical improved nutrition and lifestyle changes, should result in significant improvement in this patient group.

References

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2. Powell,Carnegie,Burke, Reversal of diabetic peripheral neuropathy and new wound incidence, Advances in Skin and Wound Care 17, 295-296, 298-300