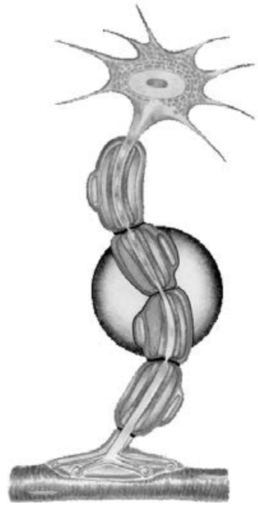


CHAPTER TWO

Peripheral Nerve Entrapment

Introduction

Peripheral nerve entrapment — sometimes called pressure neuropathy **(Akuthota & Herring, 2009; Pham & Gupta, 2009; Guyton & Hall, 2005)** — relates to some form of pressure or irritation on a peripheral nerve that changes its ability to function normally. It is generally considered to cause injury and an inflammatory response in the nerve. Peripheral nerve entrapment ranges from causing severe pain and muscle atrophy to mild, intermittent paresthesia and/or muscle weakness. As observed in applied kinesiology, the muscle dysfunction may not even be noticed by the patient, but it may be adversely influencing body posture, locomotion, or in some other way contributing to dysfunction.

Peripheral nerve entrapment was introduced into applied kinesiology in Goodheart's discussions of the carpal tunnel **(Goodheart, 1967)** and tarsal tunnel syndromes. **(Goodheart, 1971)** Walther's early review of peripheral nerve entrapment in 1982 broadened the subject in applied kinesiology, **(Walther, 1982)** and has served as the initial outline of this chapter. Cranial nerve entrapment syndromes, treated with applied kinesiology methods, have also been discussed in the recent peer-reviewed literature. **(Cuthbert & Rosner, 2010; Cuthbert & Barras, 2009; Blum & Cuthbert, 2006; Cuthbert & Blum, 2005)** Since muscular weakness found in routine applied kinesiology examination may be due to peripheral nerve entrapment, it is particularly important that the physician be aware of and able to differentially diagnose different types of the condition. Failure to return muscle weakness to normal function may result from undetected peripheral nerve entrapment. Because applied kinesiologists routinely test muscles to evaluate function, it is not uncommon to come across the more subtle types of peripheral nerve entrapment. Subtle entrapment may cause major symptoms to the patient that interfere with normal function and create remote problems.

Applied kinesiology is particularly effective in finding remote factors that contribute to peripheral nerve entrapment. The diagnosis of a piriformis syndrome indicates an entrapment of the sciatic nerve as it passes under or through the piriformis muscle. Direct treatment may be needed to the piriformis muscle, but a more in–depth study that takes the total body into consideration may find remote skeletal factors causing the piriformis tension. Another factor that might cause piriformis imbalance is disturbance in the organ and/or gland associated in applied kinesiology with the muscle. Treatment may need to be directed toward an overactive meridian, or any other of the five factors of the IVF.

It is important to recognize that like Breig with the central nervous system, many researchers have put forward evidence that for the peripheral nervous system, the clinical consequences of nerve injuries are greatly underestimated. Many experts now argue that peripheral nerve problems are far more common than clinicians believe. **(Mense & Simons, 2001; Butler, 2000; Staal et al., 1999; Devor & Seltzer, 1999; Loeser, 1985; Sunderland, 1978)** It is remarkable that only 50 years have passed since Phalen's description of carpal tunnel syndrome made this the easily recognizable clinical entity that it is today. **(Phalen, 1972)**

Cyriax was able to develop without a laboratory analysis, but by paying careful attention to his patients, the notion of "dural pain". Cyriax stated in 1948 that back pain comes from pressure and irritation to the dura mater. **(Cyriax, 1948)** He also observed that dura mater does not obey the rules of segmental reference, i.e. does not follow the familiar myotomal nor dermatomal pattern. In retrospect, and judging by the number of recent citations in the literature, Cyriax and Breig were far ahead of their time with their concepts on the biomechanics of the central nervous system and with their insistence that we are only beginning to realize the neurophysiological consequences of adverse tension in the central nervous system – and the peripheral nervous system is similarly prone to substantial adverse tensions that impair human function. Sunderland's work on the internal structure of the peripheral nerve and the role of ischemia in entrapment neuropathies also stands out. His classic text, *Nerves and Nerve Injuries* is as relevant for manual physicians as it is for surgeons. **(Sunderland, 1978)**

Much of this and the following chapter is directed at the peripheral nervous system (PNS), probably mirroring the available research work and our present understanding of the central nervous system (CNS). More is known about the PNS. It is more accessible, has far better regenerative powers and is more amenable to movement than the more protected CNS. However, despite intensive research attention to the PNS, due respect must be paid to the CNS as a contributing factor to symptoms, signs and treatment responses for PNS disorders. It is a sobering thought that, for every axon in the PNS, there are one-thousand in the CNS.

Butler has elegantly extended the early work of Breig to encompass the peripheral nervous system. **(Butler, 2000, 1991)** Butler and others have exhaustively documented how soft tissues which surround neural structures (muscle, tendon, disc, ligament, fascia, skin, or direct osseous pressure like an arthritic spur, for example), and which move independently of the nervous system, have a critical influence on neural function. These tissues have been labeled the "mechanical interface" of the nervous system and have been evaluated specifically in applied kinesiology from the very beginning. The concept, examination and treatment of "dural tension" have been presented by Goodheart and Walther for several decades. **(Walther, 2000; Goodheart, 1983)** Butler and Maitland have shown how any pathophysiology in the mechanical interface may produce tension on neural structures, with unpredictable results (motor, sensory, and autonomic). **(Maitland, 2001)**

Travell and Simons **(1999)** note that a degree of nerve compression that causes identifiable neuropathic and electromyographic changes may be associated with an increase in the number of active MTrPs. Travell and Simons **(1992)** have described how peripheral nerve entrapments can occur due to myofascial trigger points (MTrPs) in the following muscles:

Abductor hallucis
Adductor magnus
Extensor digitorum longus
Gastrocnemius
Gluteus maximus
Iliopsoas
Obturator externus and internus
Paraspinal muscles
Peroneus longus
Piriformis
Plantaris
Quadratus plantae
Soleus

Although the cause of peripheral nerve entrapment can usually be determined in an applied kinesiology examination, the precise diagnosis may never be exactly made, even though the entrapment is effectively treated. Visual observation of the entrapment at surgery is sometimes necessary for correct diagnosis. This is particularly true in conditions such as the thoracic outlet syndrome, in which various levels and causes of entrapment may be present.

Entrapment neuropathy may be subclinical, i.e., not causing symptoms of which the patient is aware. **(Akuthota & Herring, 2009; Russell, 2006)** Neary et al. **(Neary 1975)** studied the median and ulnar nerves obtained by routine autopsies from patients without known disease of the peripheral nervous system. There was enlargement in the ulnar groove at the elbow compared with the nerve proximal and distal to the site. At the carpal tunnel there was no visible swelling, but the median nerve was indented at the flexor retinaculum. Localized change of the nerve was found under the flexor retinaculum in some of the cases. Although frank clinical entrapment was not listed in the case histories of the subjects who died from unrelated factors, one wonders what an applied kinesiology examination prior to death would have revealed. Although manual muscle testing results are often confirmed by nerve conduction studies, it appears that some entrapment not documented by nerve conduction studies is observable by manual muscle testing. This is supported by the change in function after manipulative efforts to bony, ligamentous, or muscle structures, or in some other way treating the patient to release entrapment. Clinically the idea of subclinical entrapment arises when a patient complains of an old symptom 'reoccurring' by a new injury.

Today it is rare that an advanced applied kinesiologist cannot find the reason a muscle tests weak. Proper function is usually easily restored. Occasionally all efforts to locate the cause of dysfunction fail until one observes peripheral nerve entrapment affecting the muscle. This, too, usually responds, but sometimes conservative treatment to the area of entrapment fails to bring back normal muscle function. Failure may indicate a nerve entrapment needing surgical release.

The cause of muscle weakness can sometimes be enigmatic. Spinner and Spencer **(Spinner & Spencer, 1974)** state, "It is not satisfactory to consider muscle paralysis with or without sensory loss as idiopathic, spontaneous or viral, when it can be explained on the basis of a nerve entrapment." Staal et al. **(Staal et al., 1999)** expand on this and say "In the last decades some (orthopaedic) surgeons have developed a deplorable tendency to advocate operation for pain in the absence of any neurological abnormality whatsoever, on the nebulous assumption that the symptoms should be attributed to compression of a nerve that happens to be in the vicinity of the painful area. As a general rule these are 'pseudo-entrapment syndromes', the actual cause being overused muscles, bands, ligaments, joints, or a somatisation syndrome on the basis of a depressive illness or personality disorder. Of course several of these factors may coexist and interact." Applied kinesiology, with its many methods for evaluating muscle, joint, and other soft-tissue dysfunctions, can usually find the cause even prior to the development of many of the classic neurologic signs and symptoms. Using this method, conditions can be corrected before they advance to the point of paralysis, atrophy, or major sensory loss.

Cranial nerve entrapments have also been found in applied kinesiology to effect the function of cranial nerves. The cranial nerves carry dural sleeves with them for some distance; therefore any abnormal meningeal tension may be transmitted to a nerve and affect its function. Tension anywhere along the contiguous meninges can therefore be transmitted to the cranial nerves. This is because the peripheral and the central nervous systems are a continuous tissue tract, a fundamental insight of the work of Breig, Sutherland, Goodheart, DeJarnette, Chaitow and others. **(Chaitow, 2005; Goodheart, 1998-1964; Sutherland, 1998; DeJarnette, 1979; Breig, 1978)**

The neuropathies that may result from cranial bone dysfunction can be motor and/or sensory, and their severity depends on the amount of compression and neural irritation as well as the amount of ischemic radiculopathy. Breig has shown that problems come about primarily because of the entrapment neuropathy's effects on the vasculature of the nerve root. **(Breig et al., 1966)** "Pain is the cry of the nerve deprived of its blood supply", Sir Henry Head wrote. The effects of ischemia on cranial and peripheral nerve tissue have been well studied, and increasing interest in the pathophysiology of nerve compression has indicated that any rise in intrafascicular pressure – as a result of edema, compression, or torsion of the nerve root, for example – can also be damaging to neural tissue and function. **(Pham & Gupta, 2009; Patten, 1995; Moller, 1991; Dyck & Thomas, 1993; Lundborg, 1988)**

Throughout the cranium there are a number of sites where cranial nerves may be impinged upon by soft tissue at bony ridges or foraminal openings. **(Walther, 1983)** These sites may reflect mechanical or physiological changes in neural function, leading to a mechanical subset of cranial neuropathies that have been or can be successfully treated clinically by cranial practitioners. **(Blum & Cuthbert, 2006)**

Impingement of a nerve usually occurs where the nerve traverses a confining space such as the osteofibrous carpal tunnel, through a muscle such as the supinator, or between a muscle and a stable structure such as bone, as in the pectoralis minor, ulnar nerve, and piriformis syndromes. There are also a number of other, more individualized, types of impingement. **(Russell, 2006; Staal et al., 1999)**

Case Report: AK management of Parsonage-Turner Syndrome

A 30-year-old male with Parsonage-Turner Syndrome – acute brachial neuritis – presented for AK treatment involving right arm contracture, atrophy of the right arm and thenar eminence, and weakness producing a general paralysis of the forearm and index finger. **(Charles, 2011).** Electromyographic investigation led his orthopedist to recommend surgical release of the nerve entrapment in the pronator teres muscle, with no improvement; in fact the arm went from being immobilized at 45% flexion, and able to make only small rocking movements, to being paralyzed at 90% elbow flexion, unable to move his arm in any direction. The patient responded favorably in 8 treatment sessions guided by AK examination for the spinal manipulation, soft tissue treatment to myofascial trigger points, exercises and prescribed stretches, whereas previous surgical and pharmacological interventions had failed. Improved range of motion was experienced after the first treatment session, and by the eighth treatment, the patient was able to fully straighten the arm. Three years later, the patient was able to mountain climb with his arm fully functional and pain-free.

Common entrapment syndromes are:

- Median and radial nerves at the elbow, forearm, and wrist **(Saratsiotis & Myriokefalitakis, 2010; Leaf, 2010; Hogg, 1995; Walther, 1982)**
- Ulnar nerve at the elbow and wrist **(Robertson & Saratsiotis, 2005; Alis, 2004)**
- Interdigital nerves in the hands **(Heidrich, 1995)**

- Suprascapular nerve at the coracoid process **(Kharrazian, 2001)**
- Iliohypogastric nerve in the inguinal canal **(Leaf, 2010; Goodheart, 1998)**
- Lateral femoral cutaneous nerve in the pelvis **(Leaf, 2010; Skaggs et al., 2006)**
- Saphenous nerve at Hunter's canal in the leg **(Leaf, 2010; Cuthbert, 2003; Goodheart, 1998)**
- Peroneal nerves in the soleus muscle and ankle **(Walther, 2000; Travell & Simons, 1992; Heidrich, 1993)**
- Tibial nerve in the tarsal tunnel **(Walther, 2000)**
- Intertarsal nerves in the foot **(Travell & Simons, 1992)**
- Intercostals nerves at the ribs and from the intercostals muscles **(Bronston & Larson, 1990)**
- Pudendal nerve in Alcock's canal of the pelvis **(Cuthbert & Rosner, 2012; Browning, 1995)**
- Double or Triple Crush syndromes **(covered later)**

Entrapment may begin with specific trauma, but often it is very subtle; the patient may not be aware of when the problem actually began. Sometimes an acute episode will be blamed on a hard day's work, with the expectation that rest will "cure" the problem. The symptoms go away but the basic fault continues to be present, with exacerbation of the condition occurring with the next phase of heavy activity.

The actual extent by which surgery is avoided by correcting these conditions in their early developing state is unknown. However, much of the suffering from chronic pain is preventable if the acute pain is controlled promptly and effectively. Clinical examples of the importance of this principle are increasing rapidly. Specifically with regard to myofascial trigger points, Hong and Simons demonstrated that the length of treatment required for patients who had developed a pectoralis myofascial trigger point syndrome as a result of whiplash injury was directly related to the length of time between the accident and the beginning of trigger point treatment. **(Hong & Simons, 1993)** With longer initial delay, more treatments were required and the likelihood of complete symptom relief was decreased. Certainly, on a clinical basis, many applied kinesiologists obtain symptomatic relief in peripheral nerve entrapment conditions, as discussed in the next chapter and the subsequent volumes of this series. Those patients who obtain relief rarely have exacerbations that end in need of surgical release. Unfortunately, there is no method for determining what future problems are prevented by obtaining correction in the earlier states of the condition.

Entrapment neuropathies can have an exogenous or an endogenous etiology. An example of an exogenous etiology is trauma to the wrist, which causes subluxations of the carpal bones and perhaps separation of the radius and ulna. This stretches the flexor retinaculum, narrowing the carpal tunnel and producing entrapment of the median nerve. Muscle strain from overuse can also cause entrapment. Repeated forceful pronation of the forearm may cause hypertrophy and hypertonicity of the pronator teres muscle, in turn causing irritation on the median nerve as it travels through the pronator teres muscle. **(Discussed in subsequent volume)**

An endogenous involvement may be a systemic health problem that makes a person more susceptible to nerve entrapment. One should specifically look for peripheral nerve entrapment when patients have a condition with widespread effect on the small vessels, such as diabetes mellitus, periarteritis nodosa, certain types of amyloidosis, and severe arteriosclerosis with peripheral vascular insufficiency. **(Akuthota & Herring, 2009; Kopell 1980; Asbury, 1970)** Diseases like diabetes can raise the intrafascicular pressure throughout the body. **(Veves et al., 2002; Myers & Powell, 1981)**

In addition to disease processes that affect the small blood vessels, control of the blood vascular beds by the autonomic nervous system must be considered. There may be an imbalance of the autonomic nervous system affecting the peripheral circulation, causing the person to be more susceptible to peripheral nerve entrapment. Lombardini et al, **(Lombardini et al., 2009)** at the University of Perugia, conducted a study that assessed the effects of OMT (osteopathic manipulative treatment) for patients with peripheral artery disease (PAD) - and with symptoms of intermittent claudication.

> "Peripheral arterial disease (PAD) is a manifestation of systemic atherosclerosis associated with impaired endothelial function and intermittent claudication is the hallmark symptom. Hypothesizing that osteopathic manipulative treatment (OMT) may represent a non-pharmacological therapeutic option in PAD, we examined endothelial function and lifestyle modifications in 15 intermittent claudication patients receiving osteopathic treatment (OMT group) and 15 intermittent claudication patients matched for age, sex and medical treatment (control group). *Compared to the control group, the OMT group had a significant increase in brachial flow-mediated vasodilation, ankle/brachial pressure index, treadmill testing and physical health component of life quality*."

This study suggests that practitioners of manual approaches may be able to offer safe, effective, integrated health care even for patients with peripheral arterial disease, over and above the attention they offer for musculoskeletal pain and dysfunction. Johnson et al. **(Johnson et al., 1984)** discuss the influence of nervous system dysfunctions upon the vascular and cardiovascular systems specifically. Watanabe has demonstrated the clinical and neurophysiological responses of the cardiovascular system to chiropractic treatment. **(Watanabe & Polus, 2007)**

Other clinical examples would be in cases of resolution of the vascular component of cervicogenic headaches, **(Cramer & Darby, 1995)** or the positive influence upon a dysfunctional lumbar spinal segment associated with premenstrual syndromes or dysmenorrhea. – conditions that are functional in nature (pathophysiological) rather than pathological. **(Walsh & Polus, 1999; Browning, 1995)**

The "double crush" condition is discussed later, in which an individual has two problems, such as a circulatory deficiency and entrapment of the nerve. Either condition may be minimal and incapable of producing symptoms, but when found together, symptoms occur. On the other hand, circulatory problems may be secondary to entrapment. Certain types of entrapment may produce Raynaud's phenomenon. This secondary circulatory deficiency is not contributing to the peripheral nerve entrapment; rather, it is caused by it.

Premenstrual and pregnancy fluid retention are systemic problems that can contribute to entrapment. **(Wetz et al., 2006)** The excess fluid appears to cause additional pressure in confined areas through which the nerve must pass. Studies have documented carpal tunnel syndrome **(Smith et al., 2008)** and tarsal tunnel syndrome due to pregnancy by electrodiagnostic methods. The entrapment was sufficient to cause some complaint of leg pain in 69.5% of the women. **(Helm et al., 1971)** Of those studied following pregnancy, 92.4% reverted to normal values at postpartum follow–up. Peripheral nerve entrapment is often associated with an ileocecal valve syndrome in applied kinesiology. This condition causes toxicity to which the body has a natural reaction of retaining fluid to dilute the toxicity. **(Walther, 2000; Goodheart, 1998-1964)**

Applied kinesiology methods for evaluating peripheral nerve entrapment have revealed the importance of studying the entire postural balance, muscle organization, and movement patterns. The basic underlying cause of an entrapment may be remote from the actual area of involvement, e.g., a pelvic fault can cause a compensating shift in the shoulder girdle that results in a thoracic outlet entrapment. Using electromyography Mooney et al. **(Mooney et al., 2001)** found that in cases of sacroiliac joint dysfunction, hypertonicity of the gluteus maximus was found ipsilaterally and in the latissimus dorsi contralaterally. The integration between the shoulder girdle and sacroiliac joint was obvious. Giggey & Tepe **(Giggey & Tepe, 2009)** report that treatment of sacroiliac dysfunction using the sacro-occipital technique's padded-wedges (as used in applied kinesiology as well) showed significant improvements in strength of the cervical extensor muscles pre- and post-treatment; once again indicating the functional connection between the pelvis and the neck and shoulder muscles.

Failure to correct peripheral nerve entrapment by conservative methods often results from diagnosing only the area of local entrapment and failing to observe remote factors causing the problem. In this case, treating the local area of entrapment is just treating the symptoms.

Some peripheral nerve entrapment may be the result of old fractures, surgical adhesions, or congenital anomalies. Moncayo & Moncayo has recently demonstrated the usefulness of the AK treatment approach for scar-tissue. **(Moncayo & Moncayo, 2009)** Kobesova **(Kobesova, 2007)** suggests that scars may develop adhesive properties that disturb tissue tension, alter proprioceptive input, and create functional changes similar to active myofascial trigger points. This may create faulty sensory input that can result in disturbed motor output leading to adaptive postural patterns, impaired muscular strength, disturbed neurovascular activity, and pain syndromes. The diagnosis of the muscular consequences of scar-tissue has been described in the reports of Moncayo, Goodheart, Garten and Gerz. **(Moncayo, 2007; Goodheart, 1998; Garten, 2004; Gerz, 2001)**

Repeated trauma to a nerve can produce a tumor–like mass composed of dense, fibrous tissue adjacent to the nerve. This may cause symptoms of nerve entrapment. If the nerve is superficial, the mass can be palpated and sometimes mistaken for a neuroma. An example of this is continued irritation to the thumb from the trauma of a bowling ball on the bowler's thumb. **(Swanson et al., 2009; Marmor, 1966)**

The term "occupational nerve lesions" refers to pressure neuropathies caused by repeated trauma, which is often microtrauma. The Panel on Musculoskeletal Disorders in the Workplace **(National Research Council, Institute of Medicine, 2001)** states that nerve lesions caused by a single accident should not be called occupational nerve lesions. These injuries may not be noticed by the worker until there is significant disability because of the insidious nature of the developing condition. The correlation between the work and the illness is easily overlooked. Efforts are being made to study occupational causes of peripheral nerve entrapment, **(Ian'shina et al., 2009; Punnett, Keyserling 1987, Punnett, 1985)** but considerable work remains to be done. An example of this research was a study where electromyography examinations were performed on 102 patients with occupational hand disorders. 87% of the patients showed signs of tunnel syndromes: 44% of those had one tunnel syndrome, 36% two syndromes, and 7% multiple syndromes. The study concluded that "peripheral nerve disorders in residual defects with occupational hands diseases are persistent due to compression within physiologic tunnels." **(Ian'shina et al., 2009)**

The term "peripheral nerve entrapment" includes the radix of the nerve at the intervertebral foramen. Other than for differential diagnosis, radiculitis is not considered here. It is very important to correlate the material presented in this section on peripheral nerve entrapment with that in previous applied kinesiology textbooks on the spine and pelvis. **(ICAK Bookstores, 2012)** The "pseudoradicular syndrome" is peripheral nerve entrapment in the lower extremity that appears to be a lumbar radicular syndrome, but the actual causes are muscular, ligamentous, psychosomatic, and/or joint dysfunction. **(Barral & Croibier, 2007; Staal et al., 1999; Simons et al., 1998)**

Saal et al. **(Saal, 1988)** studied thirty–six cases that had peripheral nerve entrapment of the lower extremity instead of lumbar radiculopathy, for which they were referred. Forty–four percent of the cases had a positive nerve root tension sign, such as straight leg raise or femoral stretch test. Twenty–four percent had spinal range of motion abnormalities. History of lower extremity injury helped direct attention to the possible peripheral nerve lesion. Most helpful was local nerve tenderness at the lesion site. Electrodiagnostic studies and peripheral nerve blocks were used to make a final decision regarding the peripheral entrapment.

A similar situation is often present in the upper extremity. **(Discussed in the subsequent volume of this series)** Imaging studies may show spondylosis in the cervical spine, and one quickly attributes the patient's arm or hand problem to radiculitis. Many asymptomatic individuals from mid–life on have spondylosis. The symptoms may be due to double crush, or a thorough investigation may reveal a localized peripheral nerve entrapment without the spondylosis contributing to the pain.

When an area of entrapment is found, the assessment should consider all other possible factors. The patient may have more than one condition. In a study of 140 patients diagnosed with carpal tunnel syndrome and surgically decompressed, it was found that some of the symptoms were relieved in 20% of the patients, but continued pain was present. Upon close examination it was determined that a second condition was present. DeQuervain's stenosing tendinitis, ulnar compression neuritis, acute tenosynovitis, and epicondylitis were diagnosed. **(Crymble, 1968)** This is a cautionary tale about not plunging into surgery too soon without knowing the complete picture of a patient's inter-related pathophysiologic process.

Another example illustrating a secondary area of involvement is dysfunction in the foot that causes an imbalance in the piriformis. This in turn may create a sciatic neuralgia and also cause a sacral subluxation because of the piriformis' role in sacral stabilization. **(Lee, 2004; Retzlaff et al., 1974; Goodheart, 1972)** One cannot assume that the problem is local until remote components have been evaluated. If the piriformis muscle is not being influenced remotely, it may have been traumatized by an exogenous source, **(Steiner et al., 1987; TePoorten, 1969; Edwards, 1962; Robinson, 1947)** creating muscle imbalance and making it the primary cause of the peripheral nerve entrapment. Another advantage of the applied kinesiology approach to muscle system evaluation is that the piriformis muscles can be assessed in the prone, supine, and side lying as well as the standing positions; this position challenges the piriformis muscles' function with the positive support mechanisms in the feet and assesses the muscle's functionality in the positions in which the patient actually lives. The applied kinesiology approach of manual muscle testing patients while they are moving, in positions of daily living, or in positions of physical stress greatly adds to the evaluation of muscle function.

The amount of nerve compression and its relationship to symptoms has a wide range. On the one hand, as noted previously, entrapment can be observed at necropsy when there was no history of symptoms during life. **(Pham & Gupta, 2009; Guyton & Hall, 2005; Staal et al., 1999; Patten, 1995; Neary, Ochoa, Gilliatt, 1975)** On the other hand, Lusskin **(Lusskin, 1982)** states, "…the insidious and mild nature of the compression phenomenon is out of proportion to the annoying and often disabling symptoms." In severe entrapment, such as caused by space–occupying lesions or bony or fibrous–band congenital anomalies, the compression may become severe, requiring surgical intervention. This type of involvement is out of the scope of this text, other than for purposes of differential diagnosis. The thorough diagnostic evaluation taught in applied kinesiology attempts to eliminate these conditions from conservative treatment. When in doubt, a conservative approach is justified if there is no evidence of conditions such as space–occupying lesions and severe vascular or nerve compression. In general, the condition should improve within a maximum of three to four months. It is possible that continued delay in the presence of nerve entrapment can cause additional nerve damage, **(Butler, 2000; Lundborg, 1975; Sunderland, 1945; Denny—Brown & Brenner, 1944)** with lessened results from surgical decompression. This is not a universal rule. In a study of 313 patients who received carpal tunnel surgery, the duration of symptoms had little bearing on the degree of recovery. When the symptoms had been present for less than one year, there was 84% recovery; when present more than five years, 79% recovered. **(Cseuz et al., 1966)** The consideration of surgical decompression must be on an individual basis when a therapeutic trial fails to produce results. **(Kline & Hudson, 2007)**

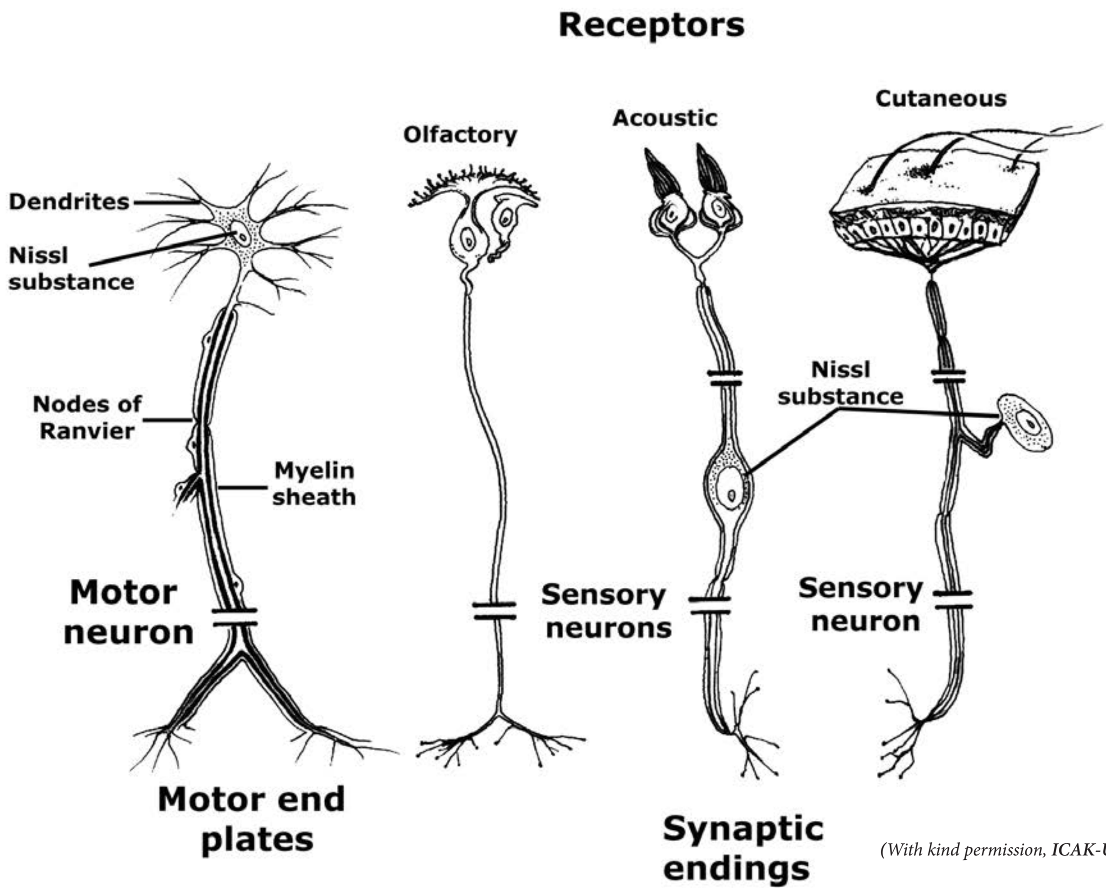
Peripheral nerve entrapment must be differentially diagnosed from a spinal involvement such as a subluxation, intervertebral disc, or foraminal encroachment from degenerative joint disease. This requires diagnosing the level of nerve involvement. In order to make a differential diagnosis, numerous areas where the nerve can be irritated must be evaluated. Various levels can cause a similar set of symptoms that must be objectively evaluated.

Disease of the nerve — such as a neuroma — must also be differentiated. It becomes obvious that all these differentiations are necessary, because failing to find the basic underlying cause of the problem and directing attention to the wrong pathological or functional process or to the wrong area of the nerve leaves the patient essentially untreated.

Entrapment Neurophysiology

Nerves are typically classified as motor, sensory, or mixed. A nerve may be classified as purely motor or sensory, which is erroneous since there are no "pure" nerves. Every muscle is supplied by both motor and sensory fibers. In this way the CNS is aware of the state of tension in muscle fibers and tendons. The automatic and coordinated interaction of agonist and antagonist muscles during movement requires innervation from the sensory nerve fibers. Because every nerve needs a certain portion of sensory fibers, there are no pure motor nerves. Motor nerves contain afferent supply from the neuromuscular spindle cell, and sensory nerves contain autonomic fibers. Within a peripheral nerve trunk there may be three main groups of fibers. (1) Motor (efferent) fibers supply the skeletal muscles for voluntary control of their activity. Their cell bodies are located in the gray matter of the spinal cord and brain stem. (2) Sensory (afferent) fibers are stimulated by various types of receptors, such as in skin, muscles, and special sensory organs. These impulses are interpreted in the central nervous system as sensations. (3) Autonomic fibers (efferent) control the smooth muscle of organs, glandular activities, and certain trophic functions of the body. **(Guyton & Hall, 2005)**

Peripheral nerves are made up of fibers from dorsal and ventral roots. On each side of each spinal segment these join together to form spinal nerves, which after only a short distance across the intervertebral foramina again divide into dorsal and ventral rami; the dorsal rami innervate the paraspinal muscles and skin, and the ventral rami of the cervical and lumbosacral spinal nerves combine to form the



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Types of Neuron

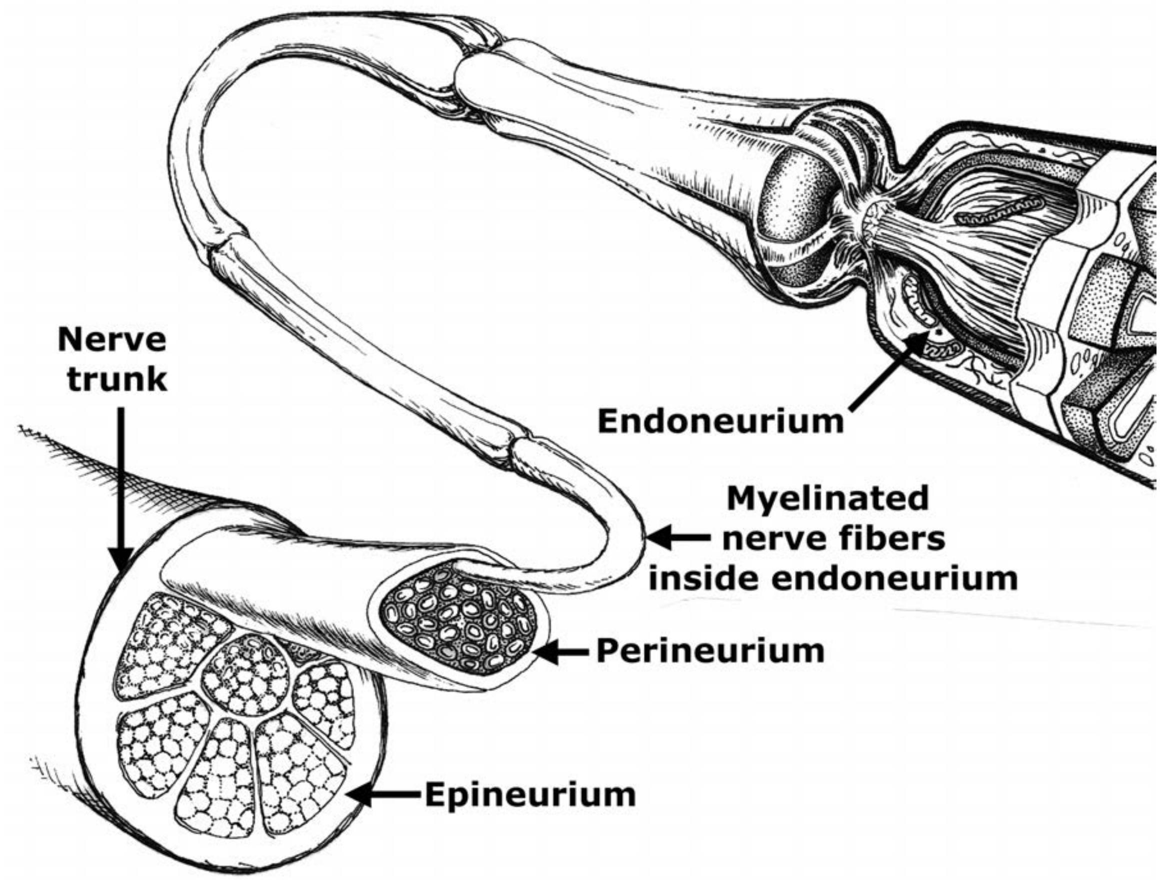
brachial and lumbosacral plexuses. The individual nerves to the limbs originate from these plexuses. The ventral rami of the thoracic origin form the intercostal nerves, except that from the first thoracic nerve, which joins the brachial plexus.

Many receptors and their axons have a lower tensile strength compared to the tissues in which they are embedded. Physical trauma to tissues and nerve trunks can damage the mechanoreceptors and their axons resulting in localized proprioceptive losses. **(Kolosova, et al., 2004)**

Reduced physical activity may result in a kind of sensory disuse, affecting the entire sensory apparatus from the receptors to their representation in the brain. In the peripheral nervous system, immobilization can lead to muscle spindle atrophy and changes in its sensitivity and firing rate. **(Halasi et al., 2005)** More centrally, it has been shown that tactile impoverishment and sensorimotor restriction of an animal's paw causes deterioration in the cortical sensory map representing that area. **(Coq & Xerri, 1999)**

Each nerve fiber has a cell body, a greatly elongated fiber called the axon, and — finally — the initiating and terminal aspects, which are the dendrites and teledendria, respectively. The dendrite or dendritic zone is the receptor membrane of a neuron. The cell body is usually located here, although it may lie within the axon (e.g., auditory neurons) or be attached to the side of the axon (e.g., bipolar neuron). The cell body containing a nucleus is the controlling metabolic center of the nerve cell. Severe injury of a nerve fiber causes degeneration of the distal segment no longer in contact with the cell body. The nerve fiber may be myelinated or unmyelinated. In myelinated nerves, the myelin sheath envelops the axon except at the endings and at periodic constrictions known as the nodes of Ranvier. The axon is maintained by materials formed in the cell body. Some of these materials are enzymes, polypeptides, polysaccharides, free amino acids, neurosecretory granules, mitochondria, and tubulin sub–units. **(Pham & Gupta, 2009; Butler, 2000; Hurst, Weissberg & Carroll, 1985)** The movement of these materials along the axon is called axoplasmic transport. Efficient transport of the material to the nerve fiber is essential for its normal structure and function, including normal growth, regeneration after injury, normal conduction, and normal transmission at the neuromuscular junctions and sensory receptors.

Nerve conduction is not explicitly an electrical current but instead continues along myelinated fibers in the form of consecutive depolarizations of one node of Ranvier to the next. If the nerve or axons are injured, transmission along the nerve is disturbed; if the chemical environment around the nerve is lacking sufficient electrolytes and other substances necessary for repolarization, nerve transmission and function will also be disturbed. Schmitt **(Schmitt, 1986)** has elegantly shown why this part of functional neurological assessment in applied kinesiology is so necessary to the complete diagnosis and treatment of nerve injury: the detection of the chemical milieu in the patient may be critical to the transmission of nerve impulses throughout the organism.



Nerves are subject to direct pressure, stretch, angulation, friction, torsion, ischemia, edema, and malnutrition. **(Maitland, 2001; Butler, 2000; Dawson, Hallett, Millender, 1983; Korr, 1976)** The strength and stability of peripheral nerves is incredible. It must be remembered that axons can be over 3 feet long, have differing sources of blood supply, bend and move constantly, rub on various tissues hard and soft and yet the axon is just one cell! Nerves can also be injured by cutting, too much squeezing and pulling, by irritating chemicals around the nerve, and by sustained reduction in blood supply. **(Pham & Gupta, 2009; Butler, 2000)** All around the body, nerves slide as the body moves. Injury or disease which alters posture or movement may lead to pain with movement. Real-time ultrasound imaging of fascial planes sliding and gliding upon each other during normal movement (flexion and extension) in asymptomatic individuals has shown how this smooth gliding motion is impaired or stuck in individuals with nerve pain. One such film was made by Dr Guimberteau, a plastic surgeon who works on hand reconstruction, taken with micro-cameras inserted during surgery, and was presented at the 7th World Low Back and Pelvic Pain Congress in Los Angeles. **(2010)**

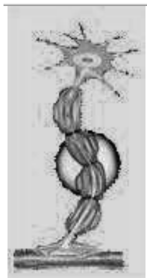
Films like these showing the free-moving nature of myofascial motion in living human tissue are revelatory of how the body actually works. These myofascial planes can get stuck (suffer myofascial gelosis) as a result of trauma, overuse, inflammation, and aging among others, and this corresponds with measurable densification, pain and limitation of movement. **(Myers, 2001; Mense & Simons, 2001)**

It appears that at least a part of the work that clinicians do achieves its results via restoring gliding potentials to these superficial layers of densely innervated (mechanoreceptors, nociceptors, etc.) myofascial tissues.

Any of these factors can cause peripheral nerve entrapment or cause the nerve to be more susceptible to entrapment. The peripheral nerve, unlike a blood vessel, does not easily stretch. It must be able to move freely in relation to its neighboring structures to avoid irritation. **(Butler, 2000; Kopell and Thompson, 1976)** Another potential mechanism that may lead to long-term sensory and motor losses is a physical change in injured tissues in which the receptor is embedded. The proprioceptive receptors and nerves may be fully intact, but their ability to detect movement may be disrupted by changes in the surrounding tissues, in the form of adhesions or shortening. **(Kolosova, et al., 2004; Brumagne et al., 2000)**

From its point of leaving the spinal cord to its destination, the nerve is vulnerable to entrapment or to factors that make it more susceptible to entrapment. Korr **(Korr, 1976)** summarizes these factors as "…compression by narrowing of the foramen; adhesions between roots and sleeves, causing angulation, shearing and constriction; shearing forces acting upon nerves passing through fascia; compression (for example, of posterior rami of spinal nerves) by sustained contraction of the paravertebral muscles through which the nerves pass; constriction at duroarachnoid junctions of root pouches; compression within foramina secondary to venus congestion (compression of spinal and radicular veins). Hypoxia, pH shifts, and other chemical changes in the environments of the nerves due to ischemia (compression of spinal arteries, sustained contraction of muscles through which nerves

Nerve Trauma

	**Normal --** Normal nerve transmission may be disturbed from high pressure exerted for a short time, from moderate or low pressure exerted for intermittent or long periods. The subtlety of pressure that can cause entrapment neuropathy is illustrated by fluid accumulation causing entrapment at the carpal or tarsal tunnel, in pregnancy, premenstrual syndrome, or the ileocecal valve syndrome. The irritation creating symptoms is often long-standing; it causes an inflammatory response in the nerve and maintains its improper function. Short-term, subtle irritation on a nerve does not appear capable of changing the characteristics of its function on a constant or permanent basis. It is necessary to diagnose the entrapment early so that conservative treatment will be effective. Chronicity increases the chance that surgical decompression may be necessary.
	**Neurapraxia** is the lesser of the types of nerve involvement. It is a segmental block of axonal conduction due to a focal region of demyelinization of the nerve. The nerve has continuity, but conduction cannot be carried out over the demyelinated area. With only slight myelin damage there may be conduction, but it is slowed, requiring greater time to activate the widened nodal region. This is probably the type of nerve involvement present in the more subtle types of entrapment often observed in routine applied kinesiology examination, when the patient is not aware of symptoms.
	**Axonotmesis** is the loss of continuity of nerve axons, but with continuity of the connective tissue sheath. This leads to wallerian degeneration of the distal part of the nerve. Following Wallerian degeneration, the proximal part of the nerve attempts to regrow. “This growth occurs at the rate of approximately 1 mm per day, 1 cm per week, or 1 inch per month.” Obvious symptoms and signs of peripheral nerve entrapment are present in this condition. When the entrapment is released, repair begins and the patient can expect to return to normal or nearly normal. The main factor is time for regrowth and maintenance of an entrapment-free condition.
	**Neurotmesis** is the separation of the axon and is the most advanced involvement. The separation of the nerve from its nutritive sources causes Wallerian degeneration, the fatty degeneration of the nerve fiber. Regeneration may still take place and will result in regaining continuity or in forming a neuroma illustrated below.
	**Neurotomesis** regaining continuity. Because of the loss of continuity of the nerve's supporting elements there is only a chance that the nerve will connect with the proximal portion. If it does the slow process of regeneration can take place. The amount of regeneration to normal is variable.
	**Neurotomesis** failing to regain continuity is illustrated. The continued nerve regeneration causes a neuroma to develop.

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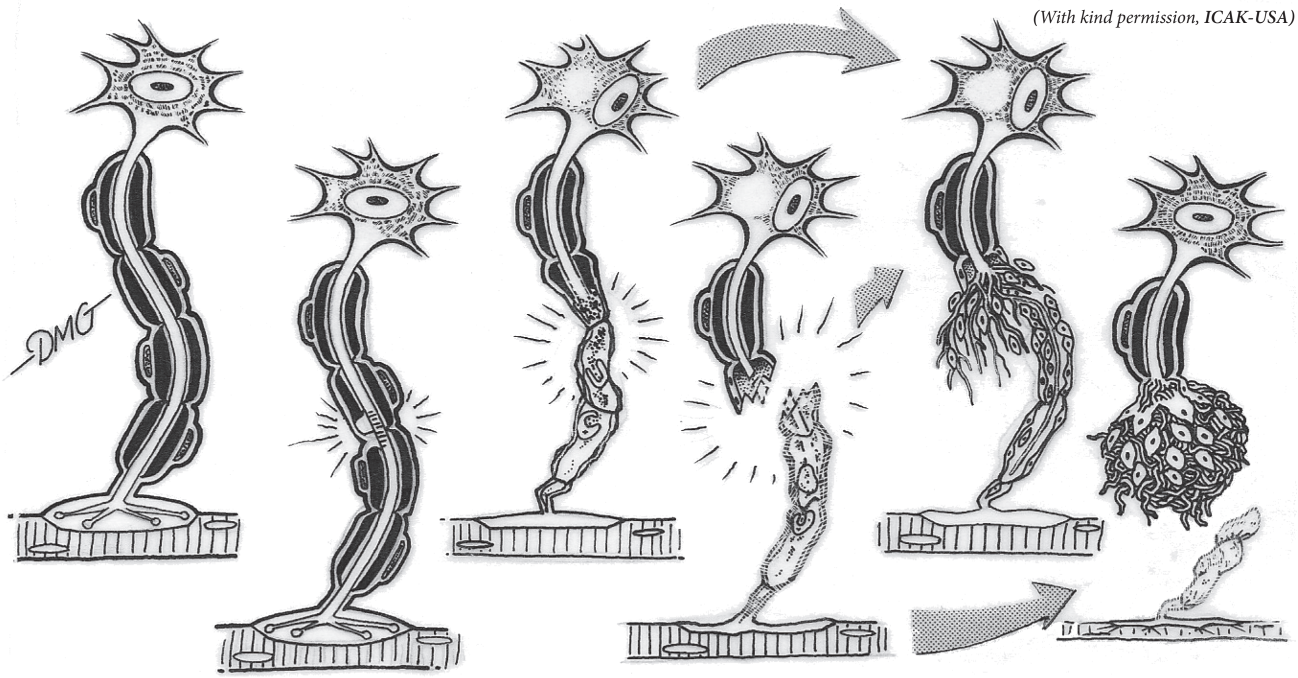
(With kind permission, **ICAK-USA**)



Sequence of peripheral nerve injury

pass, et cetera) are also important factors in the alteration of axonal excitation and conduction."

There may be damage from high pressure exerted for a short time, or from moderate or low pressure exerted for long periods or intermittently. The subtlety of pressure that can cause entrapment neuropathy is illustrated by fluid accumulation causing entrapment at the carpal **(Akuthota & Herring, 2009; Wetz et al., 2006; Spaans, 1970)** or tarsal **(Smith, 2008; Helm, Nepomuceno, & Crane, 1971)** tunnel in pregnancy, premenstrual syndrome, or the ileocecal valve syndrome. The irritation creating symptoms is often long–standing; it causes an inflammatory response in the nerve and maintains its improper function. Short–term, subtle irritation on a nerve does not appear capable of changing the characteristics of its function on a constant or permanent basis. It is necessary to diagnose the entrapment early so that conservative treatment will be effective. Chronicity increases the chance that surgical decompression may be necessary, and that more permanent long term damage may result.

The three types of nerve injury are neurapraxia, axonotmesis, and neurotmesis. Neurapraxia is the lesser involvement. It is a segmental block of axonal conduction due to a focal region of demyelinization of the nerve. The nerve has continuity, but conduction cannot be carried out over the demyelinated area. With only slight myelin damage there may be conduction, but it is slowed, requiring greater time to activate the widened nodal region. This is probably the type of nerve involvement present in the more subtle types of entrapment often observed in routine applied kinesiology examination, when the patient is not aware of symptoms. Treatment of this type of nerve conduction injury can speed up the recovery time and facilitate a more complete recovery.

Axonotmesis is the loss of continuity of nerve axons, but with continuity of the connective tissue sheath (the neurolemma or sheath of Schwann remains intact). This leads to Wallerian degeneration of the distal part of the nerve. Following Wallerian degeneration, the proximal part of the nerve attempts to regrow. This growth occurs at the rate of approximately 1 mm per day, 1 cm per week, or 1 inch per month. Obvious symptoms and signs of peripheral nerve entrapment are present in this condition. When the entrapment is released, repair begins and the patient can expect to return to normal or near–normal. The main factor is time for regrowth and maintenance of an entrapment–free condition. **(Chien et al., 2003; Staal et al., 1999; Dawson, 1983)**

The most advanced involvement is neurotmesis, in which the connective tissue and the axons are destroyed. Following the Wallerian degeneration of the axons, there is less chance for regrowth in the appropriate direction due to the loss of continuity of the nerve's supporting elements. This failure of direction may cause the nerve regrowth to ball up into a neuroma. This injury goes beyond the possibilities of applied kinesiology therapy; a surgical procedure is usually required for repair here.

In peripheral nerve entrapment that is applicable to applied kinesiology's conservative treatment, we are not concerned with the catastrophic situations in which whole nerves or roots are crushed, or even in which conduction has been blocked in all or most of the axons. This complete — or almost complete — interruption of the axoplasmic continuity would cause a near–total or total loss of neural function, with Wallerian degeneration distal to the disruption. In severe injuries the axoplasm will actually leak out of the damaged or torn nerve. Research has shown that even minor injury to a peripheral nerve as well as to the environment surrounding the nerve will have important consequences for the rate of axoplasmic flow and the quality of the axoplasm. **(Butler, 2000; Dahlin et al., 1986; Rydevik et al., 1980)**

In the more moderate situation in which conservative treatment is effective, there is a conduction block in some of the fibers in a nerve, causing a corresponding loss of sensory and motor function. This loss may be transient or fluctuating; in some cases, the sensory or motor deficits would not even be perceived by the patient. Referring to this type of entrapment neuropathy, Korr **(1976)** states, "However, since some types of fibers are more susceptible to deformation block than others, garbled sensory input and incomplete and uncoordinated efferent output may be the clinically more significant consequences." This correlates well with some of the unusual functional findings observed in applied kinesiology. Sometimes the dysfunction does not seem to follow the expected neurologic pathways. Release of an entrapment may increase range of motion in remote muscles as they improve their strength or as they relax. This may take place as muscles supplied by the pathway strengthen. Intermingling of impulses between neurons appears to be a common occurrence in peripheral nerve entrapment.

Cross Stimulation and Light Pressure

There is a great variation in impairment due to nerve compression. **(Barral & Croibier, 2007; Russell, 2006; Denny—Brown and Brenner, 1944)** Light forces can cause disturbance in peripheral nerves, even though the nerves can absorb large forces with their associated structures. Nerve roots, in comparison, are mechanically frail due to lack of perineurium. **(Barral & Croibier, 2007; Luttges, Stodieck, & Beel, 1986)** The epineurium proves to be a particularly reactive tissue that is not difficult to injure. Slight trauma such as mild compression or intermittent irritation to a peripheral nerve can cause non–degenerative, inflammatory changes and epineural edema that result in decreased nerve conduction velocity and progressive facilitation during early refractory periods. **(Butler, 2000, 1991; Rydevik et al., 1984; Triano & Luttges, 1980)** The nerve may be even more susceptible to entrapment at the radix.

In simple compression trauma to a nerve, the motor fibers are more vulnerable than the sensory ones. **(Russell, 2006; Haldeman & Meyer, 1970; Sunderland, 1945; Gasser & Erlanger, 1929)** If significant sensory defects appear, there will be delayed restoration of full motor function. In compression injuries, such as sleep paralysis of the radial nerve, an individual is more vulnerable if general health is reduced, or other factors — such as undue fatigue, drugs, or alcohol — are involved.

Pressure applied to a nerve can cause an artificial synapse between nerve fibers. This is referred to as "cross stimulation." **(Russell, 2006)** Gardner **(Gardner, 1967)** relates to Hering's experiment in which "Cross stimulation was produced by a pressure applied so gently that it did not impair conduction of the original impulse. The investigators then relieved the pressure (i.e., decompressed the nerve) and irrigated the nerve with a saline solution, which caused interruption of the artificial synapse…."

In a similar study by Granit et al. **(Granit, Leksell, & Skoglund, 1944)** pressure was applied to a nerve, causing an artificial synapse between motor and sensory fibers of the nerve. In addition to the cross stimulation between motor and sensory fibers, the original impulse was maintained. They then removed the pressure and irrigated the nerve with a saline solution; after a brief time the nerve returned to normal function.

In normal situations, nerve impulses begin with stimulation at the central or cellular ends of the nerve and travel in only one direction; that is, efferent impulses begin at the central nervous system and travel toward the periphery, and sensory impulses begin at the periphery and travel toward the central nervous system. In peripheral nerve entrapment, there may be impulses generated at the deformation site that travel in both directions. Impulses that do not begin at the end of the nerve fiber are additional ones superimposed on the usual nerve transmission. As normal impulses pass through the area of nerve deformation, there may be an amplification and prolongation of the impulse, changing the effect centrally or peripherally. Cross–talk between fibers may take place, sending an originally propagated impulse to an area different than is normally intended. "This lateral, side–to–side (ephaptic) transmission is usually from large fibers to small fibers." **(Korr, 1976)**

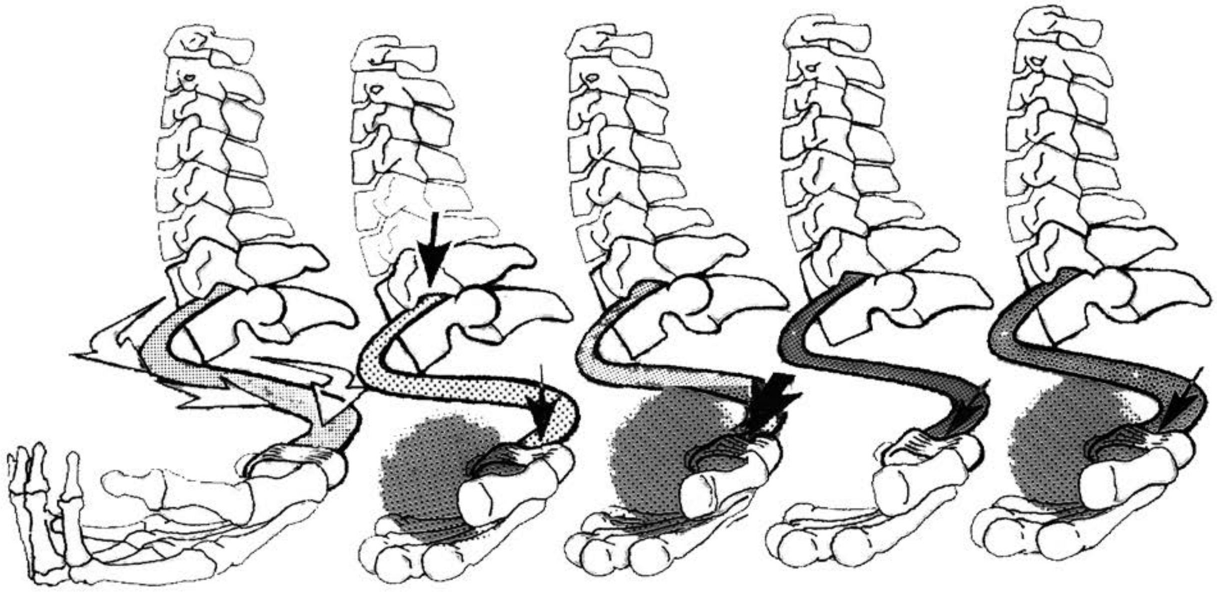
An example of cross stimulation resulting from nerve compression is meralgia paresthetica. **(Travell & Simons, 1992)** Stimulating a small area of skin may result in widespread burning pain in the distribution of the lateral femoral cutaneous nerve. This is referred to as "cross talk." "Back talk" is a result of cross stimulation that travels in the opposite direction, that is, efferent supply due to cross stimulation affecting an afferent fiber. **(Russell, 2006; Butler, 1991; Gardner, 1967)**

Double Crush

Some patients have multiple nerve compression syndromes in the same extremity, indicating that there may be a patient population that is at high risk for the development of compressive neuropathies. **(Butler, 2000, 1991; Staal et al., 1999; Ritts, Wood, & Linscheid, 1987)** This may be explained by "double crush," which consists of two entrapments or one entrapment with some other disturbance that interferes with normal nerve transmission, such as diabetes, malnutrition, or ischemia. Metabolic and endocrine disorders have been implicated in neuropathic syndromes, including hypo- and hyperthyroidism, acromegaly, and others. **(Staal et al., 1999)** The factors may be insignificant individually, but together there are significant symptoms at the distal area.

The recognition of double crush syndromes encourages clinicians to undertake a more global investigation of peripheral nerve injuries and pain syndromes, and requires the clinician to employ methods that can evaluate many tissues and their mutual influences.

In a series of 115 patients with an electrophysiologically–proven entrapment neuropathy, Upton and McComas **(Upton, & McComas, 1973)** found evidence of a cervical root lesion in addition to the one in the extremity. 70% of these patients with either carpal tunnel syndrome or lesions of the ulnar nerve at the elbow showed clear electrophysiological and clinical evidence of neural lesions



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Double crush

***A.** Normal nerve function at the nerve root and carpal tunnel syndrome. **B.** Light entrapment at both the nerve root level and carpal tunnel causes symptoms. **C.** Heavy entrapment at the carpal tunnel causes symptoms. **D.** Light entrapment at the carpal tunnel causes no symptoms. **E.** Vitamin B6 deficiency, ischemia, or some other factor that causes reduced nerve function and light entrapment at the carpal tunnel produces symptoms. Heavy entrapment at any one area can produce symptoms, whereas light entrapment at two locations causes symptoms, but the light entrapment at any one area would be symptom-free.*

in the neck. Upton and McComas developed the double crush hypothesis, indicating that the proximal lesion in the cervical spine reduced the axoplasmic transport. They proposed that the functional integrity of the axon and its target structures is impaired by the proximal disturbance, making the distal peripheral nerve more vulnerable to entrapment. Amadio **(Amadio, 1987)** comments on this work, "In the double crush there are two separate compressive foci on the same nerve, each of which individually might cause only minor symptoms, but which in combination cause a significant neuropathy at the distal focus. The most classic example of this is the double crush which occurs with a combined cervical spondylosis and carpal tunnel syndrome." In a retrospective study of 1,000 cases of carpal tunnel syndrome, Hurst et al. **(Hurst, Weissberg & Carroll, 1985)** found that the double crush effect was statistically significant in patients with carpal tunnel syndrome and either diabetes or cervical spondylosis. It is unknown whether the diabetic correlation is due to circulatory disturbance or reduced health of the nerve cell body in producing the necessary growth factors to be distributed to the nerve fiber by axoplasmic transport.

The reduction of axoplasmic transport is observed by the significant degree of narrowing of axons distal to the area of entrapment. Proximal to the entrapment, there is considerable swelling due to the damming of axoplasm. **(Korr, 1976)** Lundborg **(Lundborg, 1988)** proposed a reversed double crush syndrome where nerve injuries at the wrist could predispose proximal nerve tissues to injury. Dahlin **(Dahlin, 1987)** extended the double crush hypothesis by showing that compression as seen in carpal tunnel syndromes will block the retrograde axonal flow and induce changes in the cell bodies in the dorsal root ganglia. It must be recognized that multiple nerve compressions, including nerve roots, will significantly influence the CNS, causing facilitation and upregulation, and will produce antidromic CNS originated reflexes. Antidromic impulses travel from proximal to distal along a C fiber (i.e. the wrong way).

Since the introduction of the double crush concept it is no longer sensible to examine patients only at the site of pain or only the nerve that is capable of referring to the anatomical site of pain. The double crush concept also helps us understand patients who have a poor result after surgical release for neuropathy. **(Idler, 1996)**

Ischemia

Although this section is primarily on peripheral nerve entrapment, circulatory dysfunction plays a primary role. Everyone has had the common experience of having a leg go to sleep after crossing one's knees. Within a relatively short period, heaviness and a strong pins–and–needles sensation develop in the limb. The condition is rapidly reversible with movement, and there is no recognizable pathology. "Ischemia may play some role in chronic entrapment and may well be responsible for some of the frequently seen axonal loss and the connective–tissue changes." **(Staal et al., 1999; Dawson, Hallett, & Millender, 1983)** The symptoms of a chronic entrapment can be worsened by ischemia, which may be the factor that causes nocturnal symptoms in carpal tunnel syndrome **(Staal et al., 1999; Lundborg, 1975; Fullerton, 1963; Gilliatt & Wilson, 1954)** because during sleep the arterial blood pressure falls, pulse rate decreases, and the metabolic rate lowers. **(Guyton & Hall, 2005)**

This is seen in arterial embolism and accompanying small vessel embolism, as well as in other systemic or localized conditions that block the arterial flow. **(Veves et al., 2002; Lusskin, 1982)** A major consideration in the past has been evaluation of the thoracic outlet by provocative tests that decrease the radial pulse. As will be discussed later, these tests are not as important today in evaluating peripheral nerve entrapment; however, one should consider and do the appropriate tests for circulatory dysfunction in all cases appearing to be peripheral nerve entrapment. Symptoms of vascular occlusion should never be overlooked. **(Butler, 1991; Rob & Standeven, 1958)**

Ischemia can cause a true neuropathy independently as nerve fibers are dependent upon an uninterrupted supply of blood for normal function. **(Butler, 1991; Sunderland, 1978)** Large nerve fibers suffer earlier than smaller fibers from compression and ischemia. **(Ochoa, 1980)** The cells of the central nervous system are extremely sensitive to oxygen deprivation; however their branches in the form of peripheral nerves are less so. However if the ischemia lasts too long, the nerves are in danger of real damage. This can manifest as permanent motor or sensory dysfunction. **(Lundborg, 1988)** Anoxia also has a destructive impact on the endothelium of the endoneural blood vessels.

Commonly occurring with peripheral nerve entrapment is circulatory deficiency at the capillary level. If ischemia causes the nerve to be more susceptible to peripheral nerve entrapment, the capillary deficiency can be due to a local pressure effect that is not only on the nerve itself but also on its intrinsic blood supply. **(Barral & Croibier, 2007; Staal et al., 1999; Lusskin, 1982)** Peripheral neuropathy at a more proximal location can cause capillary constriction due to the post–ganglionic sympathetic fibers being stimulated as they pass intermingled with the myelinated nerve fibers. Adson **(Adson, 1951)** considers this the cause of vasospasm and hyperhidrosis that sometimes predominate over other neurologic symptoms in thoracic outlet syndrome.

Both mechanical compression and ischemia are responsible to varying degrees in peripheral nerve entrapment syndromes. It is critically important that the applied kinesiologist remember a fundamental rule of neurophysiology: alpha fibers are more susceptible to mechanical compression; beta fibers are relatively more resistant, but they are more susceptible to anoxia from ischemia. For this reason, when one develops paresthesia from crossing his knees, *motor function is not greatly impaired*; *however, when there is peripheral nerve compression, muscle function is impaired to a greater degree than the paresthesia that develops.*

For practical purposes, peripheral nerve entrapment is compression or other mechanical disturbance to the nerve combined with impairment of blood circulation. The peripheral nerve is normally highly vascularized, with an abundance of collateral sources of flow. Experimentally it is difficult to adequately shut off the flow to cause ischemic changes in a peripheral nerve by ligation of nutrient vessels. It takes very high pressure applied to a nerve alone to diminish conduction. When the pressure is applied to cause anoxia by diminishing local blood flow, much less pressure is needed. **(Akuthota & Herring, 2009; Butler, 1991; Asbury, 1970)** When there is prolonged ischemia, recovery from entrapment is slower. **(Staal et al., 1999; Kopell, 1980)** Ischemia without pressure can produce Wallerian degeneration and paranodal demyelination, the former being more extensive. **(Hess, 1979)**

Compression ischemia, in general, first causes mild dysesthetic sensation, followed by loss of touch, then pain sensation and finally — much later — loss of motor power. Asbury **(Asbury, 1970)** observes that this pattern of symptoms does not correlate with electrophysiologic findings, which indicate that heavily myelinated fibers providing motor supply stop conducting before the lightly myelinated and unmyelinated fibers. This indicates that motor loss would be first. *Clinical observation in applied kinesiology indicates that motor weakness, as observed by the manual muscle test, is almost always present with sensory or ischemic disturbance with entrapment of mixed nerves.* One must recognize that the manual muscle test does not evaluate muscle function in the same manner as the muscle producing power against a dynamometer; **(Nicholas et al., 1976)** however the concurrent validity of the manual muscle test has been found to be excellent in 11 controlled clinical trials when compared to muscle strength testing instruments. **(Cuthbert & Goodheart, 2007)**

The effect of ischemia in peripheral nerve entrapment is demonstrated by tests designed to block the arterial flow to the area of entrapment. These tests will be described later, but in general they cause increased symptoms more rapidly in those with entrapment neuropathy than in normal subjects. **(Butler, 1991; Fullerton, 1963; Gilliatt & Wilson, 1954)**

Further evidence that ischemia contributes to peripheral nerve entrapment is seen where measurements of the amount of pressure in a confined area in some cases are not significantly different from normal subjects, indicating that some other factor is contributing to it, such as ischemia. **(Zhao et al., 2011; Gelberman et al., 1981)** Ischemia, therefore, seems to be another factor in the double crush syndrome; the single entrapment may not be adequate to cause symptoms, but when the health of the nerve is impaired for some reason, an additional mechanical factor can cause neuropathy.

Although ischemia contributes to peripheral nerve entrapment, the mechanical factors are the critical ones in chronic entrapment lesions. **(Staal et al., 1999; Butler, 1991; Dawson, Hallett, Millender, 1983; Kopell, 1980)** In the paresthesia that develops from crossing one's knees or from the application of a suprasystolic cuff to a limb, the lack of nerve function is almost immediately reversible upon release of the cuff or uncrossing the legs; there is no recognizable microscopic or ultrascopic pathology. This failure of nerve conduction is strictly due to ischemia. **(Kopell, 1980)**

The sympathetic nervous system should be evaluated when examining for peripheral nerve entrapment. Vasoconstriction of blood vessels may be caused by irritation of the sympathetic trunk. The perineurium and epineurium are sympathetically innervated. **(Lundborg, 1988)** Selander et al **(Selander et al., 1985)** measured blood flow in the sciatic nerves of rabbits and found that by stimulation of the lumbar sympathetic trunk, intraneural blood flow could be reduced to 10% of control values. It was also found that an injection of noradrenalin into the aorta reduced blood flow by 40%.

It should be noted that muscle spindle intrafusal fibers receive innervation from branches of the sympathetic nervous system, an idea which has found experimental verification specifically regarding TMD. Sympathetic stimulation has been shown to reduce type Ia and II motor output. **(Passatore et al., 1985)** This research indicates that sympathetic action on muscle spindles – including the fight-or-flight reaction involving Selye's General Adaptation Syndrome and adrenal stress disorders – may be one of the mechanisms by which physical and emotional stress inhibit the muscular system. **(Wilson, 2002)** Patients with chronic pain syndromes often assume protective postures that may tense the sympathetic chain.

Interruption of the sympathetic supply changes sweat gland secretion, depending on the level and type of nerve involvement. If there is severance of the nerve root proximal to the sympathetic ganglia, an absence of sweating after central physiologic stimulation will result. An interruption distal to or within the ganglia will cause no sweat response to stimuli or to cholinergic substances. A peripheral irritation will cause no change or excessive sweating. **(Korting & Denk, 1976)** When the autonomic nervous system is irritated, a true causalgia may develop. In the early stage, the extremity is red, warm, dry, and swollen, later becoming cool, clammy, hot with pallor, and/or cyanotic. Raynaud's phenomenon may occur. **(Mondelli et al., 2009; Sternschein et al., 1975)**

Examination

History

Differential diagnosis of peripheral nerve entrapment is sometimes simple and clear–cut; on the other hand, it may be difficult, with many potential traps for the unwary physician. One must be constantly alert for the double crush syndrome. This may include two areas of entrapment, or one area of entrapment plus a general neuropathy from nutritional deficiency or drug side effects, or other factors. Finding only part of the problem leads to disappointing treatment results. It is an old maxim of neurology that the cause of a disorder is hidden in the patient's history and that the site of the dysfunction is detected by examination. Proper diagnosis begins with an in–depth patient history and report of current status.

The characteristic symptoms, signs, and dysfunctions of peripheral nerve entrapment provide the first clues that this may be the cause — or partial cause — of the patient's problem. The physician's clear mental picture of various areas in the body that are susceptible to peripheral nerve entrapment, and the distribution of the nerve involved, should indicate the possible areas of entrapment. Most peripheral nerve entrapment is located in more common places, such as at the carpal tunnel, tarsal tunnel, and piriformis. The less common entrapments often relate with sports or occupational activities. Often one can determine the site of the entrapment by asking about habits or posture that is relevant to the nerve or anatomical region in question. It is important to locate the area of entrapment and determine the cause. Often analysis of the activity will reveal changes that can help eliminate future problems. **(Leaf, 2010; Russell, 2006; Staal et al., 1999; Goodman, 1983)**

Etiology of peripheral nerve entrapment often relates to sports, occupational activity, and habit patterns; thus, history and consultation should delve deeply into the way the patient lives. Sports that can cause entrapment neuropathy are hiking with backpacks, gymnastics, baseball, volleyball, horseback riding, and skiing, among others. **(Toth, 2008; Hirasawa & Sakaida, 1983)**

Activities performed during one's occupation are common causes of peripheral nerve entrapment, often called occupational or craft palsies. **(National Research Council, 2001)** Throughout the discussion of various types of peripheral nerve entrapment, comments will be made about particular occupations or sports that may cause entrapment. The list of these contributing factors is exhaustive, **(National Research Council, 2001; Murphy, 1955)** and not all can be considered. The important factor is to examine the patient in the manner in which he lives and works. Consider localized factors, such as how an individual holds the tools with which he works, **(National Research Council, 2001; Charash, 1982]** the use of vibrating tools, and whether there is jerking and jostling of the body or constrained postural work positions.

When neuropathy develops rapidly, the patient usually notices the change and seeks help. Symptoms of peripheral nerve entrapment may be evident early, or become prominent many years after the initial injury. **(Staal et al., 1999; Kopell, 1980)** Often craft, occupational, and some sports activities cause conditions to develop insidiously, with slow loss of sensation and muscle function. This is often ignored by the patient, and treatment is sought for some functional deficit such as tripping, turning an ankle, or catching a toe on small rises in the substrate. Poor weight distribution and structural deformity in the foot may cause corns, calluses, alterations, or painful areas. **(Langer, 2007; Maffetone, 2003; Greenawalt, 1988; Lusskin, 1982)** The patient may first notice a problem in the upper extremity because of dropping items, poor dexterity of the hand, and inability to get the arm into certain positions. **(Goodheart, 1971)** Often no specific traumatic episode will be described during consultation because many peripheral entrapment syndromes are chronic, having developed symptoms insidiously.

The symptomatic picture presented will depend greatly on the type of nerve involved. The three potential types are cutaneous sensory, motor, and mixed. Although the first two classifications are often referred to as "pure," there are no truly pure peripheral nerves. The cutaneous sensory nerves carry afferent impulses and also supply efferent fibers to various structures in the skin. The motor nerves return afferent impulses from the proprioceptors of the muscle, joints, and associated connective tissues. The motor nerve to a muscle returns sensation from the joint(s) upon which that muscle acts. A mixed nerve is a combination of cutaneous sensory and motor nerves in one trunk. Complicating the picture is Hilton's Law which states that the nerve supplying a joint also supplies the muscles which move the joint and the skin covering the articular insertion of those muscles. A good way to think of the symptomatic pattern of peripheral nerve entrapment is sensory, motor, and sympathetic. **(Lusskin, 1982)**

Patients with acute or chronic entrapment neuropathies will often complain of feeling that their joints or muscles are weak and they fatigue easily. This experience may sometimes persist long after the pain has been alleviated and repair seems to be fully resolved. There is a biological reason for this mechanism. Forceful muscle activation will raise the intramuscular as well as the intracapsular pressure and may result in further damage to the nerve and its surrounding tissues. **(Racinais et al., 2008)**

In cutaneous sensory nerve involvement, the paresthesia is usually precisely defined by the patient. It may be a sharp, well–described pain, numbness, or altered sensations such as pruritus.

Motor nerve involvement will affect muscle activity which, if chronic, may be demonstrated as atrophy. The associated pain is more general and less well–defined by the patient. It will usually involve the muscle, its associated joints, and connective tissue in agreement with Hilton's Law.

Most neuropathies of either a motor or cutaneous sensory nerve will have some characteristics of both types. The often–involved mixed nerve may have characteristics of only the motor or cutaneous sensory fibers involved. There may be chronic sensory disturbance with no muscle atrophy, or vice versa.

Differential Diagnosis

There are two concerns in differential diagnosis of peripheral nerve entrapment. Obviously one is to differentiate conditions that simulate peripheral nerve entrapment, such as other forms of peripheral neuropathy, reflex sympathetic dystrophy, and radiculopathy. Sometimes the differentiation is to find the entire problem. This is what has already been discussed as the double crush syndrome. Double crush was originally related to two levels of peripheral nerve entrapment. **(Upton & McComas, 1973)** The same basic principle of reduced axoplasmic transport relates to conditions that cause the nerve cell body to have a lowered level of health. This can be diabetes, **(Veves et al., 2002; Hurst, Weissberg, & Carroll, 1985)** malnutrition, or other systemic conditions. Sometimes a local peripheral nerve entrapment is diagnosed, but a proximal one or a systemic problem is missed. The area primarily diagnosed may be a low level of entrapment that would not cause symptoms if the more proximal or systemic problem were not present.

When numerous factors are involved in peripheral nerve entrapment, the multiple diagnosis may be rather difficult. When two or more factors contribute to cause a symptomatic peripheral nerve entrapment, the individual factors making up the combined problem may be rather insignificant individually. For example, when there are two levels of nerve entrapment, either may be insufficient to cause symptoms but combined they do. **(Butler, 1991)** Spinner and Spencer state, "EMG studies are not always sufficiently sensitive to localize a double lesion." **(Spinner & Spencer, 1974)** When simultaneous entrapment or a diffuse peripheral neuropathy is suspected, one can evaluate other peripheral nerves to help differentiate a localized process from a systemic one. In applied kinesiology, various methods such as challenge, therapy localization, and nutritional testing can help discover all components responsible for the patient's condition.

Peripheral nerve entrapment must be differentiated from other forms of peripheral neuropathy. Metabolic disorders, such as diabetes mellitus, alcoholism, uremia, malnutrition, or vitamin deficiency may be confused with peripheral nerve entrapment or be contributing to a double crush condition. **(Travell & Simons, 1983)** These metabolic problems usually cause a symmetrical polyneuropathy, but an asymmetrical one may be present. In symmetrical neuropathy, the distal areas are involved first, with tingling and numbness beginning in the feet and progressing up the legs. The tips of the fingers may become involved. If there is muscle weakness it follows the course of the paresthesias, beginning in the feet.

Peripheral entrapment neuropathy is usually asymmetrical and localized, confining itself to a particular nerve and its branches. The complicating factor in differential diagnosis is that one who has a polyneuropathy is more subject to entrapment than a patient without the generalized problem is. Diabetics are good examples of this, as they are more susceptible to classic entrapment such as the carpal tunnel syndrome. **(Akuthota & Herring, 2009)**

Two common causes of vascular neuropathy — diabetes and vasculitis — should be easily diagnosed. Most patients with vasculitis are systemically ill with malaise, fever, weight loss, or an active disease process such as arthritis or renal disease. This can usually be easily distinguished with blood and urine laboratory tests.

Peripheral nerve entrapment must be differentiated from radiculopathy. There are usually spinal symptoms and signs indicating the possible involvement. The usual testing procedures described in Walther's ***Applied Kinesiology -- Synopsis*** **(Walther, 2000)** on the vertebral column help make the differential diagnosis, but keep in mind the double crush syndrome.

Dawson et al. **(Dawson, Hallett, Millender, 1983)** describe the following paresthesias and muscle weaknesses with levels of the cervical spine: C5 — deltoid and infraspinatus; C6 — biceps and wrist extensors, with paresthesias in the thumb and index finger; C7 — triceps, long finger flexors, and finger extensors, with paresthesias in the forearm and dorsum of the hand; C8 — intrinsic muscles of the hand and wrist flexors, with paresthesias in the little finger. L4 radiculopathy correlates with quadriceps muscle weakness and a reduced knee reflex. This must be distinguished from femoral neuropathy. L5 radiculopathy may resemble peroneal nerve entrapment. The tibialis posterior will be strong in peroneal nerve entrapment since it is innervated by the tibial nerve. "Tarsal tunnel syndrome and S1 radiculopathy may both cause pain in the heel and sole of the foot, whereas weakness of the knee flexors and foot plantar flexors points to the presence of radiculopathy." **(Dawson, Hallett, Millender, 1983)**

Amyotrophic lateral sclerosis may show its first symptoms in a localized area, such as the hand, causing it to be confused with peripheral nerve entrapment. There may be weakness of the hand, but muscle fasciculations are usually widespread and occur in muscles not yet clinically weak. Although they may not be seen by the examiner, the patient can usually describe and locate them. "A feature of the illness that is particularly pathognomonic is increased tendon reflexes in a muscle group that is fasciculating and atrophic." **(Dawson, Hallett, Millender, 1983)**

Reflex sympathetic dystrophy (RSD) has a similar local pain, tingling numbness, and atrophy as peripheral nerve entrapment. Early vasomotor changes, in most cases, include hyperhidrosis, hyperthermia, and erythema. These changes depend on the degree of sympathetic outflow. Mense and Simons **(Mense & Simons, 2001)** have summarized the basic science and clinical aspects of RSD, and Muir and Vernon have reviewed the chiropractic approach to RSD. **(Muir & Vernon, 2000)**

Drug toxicity can cause peripheral neuropathies that result in paresthesias and hypoesthesias. **(www.drugs.com www.)** The drug companies have listed the possible side-effects of their products. You can check these side-effects by reading the drug companies' literature at www.drugs.com or www.rxlist.com. A wide variety of drugs have been implicated in peripheral neuropathies.

Finally, long-term exposure to statins may also substantially increase the risk of polyneuropathy. **(Gaist et al., 2002)** Gaist et al's report in the journal *Neurology* expressed concern for the increased susceptibility to neuropathy among diabetics placed on statin drugs. They estimated that diabetics had as much as a sixteen fold increase in risk of neuropathy when statin drugs are used but suggested that non-diabetics are also susceptible.

There is a widely-recognized "drug side-effect epidemic" at work among all the patients physicians see today. The *American Society of Clinical Pharmacology and Therapeutics* stated **(2000)** that the percentage of elderly patients receiving nine or more medications was 27%, compared to 17% in 1997. This figure continues to mushroom. Most drugs have simply not been tested for use with other drugs, so that the "polypharmacy" seen in many patients must be confronted by the holistic physician. A recent article in ***Current Psychiatry*** **(Werder, 2003)** states "most individuals who are prescribed five or more drugs are taking unique combinations, representing an uncontrolled experiment with effects that cannot be predicted in the literature." The problem is so prevalent that some physicians "seem indifferent to so-called 'minor' side effects such as headaches, abdominal pain, fatigue, ringing in the ears, joint pain, insomnia, constipation, and hundreds of others that make people miserable." **(Cohen, 2001)**

It should be obvious that with a formidable list of such possible causes, it is necessary to discover what prescribed and over–the–counter medications the patient may be taking in any apparent peripheral nerve entrapment problem.

Palpation and Inspection

Palpation and inspection primarily deal with three types of evaluation for a specific nerve suspected of involvement, including inspection of the dermatome, muscles supplied by the nerve and of the nerve along its course.

Cutaneous

Deane Juhan **(Juhan, 1987)** writes about the importance of the skin:

> "The skin is no more separated from the brain than the surface of a lake is separated from its depths; the two are different locations in a continuous medium. 'Peripheral' and 'central' are merely spatial distinctions, distinctions which do more harm than good if they lure us into forgetting that the brain is a single functional unit, from cortex to fingertips and toes."

2

Table 1
Medications with neuropathy as a side-effect

Medications	Antibiotics Blood Pressure	Statins	Anti-Anxiety Anti-Depressant
Cymbalta®, Duloxetine hydrochloride®, Lyrica®, Neurontin®, Pregabalin®, Allopurinol®, Aminodipinberglate®, Amiodarone®, Amiodipine®, Amiodarone HCL®, Amitriphyline®, Besylate®, Cordarone®, Flagyl®, Lipitor®, Lotril®, Metrogl® Metrinidosole®, Metrofuranton®, Metronidazole®, Norvaso®, Pechexiline®, Vitorin®, and Zyloprin®.	Cipro®, Flagyl®, Metronidazole® __B.P.__ Amiodarone®, Atenolol®, Aceon®, Altace®, Cozaar®, Hydralazine®, Hydrochlorothiazide (HCT)®, Hydrodiuril®, Hyzaar®, Lisinopril®, Micardis®, Norvasc®, Perindopril®, Perhexiline®, Prazosin®, Prinivil®, Ramipril®, Zestril®.	Advicor®, Altocor®, Atorvastatin®, Baycol®, Caduet®, Cerivastatin®, Crestor®, Fluvastatin®, Lescol®, Lescol XL®, Lipex®, Lipitor®, Lipobay®, Lopid®, Lovastatin®, Mevacor®, Pravachol®, Pravastatin®, Pravigard Pac®, Rosuvastatin®, Simvastatin®, Vytorin®, and Zocor®.	Ambien (Zolpidem)®, BuSpar®, Klonopin (Clonazepam)®, Xanax®, Celexa (Citalopram)®, Cymbalta (Duloxetine)®, Effexor (Venlafaxine)®, Effexor XR®, Nortriptyline®, Zoloft®

Evaluation of the skin deals first with observation of its texture. There may be a palpable change in the area of nerve distribution, or the change may be only visually observed. Texture change gives some indication of how much involvement there is of the sympathetic fibers. **(Chaitow, 1987)** This may influence perspiration and circulation into the area, as the sweat glands are controlled by the sympathetic portion of the autonomic nervous system. If the nerve is severed proximal to the sympathetic ganglia, there will be an absence of sweating with central physiologic stimulation. Severance of a nerve on a peripheral basis causes lack of response to any stimulation, and there will be no response to cholinergic drugs. A peripheral irritation causes undiminished or excessive sweat reactions. **(Guyton & Hall, 2005)** The secretory pores of the sweat glands are controlled by acetylcholine, a neurotransmitter which also influences the contraction and relaxation of muscles.

The skin is also evaluated for varying temperatures by visualization and palpation. Accurate palpation also depends upon the experience of the examiner, his hydration, peripheral circulatory efficiency, sympathetic nervous system activity, and the ambient humidity and temperature of the room. The physician can best feel for cool or warm areas by running the dorsal aspect of his hand over the various dermatomes being evaluated. When evaluating the skin for excessive or diminished thermal areas, remember that imbalance of the meridian system can create hot (Re) and cold (Han) spots in specific locations. **(Walther, 2000; Stux & Pomeranz, 1987)** Barral has written a book on manual thermal diagnosis and it is recommended for further refinements of this approach. **(Barral, 1996)**

There is a wide range of electronic thermometers available that readily measure skin temperature. The temperature of a dermatome can be used to initially diagnose the cause of involvement, or to evaluate treatment effectiveness. Dermatomes with abnormal temperature can be identified by comparing the symptomatic and asymptomatic sides. When a dermatome of involvement is identified, tape the thermometer in place and allow the recording device to stabilize. If a two–thermometer recording device is available, tape the second thermometer to the normal bilateral counterpart in unilateral conditions. Make a comparison between the two sides and then put the patient through various provocative tests, which will be described with many of the conditions in the following chapter. Allow 2–3 minutes in the position to observe for temperature change.

In a similar manner, thermal recording can be used to evaluate the effectiveness of treatment. Place the thermometer either unilaterally or bilaterally as described above. After allowing the recording device to stabilize, apply the therapeutic effort, which may be structural, nutritional, or meridian system balancing, among others. Allow a few minutes to pass and observe temperature change. This is most effective as an evaluation method when both the normal and abnormal sides are being recorded. Ordinarily one expects no change in the normal side, and normalization of the involved side.

Plethora can be observed by digital pressure on the skin to blanch it, and then noting the length of time it takes for balanced color to return. The plethora may be systemic or localized in an extremity. If localized, areas of possible venous entrapment must be evaluated. Thorough evaluation of the circulation and sensibility of the dermatomes is part of the skin evaluation; this will be discussed later in the specific areas.

Methods for evaluating structural abnormalities in the skin have been developed in applied kinesiology. **(Leaf, 2010; Walther, 2000; Goodheart, 1986, 1983)** The cutaneous receptors are stimulated with joint motion. For example, when the knee flexes, the skin over the quadriceps and anterior knee stretches. When the knee extends, the skin over the hamstrings and popliteus muscle stretches. The muscle underlying the area of stretched skin is normally inhibited with the joint motion. The contribution of the cutaneous receptors to the neurologic organization of muscles in joint movement can be observed by manual muscle testing in applied kinesiology. For instance, the examiner can stretch the skin over the quadriceps muscle in alignment with the muscle fibers. This may be done by simply taking a pinch of skin between the thumb and forefinger of each hand and pulling the skin apart, taking care not to heavily pinch the skin with the fingers. Immediately after stretching the skin, manual muscle test the quadriceps muscle group. Under normal circumstances, the muscles will test weak for a variable length of time. Stretching the skin in this manner simulates knee flexion, as if the hamstrings had contracted. This would cause reciprocal inhibition of the quadriceps muscles, which is what is observed when the muscle tests weak.

Clinical evidence in applied kinesiology indicates that in some patients the cutaneous receptors may be inappropriately stimulated or react inappropriately to stimulation, sending information not in keeping with the joint motion. When a muscle tests weak as a result of improper signaling from the cutaneous receptors, it can immediately be made to test normal by skin stretching. For example, if the hamstring muscles test weak, stretch the skin over the quadriceps group in the direction in which it would normally be stretched if the hamstrings were contracting. The amount of stretch is the amount the skin will yield without damage or severe pain. If dysfunction of the cutaneous receptor is associated with the dysfunctioning hamstring muscles, they will test strong immediately after the skin stretch. Since no structure other than the skin is manipulated or stimulated, it appears that the improved muscle function results from stimulation of the cutaneous receptors. Making a muscle strong in this manner is only a diagnostic test for apparent cutaneous receptor dysfunction. The strengthening of the hamstrings will only last for twenty or thirty seconds, regardless of how vigorous or long- lasting the skin stretch is. Treatment of the cutaneous dysfunction (soft-tissue, instrumental, nutritional, cold-laser and other techniques in applied kinesiology) will correct this problem. **(Kharrazian, 2002; Ramsak, 1997; Dauphine, 1994; Burstein, 1990)**

Muscle.

The muscles innervated by the nerve being evaluated should be inspected and palpated for consistency, pain, and size. When a motor nerve is involved, the pain will typically be throughout the muscle and in the joints served by that muscle. The patient will usually complain of a dull, deep, general–type pain rather than the sharper, delineated pain of skin sensory nerve involvement. The muscle may have atrophy that is observed only by astute palpation, especially in the earlier stages of entrapment. In this case the muscle will feel like it has lost consistency, which is often called tone. Comparison with the bilateral counterpart helps determine this loss.

The circumference of the extremity should be measured bilaterally. Standard levels of measurement are established, such as 4" above the olecranon process with the arm extended, so that repeated measurements will be accurate. It is important to document muscle size on an initial examination, especially in personal injury and worker's compensation cases. Progressive atrophy from the time of injury to maximum medical improvement is an important documentation in permanent impairment rating. Muscle wasting can begin surprisingly quickly in patients with chronic low back and neck pain. The cervical and lumbar multifidus, as well as the psoas muscles have been observed to experience muscle wasting within 24 hours of the onset of pain. **(Danneels et al., 2009; Fernandez-de-las-Penas et al., 2008; Hides et al., 1994)**

There may be muscle dysfunction that is not observable by palpation and inspection, but it is observed by manual muscle testing as used in applied kinesiology. Specific challenge, therapy localization, and types of muscle tests will be explained with the various syndromes.

Nerve.

Palpation and inspection of the course of the nerve provide much useful information. In many areas, the nerve is rather superficial and can easily be palpated, which may reveal tumors or pain along the nerve.

Pain along the nerve may be proximal or distal to the point of entrapment. This tenderness is known as the Valleix phenomenon. **(Barral & Croibier, 2007; Kopell & Thompson, 1976)** Nerves have been described as small, thin, slightly twisted strings or cords. The site of involvement may be localized by observation of swelling at a potential area of entrapment. The swelling may be a result of trauma to the area, or it may be systemic from a toxic condition, hormone imbalance, or disease process.

Tinel's sign **(Evans, 2008; Wilkins & Brody, 1971)** refers to pain or tingling of a nerve when it is struck over an area of neuropathy. Tinel's sign was originally discovered almost simultaneously and independently by a German doctor, Paul Hoffmann, and a French doctor, J. Tinel, during World War I. Tinel's description indicates that pressure on a damaged nerve trunk often produces a tingling sensation projected to the periphery of the nerve and localized to a very exact cutaneous area. He differentiates this tingling sensation from that of pain produced by pressure on an injured nerve. Pain is a sign of nerve irritation; tingling is a sign of regeneration or, more precisely, it indicates the presence of young axons in the process of growing. When a nerve is relatively superficial, Tinel's sign should only be elicited by a gentle tap or pressure over the nerve. A sharp blow over the nerve of a normal individual will cause pain or tingling and should not be interpreted as a positive sign. **(Phalen, 1972)** Today Tinel's sign is usually elicited by tapping with a reflex hammer over the nerve site being evaluated, instead of the tapping finger described by Hoffmann and Tinel. **(Evans, 2008)**

General Physical Examination

A general physical examination is required to determine if there are any systemic health problems. Toxicity from conditions such as the ileocecal valve syndrome seems to concentrate swelling in areas of generalized weakness. If a patient has subclinical disturbance of structure — a tight osteofibrous tunnel or weak intervertebral disc — swelling may cause an additional enlargement of the structures that creates a symptomatic involvement; thus, both the systemic and localized problems must be treated. Other conditions such as hypothyroidism, pregnancy, rheumatoid arthritis, acromegaly because of proliferations of tissues narrowing the osteofibrous tunnel, **(Bastron, 1975)** hypoadrenia, **(Wilson, 2002)** and generalized structural imbalance can cause an individual to be more susceptible to peripheral nerve entrapment. In systemic problems such as nutritional deficiency, the distal peripheral nerves are more vulnerable to dysfunction. In these cases the entrapment is more likely to be found in distal areas such as the carpal tunnel, tarsal tunnel, and intermetacarpal tunnel.

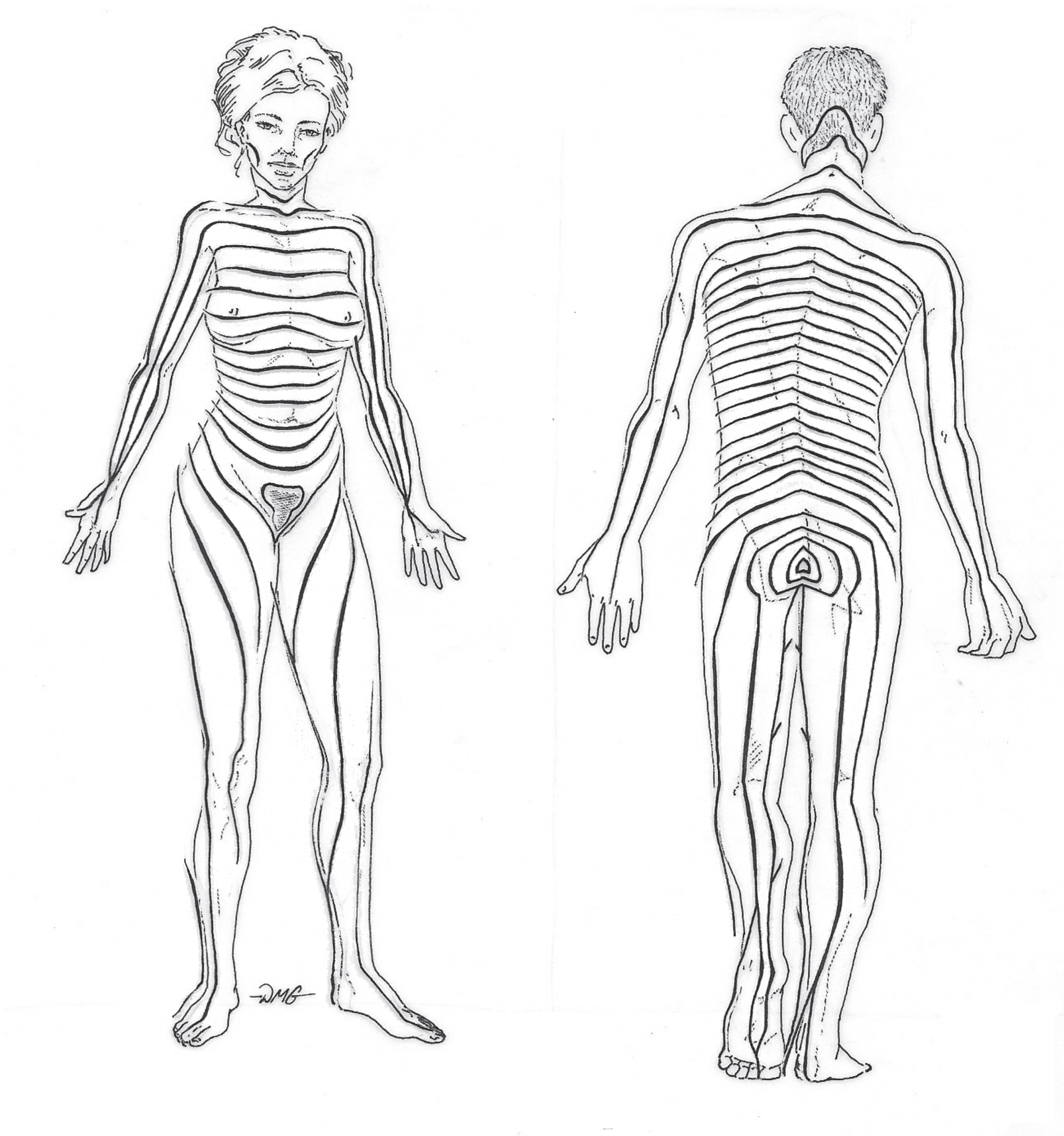
Sensibility Studies

Sensory examination can help define cortical and thalamic lesions and cord involvement, as well as peripheral neuropathies. Here we will deal primarily with peripheral nerve entrapment.

The patient must be cooperative and alert, and the physician must be careful to avoid suggestions that influence the patient's response. If a thorough sensory evaluation is made, it can be very time–consuming and tiring to both the physician and the patient. It is best, then, to intersperse this activity with other examination procedures to break up the monotony of the examination.

The tools of examination include a wisp of cotton for evaluating soft touch, a pin with a sharp point and dull head mounted on an applicator or a Wartenberg pinwheel for pain, vials containing both hot and cold water for thermal testing, a tuning fork for deep sensation, and a compass with two dull points for two–point discrimination.

In addition to the general examination tools mentioned above, there are very sensitive, graded–hair test kits to evaluate sensory loss. In general, a relatively gross method of examination for sensory loss is adequate. In a study by Gilliatt and Wilson, **(Gilliatt & Wilson, 1953)** the very sensitive, graded–hair method was used to determine sensory loss in comparison with massive stimulus, such as the examiner's finger touching the patient's skin. It was found that there was little advantage in using the sensitive approach, such as a wisp of cotton or graded hairs. They concluded, "From the practical point of view there is every advantage to be gained from recording merely the subjective sensation in response to a massive stimulus, as this is easily determined in unintelligent patients, and is much less affected by horny skin or cold hands than more elaborate methods of sensory testing."



Dermatone distribution

It is necessary for the examiner to have a general knowledge of dermatome distribution and the congenital variations of the peripheral nervous system. A chart should be available for mapping the examination findings. When a test is to be done, the patient should be familiar with its procedure and how to respond to the stimulus to avoid any misunderstanding. To ensure that the patient does understand the test, go through a demonstration and have him or her respond to the stimulus.

When the testing procedures actually begin, it is best for the patient to close his eyes and relax, concentrating on the sensation testing. Various procedures should be alternated and timed at different rates. This not only helps break the monotony of the examination, it also keeps the patient from responding to an expected stimulus without the sensation being perceived. Although many types of responses are suggested for perceived sensation, a simple "yes" is satisfactory. It also adds another depth that may sometimes be very revealing when the neurotic, unsophisticated, or malingering patient answers "no" to a stimulus.

It is necessary to evaluate the body bilaterally when testing for peripheral nerve involvement. Usually there is partial rather than total loss of sensation. This can best be observed by comparing the two sides. Bilateral comparison is easily done by simultaneously stimulating both sides, using two Wartenberg pinwheels or wisps of cotton and asking the patient to make a comparison.

When areas of sensation loss are located, the borders must be defined. In typical peripheral nerve entrapment, the borders will be abrupt and will correspond in general to the appropriate dermatome. In peripheral neuropathy resulting from ischemia, the border is ill–defined; the hypoesthetic area will probably cross many dermatomes. The same is often true when a large number of nerves is involved, such as the neurovascular bundle in certain types of thoracic outlet syndrome. When there is hypoesthesia as a result of malnutrition, the distal aspects of the extremities are most involved, causing a general loss of sensation that is greatest distally and decreases proximally. This is the so–called "glove" or "stocking" type of sensory loss. It must be differentiated from sensory loss due to ischemia.

The important aspect of the sensibility of the hand is how it can do the many tasks required in performing necessary functions. It is also important to be able to evaluate increasing or decreasing loss of function. Moberg **(Moberg, 1958)** developed a test he calls the "picking–up test." The patient is asked to pick up numerous small, familiar objects from a table and put them in a container, first with one hand and then the other. After becoming familiar with the task, the subject is then asked to repeat the process blindfolded. During both visual and blindfolded performances he is timed, and his efficiency in picking up the objects is evaluated. Under normal conditions of stereognosis, he is able to identify the objects when doing the task blindfolded. Usually the objects are picked up with median nerve–innervated fingers. If there is median nerve loss, the subject will often divert the task to ulnar–innervated fingers combined with the thumb.

Loss of sensory peripheral nerve function is accompanied by loss of sweat gland function. Moberg **(Moberg, 1958)** also devised an objective test that depends on the sweating of the skin of the fingers — the ninhydrin fingerprint method — and with it he was able to objectively show the parts of the skin with intact sensation and those that were defective. He also observed that "…it was possible to see changes in an area of defective sensibility with the naked eye. Thus it could be seen that the pulp was atrophied, the ridges were more or less eradicated and the colour was changed." One can often see these changes when comparing the normal with the abnormal hand.

A simple test to objectively document sensory loss has been described by O'Riain. **(O'Riain, 1973)** The characteristic wrinkling of the skin of the fingers when immersed in warm water is absent in the presence of sensory denervation. The procedure is especially valuable in examining young children who may not have the ability to cooperate. The extremity is immersed in 40°C (104°F) water for thirty minutes. Normally the skin of all fingers wrinkles or "shrivels"; denervated fingers remain smooth.

Circulatory Evaluation

Disturbance of blood circulation may be a result of peripheral neuropathy or a cause of it; consequently, differential diagnosis of circulation must be done when considering peripheral nerve entrapment. A disturbance in circulation may be secondary to peripheral nerve entrapment as a result of involvement of the sympathetic fibers; this is neurogenic ischemia. On the other hand, nerve tissue is very susceptible to ischemia; consequently, entrapment may be more significant secondary to a diminished vascular supply.

It must be remembered that the diameter of the arteries and veins is controlled by the nerves and nervous system. The vasomotor effect of manipulative treatment is achieved either by the surrounding nerves of the blood vessels or by the sympathetic nervous system. After chiropractic treatment to the cervical spine and brachial plexus, we have consistently seen changes in the terminal branches of the brachial plexus which can be recorded on the sphygmomanometer. **(Mc Knight, DeBoer, 1988; Einaudi, 1959; Wright, 1956, 1955)**

There is a wide range of diminished circulation. Some conditions require the immediate attention of a vascular surgeon; others are effectively treated with applied kinesiology conservative methods. Obviously, if the ischemia is due to an occlusion of the blood vessel, circulation must be returned to normal before the nerve will function properly. The ischemia may be a result of thrombosis, embolism, neoplasms, orthopedic faults, or other factors that occlude the vessel. Also, consideration should be given to vascular diseases, such as arterio– and atherosclerosis, and systemic health problems such as diabetes.

When there is evidence of circulatory disturbance, a thorough work–up must be done. One should not jump to the conclusion that a cervical rib is causing circulatory deficiency. Many people have cervical ribs that are asymptomatic. A patient may have both a vascular condition and a cervical rib that is no more than coincidental. Symptoms of vascular occlusion should never be overlooked. "The end–result may be loss of fingers, ischaemic contracture of forearm and hand, or a major amputation, following occlusion of the main vascular channels by embolism or thrombosis.

Physical tests must be put into proper perspective relative to what they are capable of showing and what they fail to reveal. In general, there are no pathognomonic provocative tests for peripheral nerve entrapment or vascular deficiency.

Measurement of the extremities provides information regarding venous or lymph drainage. Comparison can be made after an individual has been recumbent for a period of time, such as before rising in the morning, with the end of the day. Continued comparison over days, weeks, or months indicates treatment effectiveness.

It takes very high pressure applied to a nerve alone to diminish conduction, but when pressure is applied to cause anoxia by diminishing blood flow, much less pressure is needed. Compression ischemia, in general, first causes dysesthetic sensations, followed by loss of touch, then pain sensation, and finally — much later — loss of motor power. With motor weakness appearing last, it is last to return in persistent deficits. **(Russell, 2006; Asbury, 1970)**

There are several variations of the "tourniquet test," in which a blood pressure cuff is inflated above systolic level to create ischemia or additional ischemia in the area being evaluated. **(Nitz & Dobner, 1989)** In patients who have peripheral nerve entrapment, the increase in paresthesia or pain will develop more rapidly than in those who do not. Fullerton **(Fullerton, 1963)** studied nerve conduction in individuals diagnosed as having carpal tunnel syndrome when artificial ischemia was produced by a supra–systolic blood pressure cuff applied to the upper arm. The pressure was maintained for 30 minutes at 200–220 mm Hg. In control subjects the action potential did not change after 20 minutes ischemia. After 25 minutes there was still more than 70% of the initial size in all subjects. At the end of 30 minutes, all subjects had at least 50% of initial value. Patients with carpal tunnel syndrome were more susceptible to the ischemia. Occasionally the action potential had almost disappeared after 10 minutes; in some it had fallen to less than 40% after 25 minutes. In this study there did not appear to be any difference in recovery between the control subjects and patients after release of the blood pressure cuff. Both ischemia and direct pressure appeared to contribute to the abnormality of nerve function. The recovery rate here is different from nerves that suffer from prolonged ischemia, which has a longer recovery period. **(Staal et al., 1999; Wilgis, 1971)**

Another study that evaluated the rapidity of sensory loss when artificial ischemia is induced was done by Gilliatt and Wilson. **(Gilliatt & Wilson, 1954)** In a study of 40 patients with peripheral nerve and dorsal root lesions affecting the hand, compared with 50 control subjects, they found that the controls had sensory loss within an average of 14 minutes. The patients with peripheral nerve lesions developed sensory loss usually within 5 to 10 minutes of the onset of ischemia.

There are specific characteristics that one can look for in the tourniquet test in different areas. For example, in carpal tunnel syndrome the paresthesia that develops is of median nerve distribution, whereas in normal subjects the tourniquet test causes ulnar or diffuse paresthesia. **(Nitz & Dobner, 1989; Gilliatt & Wilson, 1953)** Patients evaluated for unilateral disturbance are best studied by applying the tourniquet test to the suspected abnormal limb and then the normal one, comparing the time and amount of pain and paresthesia.

There are orthopedic tests designed for evaluating the vascular system at the various areas where blood flow restriction may occur. These include maneuvers such as Adson's and Wright's tests for thoracic outlet syndrome, which will be discussed when the anatomical area is considered in the following chapters.

Several types of non–invasive instrumentation are available to provide specific data regarding the circulatory system. By themselves they are incapable of making a diagnosis, but they are significant diagnostic aids. Usually, the best approaches are non–invasive because they lack the potential side effects present in angiography. **(Benya et al., 1989; Kopell, 1980)** With non–invasive equipment, a diagnosis of patency or occlusion can usually be made; if the vessels are patent, the potential hazards of angiography can be avoided.

Plethysmography

In general, plethysmography relates to measuring blood flow volume, usually at the distal extremities. There are many types of plethysmographs available. Some are transducers that convert the mechanical pulsation of blood circulation to electricity. The mechanism may be a strain gauge directly contacting the skin or using a conductive medium that is hydraulic or pneumatic to couple with the transducer. **(Higashi & Yoshizumi, 2003)** Other plethysmographs are primarily electrical, measuring the impedance and inductance. In a class by itself is the photoelectric cell. The latter shines a light into the capillary bed that is reflected back in various intensities, depending on the bed's engorgement. The reflected light is picked up by a photoelectric cell. All modern plethysmographs display the electrical current on an oscilloscope or on a strip recorder. The latter has the advantage of making a continuous recording while the patient is maneuvered into provocative test positions. When one can look at the recorded strip rather than remembering what was on the oscilloscope, comparisons are easier to make.

The plethysmograph can be used to simply record the pulse wave in the various digits on a comparative basis. The pulse wave can also be recorded as the patient goes through various orthopedic maneuvers, such as Adson's and Wright's tests. This gives an improved evaluation of blood flow change over simply monitoring the pulse at the radial artery by the examiner's digital sensation. The provocative tests and their use with the plethysmograph are discussed with the various syndromes. Current interpretation of many of these tests, especially at the thoracic outlet, is modified over that of the past.

Another test done with plethysmography is called reactive hyperemia, which helps determine the patency of the blood vessels and whether the nervous system reacts to the body's physiologic needs. First, a recording is made of the peripheral circulation in the resting state; then the vessel is occluded proximal to the area being evaluated. This is accomplished with a blood pressure cuff pumped up above the systolic blood pressure. Complete occlusion is indicated by eliminating the pulse wave display on the plethysmograph. The restriction of blood flow is maintained for 3–5 minutes to produce ischemia. Blockage of the blood flow during this time is not harmful, but it must be pointed out that over 30 minutes of complete occlusion causes microscopic changes in human muscle. **(Wilgis, 1971)** The blood flow is then released while a recording is made of the pulse wave. In a normal reactive hyperemia test, the pulse wave will increase over the resting state. **(McGrath et al., 1980)** This is a normal body reaction to increase blood flow into the area to eliminate the ischemia resulting from the restriction. This activity is moderated by the nervous system. A positive test is one in which there is no increased blood flow after the restriction; in some cases, the blood flow may even be reduced from that of the resting state. A positive test indicates a disturbance in the nervous system's regulation of blood flow or an occlusion of the blood vessel that will not allow adequate increased flow. This test is valuable as a before and after treatment evaluation when a diagnosis of peripheral nerve entrapment has been made as the primary cause of ischemia. After the therapeutic effort to return the nerve to normal, there should be improvement in the reactive hyperemia test. Improvement of the reactive hyperemia test is often seen immediately after applied kinesiology treatment to release a peripheral nerve entrapment. In chronic conditions, there may be a delay of one or two weeks before the improvement is seen, even though correct therapy has been applied. This may be due to the fibers responsible for improving the circulation needing time for their own recovery before they can control circulation normally.

The control of the vascular bed can also be evaluated by a plethysmograph recording made at room temperature, after which the patient's extremity is placed in ice water for 3 minutes. The blood flow volume is then recorded after removal of the hand from the bath and should return to near–normal within 5 minutes; if it does not, there is evidence of vasospasm. Foster's test is similar, but rather than blood flow volume as an indicator, the extremity temperature is taken before and after the ice stress. **(Wilgis, 1980)**

Disturbed circulation observed by the plethysmograph must be differentially diagnosed. It has been shown that radiculopathy treated by a chiropractic vertebral adjustment can be improved with no additional treatment. **(Figar & Krausova, 1965; Figar et al., 1967)** Budgell & Sato have also demonstrated that regional blood flow can be affected by cervical manipulation aimed at the correction of vertebral subluxations. **(Budgell & Sato, 1997)**

Doppler

The Doppler instrument — an ultrasonic generator and flow detector — is named after the Doppler effect. The transducer emits ultrasound waves through piezoelectric crystals that are transmitted through the skin directly to the blood vessel. They are reflected back to the probe, which has a receiving unit. Ultrasound waves that strike moving tissue are reflected back differently from those that strike stationary tissue. The greater the intensity of movement, the greater the change. The waves that are reflected back are amplified and demonstrated by audible sound or by demonstration of the pulse waveform on an oscilloscope, or on a strip recorder. Arterial blood flow has an alternating high–pitched sound as the blood pulsates in the vessel, while venous blood flow has a slow, waving sound much like that of the wind. Doppler evaluation can be used to locate areas of occlusion in the artery or vein. Various methods are used in Doppler evaluation to locate an occlusion. The method is not limited to the extremities; it is also used in studying disturbances in cranial circulation.

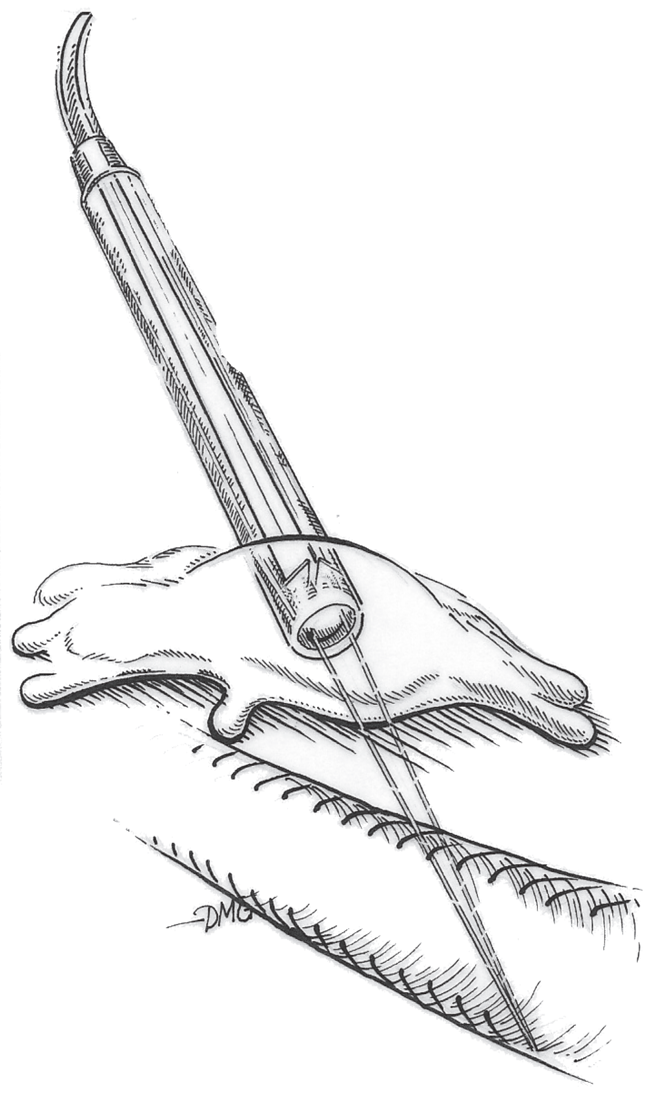
Blood pressure in the lower extremity can be accurately taken by Doppler testing. A standard blood pressure cuff of the proper size for the limb is applied at the ankle, below the knee, or above the knee. The pulse is usually monitored at the posterior tibial artery by the Doppler. This means of accurately obtaining the arm and ankle systolic blood pressures gives the ability to compare them. This is called the ankle–to–arm index. Pressure at the ankle should be at least equal to, but preferably higher than, that of the arm. When there is a diminished ankle–to–arm index, it indicates that there is some type of vascular disturbance capable of producing symptoms. Less than .9 is considered abnormal; .9–.7 is consistent with intermittent claudication; .4–.3 is consistent with resting pain; below .3, gangrene may develop.

X–ray

X–ray is used for evaluating the circulatory system and for peripheral nerve entrapment syndromes. Only a brief description of its use is within the scope of this text.

Angiography is the injection of a contrast medium into the circulatory system to study its anatomy, areas of occlusion within the vessels, and impinging structures such as neoplasms and tortuous routes. The procedure is not without its hazards. There may be a reaction to the iodides used in the contrast medium or complications at the site of arterial puncture. It is primarily an anatomical study providing no information about the dynamic state of circulation. It is not recommended for routine or repetitive use. **(Kopell, 1980)**

X–ray is more routinely used to evaluate for bony spurs, congenital anomalies, and trauma that can impinge upon a nerve or occlude a vessel. X–ray is also valuable in locating neoplasms and other pathologies that may be involved with the condition.



Schematic of Doppler

References

- Adson AW. Cervical ribs: Symptoms, differential diagnosis and indications for section of the scalenus anticus muscle. J Int Coll Surgeons. 1951;16(5):546-559.
- Aguayo AJ. Neuropathy due to compression and entrapment. In: Peripheral Neuropathy, Vol I. Eds. Dyck PJ, Thomas PK, Lambert EH. W.B. Saunders Co.:Philadelphia; 1975.
- Akuthota A, Herring SA (Eds). Nerve and Vascular Injuries in Sports Medicine. Springer: Dordrecht; 2009.
- Alis GP. Medial epicondylitis: a case study. Collected Papers International College of Applied Kinesiology, 2003-2004;1:3-4.
- American Medical Association: Guides to the Evaluation of Permanent Impairment, 5th Ed. 2001:510.
- Amadio PC. Discussion on carpal tunnel syndrome and vitamin B6. (article by Kasdan & Janes) Plastic Reconstruct Surg. 1987;79.
- Asbury AK. Ischemic disorders of peripheral nerve. In: Handbook of Clinical Neurology, Vol 8. Ed. Vinken PJ, Bruyn GW. Elsevier Pub Co.: New York; 1970.
- Barral JP, Croibier A. Manual therapy for the peripheral nerves. Churchill-Livingstone: Edinburgh; 2007.
- Barral JP. Manual thermal diagnosis. Eastland Press, Inc.: Seattle; 1996.
- Bastron JA. Neuropathy in diseases of the thyroid. In: Peripheral Neuropathy, Vol II. Ed Dyck PJ, Thomas PK, Lambert EH. W.B. Saunders Co.: Philadelphia; 1975.
- Benya R, Quintana J, Brundage B. Adverse reactions to indocyanine green: a case report and a review of the literature. Cathet Cardiovasc Diagn. 1989;17(4):231-3.
- Blaich RM. Manual and machine muscle testing before and after correction of 'respiratory faults. In: Selected Papers of the International College of Applied Kinesiology. Ed Markham BC. ICAK USA: Detroit, MI;1981.
- Blaich RM,. Mendenhall EI. Manual muscle testing and Cybex machine muscle testing, a search for a correlation. In: Selected Papers of the International College of Applied Kinesiology (1984). Ed. Schmitt WH, Jr. ICAK USA: Park City, UT; 1984.
- Blum CL, Cuthbert S. Cranial Therapeutic Care: Is There any Evidence? Chiropractic & Osteopathy 2006;14:10.
- Breig A. Adverse Mechanical Tension in the Central Nervous System. John Wiley & Sons: New York; 1978.
- Breig A, Tumbull I, Hassler, O. Effects of mechanical stresses on the spinal cord in cervical spondylosis. A study of fresh cadaver material. J Neurosurg 1966;25:45-56.
- Bronston LJ, Larson KM. A new approach to upper rib fixation mobilization. Chiropr Tech. 1990;2(2):39-44.
- Browning JE. Distractive manipulative protocols in treating the mechanically induced pelvic pain and organic dysfunction patient. Chiropractic Tech 1995;7(1):1-11.
- Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine. 2000;25(8):989-994.
- Budgell B, Sato A. The cervical subluxation and regional blood flow. J Manipulative Physiol Ther. 1997;20(2):103-107.
- Burstein EM. Raynaud's phenomenon secondary to scleroderma: a case study. Collected Papers International College of Applied Kinesiology. 1989-1990;1:17-19.
- Butler DS. The Sensitive Nervous System. Noigroup: Adelaide; 2000.
- Butler DS. Mobilisation of the Nervous System. Churchill-Livingstone: Melbourne, 1991.
- Chaitow L, et al. Naturopathic Physical Medicine: Theory and Practice for Manual Therapists and Naturopaths. Churchill Livingstone: Edinburgh; 2008.
- Chaitow L. Cranial Manipulation: Theory and Practice. Churchill-Livingstone; 2005.
- Chaitow L. Soft-Tissue Manipulation. Healing Arts Press: Rochester, VT; 1987.
- Charash R. The human factor: Tools need not harm the hands that use them. Industrial Design Mag; 1982.
- Chien AJ, Jamadar DA, Jacobson JA, Hayes CW, Louis DS. Sonography and MR imaging of posterior interosseous nerve syndrome with surgical correlation. AJR. 2003;181:219–221.
- Christiansen JA, Meyer JJ. Altered metabolic enzyme activities in fast and slow twitch muscles due to induced sciatic neuropathy in the rat. J Manip Physiol Ther. 1987;10(5):227-231.
- Cohen JS. Overdose: The case against the drug companies. Prescription drugs, side effects, and your health. Putnam: New York; 2001.
- Conable K, Corneal J, Hambrick T, Marquina N, Zhang J. Investigation of Methods and Styles of Manual Muscle Testing by AK Practitioners. J Chiropr Med. 2005;4(1):1-10.
- Coq JO, Xerri C. Tactile impoverishment and sensorimotor restriction deteriorate the forepaw cutaneous map in the primary somatosensory cortex of adult rats. Exp Brain Res. 1999;129(4):518-531.
- Cramer GD, Darby SA. Basic and Clinical Anatomy of the Spine, Spinal Cord, and ANS. Mosby-Yearbook: St. Louis; 1995.
- Crymble B. Brachial neuralgia and the carpal tunnel syndrome. Br Med J. 1968;3(5616):470-471.
- Cseuz KA, et al. Long—term results of operation for carpal tunnel syndrome. Mayo Clin Proc. 1966;41(4):232-241.
- Cuthbert S, Rosner A, McDowall D. Association of manual muscle tests and mechanical neck pain: Results from a prospective pilot study. J Bodyw Mov Ther. 2011;15(2):192-200.
- Cuthbert S, Rosner A. Applied Kinesiology Management of Long-Term Head Pain Following Automotive Injuries: A Case Report. Chiropr J Aust. 2010;40:109-16.
- Cuthbert SC, Barras M. Developmental delay syndromes: psychometric testing before and after chiropractic treatment of 157 children. J Manipulative Physiol Ther. 2009;32(8):660-9.
- Cuthbert SC, Goodheart GJ Jr. On the reliability and validity of manual muscle testing: a literature review. Chiropr Osteopat. 2007;15:4.
- Cuthbert S, Blum C. Symptomatic Arnold-Chiari malformation and cranial nerve dysfunction: a case study of applied kinesiology cranial evaluation and treatment. J Manipulative Physiol Ther. 2005;28(4):e1-6.
- Cuthbert S. The piriformis muscle and the genitor-urinary system: The anatomy of the muscle-organ-gland correlation. Int J AK and Kinesio Med. 2003;15.
- Cyriax J. Fibrositis. Br Med J. 1948;2(4569):251-5.
- Dahlin LB, Nordborg C, Lundborg G. Morphologic changes in nerve cell bodies induced by experimental graded nerve compression. Exp Neurol. 1987;95(3):611-21.

- Dahlin LB, Sjöstrand J, McLean WG. Graded inhibition of retrograde axonal transport by compression of rabbit vagus nerve. J Neurol Sci. 1986;76(2-3):221-30.
- Dahlin LB, Rydevik B, McLean WG, Sjöstrand J. Changes in fast axonal transport during experimental nerve compression at low pressures. Exp Neurol. 1984;84(1):29-36.
- Daniels L, Worthingham K: Muscle Testing – Techniques of Manual Examination, 7th Edition. Philadelphia, PA: W.B. Saunders Co.; 2002.
- Danneels LA, Vanderstraeten GG, Cambier DC, et al. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur Spine J. 2009;9(4):266-272.
- Dauphine DB. Case report of applied kinesiology treatment and allergic cutaneous vasculitis. Collected Papers International College of Applied Kinesiology. 1993-1994;1:217-221.
- Dawson DM, Hallett M, Millender LH. Entrapment Neuropathies. Little, Brown & Co: Boston; 1983.
- DeJarnette MB. Collected Writings, 1929-1984. www.soto-usa.com; http://www.sorsi.com/products-services.
- Denny-Brown D, Brenner C. The effect of percussion of nerve. J Neurol Neurosurg Psychiatry. 1944;7(3-4):76-95.
- Devor M, Seltzer Z. Pathophysiology of damaged nerves in relation to chronic pain. In: The Textbook of Pain. Eds. Wall PD, Melzack R. Churchill Livingstone: Edinburgh;1999.
- Drugs.com www. Literature produced by the pharmaceutical companies on drug side-effects.
- Dyck PJ, Thomas PK. Peripheral neuropathy, Vol 2, 3rd Ed. WB Saunders: Philadelphia; 1993:513-567.
- Edwards FO. Pyriformis syndrome. Acad Applied Osteopath Yr Bk; 1962.
- Einaudi G. Research on vasomotor disorders of the upper extremities present in patients with cervicalgia & cervicobrachialgia; changes induced by static variations of the cervical spine. Reumatismo. 1959;11(3):173-8.
- Evans RC. Illustrated Orthopedic Physical Assessment, 3rd Ed. Mosby; 2008.
- Fernandez-de-las-Penas C, Albert-Sanchis JC, Buil M, et al. Cross-sectional area of cervical multifidus muscle in females with chronic bilateral neck pain compared to controls. J Orthop Sports Phys Ther. 2008;38(4):175-180.
- Figar S, Krausova L. A plethysmographic study of the effects of chiropractic treatment in vertebrogenic syndromes. Acta Universitatis Carolinae. Supp 21; 1965.
- Figar S, Krausova L, Lewit K. Plethysmographic examination following treatment of vertebrogenic disorders by manipulations. (German translation), Acta Neuroveg. 1967;29.
- Fullerton PM. The effect of ischaemia on nerve conduction in the carpal tunnel syndrome. J Neurol Neurosurg Psychiatry. 1963;26:385-397.
- Gaist D, Jeppesen U, Andersen M, García Rodríguez LA, Hallas J, Sindrup SH. Statins and risk of polyneuropathy: a case-control study. Neurology. 2002;58(9):1333-7.
- Gardner WJ. Trigeminal neuralgia. Clin Neurosurg. 1967;15.
- Garten H. Lehrbuch Applied Kinesiology Muskelfunktion-Dysfunction-Therapie. (in German) Urban & Fischer: Munich; 2004.
- Gasser HS, Erlanger J. The role of fiber size in the establishment of a nerve block by pressure or cocaine. Am J Physiol. 1929;88.
- Gelberman RH, Hergenroeder PT, Hargens AR, Lundborg GN, Akeson WH. The carpal tunnel syndrome: A study of carpal canal pressures. J Bone Joint Surg. 1981;63(3):380-383.
- Gerz W. Lehrbuch der Applied Kinesiology (AK) in der naturheilkundlichen Praxis. (in German) AKSE-Verlag: Munchen; 2001.
- Giggey K, Tepe R. A pilot study to determine the effects of a supine sacroiliac orthopedic blocking procedure on cervical spine extensor isometric strength. J Chirop Med. 2009;8(2):56-61.
- Gilliatt RW, Wilson TG. A pneumatic–tourniquet test in the carpal—tunnel syndrome. Lancet. 1953;265(6786):595-597.
- Gilliatt R., Wilson TG. Ischaemic sensory loss in patients with peripheral nerve lesions. J Neurol Neurosurg Psychiatry. 1954;17(2):104-114.
- Goodheart GJ, Jr. The carpal tunnel syndrome. Chiro Econ. 1967;10(1).
- Goodheart GJ, Jr. Tarsal tunnel syndrome. Chiro Econ. 1971;13(5).
- Goodheart GJ, Jr. Sacroiliac and ilio sacral problems, Part 2. Chiro Econ. 1972;15(1).
- Goodheart GJ, Jr. Applied Kinesiology Research Manuals. Applied Kinesiology Systems, Inc: Grosse Point Farms, MI; 1964-1998.
- Goodman CE. Unusual nerve injuries in recreational activities. Am J Sports Med. 1983;11(4):224-227.
- Granit R, Skoglund CR. Facilitation, inhibition and depression at the ;artificial synapse' formed by the cut end of a mammalian nerve. J Physiol. 1945;103(4):435-48.
- Greenawalt MH. Feet and the dynamic science of chiropractic. Chiro Econ. 1988;30(6).
- Guyton AC, Hall JE. Textbook of Medical Physiology, 11th ed. WB Saunders Co: Philadelphia; 2005.
- Halasi T, Kynsburg A, Tállay A, Berkes I. Changes in joint position sense after surgically treated chronic lateral ankle instability. Br J Sports Med. 2005;39(11):818-24.
- Haldeman S, Meyer BJ. The effect of constriction on the conduction of the action potential in the sciatic nerve. S Afr Med J. 1970;44(31):903-906.
- Hammer W. Functional Soft-Tissue Examination and Treatment by Manual Methods, 3rd Ed. Jones and Bartlett Publishers, Inc.; 2007.
- Harms-Ringdahl K, Ed.. Muscle Strength. Edinburgh: Churchill Livingstone; 1993.
- Heidrich JM. Case history: Dupuytren's contracture and cervical disc. Collected Papers International College of Applied Kinesiology, 1994-1995;1:11-13.
- Heidrich JM. Atypical fibular head subluxation in a case of lower leg neuritis. Collected Papers International College of Applied Kinesiology, 1992-1993;1:73-74.
- Helm PA, Nepomuceno C, Crane CR. Tibial nerve dysfunction during pregnancy. South Med J. 1971;64(12):1493-4.
- Hess K, et al. Acute ischaemic neuropathy in the rabbit. J Neurol Sci. 1979;44(1):19-43
- Hides JA, Stokes MJ, Saide M, et al. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 1994;19(2):165-172.
- Higashi Y, Yoshizumi M. New methods to evaluate endothelial function: method for assessing endothelial function in humans using a strain-gauge plethysmography: nitric oxide-dependent and -independent vasodilation. J Pharmacol Sci. 2003;93(4):399-404.
- Hirasawa Y, Sakaida K. Sports and peripheral nerve injury. Am J Sports Med. 1983;11(6):420-426.
- Hogg JDW. The efficacy of applied kinesiology protocols in correcting peripheral nerve entrapment associated with carpal tunnel syndrome: An inter-examiner study. Collected Papers International College of Applied Kinesiology. 1994-1995;1:169-173.
- Hong C-Z, Simons DG. Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. J Musculoskeletal Pain. 1993;1(1):89-131.
- Hoppenfeld S. Physical Examination of the Spine and Extremities. Appleton–Century–Crofts: New York; 1976.

- Hurst LC, Weissberg D, Carroll RE. The relationship of the double crush to carpal tunnel syndrome (an analysis of 1,000 cases of carpal tunnel synrome). J Hand Surg. 1985;10(2):202-204.
- Ian'shina EN, Liubchenko PN, Ian'shin NP, Kasatkina LF, Samoĭlov MI. Multiple local involvement of peripheral nerves in workers suffering from occupational hands disorders. Med Tr Prom Ekol. 2009;(2):24-8.
- ICAK USA and ICAK International websites. www.icakusa.com; www.icak.com.
- Idler RS. Persistence of symptoms after surgical release of compressive neuropathies and subsequent management. Orthop Clin North Am. 1996;27(2):409-16.
- Janda V: Muscle Function Testing. Butterworths: London; 1983.
- Johnson RH, Lambia DG, Spalding JMK. Neurocardiology: The interrelationship between dysfunction in the nervous and cardiovascular systems. WB Saunders: London; 1984.
- Juhan D. Job's Body. Station Hill Press: New York; 1987.
- Jull G, Sterling M, Falla D, Treleaven J, O'Leary S. Whiplash, Headache, and Neck Pain. Ch. 4: Alterations in Cervical Muscle Function in Neck Pain. Churchill Livingstone: Edinburgh; 2008:41-58.
- Kendall FP, McCreary EK, Provance PG, Rodgers MM, Romani WA. Muscles: Testing and Function, with Posture and Pain. Williams & Wilkins: Baltimore; 2005.
- Kharrazian D. The role of dermal proprioceptors in reactive muscle patterns. Collected Papers International College of Applied Kinesiology, 2001-2002;1:129-130.
- Kharrazian D. The role of the transverse ligament in suprascapular nerve entrapments. Collected Papers International College of Applied Kinesiology, 2000-2001;1:81-83.
- Kline D, Hudson A. Kline and Hudson's Nerve Injuries: Operative Results for Major Nerve Injuries, Entrapments and Tumors, 2nd Ed. Saunders; 2007.
- Kobesova A. Twenty-year-old pathogenic "active" postsurgical scar: a case study of a patient with persistent right lower quadrant pain. J Manip Physiol Ther. 2007;30(3):234-238.
- Kolosova LI, Nozdrachev AD, Moiseeva AB, Ryabchikova OV, Turchaninova LN. Recovery of mechanoreception at the initial stage of regeneration of injured sciatic nerve in rats in conditions of central axotomy of sensory neurons. Neurosci Behav Physiol. 2004;34(8):817-20.
- Kopell HP, Thompson WAL. Peripheral Entrapment Neuropathies, 2nd Ed. Robert E. Krieger Pub Co: Huntington, NY; 1976.
- Kopell HP. Lower extremity lesions. In: Management of Peripheral Nerve Problems. Eds. Omer GE, Spinner M. W.B. Saunders Co: Philadelphia; 1980.
- Korr IM. The spinal cord as organizer of disease processes: Some preliminary perspectives. J Am Osteopath Assoc. 1976;76(1):35-45.
- Korting GW, Denk R. Differential Diagnosis in Dermatology. WB Saunders Co.: Philadelphia; 1976.
- Langer P. Great Feet for Life: Footcare and Footwear for Healthy Aging. Fairview Press: Minneapolis, MN; 2007.
- Leaf D. Applied Kinesiology Flowchart Manual, 4th Ed. Privately Published: David W. Leaf: Plymouth, MA; 2010.
- Lee D. The Pelvic Girdle: An approach to the examination and treatment of the lumbopelvic-hip region. Churchill Livingstone: Edinburgh; 2004.
- Leinonen V, Kankaanpää M, Luukkonen M, Kansanen M, Hänninen O, Airaksinen O, Taimela S. Lumbar paraspinal muscle function, perception of lumbar position, and postural control in disc herniation-related back pain. Spine. 2003;28(8):842-8.
- Lewit K: Manipulative Therapy in Rehabilitation of the Locomotor System, 3rd ed. London: Butterworths; 1999.
- Liebenson C. Ed: Rehabilitation of the Spine: A Practitioner's Manual, 2nd ed. Lippincott, Williams & Wilkins: Philadelphia; 2007.
- Loeser JD. Pain due to nerve injury. Spine. 1985;10(3):232-235.
- Logan AL. The Foot and Ankle: Clinical Applications. Gaithersburg, MD: Aspen Publishers, Inc; 1995.
- Logan AL. The Knee: Clinical Applications. Gaithersburg, MD: Aspen Publishers, Inc; 1994.
- Lombardini R, Marchesi S, Collebrusco L, Vaudo G, Pasqualini L, Ciuffetti G, Brozzetti M, Lupattelli G, Mannarino E. The use of osteopathic manipulative treatment as adjuvant therapy in patients with peripheral arterial disease. Man Ther. 2009;14(4):439-43.
- Lundborg G. Nerve injury and repair. Churchill Livingstone: Edinburgh; 1988.
- Lundborg G. Intraneural microcirculation. Orthop Clin North Am. 1988;19(1):1-12
- Lundborg G. Structure and function of the intraneural microvessels as related to trauma, edema formation, and nerve function. J Bone Joint Surg Am. 1975;57(7):938-948..
- Lusskin R. Peripheral neuropathies affecting the foot: Traumatic, ischemic, and compressive disorders. In: Disorders of the Foot, Vol 2. Ed. Jahss MH. W.B. Saunders Co: Philadelphia; 1982.
- Luttges MW, Stodieck LS, Beel JA. Postinjury changes in the biomechanics of nerves and roots in mice. J Manip Physiol Ther. 1986;9(2):89-98.
- Maffetone P. Fix Your Feet. The Lyons Press: Guilford, CT; 2003.
- Maffetone P. Complementary Sports Medicine: Balancing traditional and nontraditional treatments. Human Kinetics: Champaign, IL; 1999.
- Maitland G. Maitland's vertebral manipulation, 6th Ed. Butterworth-Heinemann: Oxford; 2001.
- Marmor L. Bowler's thumb. J Trauma. 1966;6(2):282-4.
- McGrath MA, Verhaeghe RH, Shepherd JT. The physiology of limb blood flow. In: Peripheral Vascular Diseases, 5th ed. Eds. Juergens J, Spittel JA, Fairbairn J, II. W.B. Saunders Co: Philadelphia; 1980.
- Mc Knight ME, DeBoer KF. Preliminary study of blood pressure changes in normotensive subjects undergoing chiropractic care. J Manip Physiol Ther. 1988;11(4):261-266.
- Mense S, Simons DG. Muscle Pain: Understanding Its Nature, Diagnosis, and Treatment. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:131-157.
- Moberg E. Objective methods for determining the functional value of sensibility in the hand. J Bone Joint Surg. 1958;40B(3):454-76.
- Moller AR. The cranial nerve vascular compression syndrome. Rev Treat Acta Neurochir. 1991;113(1-2):18-23.
- Moncayo R, Moncayo H. Evaluation of Applied Kinesiology meridian techniques by means of surface electromyography (sEMG): demonstration of the regulatory influence of antique acupuncture points. Chin Med. 2009;4:9.
- Mondelli M, de Stefano R, Rossi S, Aretini A, Romano C. Sympathetic skin response in primary Raynaud's phenomenon. Clin Auton Res. 2009;19(6):355-62.
- Mooney V, Pozos R, Vleeming A, Gulick J, Swenski D. Exercise treatment for sacroiliac pain. Orthopedics. 2001;24(1):29-32.
- Muir JM, Vernon H. Complex regional pain syndrome and chiropractic. J Manipulative Physiol Ther. 2000;23(7):490-7.
- Murphy EL. Observations on craft palsies. Trans Ass Industr Med Offrs. 1955;5/1.
- Myers JB, Lephart SM. Sensorimotor deficits contributing to glenohumeral instability. Clin Orthop. 2002;400:98-104.
- Myers TW. Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists. Churchill-Livingstone; 2001.
- Myers R, Powell H. Endoneurial fluid pressure in peripheral neuropathies. In: Hargens A (ed). Tissue fluid pressure and composition. Williams & Wilkins, Baltimore;1981.

- National Research Council, Institute of Medicine. Musculoskeletal Disorders and the Workplace. National Academy Press: Washington, DC; 2001.
- Neary D, Ochoa J, Gilliatt RW. Sub—clinical entrapment neuropathy in man. J Neurol Sci. 1975;24(3):283-298.
- Nicholas JA, Strizak AM, Veras G. A study of thigh muscle weakness in different pathological states of the lower extremity. Am J Sports Med. 1976;4(6):241-248.
- Nitz AJ, Dobner JJ. Upper extremity tourniquet effects in carpal tunnel release. J Hand Surg Am. 1989;14(3):499-504.
- Ochoa J. Nerve fiber pathology in acute and chronic compression. In: Management of Peripheral Nerve Problems. Ed. Omer GE, Spinner M. W.B. Saunders Co: Philadelphia; 1980.
- O'Riain S. New and simple test of nerve function in hand. Br Med J. 1973;3(5881):615-616.
- Page P, Frank CC, Lardner R. Assessment and treatment of muscle imbalance: The Janda Approach. Human Kinetics: Champaign, IL; 2010.
- Passatore M, Grassi C, Filippi GM. Sympathetically-induced development of tension in jaw muscles: the possible contraction of intrafusal muscle fibres. Pflugers Arch. 1985;405(4):297-304.
- Patten J. Neurological Differential Diagnosis, 2nd Ed. Springer-Verlag: Berlin; 1995.
- Phalen GS. The carpal–tunnel syndrome — clinical evaluation of 598 hands. Clin Orthop Relat Res. 1972;83:29-40.
- Pham K, Gupta R. Understanding the mechanisms of entrapment neuropathies. Review article. Neurosurg. Focus. 2009;26(2):E7.
- Punnett L, et al. Soft tissue disorders in the upper limbs of female garment workers. Scand J Work Environ Health. 1985;11(6):417-425.
- Punnett L, Keyserling WM. Exposure to ergonomic stressors in the garment industry: Application and critique of job–site work analysis methods. Ergonomics. 1987;30(7):1099-116
- Racinais S, Bringard A, Puchaux K, Noakes TD, Perrey S. Modulation in voluntary neural drive in relation to muscle soreness. Eur J Appl Physiol. 2008;102(4):439-46.
- Ramsak I. AK diagnosis and nosode therapy. Medical Journal for Applied Kinesiology. 1997;1:12-14.
- Retzlaff EW, et al. The piriformis muscle syndrome. J Am Osteopath Assoc.. 1974;73(10):799-807.
- Ritts GD, Wood MB, Linscheid RL. Radial tunnel syndrome — a ten–year surgical experience. Clin Orthop. 1987;219:201-205.
- Rob CG, Standeven A. Arterial occlusion complicating thoracic outlet compression syndrome. Br Med J. 1958;2(5098):709-712.
- Robinson DR. Pyriformis syndrome in relation to sciatic pain. Am J Surg. 1947;73(3):355-358.
- Russell SM. Examination of Peripheral Nerve Injuries: An Anatomical Approach. Thieme Medical Publishers, Inc.: New York; 2006.
- Rydevik B, McLean WG, Sjöstrand J, Lundborg G. Blockage of axonal transport induced by acute, graded compression of the rabbit vagus nerve. J Neurol Neurosurg Psychiatry. 1980;43(8):690-8.
- Saal JA, et al. The pseudoradicular syndrome — lower extremity peripheral nerve entrapment masquerading as lumbar radiculopathy. Spine. 1988;13(8):926-930.
- Saratsiotis J, Myriokefalitakis E. Diagnosis and treatment of posterior interosseous nerve syndrome using soft tissue manipulation therapy: a case study. J Bodyw Mov Ther. 2010;14(4):397-402.
- Robertson C, Saratsiotis J. A review of compressive ulnar neuropathy at the elbow. J Manipulative Physiol Ther. 2005;28(5):345.
- Schmitt WH, Cuthbert SC. Common errors and clinical guidelines for manual muscle testing: The "arm test" and other inaccurate procedures. Chiropr Osteopat. 2008;16:16.
- Schmitt WH, Jr. Muscle testing as functional neurology differentiating functional upper motorneuron and functional lower motorneuron problems. 1986 Selected Papers of the International College of Applied Kinesiology. ICAK: Shawnee Mission, KS; 1986.
- Schmitt WH. But what if there's no water in the hose? Collected Papers International College of Applied Kinesiology. 1986;Winter:125-144.
- Selander D, Månsson LG, Karlsson L, Svanvik J. Adrenergic vasoconstriction in peripheral nerves of the rabbit. Anesthesiology. 1985;62(1):6-10.
- Sharma L. Proprioceptive impairment in knee osteoarthritis. Rheum Dis Clin N Am. 1999; 25(2):299-314.
- Simons D, Travell J, Simons L. Myofascial pain and dysfunction: the trigger point manual: Upper half of body. Vol. 1, 2nd Ed. Williams & Wilkins: Baltimore; 1998.
- Skaggs CD, Winchester BA, Vianin M, Prather H. A manual therapy and exercise approach to meralgia paresthetica in pregnancy: a case report. J Chiropr Med. 2006;5(3):92-6.
- Smith MW, Marcus PS, Wurtz LD. Orthopedic issues in pregnancy. Obstet Gynecol Surv. 2008;63(2):103-11.
- Spaans F. Occupational nerve lesions. In: Handbook of Clinical Neurology, Vol 7. Eds. Vinken PJ, Bruyn GW. American Elsevier Pub Co: New York; 1970.
- Spinner M, Spencer PS. Nerve compression lesions of the upper extermity — a clinical and experimental review. Clin Orthop. 1974;104.
- Staal A, van Gijn J, Spaans, F. Mononeuropathies: Examination, Diagnosis and Treatment. WB Saunders: London; 1999.
- Steiner C, Staubs C, Ganon M, Buhlinger C.Piriformis syndrome: pathogenesis, diagnosis, and treatment. J Am Osteopath Assoc. 1987;87(4):318-323.
- Sternschein MJ, et al. Causalgia. Arch Phys Med Rehabil. 1975;56(2):58-63.
- Stux G, Pomeranz B. Acupuncture: Textbook and Atlas. Springer-Verlag: Berlin; 1987.
- Sunderland S. Nerves and Nerve Injuries, 2nd Ed. Churchill Livingstone: Melbourne; 1978.
- Sunderland S. Traumatic injuries of peripheral nerves. I — Simple compression injuries of the radial nerve. Brain. 1945;68:243-99.
- Sutherland WG. Contributions of Thought: The Collected Writings of William Garner Sutherland, D.O. Rudra Press: Portland, OR; 1998.
- Swanson S, Macias LH, Smith AA. Treatment of bowler's neuroma with digital nerve translocation. Hand (NY). 2009;4(3):323-6.
- TePoorten BA. The piriformis muscle. JAOA. 1969;69(2):150-160.
- Toth C. Peripheral nerve injuries attributable to sport and recreation. Neurol Clin. 2008;26(1):89-113.
- Travell JG, Simons DG. Myofascial Pain and Dysfunction: The Trigger Point Manual: The Upper Extremities. Williams & Wilkins: Baltimore; 1983.
- Travell JG, Simons DG. Myofascial Pain and Dysfunction: The Trigger Point Manual: The Lower Extremities. Williams & Wilkins: Baltimore; 1992.
- Triano JJ, Luttges MW. Subtle, intermittent mechanical irritation of sciatic nerves in mice. J Manip Physiol Ther. 1980;3(2).
- Upton ARM, McComas AJ. The double crush in nerve–entrapment syndromes. Lancet. 2(7825):359-362;1973.
- Veves A, Giurini JM, LoGerfo FW. The Diabetic Foot: Medical and Surgical Management. Humana Press: Totowa, NJ; 2002.
- Walsh MJ, Polus BI. The frequency of positive common spinal clinical examination findings in a sample of premenstrual syndrome sufferers. J Manipulative Physiol Ther 1999;22(4):216-220.
- Walther DS. Applied Kinesiology, Volume I — Basic Procedures and Muscle Testing (Pueblo, CO: Systems DC, 1981).

- Walther DS. Upper extremity peripheral nerve entrapment. Selected Papers of the International College of Applied Kinesiology, ed W.H. Schmitt, Jr. ICAKUSA: Detroit, MI; 1982.
- Walther DS. Applied Kinesiology, Volume II — Head, Neck, and Jaw Pain and Dysfunction — The Stomatognathic System. Systems DC: Pueblo, CO; 1983.
- Walther DS. Applied Kinesiology — Synopsis, 2nd Ed. International College of Applied Kinesiology: Shawnee Mission, KS; 2000.
- Watanabe N, Polus B. A single mechanical impulse to the neck: Does it influence autonomic regulation of cardiovascular function? Chiropr J Aust. 2007;37(2):42-48.
- Werder SF. Managing polypharmacy: Walking the fine line between help and harm. Current Psychiatry. 2003;2(2):1.
- Wetz HH, Hentschel J, Drerup B, Kiesel L, Osada N, Veltmann U. Changes in shape and size of the foot during pregnancy. Orthopade. 2006;35(11):1124, 1126-30.
- Wilgis EFS. Observations on the effects of tourniquet ischemia. J Bone Joint Surg. 1971;53(7):1343-1346.
- Wilgis EFS. Special diagnostic studies. In: Management of Peripheral Nerve Problems. Eds. Omer GE, Spinner M. W.B. Saunders Co: Philadelphia; 1980.
- Wilkins RH, Brody IA. Tinel's sign. Arch Neurol. 1971;24(6).
- Wilson JL. Adrenal Fatigue: The 21st Century Stress Syndrome. Smart Publications; 2002.
- Wright HM, Sympathetic activity in facilitated segments: vasomotor studies. J Am Osteop Assoc. 1955;54(5):273-276.
- Wright HM. The origin and manifestations of local vasomotor disturbances and their clinical significance. J Am Osteop Assoc. 1956;56(4):217-224.
- Zhao C, Ettema AM, Berglund LJ, An KN, Amadio PC. Gliding resistance of flexor tendon associated with carpal tunnel pressure: a biomechanical cadaver study. J Orthop Res. 2011;29(1):58-61.