Chiropractic Management of Peripheral Neuropathy: Pathophysiology, Assessment, and Treatment

**Purpose:** Peripheral neuropathy is one of the most common conditions treated by chiropractors in the United States. The purpose of this article is to review the anatomy of peripheral nerves and the pathophysiology, outcome measures, and chiropractic manual treatment methods used with peripheral neuropathy syndromes.

**Methods:** A qualitative review of literature in referred literature and textbooks. **Summary:** An understanding of the pathophysiology of peripheral neuropathy and the symptomatic picture associated with peripheral neuropathy is important to choose an appropriate treatment and outcome measure. Symptoms range from no symptoms, to loss of light touch/vibration sense, to chronic pain. Proper management requires the use of appropriate outcome measures so that treatment effectiveness can be determined. Low-tech, noninvasive outcome measures are the recommended evaluation tools in the management of peripheral neuropathy. At present clinical trials documenting the effectiveness of chiropractic management of peripheral neuropathy are lacking. This article reviews chiropractic treatment ranging from a variety of soft tissue treatment methods to spinal and extremity manipulation for the treatment of peripheral neuropathy. Key words: nerve compression syndromes, neuralgia, neuroanatomy, orthopedic manipulation, peripheral nerves, peripheral nervous system diseases, questionnaires.

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**THE NATIONAL Board of Chiropractic Examiners** Job Analysis of Chiropractic found that peripheral neuropathy is the second most common neurological clinical condition treated by chiropractors in the United States (headaches are the most common). A wide variety of conditions may be involved in the etiology of peripheral neuropathy (e.g., metabolic/nutritional disturbances, inflammatory conditions, connective tissue disorders, toxaemia, hereditary conditions, malignancy, plasma cell dyscrasias, infections, and entrapment syndromes). However, it is likely that the entrapment syndrome (or compressive neuropathy) is the etiology of the peripheral neuropathies typically treated by the chiropractic profession. Compressive peripheral neuropathies have many causes such as fractures, tumors, and abnormalities in musculoskeletal structures such as passages within connective tissue, bone, and muscle.

This article will focus on chiropractic management of entrapment syndrome-type peripheral neuropathies from musculoskeletal abnormalities. Management in this article means not only the types of manual treatments used by the chiropractic profession but also the continuous assessment of therapeutic outcomes. There is only one clinical trial reported in the literature that has evaluated the effectiveness of chiro-

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practic management of any of these disorders.3 The majority of the literature is anecdotal in nature—case reports. Nevertheless, it should be emphasized that lack of evidence is not evidence of lack of effectiveness. Sackett has noted that evidence-based practice is not a slavish devotion to the published randomized clinical trial. Although the weight of such evidence is great, evidence-based practice is also based on clinical experience. A review of the pathophysiology of entrapment peripheral neuropathies is included so that the rationale behind both assessment and treatment can be understood.

PATHOPHYSIOLOGY

Both assessments of outcomes and treatment must be based on an understanding of the mechanism of signs and symptoms associated with peripheral neuropathy. By comprehending the mechanisms of the signs and symptoms of peripheral neuropathy one can make reasoned decisions about which outcome measures and treatment are most appropriate in the management of peripheral neuropathy.

Within the context of clinical neurology, it is not uncommon to find inconsistencies regarding terminology. Examples include the classification of somatic receptors, spinal pain syndromes, and subluxation. Equally common are discrepancies regarding the classification and characterization of peripheral neuropathies. For some, peripheral neuropathy refers to a painful disorder caused by neurocompression, and for others, it refers to neurocompression with symptoms such as numbness and/or tingling without pain, or perhaps no symptoms at all.

More recently, pain associated with a myofascial trigger point (TrP) has been characterized as a focal neuropathy or radiculopathy rather than a myofascial insult. Although new theories are necessary to advance patient care and research endeavors, the process of putting forth ideas can often unnecessarily confuse an issue. Such is the case with the subject of peripheral neuropathies, particularly when we consider that pain need not be present for a peripheral neuropathy to exist.

Simply stated, the clinical presentation of a peripheral nerve disorder is dependent upon which structures within a peripheral nerve are compromised. A brief review of peripheral nerve anatomy will help to clarify the nature of the signs and symptoms of peripheral nerve disorders and help guide the choice of diagnostic and therapeutic methods.

PERIPHERAL NERVE ANATOMY

A peripheral nerve consists of bundles of nerve fibers/axons and their associated supporting tissues and vascular supply.3 Three kinds of connective tissue complexes surround the axons that make up a peripheral nerve trunk including the endoneurium, perineurium, and epineurium. The endoneurium consists of supporting connective tissue, a basement membrane, endoneurial fluid, Schwann cells, and myelin in the case of myelinated axons.

Axons and their endoneurial components are grouped together and contained within the perineurium, creating a fascicle of axons. The perineurium consists of collagen, proteoglycans, a basement membrane, and perineural cells that are thought to be derived from fibroblasts. Although the perineural ensheathment provides the anatomic boundary of a nerve fascicle, it perhaps more importantly acts as a diffusion barrier that regulates intrafascicular fluid. The number of fascicles within a peripheral nerve varies among nerves.

The epineurium is composed of collagen tissue, elastic fibers, and fatty tissue, which tightly bind individual fascicles of axons together. The epineurium can constitute from 30%–75% of the cross-sectional area of a peripheral nerve, indicating that connective tissue can be the major component of peripheral nerves. The epineurium also contains the vascular supply for peripheral nerves; the vasa nervorum that branches into arterioles and penetrates the perineurium.

Peripheral nerves also have a nerve supply, the nervi nervorum, which provides an innervation pattern that may be similar to musculoskeletal structures, that is, both nerves and somatic tissues are innervated by nociceptors and mechanoreceptors. Although this area of peripheral nerve anatomy and neurophysiology has yet to be examined in detail, some basic information is known. Free nerve endings, presumably nociceptors, are present in the epineurium, perineurium, and endoneurium. Encapsulated endings, presumably mechanoreceptors, are found in the epineurium and sometimes intrafascicularly. The nervi nervorum also have perivascular branches that innervate the vasa nervorum, which are referred to as the nervi vasorum nervorum.

There are many different types of axons within a peripheral nerve. Motor fibers include alpha-motoneurons, gamma-motoneurons, and postganglionic sympathetic C fibers. Sensory fibers consist of mechanoreceptor afferents and nociceptor afferents. Mechanoreceptor fibers include Ia afferents from muscle spindles, secondary endings from muscle spindles, A-beta mechanoreceptor afferents that are associated with corpulso receptors found in joints, muscles, skin, and hair follicles. Although some A-delta fibers are associated with mechanoreceptors, they are generally classified as nociceptor afferents. C fibers are also nociceptor afferents and they arise from most musculoskeletal tissues.

NERVE COMPRESSION

For years it has been understood that peripheral nerve compression can produce symptoms ranging from slight paresthesia to total paralysis, all of which depends upon the
cause, degree, and duration of compression.25 Modest pressure will compress the cytoskeletal constituents of the axoplasm and reduce the caliber of the axon, which results in conduction block that is readily reversible. Neuropaxia is the term used to describe nerve injury or compromise caused by modest pressure that result in focal conduction block but no wallerian degeneration, that is, degeneration of the axon distal to the site of injury.16 It should be mentioned that demyelination will occur in chronic neuropaxic lesions, and this is attributed primarily to mechanical compression, rather than ischemia.12

Axonotmesis is caused by greater nerve pressure caused by crush injuries or freezing and results in wallerian degeneration. The axon and myelin sheath are displaced from the site of lesion, but the surrounding basal lamina remain in continuity, which provides a route for axonal regeneration and functional restoration. Neurotmesis is more severe than axonotmesis and is associated with penetrating types of injuries that involve the transection of basal lamina and the nerve fiber itself, resulting in wallerian degeneration.16

Sunderland13 described five degrees of nerve injury; the first two are synonymous with neuropaxia and axonotmesis, respectively, whereas degrees three through five equate with neurotmesis. Sunderland also described three stages of pathology related to nerve compression. During stage 1, capillary congestion, caused by obstructed venous return, slows intrafunicular capillary circulation. The unyielding properties of perineurium results in an increased pressure inside the funiculus, which further reduces circulation, leading to impaired nutrition, hypoxia, and hyperexcitability of certain fibers. If pressure is relieved, normal circulation and conduction is restored.

Stage 2 will begin if compression is not relieved. Hypoxia/ anoxia will damage capillary endothelium. Inflammatory events such as protein leakage and edema occur, causing intrafunicular pressure to further increase. The pressure and anoxia threaten nerve survival by impairing nutrition and metabolism, physical deformation, and fibrous tissue infiltration. Segmental demyelination occurs in some fibers, whereas wallerian degeneration occurs in others. Resistant fibers conduct normally, whereas others suffer from conduction block. Reduction or elimination of compression at this time can restore circulation, resolve edema, and normal function can gradually return. However, the restoration of sensory and motor function depends on whether the nerve suffered first or second-degree injury and the extent to pathologic processes are irreversible. If compression continues, stage 3 will ensue, which involves permanent fibrotic damage.17

Based on the above descriptions, neuropaxia associated with conduction block16 would be most amenable to chiropractic management. Kimura12 suggests that short-term changes, associated with a neuropaxic lesion, are caused by anoxia secondary to ischemia. More chronic lesions, reflecting chronic nerve ischemia, may initially result in axonal degeneration of sensory fibers and then later in motor fibers. Accordingly, signs and symptoms would reflect the extent of nerve compromise and the fiber types that have been affected.

From a clinical perspective, neuropaxia may be asymptomatic or result in symptoms associated with a loss of conduction in myelinated mechanoreceptor axons such as numbness and tingling, and the clinical exam may reveal alterations in sensation, particularly light touch and vibration. For example, one of the earliest detectable signs of carpal tunnel syndrome is altered vibration sensibility, which has lead to the suggestion that at-risk workers should undergo periodic vibration perception assessments.15 In general, the sensory exam can be performed with low-tech equipment,16 and also with high-tech diagnostic devices. The incidence of peripheral nerve compromise might be more common than what is typically thought. Kimura notes:

Unexpected abnormalities of nerve conduction studies in asymptomatic subjects suggests a high incidence of subclinical entrapment neuropathies. Routine autopsies in patients without known disease of the peripheral nerve have also documented unpredicted focal anatomic abnormalities.26,27

As mentioned earlier, pain need not be present for a neuropaxic lesion to exist. More precisely, neuropaxia is not pain and should never be equated with pain, and similarly, the term peripheral neuropathy should never be equated with pain. However, in clinical practice, pain often occurs in conjunction with symptoms attributable to neuropaxia or peripheral neuropathy; a relationship that needs to be clarified.

PAIN ASSOCIATED WITH PERIPHERAL NEUROPATHY

There are two basic mechanisms by which the experience of pain can be evoked, those being somatic tissue injury or nerve injury. Far and away, somatic tissue or musculoskeletal injury is most common.8,20,21 As mentioned above, nearly every musculoskeletal tissue is innervated with nociceptors, the irritation of which usually results in pain. In short, stimulation of tissue nociceptors causes action potentials to be propagated along nociceptive axons that ultimately enter the limbic sectors of the cerebral cortex where the pain is perceived. This is referred to as nociceptive pain and is typically characterized as deep, tender, dull, aching, and diffuse. Myofascial pain associated with TrPs is typically described in this fashion, suggesting that it is nociceptive pain. Referred pain from joints and muscle is also an example of nociceptive pain. Terms that describe this nociceptive-induced pain include: myofascial pain, muscle pain, fibromyalgia, trigger points, joint pain, cervicogenic headaches, referred pain,
sclerotomal pain, sclerotogenous pain, deep pain, diffuse pain, primary disc pain, mechanical pain, simple backache, somatic pain, and somatic referred pain.\(^6\)

Pain caused by nerve injury is very different than nociceptive pain. When nerve injury is sufficient, demyelination will occur and nerve impulses can be generated at the site of the injury, which is referred to as ectopic electrogensis.\(^23\) In this situation, the nerve changes its fundamental character such that it becomes an impulse generator, instead of merely a conductor or propagator of impulses. The pain that results from nerve injury is referred to as neuropathic pain, a term that is often incorrectly used whenever a patient is considered to suffer with chronic pain.\(^6\) It should be understood that chronic pain is not synonymous with neuropathic pain. Chronic pain can either be nociceptive or neuropathic. However, specific criteria must be met for a pain to be considered neuropathic. Simply stated, "the acquisition by injured nerve fibers of ectopic pacemaker capability is among the fundamental pathophysiological changes that underlay the emergence of neuropathic pain."\(^22\) Neuromas, regenerating sprouts, and demyelination are thought to be responsible for the ectopic discharge of injured nerves. In other words, without nerve injury and ectopic electrogensis, pain cannot be considered neuropathic.

In contrast with nociceptive pain, neuropathic pain is commonly associated with abnormalities in the physical exam including changes in sensation, reduced tendon reflexes, loss of muscle strength, and, perhaps, atrophies, all of which are caused by compression of either sensory or motor axons within the nerve. The exception to this rule would be neuropathic pain that is generated by neri nervorum (discussion follows). Neuropathic pain is characterized by intermittent bouts of sharp, shooting, jabbing, and/or lancinating pain. In addition, steady burning and tingling sensations are usually present. In contrast to nociceptive pain, these neuropathic pain sensations are often entirely new and different to the patient. The terms used to describe these abnormal sensations include paresthesias, dysesthesias, and hyperpathia.\(^6\) It should be understood that this variety of neuropathic pain is rarely encountered in clinical practice,\(^6,20,21\) which is fortunate considering the fact that once neuropathic pain develops it rarely goes away.\(^23,24\)

In clinical chiropractic practice, a variety of soft tissue and manipulative techniques (see heading labelled “Treatment”) are used to reduce pain, the symptoms of neuropraxia, and to restore extremity muscle function. Although such techniques may reduce pain and neuropraxic symptoms, immediate pain reduction/resolution provides a clear indication that the pain was nociceptive in nature, not neuropathic.

Nociceptors are present in nearly all musculoskeletal tissues and within the connective tissue of peripheral nerves. Clearly, strained and injured musculoskeletal tissues can result in pain without symptoms of peripheral neuropathy. As an example, the myofascial pain associated with TrPs is typically characterized as nociceptive pain and is not necessarily associated with neuropathic signs or symptoms.\(^25\) However, peripheral neuropathy can also be present if the compromised musculoskeletal structures were to impinge upon a peripheral nerve. Although experimental evidence is lacking, clinical experience suggests that nociceptive pain frequently accompany the symptoms of peripheral neuropathy, both of which improve after manual soft tissue treatments.

Nociceptive pain associated with peripheral neuropathy may also come from the nociceptors found in the connective tissue of peripheral nerves, that is, the neri nervorum. Modest compression of a peripheral nerve may be sufficient to cause symptoms of neuropaixia, but not demyelination, ectopic electrogensis, and neuropathic pain. However, modest compression and local inflammation may be of a sufficient magnitude to stimulate the neri nervorum, resulting in nociceptive pain.\(^17\)

Confusing the mix even further is the possibility that a peripheral nerve can be compromised by a neuropathic lesion, whereas the associated neri nervorum suffer a more severe injury, resulting in the formation of a neuroma-in-continuity, ectopic firing, and neuropathic pain. Bove, et al. have pioneered the contemporary investigation of the neri nervorum,\(^26-28\) and believe that such injury to the neri nervorum may be a key missing link to better understanding the nature of neuropathic pain.\(^14\)

Bove (Geoffrey Bove, personal communication, 3/29/99) also believes that the neri nervorum play a key role in the development of myofascial pain, such that TrPs commonly manifest as focal painful neuropathies within muscle. He suggests that injury to a muscle will cause inflammation and result in the depolarization of neri nervorum, resulting in nociceptive pain. It is also possible that inflammation will encroach upon peripheral nerves as they pass through the muscle or an associated passage. Consequently, during normal movements, the nerve will experience friction injury causing the neri nervorum to discharge like other nociceptors.

Fibrous tissue deposition is a natural part of the healing process, and Bove suggests that during this process the neri nervorum may become bound to adhesions or scar tissue in local musculoskeletal tissues. Consequently, a movement may traction the neri nervorum causing them to chronically discharge. Soft tissue methods that break adhesions and restore mobility will remove the noxious irritation and allow the neri nervorum to heal (Geoffrey Bove, personal communication, 3/29/99). The point at which injury to the neri nervorum would lead to neuropathic pain is not yet understood. It is possible that traction or friction of a sufficient magnitude would injure the neri nervorum such that a miniature neuroma-in-continuity develops and neuropathic pain ensues.\(^16\)
Patients with neuropathic pain of nervi nervorum origin may not necessarily have symptoms of neuropraxia, that is, changes in the sensory or motor exam. This is because the axons in a peripheral nerve can be left undisturbed despite injury to the nervi nervorum. For a review of this subject see Greening and Lynn.39

MECHANISMS OF MYOFASCIAL ENTRAPMENT OF PERIPHERAL NERVES

A brief discussion about some of the ways in which peripheral nerves become entrapped between the various soft tissues is important. All of the peripheral nerves go through the soft tissues as they course from their spinal origins toward their final peripheral destinations. These peripheral nerves must be free to glide and move within myofascial and ligamentous canals or openings through which they travel.30,31 At various key locations in the body, peripheral nerves become vulnerable to compressive or traction forces that may disrupt their normal function.

There are four basic mechanisms by which peripheral nerve function may become altered by the soft tissues; the first three being compressive mechanisms and the last being a traction mechanism:32

1. Direct compression between the bellies or fibers of hypertonic muscles. Examples of this mechanism are the median nerve between the two heads of the pronator teres muscle,33 and the sciatic nerve between the fibers of the piriformis33 (in cases where the nerve pierces through an opening in the belly of the piriformis).

2. Direct compression between muscles and an unyielding ligamentous or fascial sheath. Examples of this mechanism are the radial nerve between the fascial opening known as the Arcade of Froesch34 and fibers of the supinator muscle, and the obturator nerve between a fascial sheath, the fibers of the adductor longus muscle, and the inguinal ligament.35

3. Direct compression between bones and hypertonic muscles. Examples of this mechanism are the lower cords of the brachial plexus (C7, 8) between the coracoid process and the pectoralis minor,36 and the peroneal nerve between the fibula and the peroneus longus or extensor digitorum longus muscles.37

4. Traction caused by adhesions. Examples of this are adhesions between the median nerve and the transverse carpal ligament or carpal bones,38 and the lower lumbar nerve roots post disectomy or laminectomy.

There are many sites in the body where peripheral nerves may become entrapped by any of the four mechanisms listed above. Some of the more common areas of myofascial peripheral nerve entrapment are listed in Table 1 for reference.25,39

For a detailed list of possible sites of peripheral nerve entrapment see Brazis,40 Patten,41 and Hammer.42

Most manual myofascial techniques would view these peripheral nerve entrapments as a secondary consequence of hypertonic muscle fibers causing the entrapment by one of the first three mechanisms noted above; direct compression between hypertonic muscle fibers themselves, between hypertonic fibers and a fascial sheath, or between a bony prominence and hypertonic fibers. Therefore, the clinician using methods such as ischemic compression (Nimmo),43-46 stretch and spray,25,39 or postisometric relaxation47-52 would chiefly be concerned with determining which specific muscle is responsible for causing the peripheral nerve entrapment, find the localized area within that muscle where the entrapment is occurring, and then apply manual treatment directly over those hypertonic fibers.

Leahy and Mock have proposed53 that repetitive strain or cumulative trauma from muscular overuse will lead to fibrous adhesion formation between muscles, fascia, and peripheral nerves. They claim to be able to determine the location of these soft tissue adhesions by manual palpation, which like palpation for TrPs and hypertonic muscle fibers, takes much skill and practice. Those using the myofascial release technique of Leahy and Mock would view peripheral nerve entrapments as caused by traction of the peripheral nerve by adhesions, rather than compression between hypertonic muscle fibers. This method of treatment consists of applying a combination of manual pressure and tension directly over the site of peripheral nerve/soft tissue adhesions, using either active or passive movement of the extremities.

It is also interesting to note that within the McKenzie system54 of diagnosis and treatment of spinal disorders, there exists a specific diagnostic category called an “adherent nerve root.” This diagnosis is reserved for those patients who are refractory to conservative mobilization and manipulation methods, and who appear to get symptoms of paresthesia or weakness at a certain point within a range of motion, such as numbness in the posterior leg during lumbar flexion. McKenzie-trained therapists use manual stretching techniques according to the work of Elvey,55 and Butler,56 which is thought to mobilize the peripheral nerves and thereby stretch apart any adhesions that have developed along the course of the peripheral nerve. This technique of mobilization of the peripheral nerves is chiefly designed for treatment of adhesions of the brachial and lumbar plexus using various positions of the upper and lower extremities as levers to exert stretching forces along the respective peripheral nerves. Although it is not a specific myofascial technique, the McKenzie adherent nerve root stretching procedures are based upon a similar mechanism of action as the work of Leahy and Mock, peripheral nerves must be free to glide through the soft tissues without tension from adhesions.
Table 1. Common myofascial entrapment sites of peripheral nerves\textsuperscript{35,39}

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Nerve Entrapment</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pectoralis minor</td>
<td>Lower brachial plexus</td>
<td>Paresthesia in ulnar aspect of forearm and digits 4/5.</td>
</tr>
<tr>
<td>Extensor carpi radialis brevis</td>
<td>Radial nerve (sensory branch)</td>
<td>Paresthesia over dorsal forearm, wrist, digits 1, 2, 3.</td>
</tr>
<tr>
<td>Supinator</td>
<td>Radial nerve (motor branch)</td>
<td>Weakness of hand and finger extensor muscles.</td>
</tr>
<tr>
<td>Flexor carpi ulnaris</td>
<td>Ulnar nerve (motor and/or sensory branches)</td>
<td>Paresthesia over digits 4/5. Weakness and/or loss of grip strength (finger flexors).</td>
</tr>
<tr>
<td>Pronator teres</td>
<td>Median nerve</td>
<td>Paresthesia of digits 1, 2, 3 and weakness of thumb adductor muscles. Mimics carpal tunnel syndrome.</td>
</tr>
<tr>
<td>Peroneus longus</td>
<td>Peroneal nerve (common or deep branches)</td>
<td>A foot drop or weakness of dorsiflexor muscles (deep br)</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Deep peroneal nerve</td>
<td>Paresthesia over dorsal web of digits 2/3 (common branch)</td>
</tr>
<tr>
<td>Abductor hallucis</td>
<td>Posterior tibial nerve</td>
<td>A foot drop\textsuperscript{30}</td>
</tr>
<tr>
<td>Semispinalis capitis and upper trapezius</td>
<td>Greater occipital nerve (dorsal primary ramus of C2)</td>
<td>Symptoms of tarsal tunnel syndrome; paresthesia along medial foot and toes.</td>
</tr>
<tr>
<td>Sternocleidomastoid (SCM)</td>
<td>Spinal accessory nerve (C.N.XI)</td>
<td>Paresthesia over the base of the occiput.</td>
</tr>
<tr>
<td>Anterior and middle scalenes</td>
<td>Brachial plexus (upper or lower)</td>
<td>Weakness or paresis of ipsilateral trapezius.</td>
</tr>
<tr>
<td>Teres major/minor and long head of triceps</td>
<td>Axillary nerve</td>
<td>Paresthesia in ulnar (lower) or radial (upper) distributions.</td>
</tr>
<tr>
<td>Piriformis</td>
<td>Sciatic nerve</td>
<td>Paresthesia over deltoid region and weakness of deltoid muscle.</td>
</tr>
<tr>
<td>Iliopsoas</td>
<td>Genitofemoral, ilioinguinal, iliohypogastric, or lateral femoral cutaneous nerves</td>
<td>Paresthesia over posterior thigh, calf, and sole of foot.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paresthesia in groin, scrotum, labia, etc/or anterior thigh.</td>
</tr>
</tbody>
</table>

Another potential etiology of peripheral neuropathy symptoms is the "double crush syndrome," which was coined in 1973 by Upton and McComas. This term is intended to describe the clinical manifestation subsequent to nerve compression or entrapment at two sites, both asymptomatic in isolation, that result in a summation of effects producing symptoms. This condition is generally accepted as valid in clinical practice.\textsuperscript{35,36} However, Swenson\textsuperscript{34} in his review concludes that although there is some basic science, there is a lack of compelling evidence to support the conclusion that two clinically insignificant lesions can cause neurologic impairment. One must wonder if the general acceptance of this condition is premature or if the scientific evidence is just delayed.

OUTCOME MEASURES

Management of peripheral neuropathy should be based on the model of the continuous feedback loop. Treatment protocol should change as a reaction to the responsiveness (positive or negative) of the patient's condition to treatment. Change in the patient's condition needs to be established by objective outcome measures.\textsuperscript{37,38} In addition, those same outcome measures may also be used to suggest at an early stage that a patient has a problem – early screening. This would lead to diagnostic testing and an early diagnosis that should minimize the impairment and provide for a more complete and rapid recovery.\textsuperscript{39}

The objective outcome measures used in peripheral neuropathy can be either low-tech or high-tech. These tests can also be thought of as being along a continuum: from tests that measure neural potential (eg, nerve conduction velocity) to those that measure a patient's actual ability to function (eg, two-point discrimination).\textsuperscript{60} Dyck\textsuperscript{37} suggests that a patient with low nerve conduction may be categorized as having a severe neuropathy even though they are asymptomatic. Although these patients will usually develop symptoms of neuropathy, the severity must be based on the type and degree of the severity of the neuropathic symptoms and the neurologic deficits.

A definitive diagnosis may be best made with the use of the high-tech procedures. However, because the high-tech procedures often involve a high level of invasiveness, are difficult to interpret, and are costly,\textsuperscript{61,62} they may not be suited as tools of treatment management, that is outcome measures. Some of these tests (ie, current perception threshold\textsuperscript{63} or motor nerve conduction tests\textsuperscript{64}) may not be sensitive enough to be of value,
either. If the patient’s symptoms are not consistent with a differential diagnosis of demyelination or axonal degeneration (causing neuropathic pain), high-tech tools like nerve conduction studies may not be appropriate. Because low-tech outcome procedures are usually minimally invasive and low cost, they are properly used at regular intervals during treatment for the continuous monitoring of outcomes. Only through tracking outcomes can it be determined whether:

1. The treatment is effective and should be continued.
2. The treatment is not effective and should be modified or the patient referred for surgical consult.
3. The treatment has been effective; however, the patient has reached their maximum improvement and should be discharged from care.

High-tech procedures typically used in the diagnosis of peripheral neuropathy include nerve conduction, electromyography, vibrometry, thermal sensitivity testing, and dynamometry. Low-tech procedures typically used to manage cases of peripheral neuropathy as outcome measures include dynamometry (eg, Jamar hydraulic type dynamometer and pinch gauge), two-point discrimination, light touch assessment with monofilaments, questionnaires, and pain assessment instruments. For the purposes of discussing management of peripheral neuropathy, diagnosis is assumed and only outcomes will be discussed.

CUTANEOUS SENSATION

Given that the first symptom of peripheral nerve compression is the loss of light pressure sense, tests of two-point discrimination, vibration, or light touch appear to be logical outcome measures. In fact, the Weber two-point discrimination test has been recommended for use as an outcome measure in peripheral neuropathy. However, the test has been found to be unreliable. Likewise, although vibration tests have also been recommended as an outcome in peripheral neuropathy, these tests also have been found to be neither reliable nor very sensitive (abnormalities only detected when nerve conduction time was at least three standard deviations above control mean). The problem with both vibration and two-point discrimination is that the clinician is not able to apply a consistent force to either the two points or to the vibrometer when placing them on the patient’s skin. Another problem with vibrometers is that they cause vibration that goes beyond the site being tested and thus could be felt at some point distant to the point of application.

These problems are eliminated with monofilament testing of light touch. Von Frey first evaluated light touch threshold in the 1800s using horsehair in a device that allowed the examiner to change the length of the hair. The longer the hair the lower the force applied before the hair bent. Calibration problems lead to the development of the Semmes-Weinstein monofilament test. The Semmes-Weinstein monofilaments have been studied extensively and found to be reliable, sensitive, and specific when used appropriately. An updated version of the Semmes-Weinstein monofilm test has been developed, the Weinstein Enhanced Sensory Test (WEST), which appears to be an advance over the older test. The WEST monofilaments are calibrated for applied force in milligrams. Each Semmes-Weinstein monofilm is labeled with the logarithm of the force in tenths of a milligram. The tips of the WEST are designed to prevent slippage from the site of application and to prevent noxious stimuli even at higher forces. Normative data has been published for Semmes-Weinstein monofilaments and for the WEST.

Monofilaments are used by starting with higher force filaments and moving to the lower forces. The filament is slowly brought down onto the patient’s skin until the filament bends. The subject is told to report when they note they have been touched. The lowest force that can be felt is the patient’s threshold. Care must be taken to avoid touching body hair. A study by Gillenson et al found that light touch threshold does not change in normal subjects as wrist position changes. It was suggested that any change in threshold as a result of changing position of the wrist is probably caused by the pathology and not exclusively because of the wrist position.

PAIN ASSESSMENT INSTRUMENTS

The most commonly used measures of pain are the visual analog scale (VAS) and the McGill Pain Questionnaire. The advantages to using the VAS are the speed and relative ease in using the scale. Another advantage is its ability to be used to assess symptoms other than pain. Accordingly, the VAS could be used to assess patient’s perception of the degree of numbness, paresthesia, or weakness associated with their peripheral neuropathy. However, the VAS is less useful in nonliterate, elderly, or less educated populations. It appears that the metaphor of pain as a continuum is difficult for this population to understand and a numerical scale rating pain intensity from 0 to 10 is more reliable for them. With literate groups, the VAS has a high reliability ($r = .94$, and $r = .71$ in the nonliterate groups).

The McGill Pain Questionnaire (MPQ) is the leading instrument for describing the various characterizations of pain. The whole questionnaire includes questions about the patient’s diagnosis, medication, medical history concerning pain, present pain pattern, accompanying symptoms and modifying factors, effects of pain, and a list of words describing pain. The lists of words is the typical part of the questionnaire that is used. There are 102 words in the list divided into three major classes of pain proposed in Melzack’s theory of pain. The classes are sensory qualities of pain, affective qualities of pain, and the totality of the pain experience.
Within the classes are 20 subclasses of words grouped by their qualitative similarity. Scoring of the MPQ is done by one of the four methods. The easiest is to count the number of words chosen that correlates well with the two methods of Pain Rating Intensity (.97 for PRI (S) and .89 for PRI). The MPQ can be obtained from, and voluntary donations (50.10 per use) to pay for its use should be sent to, the International Association for the Study of Pain: IASP Secretariat, 909 NE 43rd St., Suite 306, Seattle, WA 98105-6020, USA.

QUESTIONNAIRES

It has been suggested that generic and disease-specific instruments should be used to provide a comprehensive outcome measure. Atroshi et al. recommend, as the generic instrument, the Short-Form-36 Health Survey (SF-36). The SF-36 was developed for use in population surveys and in the evaluation of health policy. It has been studied extensively, shown to be both valid and reliable and population norms have been published. At this time it is the preeminent generic overall health instrument. The forms can be obtained from: The Medical Outcomes Trust, PO Box 1917, Boston, MA 02205

Few disease-specific instruments have been developed. Levine et al. developed the carpal tunnel specific Symptom Severity Scale and Functional Status Scale. They determined that these scales measured some element of the patient’s clinical presentation not measured by other instruments and was reliable, internally consistent, and responsive to clinical change. These scales have been used as the outcome measure in studies of treatment for carpal tunnel syndrome.

Medline searches and a review of various textbooks were unable to identify any other disease-specific instruments for peripheral neuropathy type syndromes.

ASSESSMENT OF MUSCLE STRENGTH

Grip strength or pinch strength (between the thumb and index finger) has been used as an outcome measures in carpal tunnel syndrome. However, loss of muscle function is a late effect of neuropaxia and does not usually appear until overt sensory changes have occurred. The Jamar dynamometer is the most commonly used instrument to assess grip strength. The validity and reliability of this instrument and norms have been published. When assessing clinical improvement, Nitschke et al. found that a difference of greater than 6 kg is necessary to detect a real change in grip strength when using a Jamar dynamometer.

TREATMENT

There are many manual techniques used by practitioners to treat soft tissue lesions, some with “brand” names associated with an entrepreneurial developer and others with “generic” names based upon anatomic or physiologic descriptions of the procedures. For the purposes of this review article, three generic manual myofascial techniques will be outlined and discussed that are used regularly in one of the author’s (MJS) practices to treat peripheral nerve entrapments. There are many other manual soft-tissue techniques that are more focused on the treatment of tendons and fascia, such as “Transverse Friction Massage,” a newly described technique “Intraventricular Fascial Release,” and others that will not be covered in this article for the sake of brevity.

Manual myofascial techniques

The first manual myofascial technique has been commonly referred to as ischemic compression. With this method, the clinician applies deep, firm manual pressure directly over the TrP nodule or hypertonic fibers, and holds the manual pressure for a specified period of time. In chiropractic circles this generic method is commonly known as the Nimmo technique named for the chiropractic pioneer who first developed the procedure, Dr. Raymond Nimmo. Nimmo actually coined the name receptor tonus control method (RTCM) for his technique, but common usage of the term Nimmo technique overshadowed his preference for RTCM. Massage therapists, physical therapists, and other body workers may call variations of this method myotherapy or neuro-muscular therapy.

Simons has proposed some theories of the mechanism of action of deep manual pressure applied to TrPs, including mechanical disruption of the locked actin-myosin cross links by the deep localized pressure on the TrP nodule. It is not clear that ischemia is necessarily involved with the release of TrPs by manual pressure, and therefore Simons now suggests that the term ischemic compression be discontinued in the literature, and a new term, trigger point pressure release be used. Because of the lack of standardization of the terminology, we will use the term ischemic compression for the remainder of this article.

Ischemic compression is a relatively simple treatment method. The examiner finds a TrP or hypertonic band by manual palpation of the muscle. Deep pressure is applied directly over the lesion for about 7–10 seconds, then slowly released. The pressure is held steady and not subject to rubbing or stroking movements. The same TrP would typically be treated with deep pressure for a maximum of three to four applications on that day’s visit. It is thought that too many applications in one visit, too much manual pressure, or too frequent treatments may cause the patient to become bruised posttreatment. Table 2 outlines the method of ischemic compression treatment as originally described by Nimmo.

The second form of manual soft-tissue technique involves the use of patient generated isometric muscular forces fol-
Table 2. Summary of ischemic compression

- Use scanning palpation (gentle cross fiber palpation) to locate trigger point TrP nodules and taut bands.
- Snapping palpation of taut bands may elicit a local twitch response, confirming TrP.
- Deep manual pressure is held steady for 3–7 seconds, applied to patient tolerance and slowly released.
- While maintaining this steady manual pressure, ask patient to:
  - Rate the intensity of local pain using a 0–10 scale.
  - Describe any symptoms of referred pain.
As a rule, primary TrPs will elicit distal referred pain and be rated severely painful (>7/10), whereas secondary or minor TrPs will not evoke referred pain and will be rated mildly painful (<5/10).
- Release pressure on TrP and continue to scan remainder of muscle or other muscles for TrPs and taut bands, allowing the TrP just treated a chance to Arest®.
- Return to TrP just treated and reapply manual pressure. Each TrP may require up to 3–4 applications of manual pressure during one office visit.
- After a few applications of manual pressure, expect to feel less nodularity and decreased associated taut band activity within the muscle fibers. The patient should also report some immediate change in the symptom pattern.

Clinical caveats: There are a few reasons why some clinicians may not succeed with this method:
- Excessive manual pressure is used, which may bruise the muscles. Care must be taken especially with patients who are on anticoagulants or steroids, frail, or elderly.
- Too many applications of pressure are used during one office visit, or the pressure is held too long.
- This method should not be used directly over bruised/stained muscles, hematomas, major blood vessels or neurovascular structures.

Table 3. Summary of postisometric relaxation

- Place hands on muscle in order to pretension the fibers into a mild stretch position, taking out the “slack” to the point where a barrier of resistance is felt.
- Patient is asked to gently perform an isometric contraction of the muscle for 5–10 seconds, being told to use about 25% of their maximal strength. This is not a tug of war, but rather a gentle contract-relax method of coaxing the muscle to a greater length.
- After isometric contraction, the patient is told to “Let Go”, while the clinician gently stretches the muscle to the point of new resistance (engaging a new barrier). Eye movements and breathing are helpful adjuncts in facilitating the stretch (see Lewit47–49).
- Each time the clinician stretches the muscle, this new position of greater length is maintained as the baseline position from which the patient will perform another isometric contraction. The process is repeated about 3–4 times in one office visit.
- During successful application of this method, the clinician and patient will feel a “release” of the muscle; which can be described as a sensation of the muscle abruptly lengthening and softening.

Clinical caveats: There are two circumstances that need to be avoided with this method:
- Excessive isometric contraction or aggressive stretching may cause cramping of the muscle, or micro-tears of the muscle, both of which are wise to avoid.
- Stretching across unstable joints, damaged discs, strained muscles, or sprained ligaments is contraindicated. Also, it is wise to avoid stretching muscles in the region of radioculopathy, such as the scalenes or upper trapezius with brachial radioculopathy.
(ART)\(^2\) whereas Mock\(^3\) has continued to publish under the generic term MRT. This particular method of soft tissue therapy blends some degree of manual pressure with active and/or passive stretch of the affected muscle tissue. To a less observant observer, it would appear that this method is nothing more than ischemic compression applied to the patient during active or passive stretch. However, MRT is more related to the family of stretching procedures than deep pressure procedures because the intent of the procedure is to strip away myofascial adhesions rather than release hypertonic muscle fibers. \(^{52}\)

Leahy and Mock\(^2\) describe four levels of MRT. Level 1 is really a variation of muscle stripping, where the clinician applies longitudinal manual pressure “along the grain” with the muscle in a neutral position, and level 2 is the same procedure with the muscle positioned with passive stretch/tension. Levels 3 and 4 involve passive and active movements of the muscle to be treated, respectively. Both levels 3 and 4 of MRT use the same manual contact by the clinician, which is a broad, flat finger contact just distal to the lesion while the muscle is passively stretched by the clinician in level 3, or actively stretched by antagonist muscle contraction in level 4.

Several mechanisms of action may explain the clinical results of MRT. Although Leahy and Mock speak only of adhesions, it is possible that this method is simultaneously releasing hypertonic muscle tissue along with myofascial adhesions. First, the longitudinal nature of the muscular stretch and stripping actions may literally pull-apart actin-myosin contractures found in TrPs, or abnormal scar formations found in soft tissue adhesions. Second, the active and passive stretching may inhibit hypertonic muscle fibers and taut bands by virtue of muscle spindle or Golgi tendon organ activity. Finally, in level 4 MRT the principle of reciprocal inhibition is combined with specific stretching. Table 4 outlines the four levels of MRT.\(^{3,2-96}\)

**CLINICAL APPLICATIONS OF MANUAL MYOFASCIAL TECHNIQUES**

The chiropractic clinician is likely to encounter peripheral nerve entrapment syndromes often in practice. The clinical ability to diagnose and manage them is therefore a necessary competency. All of the previously noted manual treatment methods have their value in clinical practice, yet they each have their own respective strong and weak points. As with osseous manipulative or adjutant techniques, the type of soft tissue treatment method will depend on many factors, such as patient age, frailty, medications, pain threshold, joint instability, and so on.

Keeping in mind that every patient has unique circumstances; we must maintain an open mind to altering our treatment methods and plans to best serve our patient’s clinical needs.

<table>
<thead>
<tr>
<th>Table 4. Summary of myofascial release techniques</th>
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<tr>
<td><strong>Level 1</strong>: Patient is passive and no tension is applied. Clinician applies longitudinal gliding pressure along the grain of the muscle passing through the Anodule® or Aadhesion®. This appears very similar to a generic version of stripping massage.</td>
</tr>
<tr>
<td><strong>Level 2</strong>: Patient is still passive, but now tension is applied by positioning the muscle such that there is a moderate amount of stretch. The same gliding pressure is applied by the clinician as in level 1.</td>
</tr>
<tr>
<td><strong>Level 3</strong>: Patient is passive, and now the muscle is passively stretched by the clinician while he/she applies a superficial gliding pressure through the Aadhesion® or Anodule®. This level uses a combination of manual pressure longitudinally along the grain of the muscle, while simultaneously using a specific passive stretch of the same fibers.</td>
</tr>
<tr>
<td><strong>Level 4</strong>: Patient actively contracts the antagonist muscle, while the clinician applies superfluous pressure as in level 3. The contraction of the antagonist causes reciprocal inhibition and stretching of the muscle being treated. Levels 3 and 4 are the preferred method of treatment, as long as the patient can tolerate the intensity of pressure/stretch.</td>
</tr>
<tr>
<td>The gliding pressure used in levels 3 and 4 is not a deep stripping massage action; rather it is a more superficial tensioning of tissues that concentrates the effect of the stretching action into a very specific area of the muscle.</td>
</tr>
<tr>
<td>This method is not ischemic compression combined with active or passive stretching. Ischemic compression involves deep, steady, and constant pressure. With levels 1 and 2 of myofascial release techniques, deep gliding pressure is used and in levels 3 and 4 superficial, tensioning pressure is used to take the slack out of the tissues while the therapeutic stretching action does the work.</td>
</tr>
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</table>

**Clinical caveats**: The contraindications and cautions to the application of this method are similar to those for both ischemic compression and postisometric relaxation, since both stretching and deep manual pressure are components of myofascial release techniques.

Ischemic compression or trigger point pressure release is probably the quickest and easiest method to perform in the average chiropractic clinic. While the clinician is palpating the patient’s musculature for taut bands, spasms, and TrP activity, this method can be applied almost immediately. Once a TrP “knot” or “node” is located by palpation, deep manual pressure can be applied right then and there. Rarely does the doctor or patient have to change position to apply deep manual pressure. However, it is critical that the doctor respect the patient’s pain threshold, because excessive pressure can cause the patient to tense up and contract the muscle even more, which defeats the purpose of the therapy.
Contraindications to ischemic compression include: application over areas of recent trauma, hematoma, or bruising; application over the calf with suspected thrombosis or phlebitis; application directly over neurovascular structures; application over the skin of patients on anticoagulants, long term steroids, and other medications/nutritional deficiencies that predispose to soft tissue bruising or bleeding.

AMRT procedures are ideal for the patient who has a low pain threshold or severe muscle tightness. The patient has some control over how strongly the muscles are isometrically contracted, and can give feedback to the clinician about the degree of stretching. There is no problem applying this method with patients who are frail or whose skin would be sensitive to bruising from deep pressure techniques. The anxious patient also learns how to breathe and feel their own muscles relax, providing a form of low-tech biofeedback training.

Remember Newton’s third law, every action has an equal and opposite reaction. With respect to clinical situations, this means that whenever one structure is being stretched the joints on the opposite side are being compressed. For this reason, one must be careful not to stretch and release a hypertonic muscle and cause a reactive compressive injury to the facet or discs on the opposite side. Also, it may be contraindicated to perform vigorous stretching techniques across an unstable joint. Sometimes stretching an inflamed peripheral nerve or nerve root will worsen, rather than alleviate the patient’s symptoms. It is common knowledge not to stretch the hamstrings of a patient with sciatic radiculopathy, and therefore the same caveat applies to stretching the upper trapezius, scalenes, and so on, in the patient with brachial radiculopathy. It should be obvious that stretching techniques are contraindicated in muscles that have recently been strained or torn.

The MRT method is an excellent combination of active and passive participation by the patient. In levels 3 and 4 of this method, the patient becomes involved with movement of body parts and experiences the sensation of their muscles undergoing contraction, relaxation, and stretch. This allows for immediate patient education on the proper positioning and degree of stretch required to release the hypertonic muscles and/or adhesions. These patients are then ready to learn home stretching techniques rather quickly. An advantage to these methods over standard ischemic compression is the combination of active/passive stretching along with manual pressure, which allows for a concentrated area of stretch and pressure directly over the specific site of the TrP or adhesion. As a general rule, the contraindications for application of MRT are similar to those for ischemic compression.

The clinician who is proficient in all three of these methods, as well as other soft tissue techniques, will be better equipped to help his/her patients with myofascial peripheral nerve entrapments. Many times it will be necessary to try one of these methods, and then switch to an alternate technique if the patient is not responding as well as expected. There is no reason not to combine methods during one office visit, for example, applying MRT directly over a specific TrP location, then following up with a more regional AMRT technique such as a postisometric contraction stretch. Regardless which manual myofascial technique(s) the doctor chooses to use in the clinic, patients are best served by being educated about ways to prevent overuse syndromes and how to perform active home stretching techniques.

OSSEOUS MANIPULATION

Case reports and textbooks suggest that manipulation, often in conjunction with soft tissue treatments discussed above are an effective treatment regime for some peripheral neuropathies. Although this seems to be part of what may be termed the oral history of both the chiropractic and osteopathic professions, the scientific documentation of effectiveness of manipulative management of these conditions is sorely lacking. We have been able to find only one randomized clinical trial of manipulation for the management of any peripheral neuropathy in the indexed literature (both MedLine and MANTIS). However, conservative management is appropriate as long as regular assessment of the patient’s condition does not show a significant decline in neural status.

This clinical trial concerned the chiropractic management of carpal tunnel syndrome. Davis et al found no difference in effectiveness of chiropractic management compared with conservative medical care. However, both were deemed effective and there were fewer real or potential side effects from chiropractic care. Chiropractic care in this study included manipulation of the wrist, elbow, shoulder, cervical and thoracic spine as considered appropriate by the treating doctor. In addition, the subjects were treated with amyofascial massage and loading procedures, ultrasound treatment, and nocturnal wrist supports. Most authors recommend manipulation of the cervical and thoracic spine, as well as the upper extremity and more particularly the wrist in the treatment of carpal tunnel syndrome. Bonebrake et al used manipulation of all areas of the spine and both upper and lower extremities to treat carpal tunnel syndrome.

Recommended regions for manipulation in the treatment of thoracic outlet syndrome include cervical and thoracic spine, sternoclavicular, acromioclavicular, and rib articulations. Knutson presents a case of scalenus anticus syndrome, a form of thoracic outlet syndrome, which was treated with manipulation of the atlas only. For the treatment of meralgia paresthetica manipulation of the lumbar spine or restricted motion segments is suggested. Manipulation of the talus and calcaneus is advised in the treatment of tarsal tunnel syndrome.
CONCLUSION

When a patient presents complaining of symptoms that appear to be neurogenic, the first duty is to determine whether the symptoms are from neuropraxia or from neurotmesis or axonotmesis. Symptoms from neuropraxia are more likely to be amenable to conservative chiropractic management as neuropraxia is the typical degree of nerve injury in peripheral neuropathy presenting to chiropractic practices. Thus the symptoms are initially caused by conduction block (loss of light touch and vibration sense) or if painful the pain is nociceptive not neuropathic. Conservative management requires constant monitoring of outcomes so that effective treatment is continued and ineffective treatment is discontinued and referral is made, if appropriate, to prevent a progression of a neuropraxia to neurotmesis or axonotmesis. An understanding of the pathophysiology of peripheral nerve compression provides a more rational basis for the evaluation of the treatment outcomes in patients with these conditions.

The mechanisms that cause these nerve compression syndromes suggest that some of the soft tissue treatments that are used by the chiropractic profession should be effective. However, at this time no clinical trials have shown that these methods are effective treatments. Likewise, clinical trials are lacking that show that manipulation is an effective treatment for compressive peripheral neuropathies. A documented mechanism of peripheral neuropathy that accounts for the apparent effectiveness of manipulation in the treatment of some peripheral neuropathies is also lacking. Anecdotal evidence suggests that the use of both soft tissue and manipulative treatment may be an effective treatment for peripheral neuropathies. Conservative management, although lacking compelling evidence for effectiveness, should nevertheless be instituted as long as objective outcome measures are used. The use of such outcomes will then alert the clinician of a degeneration in the patient’s condition, suggesting the need for a more aggressive treatment. It should be obvious that the effectiveness of chiropractic management (soft tissue and manipulation) is an area ripe for clinical research.

REFERENCES


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