



Evidence for Increased Magnetic Resonance Imaging Signal Intensity and Morphological Changes in the Brachial Plexus and Median Nerves of Patients with Chronic Arm and Neck Pain Following Whiplash Injury.

This is an excellent research report by Greening et al. discussing the morphological changes in the median nerve after trauma. This research report is very relevant to the Integrative Dry Needling (IDN) treatment concept of Neurological Dry Needling (NDN), and I wanted to comment on the key points and provide my clinical perspective.

- It is estimated 50% of patients that sustain a cervical whiplash injury develop chronic symptoms. This, of course, leads to the question, if healing is expected to occur over time, where is the persistent pain generated from? They point out that patients frequently present with signs of cutaneous hypersensitivity in the cervical spine and upper limb, such as allodynia and hyperalgesia and hypoesthesia, may indicate neuropathic pain causation.
- They suggest that nerve inflammation (neurogenic inflammation) occurs without overt damage (axonal degeneration or demyelination) and can be a cause of the neuropathic symptoms experienced by patients post whiplash. Neurogenic inflammation can be diagnosed using a T2-weighted MRI. An increase in the T2 signal intensity is an indicator of nerve edema that is a direct result of inflammation and vascular changes within the nerve structure. The key point here is the peripheral nerve inflammation can exist in the absence of demyelination i.e., structural damage. Consequently, nerve conduction studies may be read as negative, and clinical neurological testing may only show a minor sensory loss because only small-diameter axons are affected. This opens the opportunity to have a therapeutic effect when providing treatment, specifically dry needling, near the nerve structure.

The study demonstrated a greater T2 signal intensity occurred not only in the roots of the brachial plexus but also in the median nerve of patients with chronic pain post whiplash. This is in line with empirical findings that when a nerve has irritation in one area, assessment (and treatment) is necessary down the entire length of the nerve.

- We understand from Shah's work 2,3 that inflammation is not a local phenomenon, especially in the context of neurogenic inflammation.
- When the axonal transport system of a nerve is disrupted at a proximal site, it may increase the susceptibility of distal nerve segments to further injury (i.e., Double crush Theory). Another way to say that is neurogenic inflammation in one area of a peripheral nerve may lead to inflammatory processes along the entire nerve. Clinically, pain responses, as a result of the neurogenic inflammation, are commonly reproduced when performing nerve assessments, either with digital pressure or with stretch to the involved nerves.

- All of the study participants demonstrated signs of diffuse sensory changes (paresthesia and dysesthesia) of the upper limb, not localized to specific dermatomes or myotomes. They state a likely explanation for the diffuse symptoms is a central mechanism like central sensitization. Development and maintenance of sensitization in the dorsal horn require ongoing peripheral input from nociceptive axons that eventually results in the reported cutaneous hypersensitivities. Since the patients did not demonstrate signs of frank nerve injury, neurogenic inflammation could be a driver to this ongoing central barrage.

This study demonstrates objective evidence for the presence of neurogenic inflammation using T2 weighted Magnetic Resonance Imaging. This provides validation, within the limitations of the study, that the nerve itself can be the perpetuator of soft tissue dysfunction/pain. Within the current construct of dry needling treatment, the emphasis is on structures that are perceived to be the pain source, i.e., muscles. In situations like chronic whiplash, persistent symptoms may not be attributable to damage to musculoskeletal tissue. The neuropathic component of ongoing pain needs to be better elucidated with improved clinical application. This will require new thoughts and techniques on how to assess and treat neurogenic inflammation within the peripheral nervous system.

Clinical Relevance: If the inflammation/edema is within the nerve itself, at peripheral nerve terminals and around the nerve, this may contribute to decreased modulation of pain locally and at the spinal cord level. Neurogenic inflammation causes nerve irritation and resultant endogenous muscle guarding that is seen clinically in the form of a myofascial trigger point. According to this study, neurogenic inflammation can exist anywhere along the length of nerves, so it would seem inefficient to arbitrarily “hunt down” local trigger points searching for the source of a patient’s symptoms. A simpler global assessment of patient condition is needed, and the peripheral nervous system may be the key. Conceivably, a therapeutic intervention that focuses on decreasing the mechanical, physical, or chemical irritation of a nerve by normalizing blood flow and releasing endogenous muscle contraction would be of greater clinical and functional benefit. With proper knowledge of neuroanatomy, clinicians could locate the sensitized areas of the peripheral nervous system and provide more efficient and effective dry needling treatment.

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2. Shah J, Phillips T, Danoff J, Gerber L. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl. Physiol* 2005;99:1977-84
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