

Diabetic peripheral neuropathy and neuro dynamics

Abstract

Diabetic peripheral neuropathy is a common, disabling, and costly complication of diabetes mellitus. In order to reach an exact diagnosis of electrophysiological studies and the evaluation of diabetic neuropathy it is crucial to obtain information about these variables. The information provided by electrodiagnosis is functional and not static, telling the practitioner how nerve and muscle are functioning. It is known that after a peripheral nerve, peripheral sensitization aberrant regeneration may occur alongside this the mobilization of the nervous system is an essential approach to physical treatment of pain. Neurodynamics encompasses interactions between mechanics and physiology of the nervous system. Alongside this, neurodynamic as a physical innovative therapy has been seldom used and in fact may be beneficial in preserving nerve function thus preventing the adverse effects of intraneural edema. That is, the rationale for the use of neurodynamic diagnosis and treatment is that it is considered capable of detecting the increased nerve structure associated with these conditions.

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Introduction

Neuropathic pain is an important problem because of its complex natural history, multiple possible etiologies, and poor response to standard physical therapy modalities. Disorders of the peripheral nerve system (PNS) are heterogeneous and may involve motor fibers, sensory fibers, small myelinated and unmyelinated fibers and autonomic nerve fibers, with variable anatomical distribution (single nerves, several different nerves, symmetrical affection of all nerves, plexus, or root lesions). Furthermore, pathological processes may result in either demyelination, axonal degeneration or both. In order to reach an exact diagnosis of any neuropathy electrophysiological studies are crucial to obtain information about these variables.¹

As a consequence of ongoing spontaneous activity arising from the periphery, Spino Thalamic Tract neurons develop increased background activity, enlarged receptive fields and increased responses to afferent impulses, including normally innocuous tactile stimuli. This phenomenon is called central sensitization. Central sensitization is an important mechanism of persistent neuropathic pain. Additionally, hyperalgesic/allodynic responses in uninjured neural tissues may be the result of alterations in central nervous system processing of afferent information (i.e. central sensitization).² It is known that after a peripheral nerve lesion, peripheral sensitization aberrant regeneration may occur. Neurons become unusually sensitive and develop spontaneous pathological activity, abnormal excitability, and heightened sensitivity to chemical, thermal and mechanical stimuli.³

The positive and negative symptoms associated with musculoskeletal presentations of peripheral neuropathic pain are produced by sensitized nociceptors in neural connective tissues, hypersensitive AIGS, a sensitized pain neuromatrix, myelin changes, and axonal degeneration (Nee & Butler, 2006). That is, some areas of the brain associated with sensory perception, emotion, attention, cognition and motor learning are activated during pain experience.⁴ Diabetic poly neuropathy is one of the most common long-term complications of diabetes affecting ~50% of all diabetic patients. For example, subclinical diabetic peripheral neuropathy can be detected by electrophysiological tests, which are useful to verify the range and extent of the nerve lesion involved in the early stage of diabetic peripheral neuropathy.⁵ Other findings suggest that thalamic

neurons can act as central generators or amplifiers of pain in diabetes.⁶ To this point and although there are multiple methods for detecting and monitoring DPN, nerve conduction studies (NCS) are generally considered to be the most sensitive and reproducible.⁷ In hereditary neuropathy, for example, electrophysiological studies are also used to distinguish axonal neuropathies from demyelinating neuropathies, though overlap and 'intermediate' patterns have become well recognized.⁸

The electrophysiological changes are not always concordant with clinical manifestations.⁵ The most common clinical and electrophysiological manifestation of diabetic neuropathy is the sensory disturbance, which is more severe in lower limbs.⁵ The information provided by electrodiagnosis has a functional character, telling the practitioner how the nerve and muscle are functioning. Nerve conduction studies (NCS) and qualitative sensory testing (QST) are important part of the complete electrodiagnostic exam. However, in pain syndromes, conventional studies may give normal results when large fibers are not involved, and the use of autonomic measures in such conditions is particularly relevant.⁸

According to Vinik et al.⁹ the main drawback of NCS is that small myelinated and unmyelinated nerve fibers, which are affected early in the disease course of diabetic neuropathy, do not contribute to the sensory action potential detected by routine NCS. Electrophysiological data must, therefore, always be evaluated in a clinical context. Evoked potentials, on the other hand have the capability of revealing clinically unsuspected pathology when demyelinating diseases are suggested.

Rehabilitation programs tend to be emphasized and combined with pharmacotherapy in daily practice.¹⁰ Traditional approaches use Transcutaneous electrical nerve stimulation (TENS) and interferential current (IFC) to relieve stiffness, improve mobility, relieve neuropathic pain, reduce edema, and heal resistant foot ulcers.¹¹ Neuro dynamic, i.e., the mobilization of the peripheral nervous system, is a physical approach to the treatment of pain; the method relies on influencing pain physiology *via* mechanical treatment of neural tissues and the non-neural structures surrounding the nervous system.^{2,13} Through clinical reasoning the nervous system seems to be the logical place for treatment and explanations, although previous descriptions of this method have not clarified the relevant mechanics and physiology,

including interactions between these two components. Within this reasoning it is important to determine and develop clinical research to ascertain the diagnostic value of neurodynamic sequencing in damaged neural tissue.

Conclusion

Neurodynamic may be beneficial in preserving nerve function by limiting intraneural fluid accumulation, thus preventing the adverse effects of intra neural edema.¹³ The rationale for using neurodynamic in diagnosis and treatment is that they are considered capable of detecting the increased nerve mechanosensitivity associated with these conditions.^{14,15}

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Conflict of interest

This manuscript has no conflict of interest with any parts.

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