Textbook of Pain Electrodiagnosis

Pain Fiber Nerve Conduction Study - pf-NCS
Evidence Based Medicine
Second Edition

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2nd Edition - Pain Battle Continues

The pain epidemic raging on. In a May 31st **Los Angeles Times** article titled **Death Tied to Painkillers Rising in U.S.** it is reported that the trend continues. More deaths are being caused by prescription pain medication than by automobile accidents. This is bound to continue until enough physicians become aware that over 50% of pain patients incorrectly localize the source of their neck and back pain. These are the patients making up the majority of pain sufferers and, without effectively detecting the nerve root involved, they require pain relief. The pf-NCS offers a way to locate nerve root injury and to weed out malingerers and drug seekers.

Reports from the **National Institute of Health show that pain** causes 40% of patients to seek medical consultation. An **NBC News Special Report** recently reported that ***"Pain is now America's leading public health problem."*** The June 2007, Newsweek reported: ***"At any given time, 25% of the U.S. population suffers from pain"*** and went on to report that on average 43% of pain patients develop chronic symptoms, while 50% to 80% of spine surgeries result in no change or worse symptoms.

The failure to deal with neck and back pain is often blamed on treatment limitations and poor surgical techniques, but it has been well known that this is not the real problem. **The Massachusetts General Hospital Handbook of Pain Management**, 2nd Edition; Saunders (2002) states on page 382: ***"In most cases of neck and low back pain, the anatomic and pathologic diagnosis remains unclear." "In most cases,"*** that's over 50%. Page 380 it states: ***"History and physical examination have a limited role in the diagnosis of back and neck pain but are important in ruling out serious pathology."***

As for conventional electrodiagnostic tests, EMG/NCV, page 353 states: ***"Most neuropathic pain syndromes are mediated by small- diameter fibers*** (i.e. A-delta and C), ***which are not evaluated by these tests*** (i.e. EMG/NCV). The text **Neurology for the Non-Neurologist**, by Werner and Goetz, Lippincott; (2005) states on page 23: ***"EMG/NCV in neck, shoulder and low back pain in the absence of deficit (motor nerve related symptoms, such as weakness) is costly, time consuming and seldom benefits the patient."*** The 5th edition of **The Spine** Vol.1, Saunders; (2006) states on page 218: are mediated by small- diameter fibers (i.e. A-delta and C), ***"Whenever a patient, whose sole complaint is pain (affecting the limbs, neck or back), is referred for an electrodiagnostic exam (EMG/NCV), there is the expectation that there has been some concomitant damage to large nerve fibers that will register on the EDX exam. As is noted later, with chronic lesions this is usually an unrealized hope."*** In Saunders **Tumors of the Spine**, page 280 states: ***"Radiculopathy: Most muscles are innervated by more than one nerve root. Sensory fiber compromise alone is the most common clinical presentation of radiculopathy. Isolated motor dysfunction is the least common."***

What about imaging? Abnormal anatomy does not necessarily equate with abnormal

nerve function. The literature is clear that most normal subjects have anatomical changes that suggest pathology though none exists. **The New England Journal of Medicine**, Vol. 331, July 14, 1994; Page 69:73: ***"In 98 normal subjects 52% had disk bulging, 27% had protrusions and 38% had abnormality at more than one level."*** **Pain Medicine & Management**, McGraw Hill; (2005) Page 28: ***"Pain cannot be imaged."***

What Causes Misdirection: After I understood the mechanism causing over 50% of pain to be referred, the literature took on new meaning. The mechanism has actually been described in medical physiology textbooks for decades. However, absent a method to measure small fiber function there was no practical way to study this phenomenon. **Guyton & Hall Textbook of Medical Physiology** explains in editions going back several decades. Soon after nerve injury, the pain fibers that are very poor localizers (C-Type - Slow Pain fibers) up- regulate. Concerning this shift to poor localization the **Guyton & Hall Textbook of Medical Physiology** 12th Edition (2011) states (pages 584- 585): ***"It explains why so many patients have serious difficulty localizing the source of some types of chronic pain."*** *The patients really don't complain of having difficulty localizing the source of their pain, s/he simply localizes to the wrong nerve. The type of chronic pain happens to be the most common type - neck and low back pain.*

Pain Fiber Nerve Conduction Study (pf-NCS)

In dealing with sensory pathology the pain fiber nerve conduction study (pf-NCS) is the ultimate in evidence based medicine. The June 2012 issue of the most widely read pain journal, ***Practical Pain Management****, published* ***neurosurgeon, Peter Carney's****, report in which he found the* ***pf-NCS correctly changed treatment in 56% of pain patients, and changed the side of treatment in 8%****. Dr. Carney's findings support the decade long consensus of AASEM pf-NCS certified physicians, which has held that at least 50% of neck and back patients incorrectly localize the source of pain with many reporting the source to be on the side opposite from the injury.*

Is That Smoke I Smell?

To understand how the pf-NCS compares to conventional electrodiagnostic exams (EDX) the analogy of a smoke detector is a good fit. A smoke detector pin points the earliest sign that a room is on fire. Likewise, pf-NCS identifies a change in sensory function within hours after nerve injury. Applying this analogy to conventional EMG, we could say that EMG tells us when half the roof is burned away, since EMG requires a loss of at least 50% of the protective myelin that covers large fibers before it can begin to detect pathologic changes. Also, the rate of regeneration is quick in small fibers, since they are 50 to 100 times smaller. Rapid recover gives the pf-NCS the added utility of effectively monitoring treatments effectiveness.

Initially I did not grasp the clinical value of my invention. I felt sure pf-NCS would be useful for research, but of limited clinical application, since patients indirectly tell us which nerve is causing a problem. The patient indicates a pattern of somatosensory symptoms, and it is a straightforward process to match this pattern to the known pattern of nerve distributions. However, it soon became obvious that over 50% of neck and back patients are incorrectly localizing the source of pain. Instead they localize the source to healthy nerves - pain is referred.

A multicenter study, reprinted in this textbook, explains that as pain increases so too does the likelihood of the patient incorrectly localizing its source. We now know that sensory injury does not alter light touch (A-beta) fiber function, so pinwheel testing has limited diagnostic value. C-Type (Slow Pain) fibers are similarly of little diagnostic help, since they up-regulate after injury and actually cause the refer pain phenomenon. Only the small A-delta (Fast Pain) fibers consistently down-regulate. Because down-regulation occurs within hours following injury, A-delta fibers are the perfect marker to identify early or chronic sensory pathology.

In the early 1900s the first attempts to use electricity to measure small fiber sensitivity began. For five decades two basic factors were unknown. After the 1950s they were mistakenly not taken into account. As a result, by the mid-1970s researches came to the mistaken conclusion that small fiber sensitivity shifts so much, and so often, that measurements were impossible. I was fortunate in that my PhD work had dealt with these overlooked factors - skin chemistry and electrical impedance. Additionally, my experience with biofeedback gave me the knowledge to develop an effective examination protocol. The final touch was an analysis algorithm using the patient as his own control. Rather than comparing measurements with population averages, which at best yield a sensitivity of 67%, using the patient as his own control boosts sensitivity to as close as 100% as possible.

Once FDA clearances were granted, independent evaluation was needed. A chance meeting with Randall Cork, MD, PhD filled that need. Dr. Cork allowed me to blind test several patients at the ***Louisiana State University Pain Center*** *in Shreveport. Dr. Cork saw how the pf-NCS could identify nerve root pathology, which convinced him to undertake a study.*

Over the next 3 years Dr. Cork completed a study published in the peer-reviewed ***Internet Journal of Pain, Symptom Control and Palliative Care*** *(2002). This class I study (all examiners were blind to results of other tests) found pf-NCS statistical sensitivity to approach 100% in detecting nerve-root pathology. More studies followed, all supporting the superior diagnostic capability of pf-NCS.*

The next challenge was to present the pf-NCS to a skeptical profession, made all the more difficult due to a similar, but ineffective, poorly devised device that had been vigorously

marketed over the preceding decade. This resulted in a very poor impression of this type of technology. However, remarkably within three years early researchers, including Dr. Cork, founded the ***American Association of Sensory Electrodiagnostic Medicine*** *to sharing data. In a short time the AASEM gained national recognition when its meetings qualified for Continuing Medical Education (CME) credit. Soon it was the first medical organization certifying physicians in pf-NCS. To gain some idea of its acceptance, the present and past AASEM directors include physicians who are or were Directors of Pain Management at Kaiser, Johns Hopkins and Louisiana State University, as well as the Veteran's Administration.*

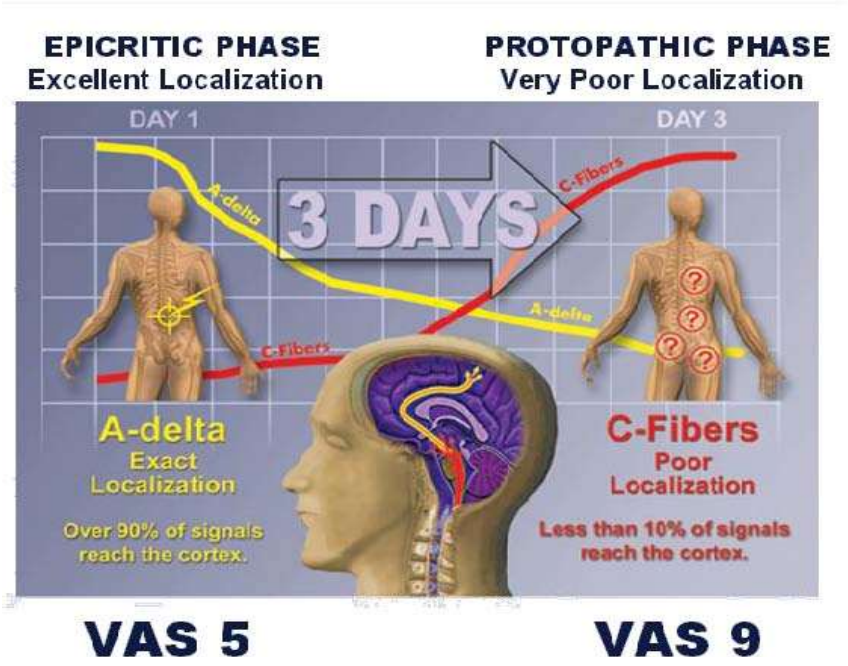
To be clear, pf-NCS will not replace conventional motor fiber EDX, physical examinations, MRI or the patient history. The pf-NCS furnishes the certainty that the involved sensory pathway has been identified or, just as important, the patient does not have a neurological component contributing to his sensory symptoms.

From a financial standpoint, the pf-NCS offers a huge savings. Early detection of pain pathology means fewer chronic pain cases. It also means conservative treatments can be instituted to avoid surgery. To put it simply, pf-NCS offers to improve the quality of our lives and reduce health care costs. After all, with 40% of patients suffering from pain this makes it the largest expenditure in medicine.

Many thanks go to my wife, friends and many colleagues who encouraged and contributed to the text. My sincere thanks, Jim.

Chapter I

Pain Physiology - Pain's Paradox



Smoke Detector vs. Roof Half Gone

In about 15 minutes a doctor or his nurse can perform the painless pf- NCS and detect the first stage of the early warning pain nerve fibers that signal injury with 100% peer-reviewed statistical accuracy.

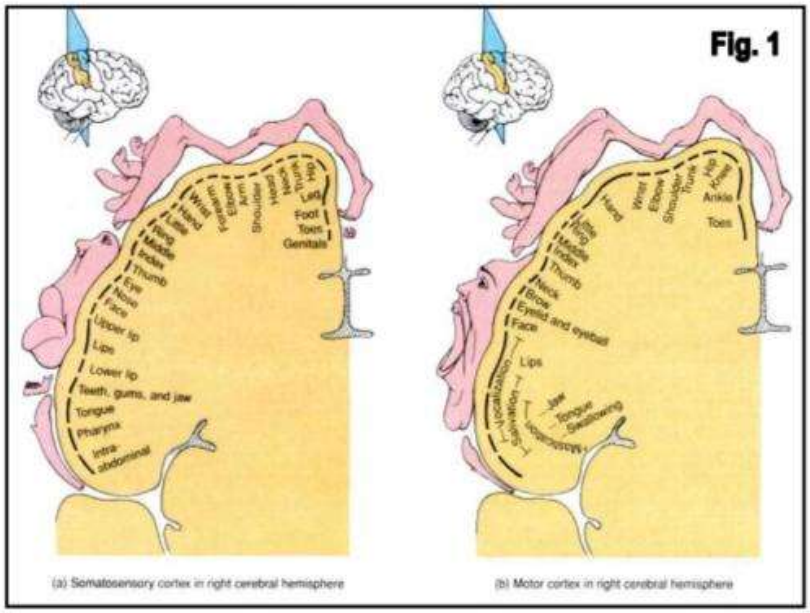
By the same analogy, EMG requires half the roof to be burned away before it can even begin to detect changes. Why? Because EMG cannot test early warning pain fibers, and it requires at least a minimum myelin loss of 50% before detectable changes occur.

Part I - Basic Anatomy & Physiology

This (Part I) reviews pertinent neurological anatomy and physiology. Part II focuses a deeper understanding of the physiology and the paradoxical physiology of pain with emphasis on the most common types of chronic pain — radiculopathic and peripheral entrapment pain. The reader learns how pain's physiological paradox confuses the patient and clinician in over 50% of the cases, and how this paradox is used to in localizing the injured nerve. **The Massachusetts General Hospital Handbook of Pain Management, 2nd Ed., Saunders, 2002,** Pg. 382: ***"In most cases (over 50%) of neck and low back pain, the anatomic and pathologic diagnosis remains unclear."***

Motor Bias: Though pain pathology is far more common than motor disorders, a bias exists towards studying motor physiology, which is understandable since until the development of the pf-NCS there was no practical method to quantify pain fiber function. This newfound ability is allowing a shift to the sensory system. What is of major importance is that the motor and sensory systems are functionally integrated. In fact pain is reflected in the motor system. For example, in an area of pain the dermal tissue is cooler than the surrounding non-painful tissue, due to localized vasospasm that occur concomitant to pain. This cooling is easily demonstrated by thermography.

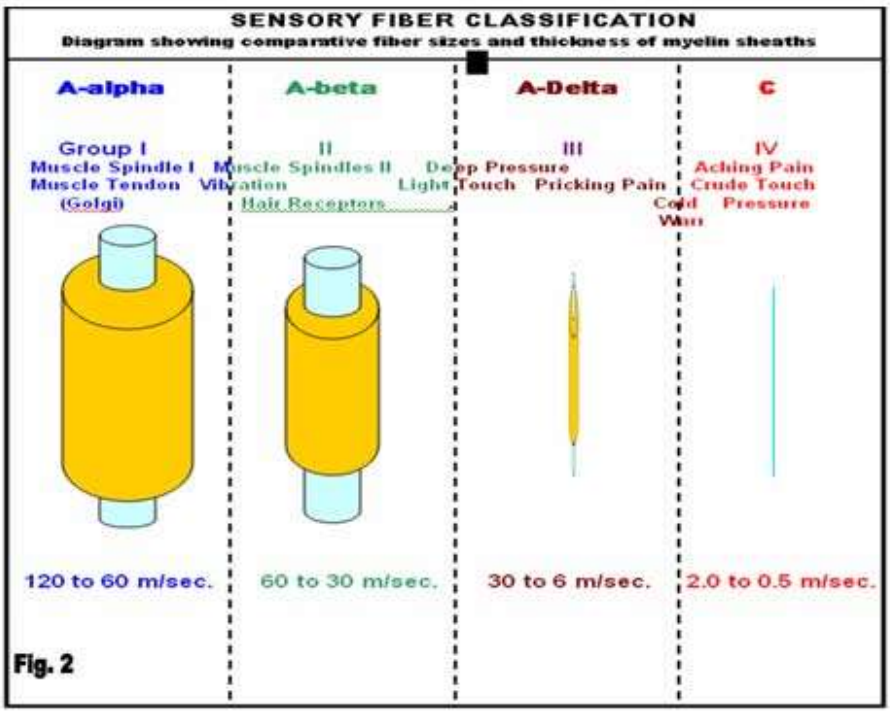
The integral relation of sensory and motor systems is significant and multi-faceted. For one, sensory information is necessary to inform the motor system so it can carry out locomotion. Sensory information is also necessary for other diverse functions, such as glandular secretions and immune responses. For example, the pituitary gland is recognized as the master endocrine gland, but the posterior half of the pituitary is nervous tissue - neurohypophysis, an extension of the thalamus. Thalamic neuro-secretions signal the neurohypophysis to send secretions to the adenohypophysis, which in turn secrets hormones, altering the function of other glands. To put it simply, every organ and gland must be monitored by the sensory system, which collects information necessary to complete the cycle of activation.[i]

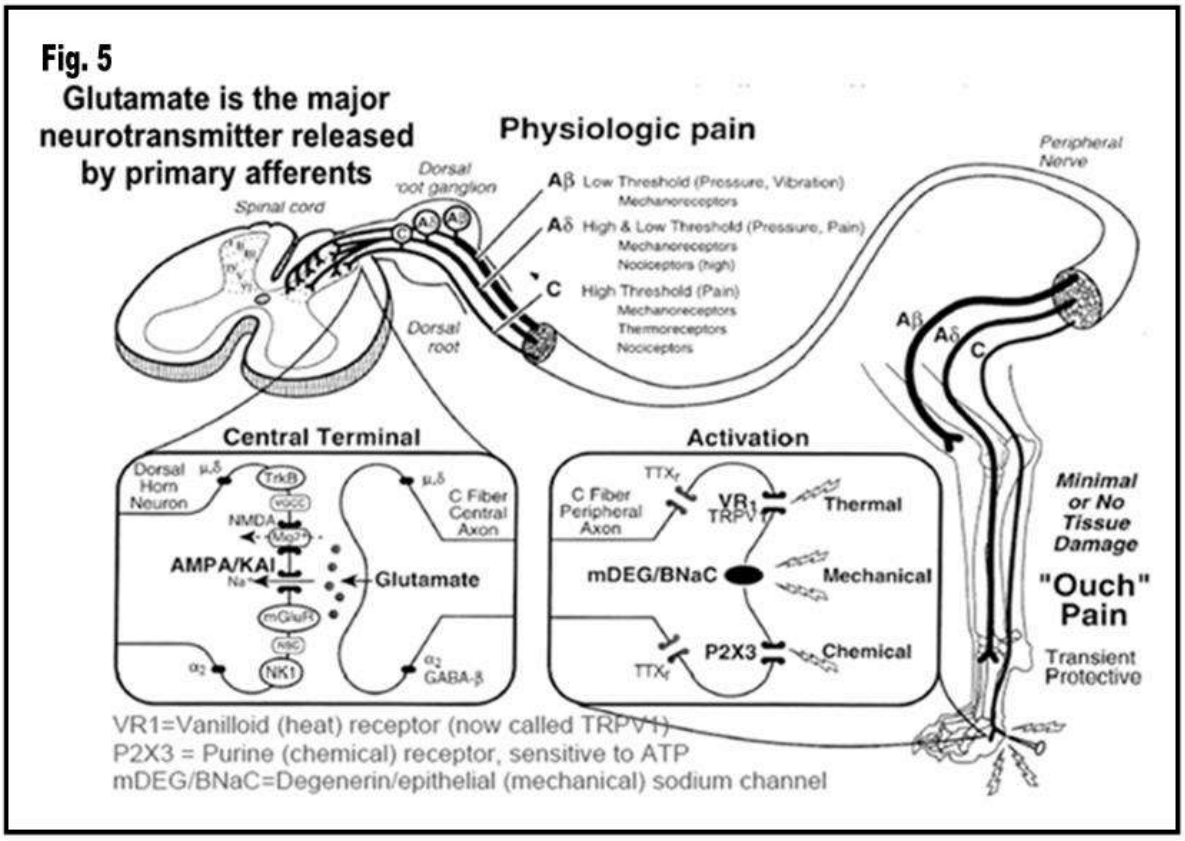


Somatosensory Cortex: A close look at the sensory homunculus (Fig. #1) clearly shows that very little neural circuitry is dedicated to processing signals from the spine and extremities. The lips, tongue, arms, legs and fingers (with the exception of the little finger) have more of the cortex devoted to each of them than the entire spine from the base of the skull to the tip of the coccyx. Only the little finger and foot have less cerebral cortex devoted to them than the spine. This meager amount of neuronal apparatus makes localization difficult, but as will be explained in Part II, there is even more reason for confusion caused by the paradoxical mechanism of pain.

Nerve Fiber Types: The major efferent and afferent fibers can be described on the basis of fiber **size**, fiber organization, **conduction speed**, and **destination**. Fig. #2 shows the relationships between the motor and sensory fibers, as to size, speed, and function.

Nerve fibers are also classified by systems and pathways. The three classifications are the spinothalamic system, the medial leminiscal system, and the spinocerebellar system. Of these three, the afferent spinothalamic system, which concerns transmission of pain, will be discussed here.





Biochemistry of Pain: This diagram shows pain's basic biochemistry.

It should be noted that conventional pain scenarios start with pain provoked by receptor stimulation. Injury involving a nerve-root or major nerve tract is different from pain induced by receptors. The pf- NCS has shown that the second phase of pain, the **Protopathic Phase**, which will be discussed in detail later in this chapter, is prolonged in chronic nerve-root and major peripheral nerve injury. This prolongation of the **Protopathic Phase** is evidenced by continued inhibition of A-delta fiber sensitivity.

Three Primary Sensory Functions: Compared to the efferent motor system, the afferent sensory system is as, or more, complex. Sensory functions can be divided into three primary functions and one special function.

1. Exteroception: Includes perception of outside stimuli such as light, chemical (i.e., caustic or acid), temperature, and sound.

2. Interoception: Includes perception of stimuli generated within the body such as oxygen concentration, gastric juice and blood pressure. Interoception is vital to maintaining homeostasis, vegetative functions, and hormonal regulation.

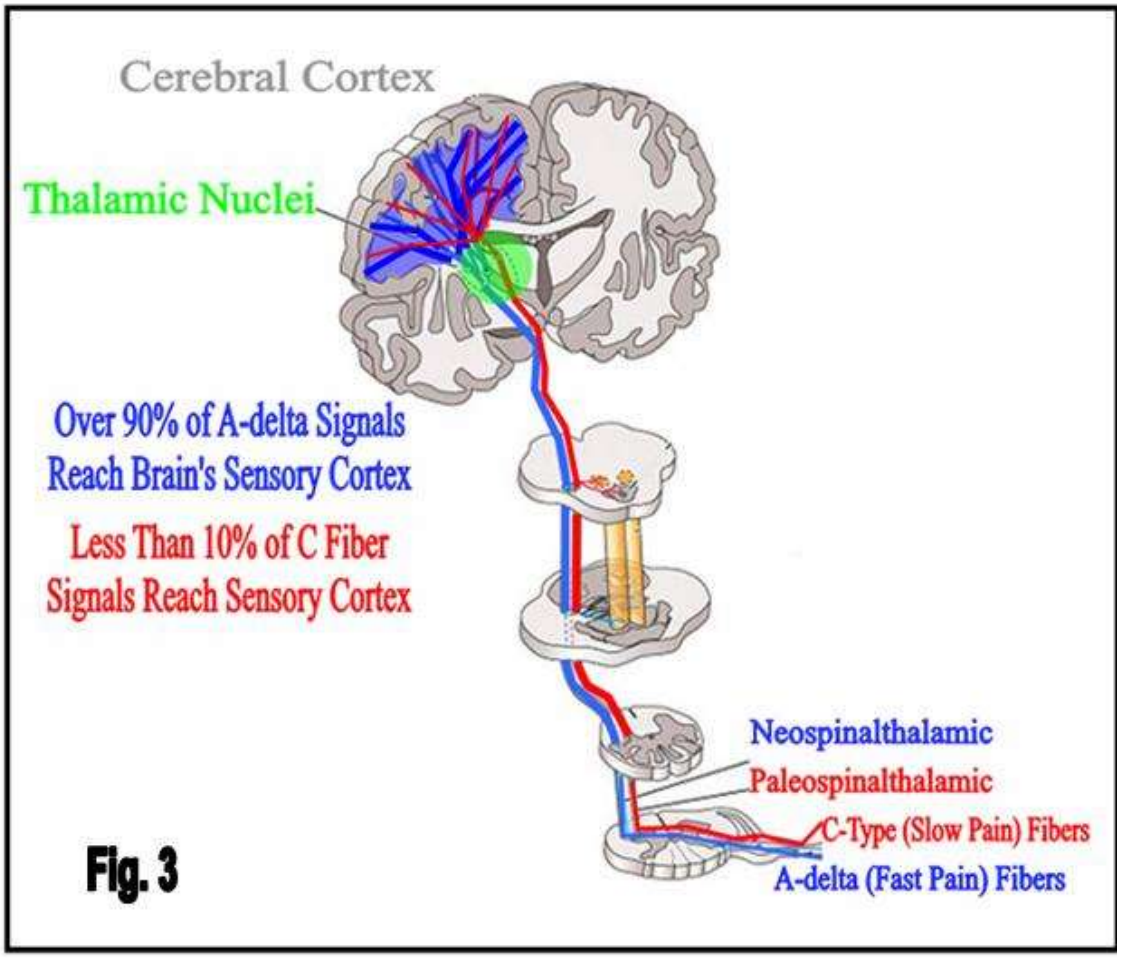
3. Proprioception: Proprioception orients the body externally in space and internally between anatomical structures. The external orientation involves input from pressure and stretch receptors, as well as sites such as the vestibular apparatus, eyes, and soles of the feet.

Internal Proprioception: Vertebral coupled motion is an example of internal proprioception. Spinal cord centers control the small muscles responsible for twisting and tilting each vertebra to accommodate gross body movements. Sensory proprioceptive feedback from the muscles, tendons, ligaments and joint capsules is necessary for these centers to effectively coordinate motor function. In PART II of this chapter, coupled motion of the vertebral segments will be discussed as it pertains to diagnosing radiculopathy.

Afferent Pathways: Each afferent pathway, from receptor to proximal central nervous system (CNS) termination point, consists of two or more neurons which are labeled in ascending order: Primary (first order) neuron, Secondary (second order) neuron, and Tertiary (third order) neuron. Additionally, there are many collaterals branching off at various levels throughout the spinal cord and the brainstem.

The majority of primary neurons synapse with secondary neurons on the same side and level where they enter the spinal cord. The next greatest number of primary neurons, cross over to the opposite side to synapse with the secondary neurons before ascending. The third set ascends one level on the same side before synapsing on the same side. Finally, there are those ascending and crossing over before synapsing with secondary neurons. Also mixed into this network are interneurons and the tertiary neurons. Though most authorities describe primary neurons as also descending 2-5 levels (spinal segments), based on results of thousands of A-delta pf-NCS examinations, it seems there are very few primary A-delta fiber neurons which descend. This will be discussed in Chapter V, which deals with the analysis and interpretation.

Paleospinalthalamic Pathway - Slow Pain: As humans evolved the slow pain C fibers dominated in forming the paleospinalthalamic pathway. This primitive pathway signals that something is wrong but, due to its poor connectivity with the later evolved cerebral cortex, C fibers cannot accurately localize the source of pain. See Fig. #3



Neospinalthalamic Pathway - Fast Pain: The more recently evolved division is dominated by A-delta fibers which developed along with the cerebral cortex. The A-delta fibers have excellent connectivity that enable localization of injury and to quickly move the body away from the source of injury, during what is termed the Epicritic Phase. Next is the Protopathic Phase, in which the A-delta fibers down-regulate leaving the primitive C fibers to signal until the damage is repaired.[iii] These phases are at the heart of the paradox that will be explored in Part II of this chapter.

Ion channels: Nerves, including sensory nerves, have ion channels embedded in their membranes. Ion channels are a basic component of all living things, including single-celled protozoa, bacteria, and yeast. The most ancient were probably mechanically-gated channels sensitive< to stretch, followed by the more complex voltage-gated potassium (K) channels. The Escherichia coli bacterium, for example, is transitional in that it has both stretch and voltage-gated channels. Voltage-gated channels are also found in the plasma membrane of some types of plants, such as the Venus Fly-Trap.[iv] The sodium (Na+) channels originated in true animals (Metazoa) during the Cambrian explosion: 550 million years ago.

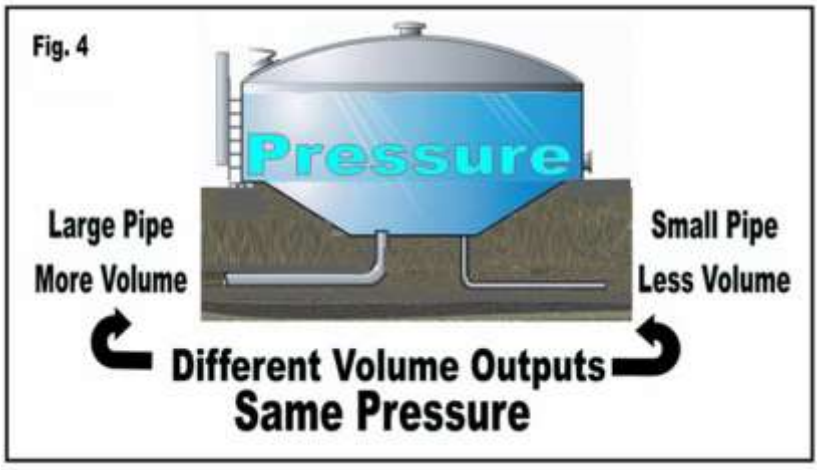
Voltage-Gated Channels: The action potential (nerve impulse) is generated by the Voltage-Gated Sodium and Potassium Channels. Keep in mind that potential and voltage are synonymous terms.

Two Components of Electricity: There are two basic components to an electrical signal: 1) Pressure/voltage/potential; and 2) Volume/current output. Of these two, only the voltage causes the action potential (nerve impulse).

Water is a good analogy for electricity. Both have volume (current output) and pressure (voltage). The pressure in a tank of water is determined by the depth of the water. In the diagram the left pipe is larger than the right so, per unit of time, a greater volume of water passes through the larger pipe than through the smaller pipe; the volume of flow is different but the pressure is the same in both pipes. The smaller pipe has greater resistance than the larger one.

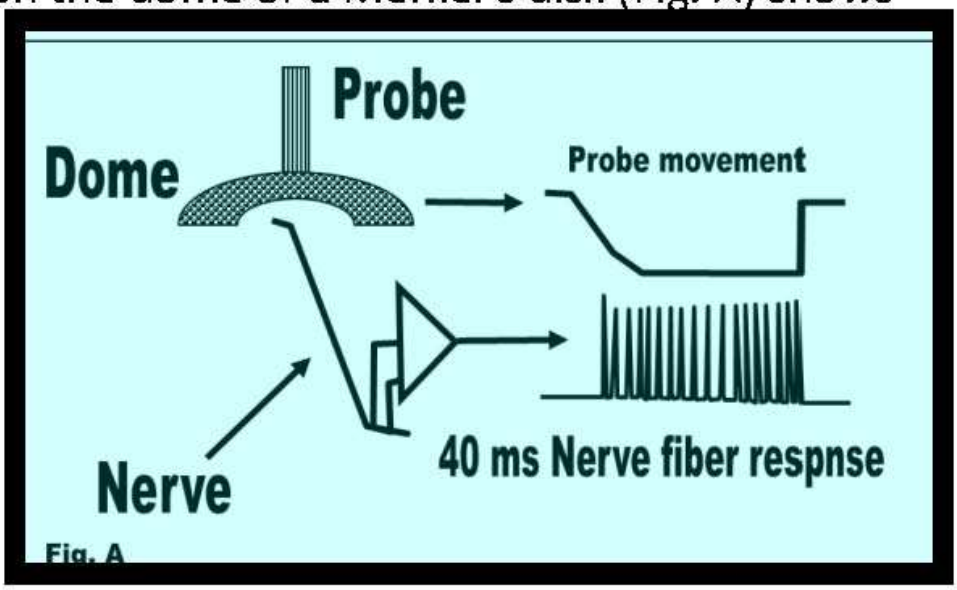
Electricity works in the same way. The volume of energy passing through the body changes because the skin's resistance constantly changes. This is analogous to the water pipe changing size; as it changes, so too does the volume of water exiting the pipe. What is important is that the pressure (voltage) is constant and unaffected by the skin's changing resistance (impedance).

The vast majority of sensory injuries do not involve receptors. The injury is to the peripheral nerve tract, which includes spinal nerve root injuries - the most common type of sensory injury. The pf-NCS was developed based on data gleaned in part from previous research findings, such as the discovery that certain stimuli frequencies selectively activate certain types of sensory nerve fibers. The key point is that no previous researcher was able to make reasonably accurate sensory functional measurement, and without this there was nothing to build on or refine. One cannot improve accuracy unless one has a technique that has some degree of accuracy to begin with.

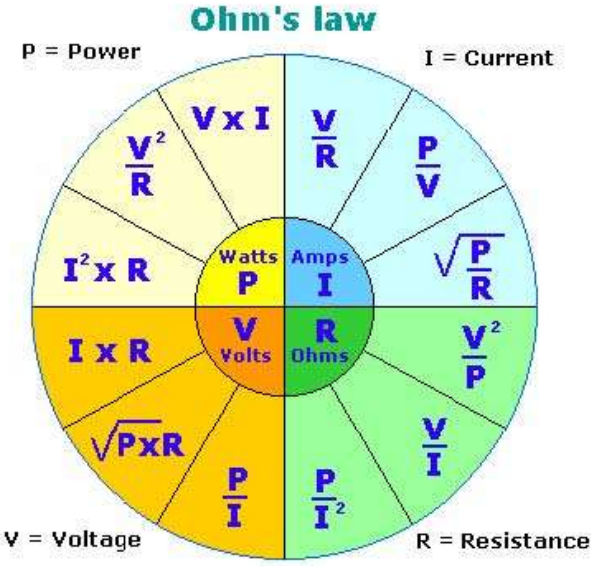


An Interesting Observation is that the nerve response caused by pressure (A-delta fibers) on the dome of a Merkel's disk (Fig. A) shows 18 spikes in 40 ms, or 350 Hz (spikes per second), the upper range of frequency attributed to the A- delta fibers (150 Hz to 350 Hz). The mid point of this range is employed in pf-NCS technology.

) on the dome of a Merkel's disk (Fig. A) shows



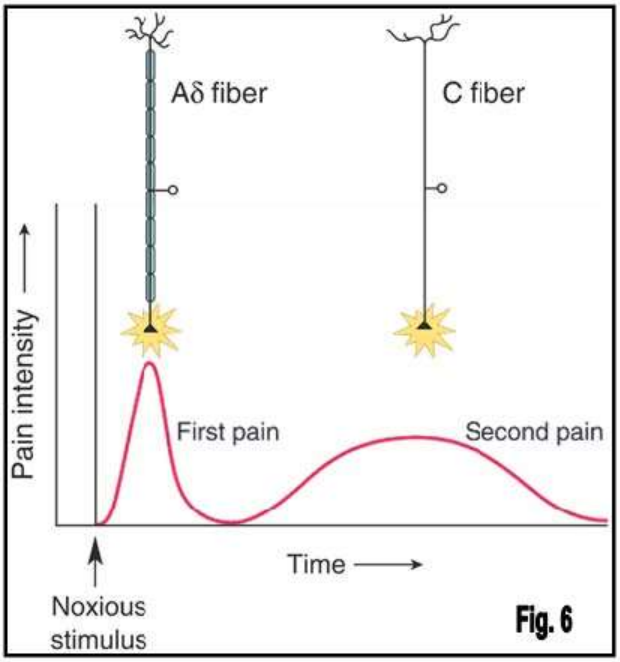
Measuring Normal Sensory Action Potential: In developing pf-NCS technology these discoveries were taken into account and what seemed to have been overlooked was corrected. Then, instead of using the prototype to look at basic physiological questions to determine, for example, if the stimulus was really selectively firing any type of specific fibers, it was used to test patients with well-defined nerve root injuries. The idea being to see if the data associated with the suspected injury differed from data of normal nerve roots. By the time 75 patients had been tested it was quite obvious that the A-delta fibers of an injured nerve root always down-regulate.



PART II - Pathological Physiology

Generally, most texts dealing with pain focus on the role of C-type (Slow Pain) fibers, and essentially ignore A-delta (Fast Pain) fibers. Here the focus will be switched, since the complex A-delta fibers have been found to be the key to diagnosing radiculopathic pain, as well as many other entrapments and sensory disorders.

Withdrawal Reflex: The withdrawal reflex is a good starting point for developing an understanding of the complex pathophysiology of pain, and to understanding the part A-delta fibers play. The first recognized historical reference to the withdrawal reflex was the 1664 **Treatise of Man** by **Rene Descartes**. Descartes attempted to explain how the body withdraws from pain by describing how particles of heat act on a spot of skin attached by threads to a valve in the brain. Descartes envisioned that the threads pulled open a valve releasing "animal spirits" which flow to the muscles, in turn causing the head to turn toward the affected body part and the body to move away from the source of injury.



Descartes' threads to the brain are the A-delta fast pain fibers. However, as the signal is traveling along these "threads" to the brain, at around 15 mph, some A-delta neurons synapse with motor neurons in the anterior (ventral) horns of the spinal cord, causing the body to pull away from the source of pain a few milliseconds before the pain signal reaches the cognitive centers of the brain. This is called the **Epicritic Phase.** The **Epicritic Phase** is quickly followed by the **Protopathic Phase** in which the A-delta fibers that almost exactly localize the source of pain, down-regulate. This down-regulation allows the withdrawal muscles to return to normal function.[v-vi]
As the A-delta fibers down-regulate the poor localizing C fibers up-regulate to keep the body aware of the injury. Regarding the shift from excellent to poor localization, ***Guyton & Hall Textbook of Medical Physiology*** states the following: ***"It explains why so many patients have serious difficulty localizing the source of some types of chronic pain."*** The ***"types of chronic pain"*** to which Guyton refers are the most common types — neck and back pain. As for ***"many patients have serious difficulty localizing the source",*** it is rare for a patient to report he is having difficulty localizing the source of his pain, rather, he simply localizes pain to the wrong nerve-root.

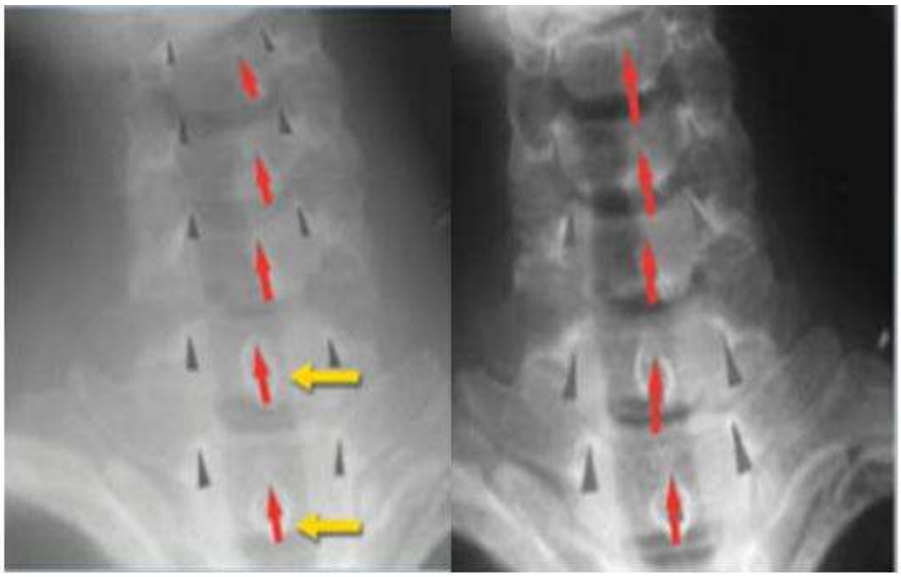
Pain's Paradoxical Physiology: The more severe pain becomes the poorer its localization. In 2010, the AASEM sponsored a retrospective multi-center study of painful cervical and lumbosacral radiculopathy cases, in which the relationship between A-delta fiber sensitivity was compared with the subjective **Visual Analog Scale (VAS)**. In these 83 pre and post-treatment studies, an inverse relationship between A-delta fiber sensitivity and the patient's subjective VAS pain rating was found. This means that as pain decreases, A-delta sensitivity increases. This suggests that the **Protopathic Phase** is prolonged in nerve-root and probably also in peripheral nerve injuries. The only way pain can increase while A-delta fiber function is decreasing is for the pain signals to be coming from C-type fibers. This paradoxical relationship shows that patients become more dependent on C-type fiber signals as pain increases, in turn increasing the probability of inaccurate localization of the source of pain.

Pain — Alarm System: The **Epicritic Phase** is not a constant ongoing process as are the sensory functions previously discussed. Essentially, it is an alarm that is helpful when needed. Continuation of the **Protopathic Phase** is pathologic. **Pathological Phase:** If an injury is minor, the **Epicritic Phase** turns off and the healing body goes about its business. If the injury is more severe, such as injury to a nerve-root or major peripheral nerve, then the **Protopathic Phase** begins. In this second phase, the A-delta fibers diminish in activity, which allows muscle contraction to subside. Simultaneously, the primitive C-type fibers of the paleospinalthalamic pathway begin signaling dull, aching pain. The body continues receiving dull pain sensations as long as the injury is unresolved. If the injury does not resolve for whatever reason, then the **Pathological Phase** begins as essentially the continuation of the **Protopathic Phase** with chronic changes added. In some cases, after it seems an injury should have resolved, painful symptoms persist into what is referred to as allodynia, sympathetic mediated pain, or reflex sympathetic dystrophy. This list may well

include fibromyalgia, because it is likely caused by an injury that has failed to resolve, and the pain has been referred to a nerve other than the injured nerve. There is a strong likelihood syndromes are associated with unrecognized ongoing radiculopathy or peripheral nerve entrapment. **Sensory Radiculopathy:** Until recently the role of small pain fibers in radiculopathy has not been well understood, in the absence of a practical method to measure small pain fiber function. Now we can say that the term **sensory radiculopathy** may be a bit of a misnomer given that all radiculopathies are essentially sensory, and are infrequently mixed (sensory and motor), and rarely pure motor.[vii] The pf-NCS has revealed a link between chronic prostatitis and lumbosacral radiculopathy. This and other discoveries will be discussed in depth later. For now, one particular discovery is at the heart of understanding the pf-NCS - paradoxical physiology of pain.

Radicular Proprioceptive Dyskinesia: A-delta fibers are unique in many ways with one being that they are the only sensory fibers whose primary neurons synapse with motor neurons in the spinal cord. As previously discussed, this connection automatically causes the body to withdraw from a source of pain. A-delta fibers are also unique in that they transmit multiple types of sensations including hot, cold, vibration, pricking, burning deep pressure and stretch. These last two - pressure and stretch - mean A-delta fibers are related to spinal segmental proprioception.

One of the first discoveries that was made following the development of the pf-NCS was that the spinous processes of the vertebra above and/or below a pathological nerve-root (as detected by the pf-NCS A-delta exam) rotate in reverse from the normal motion. Normally, when the spine is laterally bent, the spinous processes of the cervical spine rotate toward the contralateral side of head tilt (toward the convexity). In the lumbar spine, the lower spinous processes normally rotate toward the ipsilateral side (toward the concavity) - same side as lateral bending.



it the Physiologically, has been established that centers in spinal cord control the small intrinsic spinal muscles responsible for vertebral rotation (coupled motion). After consistently

seeing this phenomenon in dozens of patients it was concluded that concomitant to A-delta hypo-function there is a disruption of the proprioceptive signals coming from the adjacent vertebral joints, capsules, ligaments, and tendons. This syndrome is now known as **Reverse Vertebral Dyskinesia (RVD)**. I first described this phenomenon at the 2005 AASEM Annual Conference. Since then, literally hundreds of clinicians certified in the pf-NCS have confirmed the relationship between sensory A-delta radiculopathy and **RVD**.

A-delta Mechanoreceptivity: A-delta and C fibers are the smallest diameter (50 to 100 times smaller than motor fibers) and most numerous axons in peripheral nerve bundles. Rabbit studies using vital fluorescent dye to selectively visualize living free nerve endings in the corneal epithelium have revealed that these endings are organized. The C fiber endings terminate as short (< 50 microns) vertically directed processes clustered within the epithelium, while the A-delta fibers terminate as long (0.1 - 1.2 mm) horizontal processes running parallel to the epithelial surface. Only the A-delta fiber endings are mechanoreceptive, and the unique elongated structure imparts directional selectivity.[viii] These findings support that the A-delta fibers have a proprioceptive component. Certainly if the cornea of a rabbit has such an A-delta fiber arrangement, the same is likely true of human vertebral joints, capsules, ligaments, and tendons. **Referred Pain:** Medical texts insinuate that referred somatosensory pain occurs, but fail to expound on this issue. As an example, **Guyton & Hall's Textbook of Medical Physiology** in its 2nd Edition (1960), and in the newest 12th Edition (2011), nearly identical wording is used to describe referred pain. In the 12th Edition Guyton addresses referred pain on page 588. The wording is identical to the 1960 edition: **"Often a person feels pain in a part of the body that is fairly remote from the tissue causing the pain. This is called referred pain. For instance, pain in one of the visceral organs is often referred to an area on the body surface."** Nothing is said about somatosensory pain? The text proceeds to elaborate on visceral referred pain, such as excess gastric acid, hollow organ spasm, distention, organ diseases, sunburn, herpes and Tic Douloureux, but not a word is mentioned regarding referred nerve-root or peripheral nerve pain.

Receptor Generated Pain? Note that physiology texts consistently discuss pain generated by receptors, but injury most commonly involves the nerve-root and peripheral nerves and not the receptors.

Taking into consideration all the many sensations the sensory system processes, it is easy to see that pain only comprises a small percentage of the sensory circuitry. Pain is literally an initial flurry of reflex neural activity that moves the body away from the source of damage (withdrawal reflex), and is followed by slow pain signals, which only cease when the damage is repaired, or when a drug turns off the signal.

Painless Pathology: Numbing of A-delta fibers are often the only sign of nerve pathology/injury. A particularly memorable case is that of a physician who was found to have very severe bilateral L5 A-delta fiber hypo-function. She was incredulous because she

had no symptoms. She had experienced severe back pain about two years before. A day later she called and apologized for her skepticism. It seems that after her morning shower, while drying off, she had noticed that the tops of both her feet were completely numb, and was quite surprised she had not noticed this numbness before.

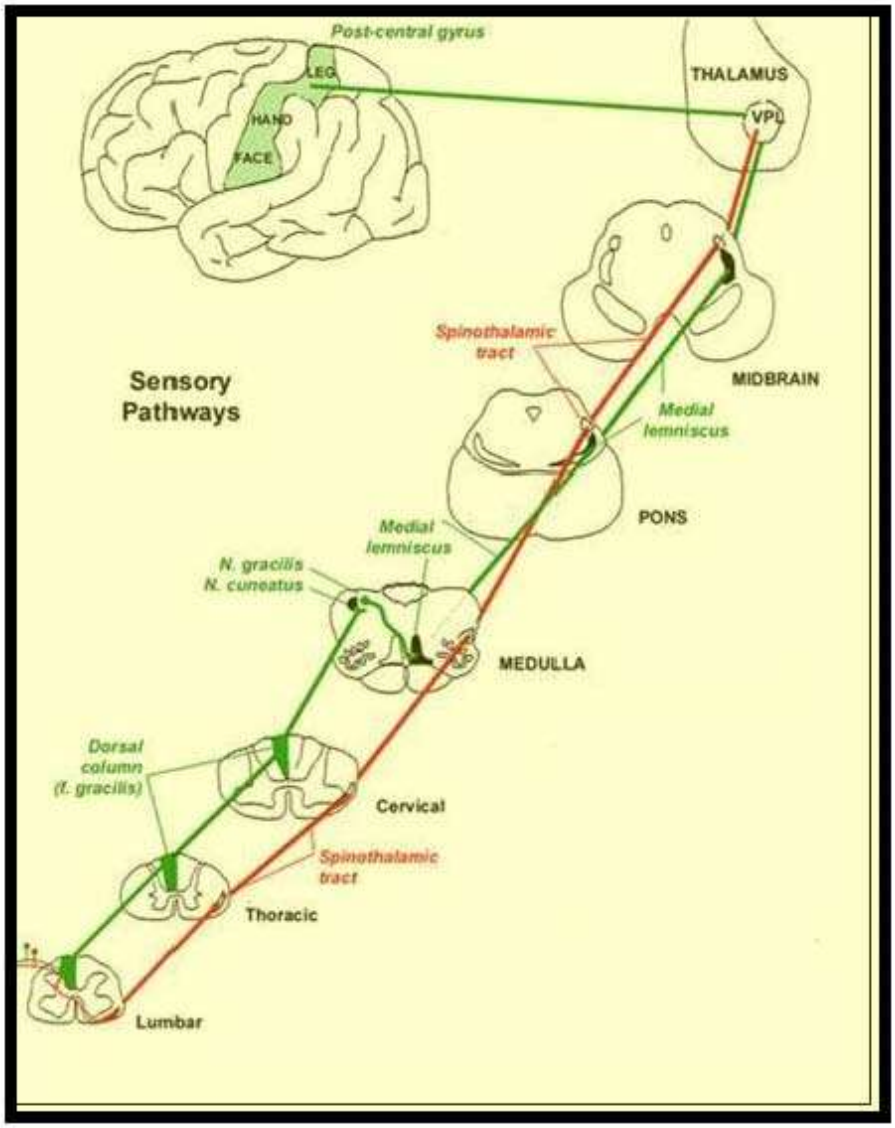
This is an example of how A-delta dysfunction does not necessarily involve pain. Numbness can go unnoticed; especially in the lower extremities, which have such a small portion of the sensory cortex dedicated to this part of the body.

Prostatitis & Vulvadynia: A recent study points out how pain can be minor or absent in the presence of sensory radiculopathy. Irving M. Bush, MD, is a professor of urology at **The Rosalind Franklin University of Medicine and Science** and **The Chicago Medical School**. Dr. Bush is also a former head of the **FDA National Scientific Advisory Committee of Gastroenterology**. Along with his colleague, M. Badruddoja, MD, their findings were presented at the **International Pelvic Pain Society's 15th Annual Scientific Meeting** in San Diego California, October 27, 2007. Using the pf-NCS, they found a correlation between chronic prostatitis and lumbosacral sensory radiculopathy. They found this same relationship in female pelvic pain. Many of these patients had little or no symptoms of concomitant lumbosacral pain. Drs. Bush and Baddruddoja suggest that these nerve entrapments may predispose the patient to such disorders.[ix] Based on Bush's vulvadynia findings and preliminary tests of vulvadynia patients at the **UCLA Department of Obstetrics and Gynecology**, a study is now underway there to look at the connection between female pelvic pain and lumbosacral sensory radiculopathy. Of the 75 patients scheduled for testing in the UCLA study, 12 have been tested and all but one has been found to have A-delta radiculopathy at L5 or S1 or both levels. **Reduced Diabetic Amputations:** A group of North Carolina endocrinologists reported to Medicare that pf-NCS has reduced the number of amputations by allowing early detection of diabetic polyneuropathies. In the past, by the time the EMG/NCV could show positive findings, it was often too late to prevent amputation. In the future it is sure that many more applications will be found for the pf- NCS.

Pain management physicians are well aware that nerve-root pain can be referred and report this finding in many of their patients. However, pf- NCS is showing that referred radicular pain is far more common than previously suspected. At all the **Annual AASEM Conferences**, a consensus is taken of physicians certified in pf-NCS concerning the percentage of patients that incorrectly localize the source of their pain. The consensus has held steady for over 5 years that 50% of patients incorrectly localize the level of nerve-root pathology, and 20% localize the source of pain to the side opposite of the nerve-root causing symptoms. This correlates with the statement in the 2nd Edition of **The Massachusetts General Hospital Handbook of Pain Management** that ***"In most (over 50%) of neck and back cases the anatomical and pathological diagnosis remains unclear."***

In the diagram following, the light touch A-beta fibers are in green and the A-delta fibers are red. Note how they converge in the medulla; both of these excellent localizers share anatomically adjacent tracts.

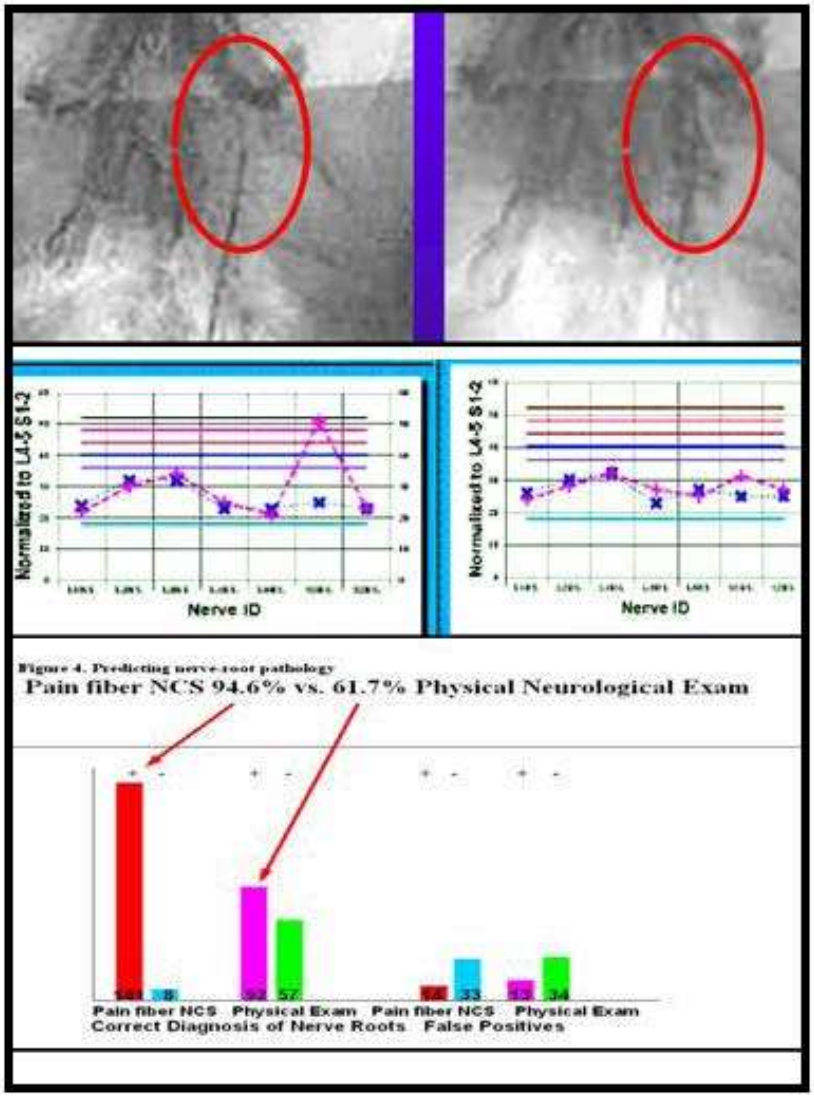
A-delta and A-beta are excellent at respectively localizing pain and light touch. Note how they have nearly identical pathways to the sensory cortex.



Chapter II

A Brief History of Electricity in Medicine

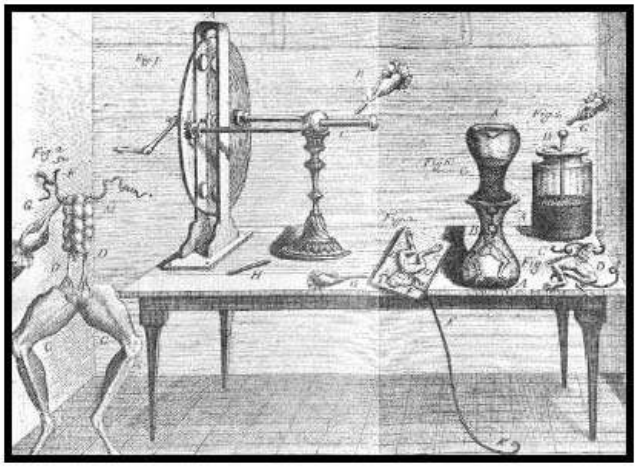
Developing pf-NCS

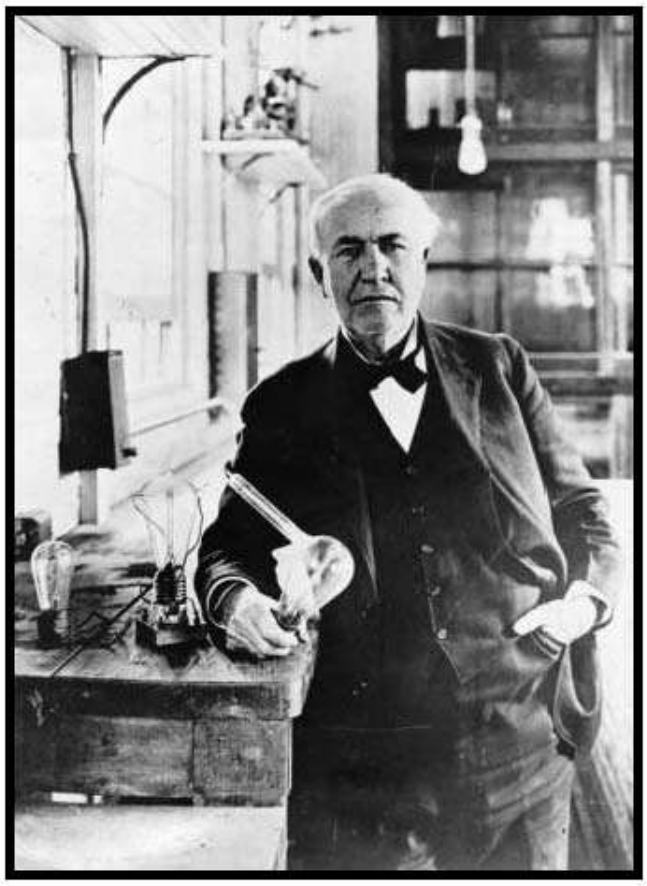


PART I - Brief History of Electricity

The first use of electricity for medical purposes can be traced back to the Egyptians, who used electric eels to treat muscle and joint pain. In 1752, the modern electric age was ushered in by Benjamin Franklin's electrical experiments, which led his invention of the lightning rod, used to prevent structural fires.

The breakthrough in using electricity for diagnostic purposes came in the 1790s with the discovery of electromotive force, better known as voltage, by Count Alessandro Volta. This was quickly followed by Luigi Galvani's discovery that a direct electric current can cause muscle contraction. For the next hundred years little progress was made toward employing electricity for diagnostic purposes.





Late in the 19th Century, while Edison was developing his incandescent light bulb, Féré devised a method to measure the electrical resistance of the body by introducing electricity at one electrode and measuring the **current output** from a second. He called his new measurement exosomatic, meaning from **outside the body**.[x] Inspired by Féré's discovery, Tarchanoff took the next step and developed a method to detect the electrical pressure (voltage) generated from inside the body. He termed this an ***endosomatic*** measurement.[xi]

In the early 1900s, as electricity was finding practical applications for such things as the telegraph, street lights, and powering vehicles, attempts were made to measure nerve sensitivity using electricity. Since electricity can be precisely measured, it was hoped that it would give a more exact quantification of nerve function and replace the less than accurate methods of the past, such as the reflex hammer, pins, feathers, and hot or cold test tubes.

By the turn of the century electricity was everywhere.

PART II - Electromyography - World War-II

The need to determine which nerves were cut and crushed in wounded service personnel during World War II prompted the U.S. Army to initiate a crash program that lead to electromyography (EMG). EMG and its sister test, nerve conduction velocity (NCV), have remained relatively unchanged over the intervening six decades. Basically, since no alternative methods existed, they have remained the gold standard in electrodiagnosis. Interestingly, it is not widely known that EMG-Type tests are limited to large motor fibers and cannot detect pathology unless there is gross anatomical damage.

Understanding Conventional Electrodiagnostic Examinations (EDX): Incredibly, through a misunderstanding of the limitations of EMG/NCV, these tests are routinely used in neck and back pain. Non-neurologists seldom receive even rudimentary training in EMG/NCV. Actually, many medical interns are taught to refer patients to a neurologist when any type of nerve problem is suspected – "just read the report". As a result, many non-neurologists are unaware that EMG/NCV cannot test pain nerve fibers. Some are under the impression that the **M** in EMG stands for **myelin (fatty covering of the nerve fibers)**, or that the **M** stands for **myelo (spinal cord)**. The result is that physicians are often working under the impression that an EMG can test all types of nerves. To be clear, the **M** stands for **myo - muscle**. **EMG/NCV Examination Protocols:** EMG/NCV is touted by many neurologists as being capable of measuring large fiber function, but this is not the case. These tests require a minimum loss of 50% of the myelin covering before conduction changes are detectable (i.e., conduction slowing or is non-existent). Therefore, EMG/NCV is a measure of anatomical integrity rather then actual functional capacity.

EMG/NCV Cannot Assess Pain Fibers: Non-neurologists seldom understand how EMG/NCV is performed or how to interpret the results. The basics are simple to understand: EMG tests the reaction of a muscle, while Nerve Conduction Velocity (NCV) measures large fiber conduction speed. NCV is complimentary to EMG, so they are generally considered as a single procedure under the term EMG. NCV rule in or out whether the nerve associated with the muscle problem the EMG found, is in fact the problem. The velocity and other conduction characteristics are compared to the population averages on a bell- shaped curve—sensitivity is around 67%.
The fatty myelin sheath supports rapid transmission of the nerve signal (action potential). In the presence of **severe** myelin degradation, large fiber conduction slows and causes abnormal muscle contraction. An EMG that reveals abnormal muscle activity leaves the question as to the cause - is the problem the muscle or the nerve controlling the muscle? This is where NCV comes into play.

EMG/NCV cannot test small pain fibers because, as the name implies, they are too small and they have little or no myelin. EMG/NCV may be the gold standard for verification of gross large fiber (motor) pathology, but the problem is that pain patients rarely have concomitant motor deficit, so EMG/NCV in such cases is ineffective. The pf-NCS does not simply verify suspected pathology, as does EMG, the pf-NCS locates pathology and is able to do this during the earliest stages of pathology.

Similar to many physicians, third-party payers do not seem to realize that these tests are ineffective in diagnosing pain and often insist on pain patients undergoing EMG/NCV studies. It is no secret that EMG/NCV cannot test pain fibers. Many neurological texts explain that using EMG/NCV in pain patients without motor deficit is costly, time consuming, and seldom benefits the patient.xii What misleads many is that EMG pain studies tend to involve "a select group of patients". More rigorous studies have shown, in cases involving pain, that EMG/NCV has sensitivity of 29% [xiii] with false positive findings, ranging up to 14% to 48%.[xiv] [xv]

Other problems are inherent in testing large nerve fibers. For example, it is common practice to reinsert the needle until the velocity measurement is consistent. However, there are vague rules such as: if a drop of blood comes out when removing the needle the results are probably false.

An even greater concern is that EMG/NCV is targeted by the patient's symptoms and physical neurological findings which, as reported in **The Massachusetts General Hospital Handbook of Pain Management**, are ***"unclear in over 50% of neck and back pain cases."***[xvi] It should be noted that these methods result in 43% of pain patients developing chronicity and up to 80% of back surgeries failing, so how can it be expected to accurately guide EMG? Meanwhile, the pf-NCS has shown sensitivity approaching 100%, so it is logical that this painless test is a far superior method to help target EMG/NCV.

Misleading Omission: It is often said that **it can take 6 weeks to 6 months for sufficient myelin degeneration to develop to allow EMG/NCV to detect pathology**. What is left unsaid is that in most pain patients sufficient myelin degeneration never develops. Neurological and orthopedic surgeons understand that EMG/NCV cannot assess pain fibers and cannot access the preganglionic dorsal nerve-root (pre-DRG) fibers associated with most spinal pain.[xvii] This explains why these specialists have been some of the first to employ pf-NCS in their practices.

Summary of EMG/NCV Limitations: Keeping in mind that until the introduction of the pf-NCS electrodiagnostic technology had not changed since the 1960s, and that it is well recognized that over 95% of neck and back pain patients have no motor symptoms, the following statements by Werner and Cavender in their 1999 **State of the Art Review** published in **Physical Medicine and Rehabilitation** sums up the marked limitations of electromyography (EMG) in diagnosing painful radiculopathies: ***"In chronic cases, particularly in individuals with predominantly sensory symptoms, it is difficult or impossible to clinically establish the type of or severity of nerve injury. Only if there is obvious muscle atrophy can one know for certain that motor axon degeneration has occurred. The EMG study can be normal in the face of known pathology depending on the timing of the study and the nerve fibers involved."*** The review finds fault with somatosensory evoked potentials (SSEP) and explains the limitations of the H wave study, which is limited to testing S1 pathology. Concerning F wave studies: "Despite the theoretical advantages of using the F response to define proximal (pre dorsal root ganglion) conduction, it is of little practical value in the evaluation of radiculopathy. Considering the fact that most radiculopathies are associated with little or no motor deficit and the biases found in selection of a gold standard for compressive radiculopathy Werner and Cavender state: "Thus, the sensitivities reported in the literature are falsely elevated and tend to lull us into thinking that electrodiagnostic evaluation of radiculopathy is both sensitive and specific."[xviii] In spite of this negative review, it has been used to support the efficacy of conventional electrodiagnostic studies solely on the basis that these tests have high sensitivity in the presence of over 50% axon degeneration in motor fiber pathology, which is found in less than 5% of neck and back cases.[xix- xx]

Surface EMG: The problem with SEMG is that it cannot differentiate between muscle contraction caused by an injured nerve, by referred pain, or by compensatory muscle contraction.

MRI: Many consider MRI to be the gold standard for diagnosing nerve- root pathology. However, regardless of MRI's excellent imaging capabilities it cannot image pain and abnormal anatomy does not prove the presence of nerve pathology. The 50% to 80% failure rate in spinal surgeries attests to the fact that matching abnormal anatomy with symptoms is less than effective. A 1994 study published in The New England Journal of Medicine reported that 52% of 98 normal subjects had diagnosable disk disorders. Protrusion and extrusion was found in 27%. Abnormality of more than one disk was found

in 38% of these subjects.[xxii]

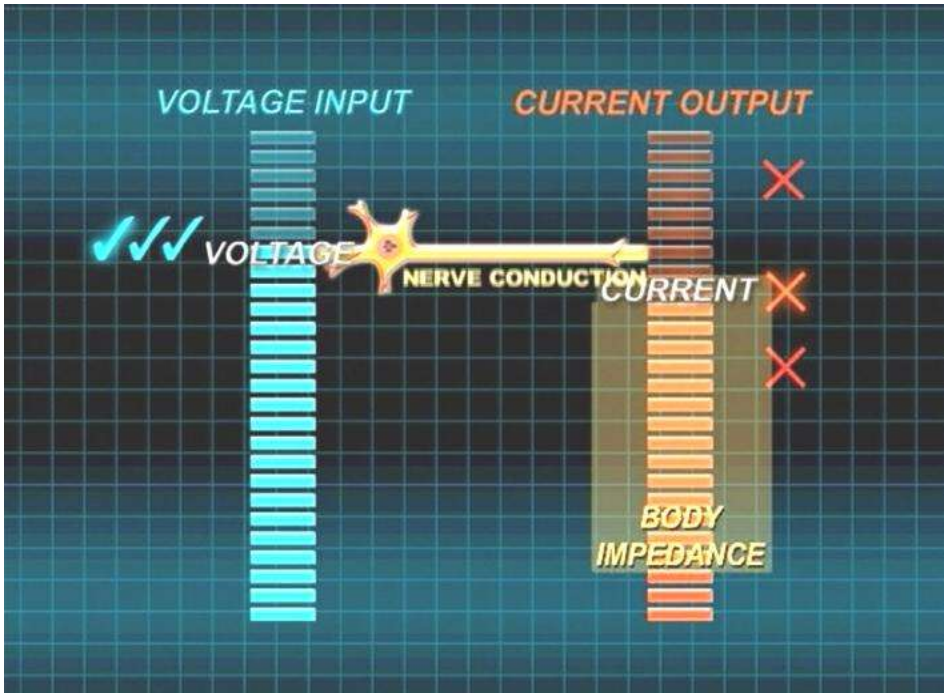
Part III - Developing the pf-NCS

To fully appreciate the pf-NCS one must fully understand the limitations of a history, physical neurological exam, EMG/NCV and MRI to make a diagnosis. The need for a direct measure of small pain fiber function is best summed up in the journal **PAIN**; **"There is an urgent need for a simple method to diagnose pain disorders."**[xxiii]
Pain's monetary cost is only eclipsed by the magnitude of patient suffering.[xxiv] The cost of pain to our economy is reflected in the U. S. Congress resolving to direct the National Institute of Health to find a more effective method to diagnose neck and back pain.

Post World War II Electrodiagnosis: Efforts to find an effective method to measure pain fiber function date back to the early 1900s. The main problem preventing a breakthrough was that early researchers started off in the wrong direction by correlating the nerve's threshold with current output - the amount of energy passing through the body at the time the nerve fired an action potential. Correlating Féré's **exosomatic measurement** (current output) with nerve conduction threshold was a mistake since voltage—the electromotive force—is the component of the electrical-signal that initiates the nerve impulse, not the current output. Current output plays no part in initiating an action potential, other than being the vehicle that delivers the voltage to the nerve membrane.

Disregarding voltage as the cause of an action potential continued into the 1960s, even though a Nobel Prize was awarded for the discovery of the nerves' voltage-gated channels. The skin's shifting resistance was found to be under the control of the autonomic nervous system, and only minimally involved with thermoregulation by sweat glands. Also, by that time current output was widely in use under the term galvanic skin response (GSR), in biofeedback training and, as early as the late 1940s, GSR was included as one of the tests in the well-known lie-detector test, the polygraph.[xxv] Fluctuations in electrical skin resistance also began to be used in therapeutic and research fields, and electro-acupuncture. However, pain fiber researchers failed to realize that the shifting impedance of the skin was the reason they could not attain repeatable measurements.

This diagram shows how using a patented modulated signal; voltage causes conduction at the same repeatable intensity, while current output results in different measurements due to the constantly shifting impedance of the skin.



PART IV - Electromotive Force = Voltage/Potential Voltage-Gated Na & K Channels

In 1963 the physiologists, Hodgkin and Huxley, won the Nobel Prize for discovering Voltage-Gated Sodium and Potassium Channels of the nerve membrane. Studying the large neurons of squid, they found that opening the voltage-gated channels allowed sodium (Na) and potassium (K) ions to switch sides of the nerve membrane, which caused a small burst of voltage that opened the next channel and the next and the next. This cascading reaction is the action potential. Potential is synonymous with voltage or electrical pressure. This discovery demonstrated voltage to be the component of the electrical signal that initiates the nerve impulse.

Curiously, even after this discovery, researchers continued measuring current output. The few who looked at the voltage failed to take into account the skin's impedance, which is most often higher than the threshold of the nerve fibers. The failure to incorporate voltage and to recognize the effect of impedance resulted in continued inconsistent measurements. The final outcome was that researchers came to the erroneous conclusion that the nerves themselves were at fault. The shifting threshold was erroneously attributed to inherent

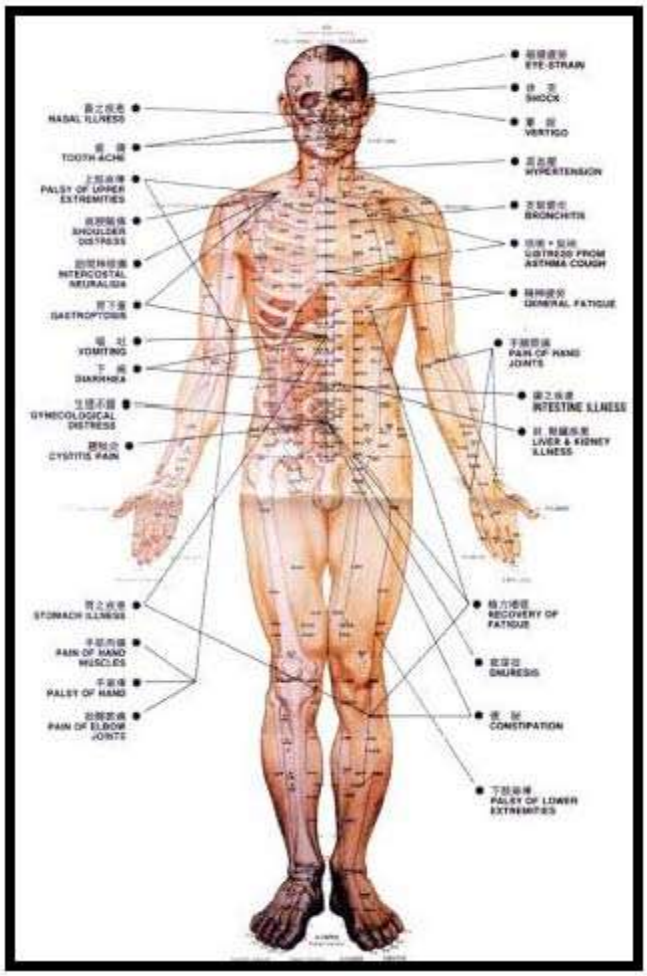
instability of the nerves, making it impossible to glean meaning from the measurements. By the mid 1970s, investigations were abandoned.[xxvii]

In the mid 1990s a method was successfully developed which took into account the two overlooked components: 1) Voltage causes the voltage-gated channels to initiate the action potential; and 2) Skin impedance shifts and is usually breached at a higher intensity than the threshold of the action potential.

Misconceptions: For decades medical physiology texts have devoted entire chapters to the voltage-gated channels and membrane potentials (voltage). But without a method for practical clinical application and with the clinical focus being on large motor fibers, a general ignorance of small pain fiber physiology has developed within the medical community. For example, as late as 2002 a panel composed of two MDs and an MD/PhD published a web site memorandum in which they attributed the "theory" that nerves are sensitive to voltage to Hedgecock, the head author of this textbook. Only after being directed to the chapters in physiology textbooks did they acknowledge their error. In 2010, two neurology professors, with combined teaching experience of 42 years at prestigious medical schools, submitted reports that demonstrate how deep the misunderstanding of sensory physiology runs.[xxviii] One professor stated that; "If you could stimulate the A-delta fibers with an electrical signal it would only result in the subject experiencing pain, since A-delta fibers only transmit pain." This is in spite of the fact that for at least 50 years physiology texts have listed that A-delta fibers transmit not only pricking pain, but also transmit hot, cold, deep pressure, stretch, touch, and itching sensations. Another statement the professors made was that "there are no electricity receptors," implying that a subject cannot feel electricity. The neurologists also were confused about the suprascapular nerve, saying it is purely motor and not sensory. Anatomical naming is the accepted form, and most dermatome charts show the suprascapular region labeled as C4, therefore, a sensory nerve in this region can be correctly named the suprascapular sensory nerve. Some would say that this is actually the Supraclavicular nerve, which is simply slitting anatomical hairs. Actually, a recent study found that the suprascapular nerve, which was thought to be purely motor, does contain sensory fibers.[xxix - xxx]

Based on such a dearth of knowledge concerning sensory physiology and anatomy, there is little wonder that neurologists question whether pain fibers can be selectively stimulated by specific electrical frequencies. Neuroselectivity will be discussed later in this chapter.

Acupuncture Connection: A key insight that led to the development of the pf-NCS was gained through the study of acupuncture. What does acupuncture have to do with nerve physiology? Actually, it has everything to do with it, because it has been proven that acupuncture points are in fact nerve centers.



Nerve Centers: In 1972, the **UCLA School of Medicine** held a conference where several faculty members from the **University of Tokyo Medical School** explained how Dr. Yoshio Nakatani, had discovered that traditional acupuncture points were in fact nerve centers. Nakatani discovered that acupuncture points have lower electrical resistance compared to the surrounding cutaneous tissues. Through experiments Nakatani learned that needling causes a release of the neural stimulants histamine and bradykinin. Further studies revealed that an electrical stimulus between 600 mA to 800 mA at 27 volts released more histamine in 10 to 20 seconds than two hours of traditional needling. The conclusion they reached is that acupuncture points are nerve centers, since nerve tissue is likely a better conductor of electricity than cutaneous tissues. The traditional method of finding acupuncture points by identifying them as more sensitive than surrounding skin also supports that they are nerve centers.

At the **University of California at Irvine (UCI)** in 2001, Z. H. Cho, PhD, E. K. Wong, MD and J. H. Fallon, PhD, published a text, **Neuro- Acupuncture**, which reports how they succeeded in settling the question of acupuncture points being nerve centers using fMRI. Cho and his colleagues demonstrated that stimulation of an acupuncture point excites brain centers. For example, stimulating an acupuncture point near the knee, known to influence vision, activates visual centers in the occipital lobes of the brain.[xxxi] Based on the recognition that acupuncture points are nerve centers these sites have been given the term; **Asia Points**. The **Asia Points** are almost identical to the sites proposed by Dr. George Riddock, a British neurosurgeon. During World War II, he determined that these points are over specific nerves associated with specific nerve-roots.[xxxii] **Asia Points** are the standardized test sites used in pf-NCS.

These revelations demonstrated that needling does not stimulate some mysterious chi (life force), but instead releases chemical mediators which in turn stimulate nerves that cause reflex activity in the central nervous system (CNS).

Who Discovered Acupuncture? There is some controversy over who first developed acupuncture. The Chinese are generally given credit based on the fact that the first acupuncture text was written in the 8th century AD by a Chinese physician at the direction of the Yellow Emperor. However, it is highly unlikely that the Chinese were the first to use this technology. Historically, it is known that acupuncture techniques were in use in India long before the Yellow Emperor's text, so it is likely that acupuncture could have been exported from India to China sometime after the 3rd century BC by wandering Buddhist monks.[xxxiii]

The controversy goes back even further in time. Over 5300 years ago, a man froze to death in the Alps. On September 18, 1999 Lancet reported on the "Iceman" and published pictures showing that he had acupuncture points tattooed on his body, which correspond to points used in the treatment of low back pain. Radiographic examination found that the Iceman suffered from advanced low back arthritis. The Iceman is thus proof that 5300 years ago acupuncture was so technologically advanced that a European had points tattooed on his body to facilitate treatment.

It will probably never be known by whom, when or how acupuncture was developed, but it is certain that acupuncture was developed in the absence of knowledge about electricity and the nervous system. Whoever the inventors, it is certain that they had no knowledge of the bio-computer contained in the human skull. Proof attesting to this ignorance is found in the fact that coinciding with the time of the Iceman's death, the most advanced civilization on earth, the Egyptians, were throwing away the brain during mummification, while saving organs like the liver and spleen in precious jars.[xxxiv] The truth is that Chi and the meridians were devised as a way to explain the acupuncture phenomenon. Chi is the voltage action potential, and the meridians are nerve pathways.

Transcutaneous Nerve Stimulation: Hedgecock attended the 1972 UCLA conference where Dr. Yoshio Nakatani's research was presented. Then traveled to Japan, Korea, and China to study acupuncture and was subsequently certified by the Hong Kong Acupuncture Federation. Though trained in needling techniques, transcutaneous electrical stimulation was found to be more effective. Meeting with success in helping with problems ranging from tennis elbow to poor hearing, the opportunity came about to treat a stroke victim, which led to a key element required for the development of the pf-NCS.

Suspecting that it might be possible to reactivate the neurons surrounding the area of stroke damage, this was attempted. It is widely considered that these neurons near the damaged brain tissues are capable of function, but in a state similar to that of shock - suspended in a physiological limbo. To the patient's delight treatment was found to be remarkably effective in patients with lesions of the sensory cortex.

The points chosen were those furthest from the brain, near the nail-bed of the fingers, called Akabane points.[xxxv] With each treatment, the patient incrementally gained function of the affected hand. The hand started out as a closed fist, but after a few treatments the patient was able to pick up coins. The delight was doubled, because as his hand improved so too did the untreated lower extremity, and within a few weeks the patient could walk without the aid of his cane. After the patient attended his church group's monthly stroke support meeting, the author was quickly inundated with stroke patients.

Electrical Impedance: Treating these stroke patients revealed some interesting observations. One was that patients with flaccid paralysis (motor cortex damage) failed to show even the slightest sign of improvement, while those with spastic paralysis (sensory cortex damage) consistently showed a positive response.

The observation, which later proved to be of importance in the development of pf-NCS, was how patients responded to the severe pain that accompanied stimulating the dormant neurons in the sensory cortex. It was observed that in anticipation of the pain, which the patients compared to being touched by a burning cigarette, patients would either hold their breath or breathed normally. It was soon noticed that in those who breathed normally, it could take several minutes for the stimulus to break the skin's impedance. However, in those holding their breath the impedance was broken in a few seconds.

For practical purposes, to speed up the treatments, those patients who breathed normally were simply asked to hold a deep breath. Fascinated by this phenomenon, I explained the probable neurological mechanism and pathways in a paper. Realizing this discovery had very previously been unobserved, the University of Pasadena Science Department faculty of the awarded me a PhD in neurophysiology. The discovery of the Respiratory Impedance Variable (RIV) would prove to be a key element in developing pf-NCS technology.

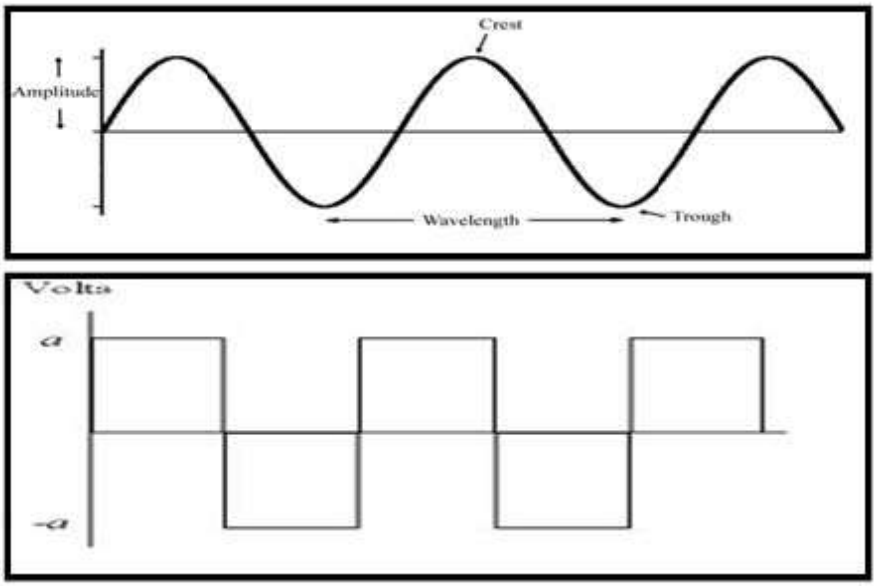
Neuroselectivity: Though researchers overlooked the significance of voltage and

impedance, they did make important discoveries. The most important of these was that different types of sensory fibers respond to different frequency ranges. The C-Type fibers were found to respond to a frequency between 1 Hz to 10 Hz (cycles per second), A-delta fibers respond between 150 Hz and 350 Hz, and A-beta fibers respond between 1500 and 3000 Hz. [xxxvi] [xxxvii] [xxxviii]

It seems obvious that the 250 Hz modulated frequency is assessing A- delta fibers since it so effectively detects pathology. However, not everyone is convinced. The most compelling empirical evidence is that this frequency causes all the sensations transmitted by the A-delta fibers: pricking, hot, cold, vibration, pressure and itch. The physiological argument is that each fiber type is known to accommodate within a certain time range. Therefore, it is logical that a stimulus with a frequency timed to that accommodation cycle would selectively stimulate that particular fiber. A demonstration conducted at the **New York University, Kriser Dental Center College** helped show neuralselectivity. Eight volunteers were tested with three neuroselective frequencies; 5 Hz C fibers, 250 Hz A-delta fibers and 2000 Hz A-beta fibers. After injecting the trigeminal nerve with Lidocaine on one side, both sides were tested at intervals. The basic concept was that as the affect of the Lidocaine wears off a comparison with these frequencies and other stimuli could be made. The main question, however, is that these tests were measuring current output and not voltage. Additionally, the testing protocol was to turn the stimulus to zero between each serial test, which means the skin impedance was being measured. Perhaps the only reason this demonstration had any correlation is because the skin's impedance is probably affected by Lidocaine.

In 2007, at the **6th Annual AASEM Conference**, the author came up with a much more straightforward experiment. It was reasoned that since the A-delta fibers uniquely synapse with motor neurons (reflex withdrawal), if the A-delta frequency was actually selective then it should cause muscle contraction. Some 100 attendees witnessed how the 250 Hz stimulus caused muscle contraction when applied over the myoneural junction, while the 5 Hz and 2000 Hz did not cause contraction even when the stimulus was turned to maximum intensity. This is objective evidence that the A-delta frequency and patented modulations do, in fact, selectively stimulate A-delta fibers.

Other Early Discoveries: Researchers also found the internal resting state voltage of the nerve fibers to be approximately minus (-) 90 millivolts. [xxxix] This finding suggests that the internal voltage of a damaged nerve fiber might then require greater voltage intensity to initiate firing than would a normal nerve fiber. Conversely, irritation might increase the internal voltage with a subthreshold potential and the nerve fiber would require less voltage to initiate an action potential.



Improved Modulated Waveform Once voltage was found to produce repeatable measurements within a reasonably narrow range, the next step was to determine if this range could be further reduced by altering the signal modulation.

Previous devices had used current output and a sinusoidal waveform, which was described as being "comfortable" (top figure). However, the patient is being stimulated with the minimum intensity causing an action potential, so there is no reason for the waveform to be "comfortable".

It was found that an uncomfortable square waveform could be detected within a far more narrow range. Interestingly, at threshold the uncomfortable waveform was found to be more comfortable than the sinusoidal waveform. At threshold the sinusoidal waveform feels like a biting sensation, while the square waveform is a tickle. This difference in sensation may be due to the rectilinear/square wave being detectable at a lower intensity. What accounts for this narrower range is apparently that when the top of the sinusoidal waveform reaches threshold there is too little energy to open the voltage-gates and initiate an action potential. By the time enough energy is present there is a slight over stimulation and, as a result, the sinusoidal first sensation is a sharp pricking sensation rather than a tickling sensation.

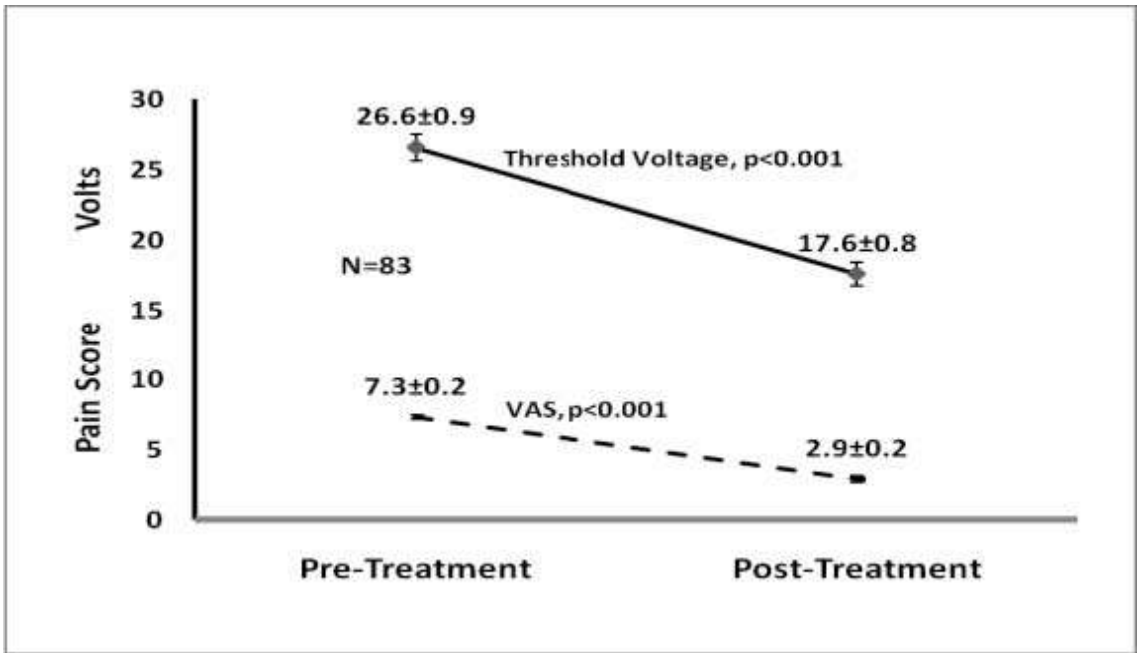
In experiments, the square and sinusoidal waveform signals were connected to an audio speaker. It was found that the square waveform could be heard at a distance of over 30 ft., whereas, at the same intensity setting the sinusoidal waveform could only be heard within less than 5 ft. from the speaker. A good analogy to explain this is to think of the difference between being blindfolded and touched by a big, soft cotton ball (sinusoidal waveform) or being touched by a brick (square waveform). The soft cotton ball may not be noticeable at

first contact, and may require some increased degree of pressure before it is perceivable. On the other hand, a brick will be noticed as soon as it makes the slightest contact. Again, the point being that the subject is not hit with a brick, he is only being touched by one.

The final outcome is that on a 100 point scale, with 100 being equal to approximately 10 mA / 50 volts, the sinusoidal waveform is detectable in a range of about 5 points, whereas, pf-NCS modulations allowed detection to be within 1 point.

Detecting Malingering: The pf-NCS recognition range is so narrow that by simply using the patient's response it is quite easy to detect malingering. This is based on the fact that a human's ability to detect a change in intensity is a wider range than the range within which the threshold sensation can be noticed. Additionally, at each site the characteristics of the sensation change with each repeated test. The sensations shift between tingling, itching, pricking, vibration, warmth, cold, and pressure. These variations add confusion. As a result, a patient who repeatedly reports he feels the threshold sensation within less than 2 points (0.2 mA or 1 volt) on the dial can only be reporting the threshold. If he is attempting to defeat the test, he cannot be within this narrow range. This is why it is important to tell the patient to report the first sensation he feels, whatever it may be. The potentiometer is the fail-safe, because it verifies the action potential independent of the patient's psychophysical assessment (perception).

Proving pf-NCS Efficacy: The medical community resists change. The most effective way to convince medicine that a new technology is efficacious is peer-reviewed studies. In 2002, a seminal peer-reviewed study was published in the Internet Journal of Anesthesiology (IJA) (see page 60). Internet journals have come to be recognized as offering excellent data to a much wider readership than paper journals. The IJA editorial board is a literal who's who in international academia.



The latest study, which has recently been submitted for peer-review publication, combined with the IJA 2002 study puts to rest any questions about pf-NCS efficacy. Both of these studies and others start on page 187. This graph is one from the newest study. It shows the direct relationship between A-delta sensitivity and the subjective pain VAS rating.

Below is a most important element discussed in the abstract from the study, **Paradoxical Relationship: A-Delta Function and VAS**, authored by Randall Cork, MD, PhD and Michael Bezel, MD: ***A significantly close relationship was found between the change in voltage required to cause an action potential (nerve impulse) in the A- delta fibers of the pathological nerve-root and changes in the subjective VAS rating. The findings support the utility of the A-delta pf-NCS for detecting the level and side of painful radiculopathy and measuring changes in pain.***

In a nutshell what this study proves are two things: 1) The more severe pain becomes, the more likely the patient will incorrectly localize its source; and 2) The A-delta pf-NCS can detect the sensory nerve injury that the patient cannot effectively localize.

PART V - Analysis Algorithm

Chapter V explains how the analysis is performed. Here, a few basic concepts of the analysis algorithm and development will be covered.

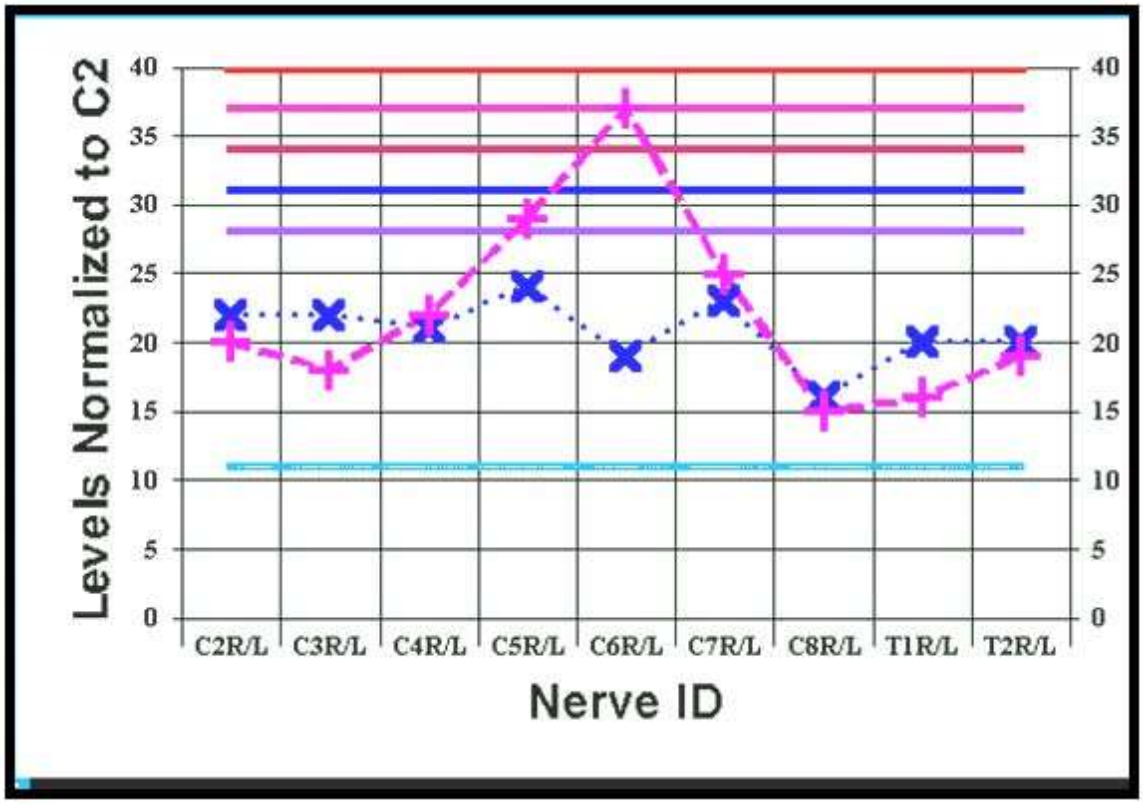
Bell-Shaped Curve vs. Nomogram: Comparing data to population averages on a bell-

shaped curve generally yields about 67% sensitivity. However, by using the patient as his own control (his own bell-shaped curve), sensitivity can approach 100%. The more data available from normal nerves the greater the sensitivity. Fortunately, time is not a factor since an experienced examiner can test all the major nerves in a region in about 20 minutes; the time needed to complete an analysis is dependent on the complexity of the patient's pathology.

In a standard examination of the cervical region, 19 nerve sites are tested. In the majority of patients only one to three nerve-roots are involved, so in most cases there are 15 or more normal controls. In the lumbosacral study, 14 nerve sites are tested, so there are usually 11 or more controls.

Initial Algorithm Study: Initially, in developing the analysis algorithm, data from 75 patients who were examined using the standardized test sites (Asia Points) was compared with the level and side of suspected nerve-root pathology. The measurement from the A-beta and C-Type fibers showed no correlation with the suspected pathology. However, the nerve with the greatest diminished A-delta sensitivity (requiring the highest voltage to cause an action potential) correlated with the suspected nerve-root pathology in all but a few cases. In those patients where the A-delta hyposensitivity did not match the suspected pathology, it was later found that the nerve-root the A-delta test identified was in fact the pathological nerve, and the suspected nerve- root was referred pain.

Sensitivity Variations: The data from the first 75 patients revealed a slight variation in sensitivity between test sites. These variations are most likely caused by differences in skin thickness or nerve depth, and perhaps even differences in nerve size. However, it seems certain that these differences are not due to sensitivity variations of the nerves themselves. For example, the T1-2 (ulnar nerve) sites, which are on the thin skin of the inside upper arm, have a lower voltage threshold than thicker skin on the back of the hand (C6-7 radial nerve). To avoid the undulating zones that these variations would give to a graphic display, it was decided to employe a nomogram in which the peaks of the bell- shaped curves are aligned on the same horizontal plane. This makes the graph a symmetrical grid, which greatly facilitates a visual comparison of the measurements.



Nomogram Rating Zones: It is safe to assume that most of the nerves in a region are normal, therefore, the measurements are averaged and that average is placed in the center of the **Normal Zone**. Pathology is identified as the highest measurement - the nerve requiring the highest voltage to cause an A-delta fiber action potential. The greater the deviation toward hypo-function (death), the more likely pathology exists.

To further develop the analysis, it was necessary to test more subjects. Eventually, over 600 patients with well defined nerve-root pathology were tested. As found in the initial group of 75, the greatest A-delta fiber hypo-function matched the major nerve-root pathology in most cases. Like the first group, the 600+ group also had about 25% whose pf-NCS findings did not match the suspected lesions. When the treating physicians were advised to consider shifting their focus to the nerve- root indicated by the pf-NCS findings, these patients began to respond favorably to various interventions.

Sympathetic Pain: Early on, it was noticed that patients diagnosed with RSD experienced severe pain at threshold. The pain these patients experienced caused them to pull away from the test electrode. In other words, instead of experiencing a tickling sensation at threshold, these patients had a painful withdrawal reflex. It was also noted that several minutes after testing, these patients complained of a continuing throbbing type pain at and around the test site. Based on these findings, the device was designed to test all three types of sensory fibers - the Slow C-Type fibers, the Fast A-delta fibers, and the large light touch

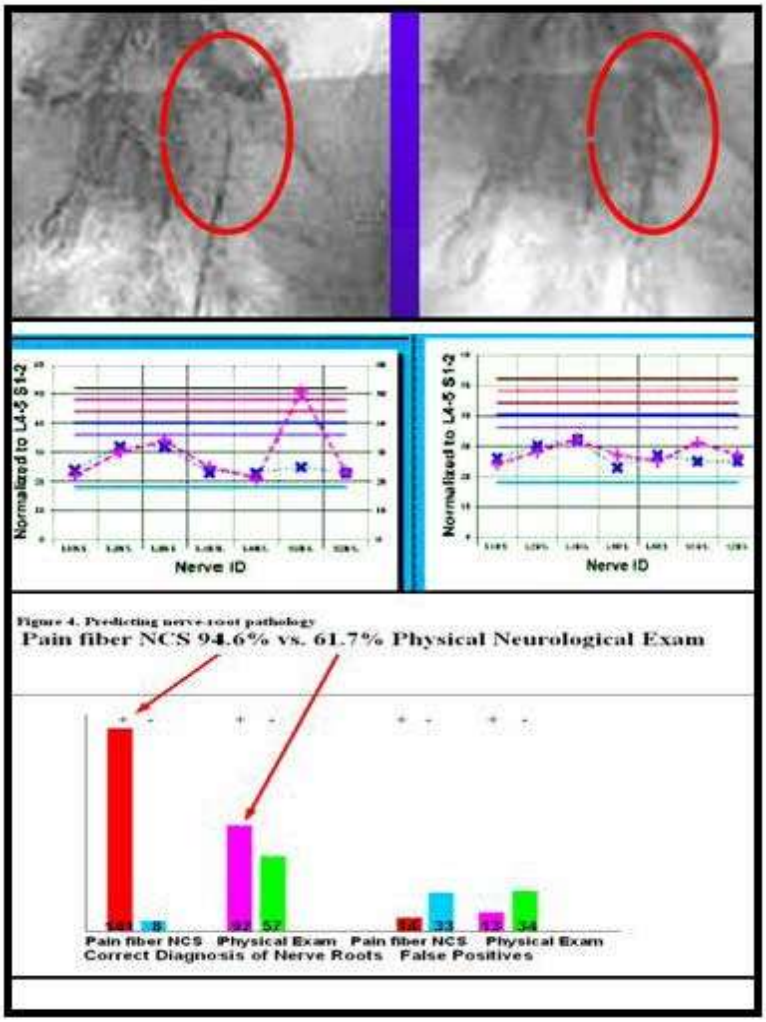
A-beta fibers. This gives the physician the capability to test suspected sympathetic pain cases with all three types of sensory fibers. It is recommended that such tests include the nerves above and below the involved nerve and the same nerves on the contralateral side because this gives the physician more data to allow closer monitoring.

Deviation Index Ratings: The analysis algorithm rates A-delta diminished function (hyposensitivity) on a scale from +1 Mild, to +5 Very Severe. Hyper-function is identified as a measure below the normal zone. Hypersensitivity indicates that conduction occurring below the normal stimulus intensity. This is probably due to irritation of the nerve by adjacent tissue, such as inflammation. Seldom is it due to direct nerve pathology.

FDA Marketing Clearance: In late 1997, the pf-NCS prototype device was granted FDA marketing clearance as being safe and effective. Patents, both U.S. and foreign, were granted in 1998 and the software algorithm was copyright protected that same year. Additional patents were granted in 2004.

Louisiana State University Pain Center Demonstration: Randall Cork, MD, PhD, Chairman of the Department of Anesthesiology and Director of Pain Management at Louisiana State University in Shreveport, was first introduced to the pf-NCS at a medical conference in February of 1998. Dr. Cork previously used a current output device, which resulted in him suspecting that this new device was just the same failed technology. Once Dr. Cork understood the difference, he asked for a demonstration and it was arranged for a group of his pain patients to be blind tested at the LSU Pain Center.

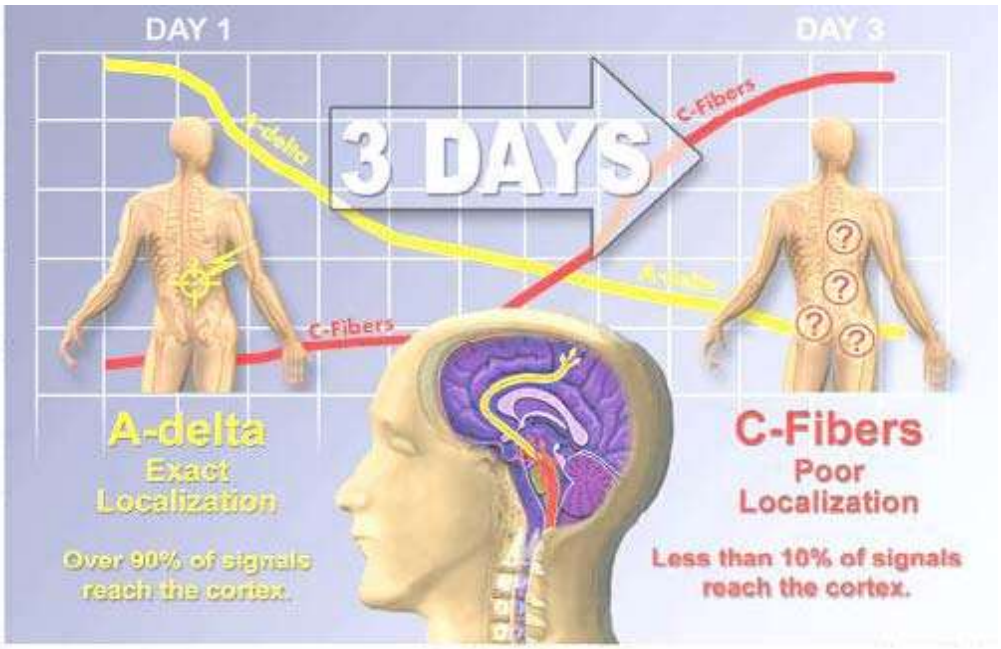
Six patients were blind tested, and within two hours the pf-NCS correctly identified radiculopathies, peripheral neuropathies and a sympathetic pain syndrome. Also, it identified the cause of bilateral hand pain and occipital headaches in one of Dr. Cork's nurses. Impressed with the results, Dr. Cork quickly designed a study.



Peer-Reviewed Radiculopathy Study: In 2002, Dr. Cork published the first peer-reviewed study of the pf-NCS. In this 3 year study, he compared the pf- NCS findings to the history and physical neurological (HPN) exam of 49 chronic patients with L5 or S1 radicular back pain. Of these, 25 were failed back surgery cases. The pf-NCS findings were compared to nerve- root adhesions visualized on an epidurogram (contrast dye study).

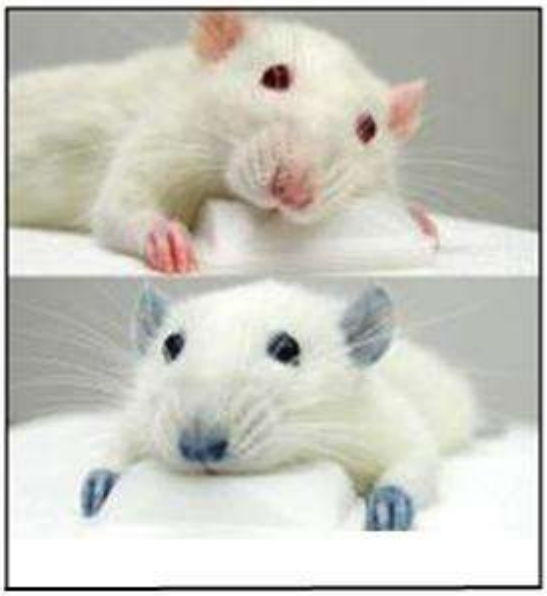
Findings: The HPN exam had 61.7% sensitivity while the pf-NCS had 94.6% sensitivity in detecting which nerve-root had adhesions on the epidurogram. Specificity was statistically equal at 71% (+ -1%). xl Lecturing at the 2005 AASEM conference, Dr. Cork explained that he considered the pf-NCS to be the gold standard, since it measures function and not simply abnormal anatomy. Cork felt that the specificity is very likely much higher than 71%, because the pf-NCS can detect pathology that the epidurogram is incapable of imaging.

A complete compilation of studies can be found starting on page 175.


DAY 1
DAY 3
3 DAYS
A-delta
C-Fibers
C-Fibers
A-delta
A-delta
Exact
Localization
Over 90% of signals
reach the cortex.
C-Fibers
Poor
Localization
Less than 10% of signals
reach the cortex.

Chapter III

Recent Advances in Pain Physiology



Part I Recent Advances in Pain Physiology

A-delta Depression: A 1997 rat study by Sandkuher, et al; published in Society of Neuroscience (Germany) shows suggests that the site of A- delta down-regulation is likely in the substancia gelatinosa of the spinal cord. In 1998 Hedgecock discovered that down-regulated A-delta fibers consistently were found concomitant with nerve root injury.

Glial Cells: Increasingly researchers' attention is now being focused on the glial cells of the brain and spinal cord, and the part they play in pain syndromes. Microglia, astrocyes and oligodendrocytes in the central nervous system have for over 100 years been considered structural and metabolic support cells. It had been thought that their primary role was to aid in the repair of injured neurons and act as a supporting framework. It has now been discovered that they have a much more active role to play. The earliest clue to this more dynamic role is found in the work of Franz Nissl, who in 1894 noted that microglia and astrocytes, in the dorsal horn of the spinal cord, become thicker and more numerous just after an injury involving the peripheral nerves that terminate in the dorsal horn.

New studies: Recent studies show that microglia, in particular, actively participate in the onset of chronic pain, while astrocyes participate in pain's perpetuation. Together these

cells monitor the chemical activity of neurons, such as the chemical activity between the incoming sensory peripheral nerves, which carry the incoming pain signal to the dorsal root ganglion (DRG) cells, and then on to the dorsal horn. There they interact with the outgoing spinal neurons, before they pass into the spinothalamic tract. Intense synaptic activity resulting from the increased neuronal signaling due to severe pain can result in reactive glia. The glia cease to perform their usual job, which is mopping up the overly abundant neurotransmitters building up excessively in the synapse, and thereby ease the job of neuronal firing. This may be important in the short run to allow healing of these incoming neurons, but in the long run hyper-sensitized secondary spinal neurons activated in this process can become a source of neuropathic pain. Neuronal mechanisms also exist involving gene regulated ion channels and other sensitizing substances. The end result of these neuronal mechanisms, and the very significant additional inflammatory cascade of cytokines and other glial factors released by reactive glia, cause depletion in the number or functioning of incoming inhibitory neurons. The additional result is the amplification of the outgoing pain signals from the DRG and dorsal horn, which are then transmitted to the spinal neurons.[xli]

The glial cells help ease the incoming neurons' continued functioning, and so continue their major role of neural support and the promotion of healing, just as a splinter provokes a cascade of inflammation and nerve sensitization in a much larger area of nearby tissue to promote rest and healing of that body part. The incoming and outgoing neurons should be down-regulated, if the person was to achieve pain control through quick artificial means. The innate mechanism of sensitized neurons described above, on the other hand, is a slow process that comprises the body's own ability to eventually heal the involved neurons. Once the sensitizing cycle described above unfolds, in which pain signals are ramped up, normally innocuous stimuli, such as skin contact with clothing, water, sunlight or wind may cause severe pain. This pain may be resistant to relief by opioid analgesics because the analgesic action of administered opioids and opioid-like chemicals are often quickly opposed by prompt glial reaction. Thus, glial activity may play an important role in the **Protopathic** Phase of pain.

A-delta Fiber Down-Regulation: It may be of interest to note that during the development of the pf-NCS technology it was found that A- delta function consistently diminished in an injured nerve. It was also found that sensitivity of the C-Type fibers seldom had a significant shift and most often was within a normal functional range. This was in spite of the fact that the subjective pain was reported to be severe. Perhaps the increased pain is not so much a C-Type fiber activity, but due to glial activity. In other words, the glia supports the C-Type fibers so they test normal, when in fact they are working overtime. Indeed studies using IBA-1 immunostaining demonstrate dorsal horn microglial activation is much less in rat pups than adults after the injection of lipopoly- saccharides (LPS) or N-methyl-D-aspartase (NMDA), which increase adult rat allodynia. This suggests the immaturity of the microglia dampens allodynia. Intrathecal injection of cultured ATP-activated microglia, known to cause mechanical allodynia in adult rats, demonstrated no

such response in rat pups until they were around 16 days old.[xlii]

Spinal Neural Receptors - Targets for Pain Relief: Over 80 years ago ATP was recognized by Nobel Laureate Otto Meyerhof as the cellular source of energy for muscle contraction. In 1959, Pamela Holton at Cambridge suggested that sensory nerves release ATP outside the cell. Its further role outside the cell was discovered 3 years after that, in 1962, by a young Australian neurophysiologist, Geoffrey Burnstock, who was studying the nerves of the autonomic nervous system that control smooth muscle tissue (blood vessels, intestines, bladder, etc.). A series of experiments showed that with other neurotransmitters blocked, the continued signaling by autonomic nerves to the smooth muscle was indisputably accomplished by the release of ATP. Based on his continued experiments, in 1972, Burnstock proposed the existence of "purinergic nerves", which release ATP as their signaling molecule or neurotransmitter. This name of course has its origin in the chemical structure of ATP, wherein the base adenine is combined with a sugar forming the purine, adenosine, with three detachable phosphate subunits, with chemical energy trapped in each of the three bonds.

Teamwork, not Agents Acting Alone: Neurotransmitters need receptors to pick up the signal at the synapse and carry the message forward. The first receptor for a neurotransmitter was discovered in 1970. Naturally the receptors for purinergic nerves were looked for, but remained elusive for over 20 years, until the molecular tools first available in the 1990's allowed many groups worldwide to isolate ATP receptors. In 2009 the latest of these receptors, the P2X4 ATP receptor subtype, was isolated as a pure crystalline structure, and it appears to have multiple roles to play in perpetuating various types of chronic pain.

In addition to the teamwork of neurotransmitters and receptors working together to get the message to the cells, there is also teamwork between neurotransmitters that work together to modulate the signal at the synapse. Purinergic nerves release ATP to work in concert with other neurotransmitters, including substance P and glutamate. This gives proper nuance to the signaling involved in transmitting the pain message. Before all these more precise molecular techniques were developed however, the "old school researchers" were able to use pharmacologic methods to identify various families of receptors.

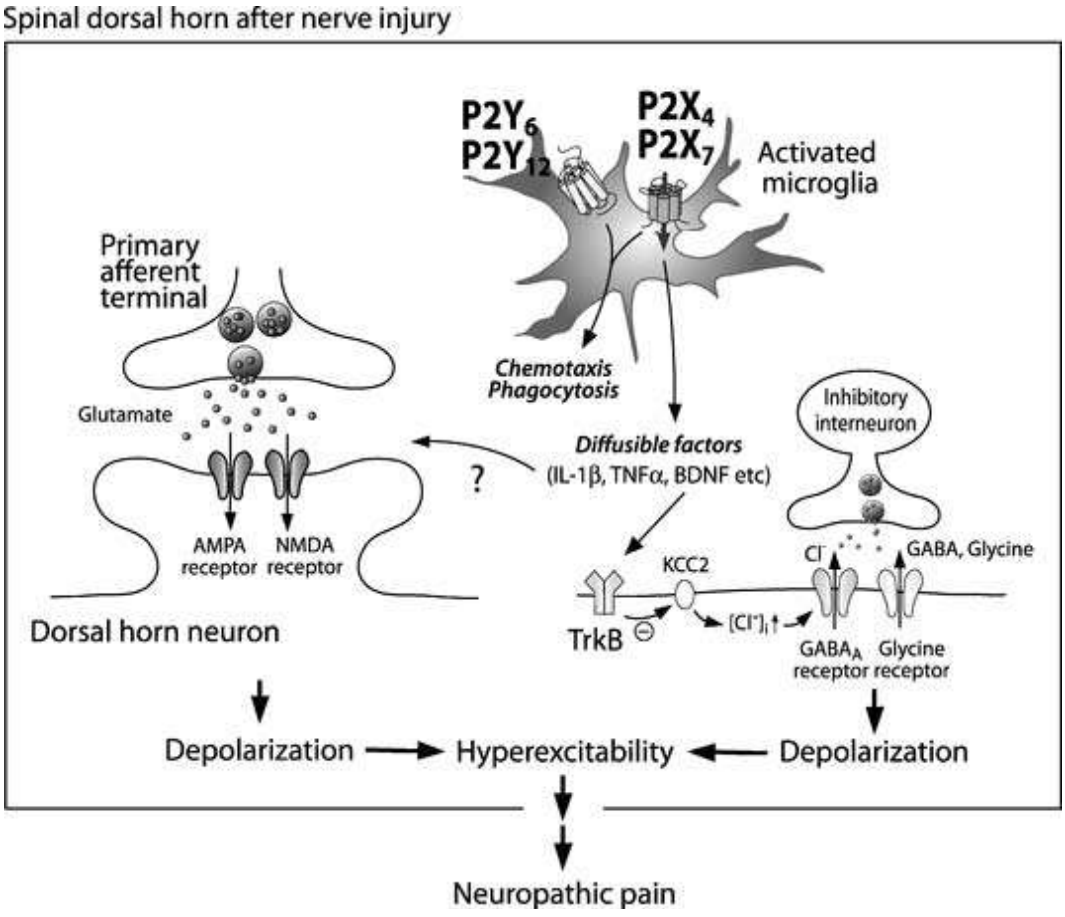
Receptor Families: The high energy ATP, with all its bonds intact, is handled by the P2 receptor class, (a "higher level family" in the organization). The lower energy AMP and final breakdown product, adenosine, still have roles to play as neurotransmitters, and are handled by the "lower level operatives", the P1 receptors. But the "higher level family" of receptors, P2, is now known to have subtypes. Both P2X, and P2Y receptor subtypes play important roles in the Dorsal Root Ganglia (DRG), and the dorsal horn where primary sensory afferent nerves pass off their signal to the second level neurons.

Individual Players in the Network (of Receptor Subtypes) Have Now Been Identified.

Introducing the Players in the P2X Subfamily: The microglial cell P2X receptor subtypes that have been identified to date as playing a role in pain modulation include P2X3, P2X2/3, P2X4 (newly identified in crystalline form), and P2X7.

A P2X5 receptor has been identified, but not found to be expressed by microglia, or have any role to play in pain modulation. (You might call it the "black sheep" of the family.) All members of this subfamily of receptors work the same way. ATP binds externally to the "cation channel", which, since the active channel thus produced allows both calcium and sodium ions to rush in, is termed "non-selective." So P2X receptors are non-selective "receptor-mediated", or "ligand-gated", or even "transmitter-gated" ion channels. Once these calcium and sodium ions rush in, they may stimulate activation of other ion channels, including voltage-operated calcium channels, and other enzyme systems may be stimulated "downstream", including tyrosine kinases, and MAP kinases.

The P2Y Subfamily: This subfamily is more surreptitious. ATP binds externally,



and an embedded "G-protein" sitting in the cell membrane, becomes excited and causes a signal to be passed to the interior of the cell, which is now known to enhance excitatory neural activity by affecting the action of glial cells at the neuronal synapse, affecting other

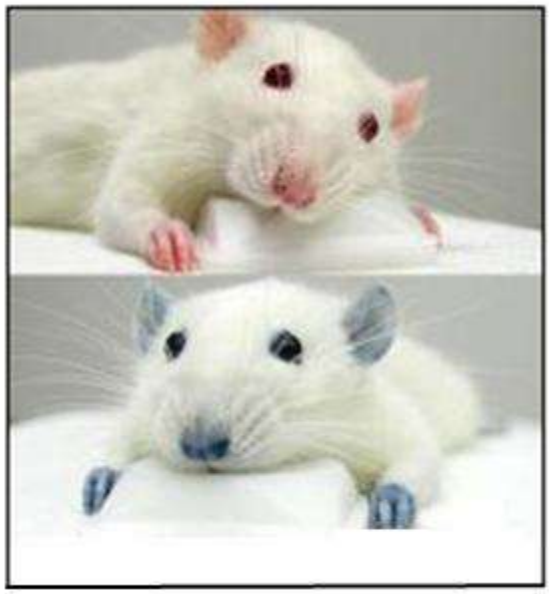
regulators of neuronal activity at the synapse, and by propagating neuronal activity that is calcium dependent. The P2Y subfamily is a little less selective than the P2X subfamily, and recognizes both extracellular ATP, and the less energetic ADP. **The P1 Family - the "Wet Blanket" Family**

AMP, a "low-energy" actor in this drama, and its final breakdown product partner, adenosine (ADO), can bind to the P1 receptor, and if this occurs it tends to quash further ATP release. So in contrast to the P2 family's tendency to be excitatory to neurons, the P1 family, tends to put the brakes on excited neuronal signaling.

P2 Family Receptors and the Many Related Signaling Mechanisms Involved in Sensation

Various research findings support the central role of ATP receptors in sensation, and the promotion of the chronic pain states. ATP Receptors are also found in taste buds (P2X2 and P2X3 subtypes, as in glia).

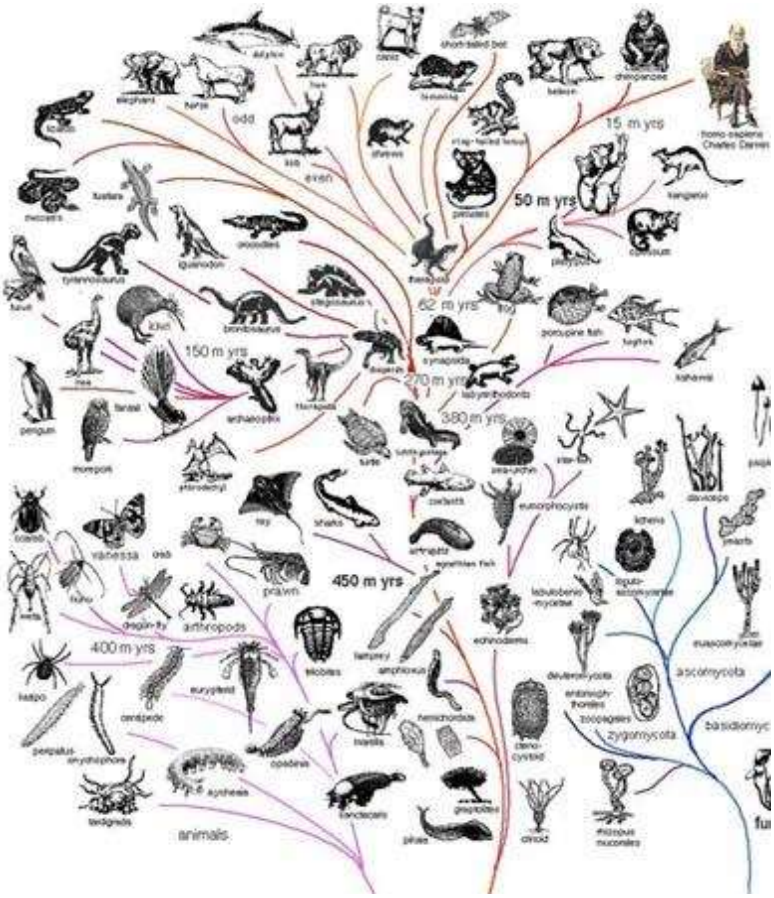
Selective P2 Receptor Antagonists, Including the "Blue Dye" Protection for Spinal Cords



Much of the research to date involves the mouse or rat experimental model, with some variation between human and rat P2X7 receptors specifically identified. Brilliant Blue G dye was used in experiments with laboratory rats, and those so treated had a quicker recovery of some spinal cord function after induced spinal section. It is a close chemical cousin of the FD & C Blue No. 1 dye used commercially, as in human sports drinks. Unfortunately this will not work in human spinal cord injury, because this agent is more selectively active for rat type P2X7 receptors.

New ***in vivo*** studies enhance our understanding of the role these specific receptor subtypes play in pain modulation. Many pharmaceutical products are currently being utilized in human safety and efficacy trials, including those for pain control, and there is still much ongoing animal pharmaceutical research.

The breakthrough drug Clopidogrel acts on P2Y12 receptors in platelets. This pharmaceutical success gave impetus to further ATP receptor subtype research, which may yet lead to a breakthrough in control of chronic pain. ATP receptor subtypes are also found in many other areas throughout the body that require very nuanced regulation and counter- regulation like the muscle in the artery wall, and the inner endothelial cell layer, in brain, heart, bone, the bowel, the immune system, and other major organs, which may well be targets for future research.



We Are Not Alone: The 2009 determination of the crystal structure of the zebra fish P2X4 receptor by researchers at the **Oregon Health and Science University** should be considered one of the most important breakthroughs in the last two decades. Their

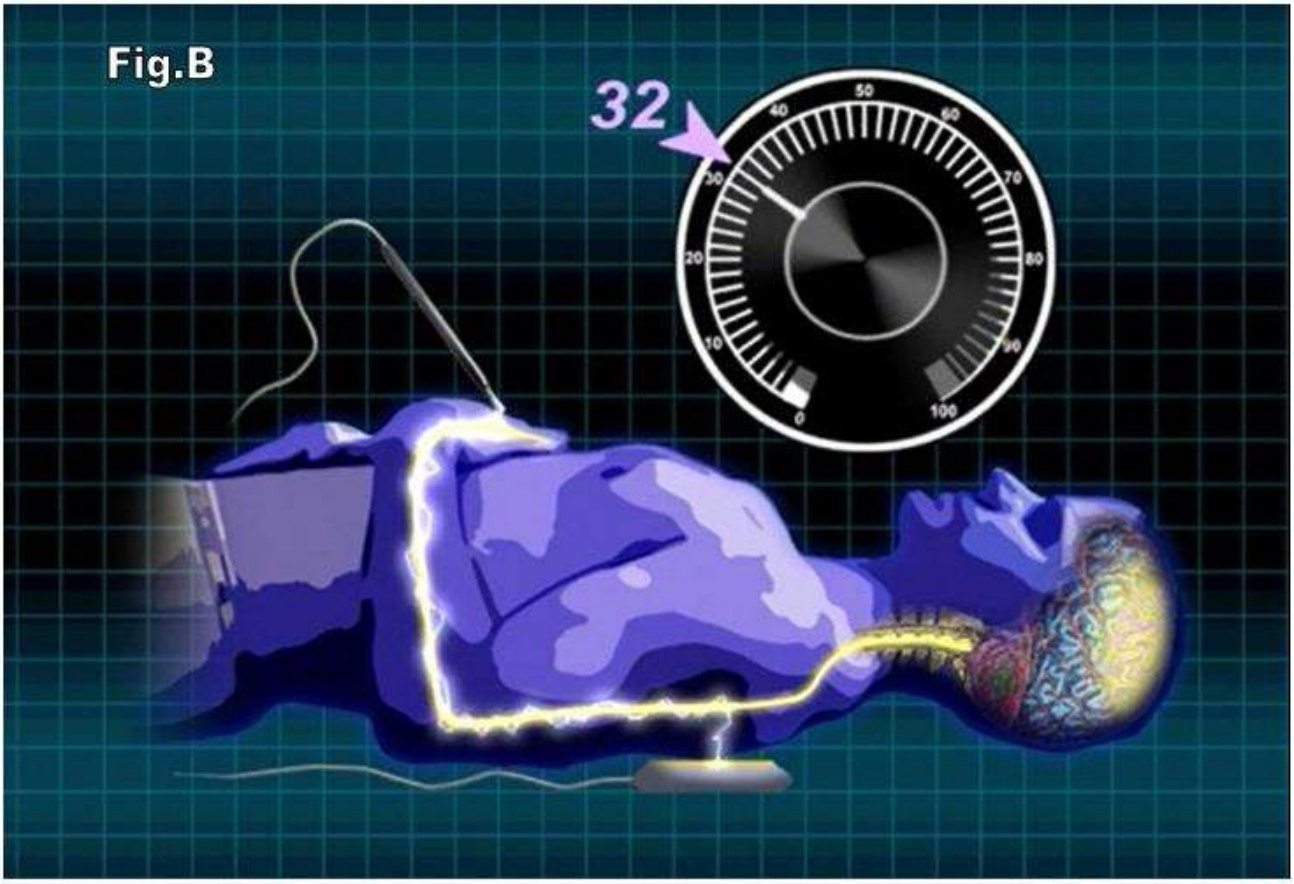
discovery that ATP receptors are also found in plants and primitive life forms, such as worms, amoebas and slime mold indicates a central signaling role early in life's evolution.

Chapter IV

Performing the pf-NCS

WAIT 3 SECONDS **AFTER TELLING PATIENT TO SAY NOW THE INSTANT HE FEELS THE SLIGHTS SENSATION AT OR DISTAL TO THE ELECTRODE. WHY? BECAUSE IT TAKES A PERSON ABOUT 3 SECONDS TO SHIFT HIS ATTENTION FROM HEARING YOU TO AND PUT HIS ATTENTION ON THE SITE.**

DO NOT TURN BELOW 5, DO NOT LIFT ELECTRODE.

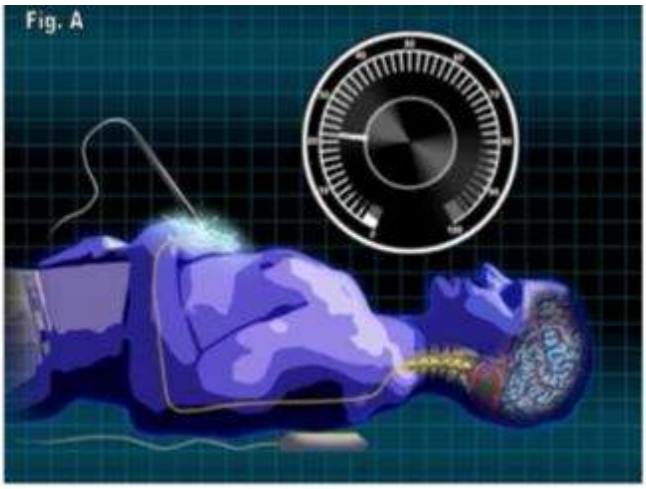


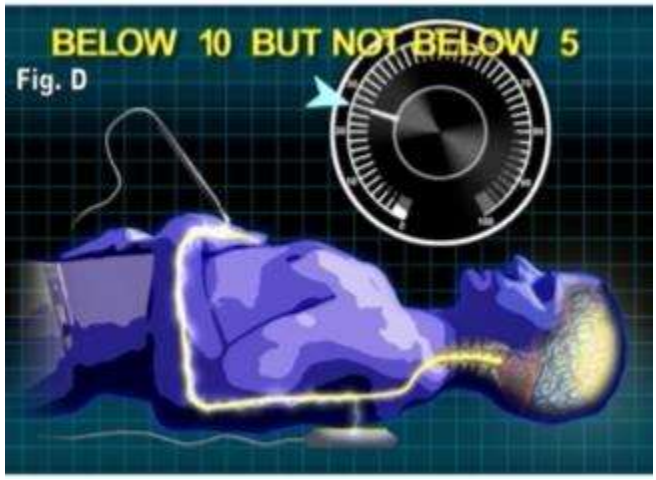
PART I - PF-NCS Basic Steps

Step #1 Breaking Electrical Impedance: By far, the most important thing for the examiner to understand is that the voltage intensity required to break the skin's resistance is usually higher than the intensity causing the A-delta fibers to fire a threshold spike (action potential). This means that until the skin's resistance is broken, the voltage cannot reach the nerve. This also means that the sensation the patient feels when the resistance is broken is stronger than what s/he will feel on the second test, when the minimum voltage causes firing. This difference in sensation should be explained to the patient so s/he understands that the second test will probably feel much weaker.

The skin's electrical resistance is overcome in the same way that the air's electrical resistance is overcome before lightning strikes. As energy builds in a storm cloud, the air becomes charged (ionized), which forms a pathway along which the electricity can flow. When the air becomes sufficiently ionized the lightning strikes, traveling between the cloud and the earth (ground).

The pf-NCS is the same as lightning. As the dial is turned up and the energy increases, an ion charged pathway is formed between the sponge ground (earth) and the saline-soaked cotton tip of the test electrode (cloud). The difference is that lightning releases all its energy when it strikes, and the ion pathway collapses. In the pf-NCS, when the skin's resistance is overcome the ion pathway persists so long as the dial is not turned too close to zero. This is why the testing protocol is to break the skin's resistance, and then immediately turn the dial down to a little below 10, but not to zero. At a setting of 10, approximately 1 mA at 2.5 volts of electricity is passing through the body to maintain the ion pathway. This allows an approximate measurement of the threshold to be made the second time the dial is turned up.





In Figure A, the dial is being turned up but the ion pathway has not as yet formed and the current is not passing through the body. In Figure B, the resistance is broken at a dial setting of 32 and electricity passes between the test electrode and the ground. Notice that the voltage has triggered the nerve membrane to fire, and a second or two later the brain feels the sensation. In Figure C, the dial is back at 10, and without firing the nerve, the trickle of electricity is maintaining the ion pathway.

Step #2 Estimating A-delta Fiber Threshold: The second time the dial is turned up, a close estimate of the voltage intensity causing an action potential can be made. He may be expecting the sensation to be as strong as the first. Therefore, It is important to say to the patient: **"This time it will feel much weaker, so pay close attention and say 'NOW' the INSTANT you feel the slightest tickle."** Wait 3 seconds to allow the patient time to focus his attention back on the test site. In Figure D, the patient feels the second stimulation at 26. Again, the dial is turned back immediately to 10, which maintains the ion pathway.

Step #3 - Close In On The Threshold: Step #3 to repeat Step #2 until you are satisfied you have found the threshold. On these test is sufficient to say; "Again" and **WAIT 3 SECONDS before starting the dial up**, since the patient is now expecting the sensation to feel weaker.

Step #4 Exact - Objective - Measurement: Same as Step #2-3, tell the patient to say NOW the INSTANT he feels the sensation, wait 3 seconds before starting the dial up. When the dial is 2 points below the setting where he has been saying NOW, step on the foot peddle while you continue to turn the dial up. When he says NOW step on the foot peddle again. The peddle starts and stops the recording of the real-time waveform.

Sensory injury causes a reduction in glutamine neurotransmitters, so a stronger than normal stimulus is needed to initiate firing of a threshold A-delta action potential (nerve impulse).

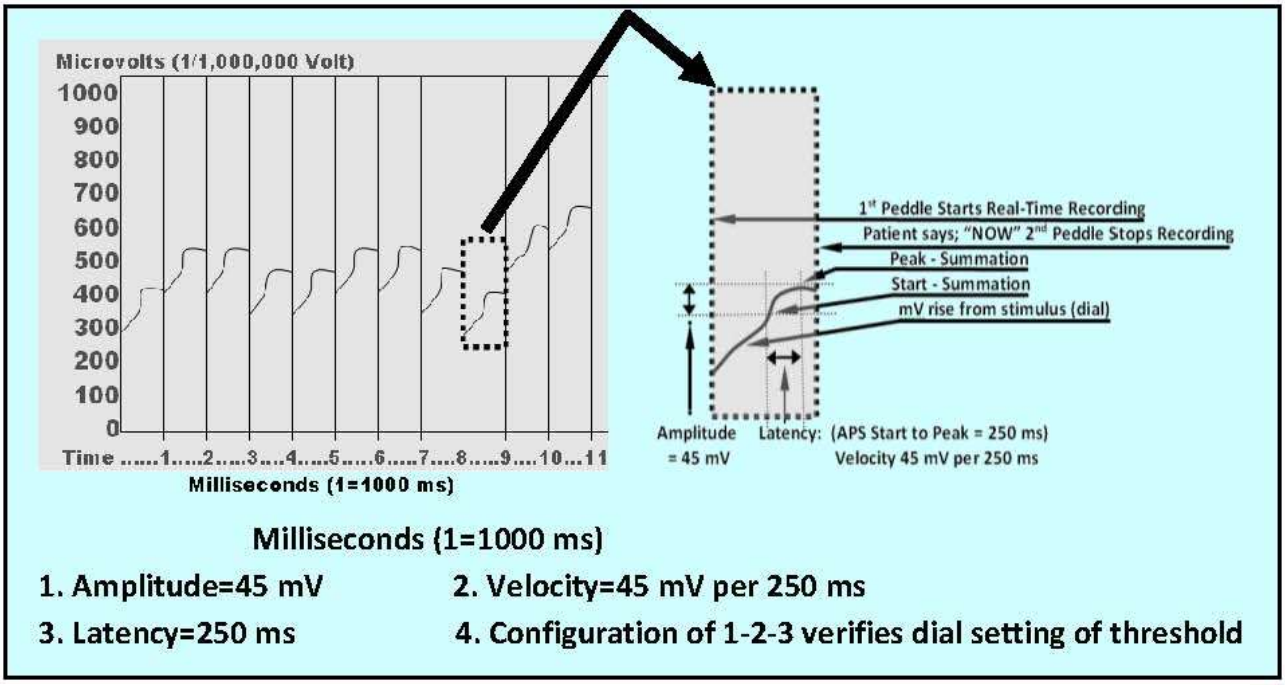
REVIEW - THRESHOLD ACTION POTENTIAL: At threshold a subject feels a slight sensation when a stimulus is strong enough to cause a sudden increase in the number of fibers that are firing. For example, near threshold the number of firing fibers ramps up, say from 200 to 400 to 1000, then, within a few milliseconds (ms) the number firing jumps to > 3 million. This spontaneous jump is termed Action Potential Summation. Potential = Voltage. Action Potential = Moving Voltage (i.e., voltage moving along the nerve fiber as each voltage-gate opens producing a burst of voltage that opens the next and the next.) Motor fibers are 50 times larger than A-delta fibers and require a loss of at least 50% of the myelin covering before EMG/NCV can begin to detect changes in velocity, latency or configuration. Therefore, these responses are of no known diagnostic value in A-delta fibers, however, combined they verify the strength of stimulus required to cause a threshold firing.

Step#1: Break the skin impedance.
1. Tell the patient; "You will feel a very slight tickling sensation. Say NOW the INSTANT you feel it here (wiggle the Q-tip) at or near this electrode." WAIT 3 SECONDS BEFORE STARTING THE DIAL UP.
2. When he says NOW, quickly turn the dial back to 10 - DO NOT LIFT THE Q-TIP - DO NOT TURN TO ZERO.

A patient's mind is wondering, so always give a warning that you are going to test again. Without a warning threshold measurements will be inaccurate.

Step# 2: Find the dial setting where he says NOW:
1. Tell the patient; "This time it will be MUCH WEAKER, Say NOW the INSTANT you feel anything." WAIT 3 SECONDS BEFORE STARTING THE DIAL UP, so he has time to refocus back on the Q-tip.
2. When he says NOW, turn back to 10 and say; "Again" WAIT 3 SECONDS before starting the dial up.
3. Repeat, and each time turn a little slower to find the dial setting where he says NOW. ALWAYS TURN BACK TO 10 IMMEDIATELY, and NEVER LIFT THE ELECTRODE or TURN THE DIAL TO ZERO.

Step# 3: Record Waveform:
The brain takes a second or two to process the sensation and say now, so firing actually occurs at a setting just below where he says NOW. For our example let's say he is saying NOW at 30.
1. After finding the setting where is says now, test one last time and press the foot-peddle at 28 (2 points below where he says NOW). Turn up slow and smooth and press the peddle as you hit the number. The first foot-peddle press starts the waveform recording.
2. When he says NOW, press the foot-peddle again to stop the recording.



PART II - Recruitment - More Objective Proof

Recruitment is a process wherein, as nerve fibers fatigue, adjacent fibers are induced (recruited) to fire. Recruitment increases total voltage output detected by the potentiometer. It is more important to prove that a nerve is abnormal than to verify that a nerve is normal. Recruitment gives the strongest evidence that abnormal A-delta fiber hypo-functioning exists.

After completing the test, return to the nerve with the highest amplitude measurement, which is likely the main pathological nerve. Break the skin's impedance as explained in Step #1 and then proceed through to Step #3. Do not expect the measurement to be exactly the same as before because testing often shifts the nerve's sensitivity. This will be explained later in this chapter. Once the threshold is detected, turn the dial down 2 points and leave the dial at this lower setting for 60 seconds. If the nerve is not firing the potentiometer number will fluctuate within a small range but will not increase. Next, turn the dial up the 2 points to where the sensation is felt and leave the dial at this setting for 60 seconds. If the nerve is firing the potentiometer number will increase. Recruitment at the higher amplitude, and not the lower, verifies that the higher dial amplitude causes conduction of an action potential.

PART III - Detect Malingering & Technical Misconceptions

Malingering: If recruitment is noted within the normal stimulus intensity and the patient reports he feels nothing, then he is trying to defeat the test. Given enough time recruitment will eventually increase to the point that the patient will experience an involuntary withdrawal reflex, which proves he is a malingerer.

There are a number of reasons why an experienced examiner can easily detect an attempt to defeat the test based on the patient's perception alone. **A) Weber & Fechner Principle:** Based on the sensory discrimination principle of Weber and Fechner, sensations innate to humans, such as temperature, pressure and etc., cannot be judged within the same narrow range that we can detect a voltage threshold sensation. Therefore, if a patient repeatedly reports that he feels the sensation within 1 dial point he can only be reporting the threshold sensation. **B) Brain Interpretation:** The cuneate nucleus is a major switching relay for somatosensory signals, and it is felt that sensory experience is influenced by the crossovers of transmissions in this region. **C) Multimodality of A-delta Fibers:** A-delta fibers are multimodal in that they transmit several types of sensations, from temperature (hot to cold) to pricking pain, tickling, vibration, pressure and tickling sensations. **D) Neuron Patterns/Ensembles:** Firing is felt to switch between ensembles of neurons that transmit different sensations. The concept is that the first firing may involve an ensemble that sends, for example, pressure signals. Once fired this ensemble is not as easily potentiated to fire as easily as a second ensemble that may transmit vibration, and so forth. Whatever the mechanism, or mechanisms involved, the fact is that the sensation changes with each threshold serial test. An attempt to defeat the pf-NCS involves the patient not reporting when he feels the first sensation, but waiting until it feels stronger of different and then repeatedly reporting that different or stronger sensation to be the threshold. The problem for the malingerer is that the sensation changes. The first threshold sensation may feel like a vibration, while the second is heat, the third pressure, etc. As a result, the malinger cannot give consistent responses as can a patient who is honestly reporting when he feels the threshold sensation.

Technical Misconceptions: There are both anatomical and physiological factors which make testing large fibers different from testing small pain fibers. The difference between

large fibers and small pain fibers is not only size, but also the fact that pain fibers have little or no myelin (see Figure #2 on page 22). Conventional EMG/NCV and NCS are based on gross myelin loss or severe nerve damage, which changes conduction velocity, latency and configuration. Pain fibers are too small for such tests and, since they have little or no myelin, the velocity, latency and configuration would not be diagnostic even if they could be tested using conventional large fiber methods. The diagnostic measurement for pain fiber function is the intensity/amplitude of voltage causing the conduction of a nerve impulse (action potential). Capturing the real- time amplitude, velocity/latency and configuration of the action potential summation verifies that conduction occurs at a specific intensity of voltage stimulus. It is not known if these measures are of any diagnostic significance in and of themselves, but they do act to verify that the threshold firing takes place at a specific stimulation intensity.

The color coded dial markings of most pf-NCS devices are separated by 2 point increments between the large numbers. These color marks allow the examiner to make a quick mental note of the dial position at a glance. This allows the examiner to immediately turn the dial back to 10 and then take his time looking at the dial to determine the exact measurement.

To allow the examiner to focus on the dial without distraction, it is important for the hand holding the test electrode to be anchored on the patient or table. Anchoring also prevents any sight motion, which the patient could mistake for the conduction sensation.

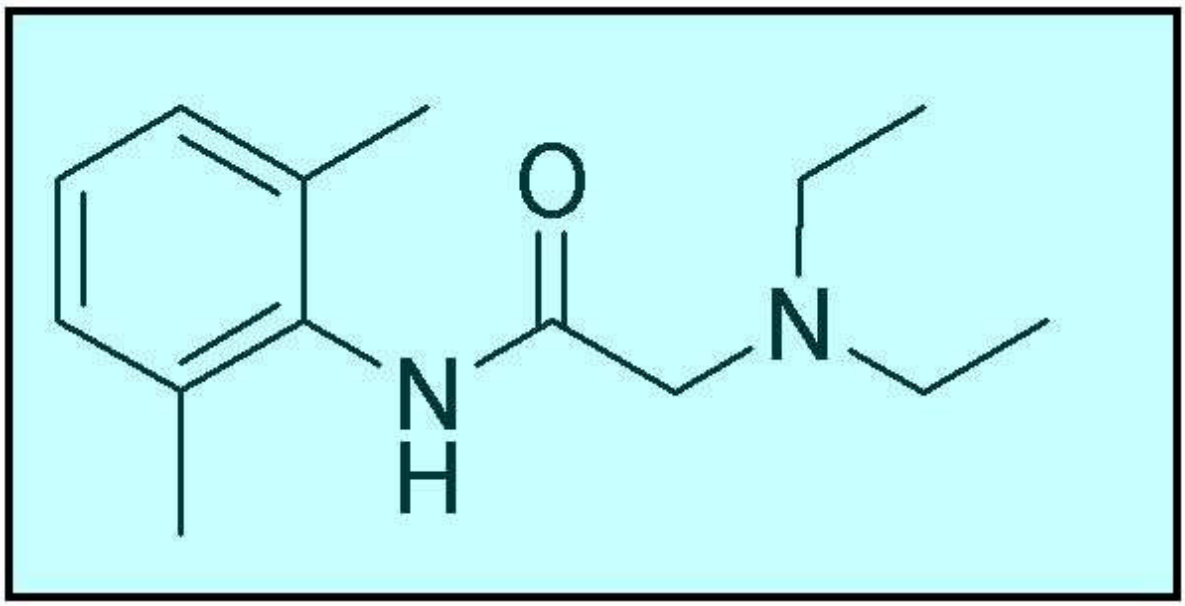
When the patient reports the sensation, the examiner immediately turns the dial down, **NOT TO ZERO**. Turning too close to zero will stop the electrical flow, in which case, breaking the skin's impedance would be again necessary. Hence, the examiner must turn the dial down to a little below 10. Meanwhile **DO NOT LET THE TEST ELECTRODE BREAK SKIN CONTACT**. Breaking contact stops the electrical flow in the same way that turning the dial too close to zero can stop the electrical flow.

Exception to turning back to 10: There are exceptions when the dial should not be turned back to 10. One exception is in cases where the skin's impedance is extremely high. This is most often seen when testing sites such as the plantar branches on the bottom of the foot. Skin thickness can cause the ion pathway to collapse at rather high settings. This can be controlled by placing the ground closer to the site being tested. For example, in testing the foot the ground can be placed under the calf of the side being tested. The closer proximity of the ground aids in maintaining the ion pathway. However, even with the ground close to the test site, the ionization pathway may collapse at a high dial setting. In such cases what indicates collapse is that subsequent measurements are high and unstable because each test is breaking the constantly shifting impedance (this is called shifting). If sifting is noticed, turn the dial back about halfway and ask the patient if he feels any sensation. You might even try turning the stimulus off to let him feel no sensation (just the cotton tip) then, once again, turn it up until he feels it and immediately back to the halfway point to let him compare this to no sensation. In this way, the test can continue with assurance that

collapse of the ion pathway is not a factor.

Inter and Intra-Operator Repeatability: The pf-NCS examiner must be efficient and fast to avoid shifting the threshold. The good news is that there is an ample window of time within which an accurate and repeatable measurement can be made - usually a minute or two. However, regardless of how fast and efficient the examiner may be, the electrical stimulation initiates either a cascading release of histamine or desensitization by accommodation. For this reason, comparing inter and intra-operator repeatability is not practicable. Conventional electrodiagnostic tests use inter and intra-operator repeatability to demonstrate efficacy, but these tests are based on fixed anatomical changes such as gross myelin loss or severe nerve fiber damage. By contrast, the pf-NCS is based on functional sensitivity which the test itself may alter. The pf-NCS should be looked upon as a snapshot of the state of the A-delta fibers. Testing a few minutes later or hours later, in most cases, cannot be expected to result in nearly identical measures. The general pattern may be similar a day or two later, but injury and entrapment change over time, making sensitivity transient and unstable.

Patient complains of pain, but pf-NCS is normal: Findings can be completely normal in spite of the fact that the patient may complain of severe pain. In such cases, the problem is not due to nerve damage; pain is due to damage of other tissues and the A-delta fibers are simply telling the patient that he has damaged tissues. Another reason for a negative test is that at that time the patient had little or no symptoms. This points out that it is important to determine how severe the patient's symptoms are when the examination is performed, so always have him grade his symptoms using the visual analog scale (VAS).



Lidocaine Paradox: Though no formal study has been conducted, it has been reported that A-delta measurements return toward normal function following epidural injection of Lidocaine. This constitutes an apparent paradoxical reaction in which the A-delta fibers become more, rather than less, sensitive in the presence of Lidocaine. This paradoxical shift has also been reported with other types of analgesics. This possible paradox is an area ripe for study.

PART IV - Patient Setup

Before discussing patient preparation for the pf-NCS examination, this is an appropriate point to address exactly what the test is not and, thereby, avoid possible confusion.

Pf-NCS vs. Dermatome Tests: There are three things that make a dermatome test different from the pf-NCS: 1) Dermatome tests evaluate sensation mediated through the receptors: 2) Dermatome tests cover an area supplied by the cutaneous branches coming from a specific nerve-root: 3) The pf-NCS does not test a large derma (skin) tome (layer), but assesses the function of A-delta fibers in a major nerve that originates from a specific nerve-root. The pf-NCS tests a single site along the nerve and evaluates that nerve tract.

Pf-NCS vs. Quantitative Sensory Testing (QST): Many aspects make the pf-NCS different from a QST. First, QST employs naturally occurring stimuli that are innate to humans, such as hot, cold, pressure, vibration, etc. This is different from pf-NCS which uses electrical stimulation, which by definition makes it an electrodiagnostic examination (EDX). QST requires the patient to make a judgment as to a change in the strength of a stimulus, such as hotter, colder, more or less pressure, etc. The pf-NCS does not require a judgment of change, but instead a recognition of any sensation and this measurement is verified by a potentiometer, which detects the amplitude of millivoltage produced by the action potential. This objectively verifies that the nerve fires at a certain voltage stimulus amplitude, which makes the pf-NCS unlike a dermatome test or QST.

Patient Preparation

History: A comprehensive history requires listening. Since the A-delta fibers down-regulate, the source of pain is incorrectly localized by over 50% of patients, so it is critical to ask the patient where he felt pain at the onset of symptoms. If the patient can accurately recall the anatomical location of his first symptoms, it is highly probable this will correlate with the pf-NCS findings. The problem is that patients cannot be trusted to remember where they first felt pain. Therefore, if you are sure the pf-NCS was performed correctly,

trust it even if the findings show the problem to be on the opposite side from where the patient reports symptoms. The pf-NCS has sensitivity approaching 100%, which is not true of any patient's memory.

Patient cooperation: We all know how a subject can be hypnotized and given the suggestion that an ink pen is a hot poker. A similar thing can happen with patients. Most physicians are unaware that establishing rapport is identical to the first stage of hypnosis.[xliii] Another word for rapport is trust. From this state of trust the patient is transferred to a nurse who proceeds to the next level of deepening suggestibility. The nurse asks the patient to do something he normally does not do for others - he removes his clothes. This may heighten the hypnotic state and in this heightened state the patient is told what to expect during the test.

Patient Instructions: The worst statement an examiner can make is to tell the patient, **"This won't hurt"**. From childhood we have learned that when we hear this, it often means exactly the opposite, it will hurt. Additionally, most people are afraid of electricity, so avoid using the term "electrical sensations" and, simply, use the term "sensation". "Electrical" will only conjure up the idea of electrocution and pain. The examiner may want to be honest and tell the patient of all the possible sensations that s/he might feel, but this would be a mistake in most cases because the majority of patients will only remember the things that make a strong impression, such as **pricking, burning** or **electric shock**. Many examiners instruct patients by saying; **"Tell me when you feel anything"**. However, this does not convey the need to say, **"NOW"** as soon as the sensation is noticed, and the patient is left to decide for himself how the sensation is going to feel. Furthermore, the patient may have already heard about other nerve tests and how painful they are, so we need to put the patient at ease.

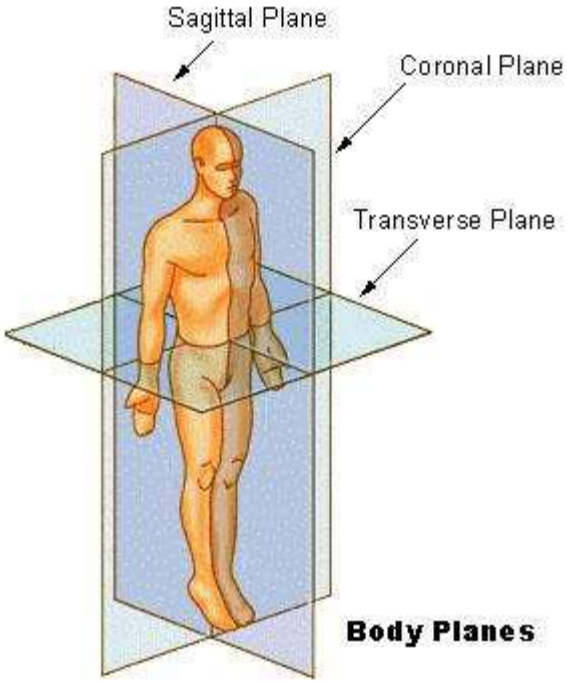
The best instruction: Let the patient form an idea in his mind as to what to expect. Hence, the best thing to say is, **"You're lucky because this test doesn't use needles and electric shocks. We call it the tickle test. We can test 5-year old children and they just laugh. I'll touch you with this cotton tip and all you do is say 'NOW' the INSTANT you feel the slightest tickle. And best, the test won't take long. The doctor does most of the work analyzing the results. OK, let's start. Say 'NOW' the INSTANT you feel the slightest tickle."** The patient has now internalized that the test is painless, it just tickles, and s/he needs to say **"NOW"** the **"INSTANT"** that s/he feels the tickle.

Test Sites: Since Benjamin Franklin's experiments and his invention of the lightning rod it has been understood that electricity follows the path of least resistance. Instead of the lightning passing through the wood house and setting it ablaze, Franklin's lightning rod conducts the electricity from a ground spike in the basement and through a wire cable to the rod in the roof. Likewise, since the test sites (Asia points) are nerve centers, they conduct electricity better than the surrounding tissues. This means that if the cotton tip of the test electrode is close to a site the electricity will travel to the site. For most sites the tip

only needs to be within 2cm to 3cm of a test site.

Blemishes: It is important to avoid scars, moles, skin abrasions and other cutaneous defects that may conduct abnormally. Generally it is best to test proximal to such defects. Remember to always test the same location on the opposite side for comparison.

Anatomical Position: The description of the test site locations are based on the anatomical position. This is especially important in the upper extremity where lateral is the thumb side of the hand and medial is the little finger side.



Part V - Examination Protocols and Test Site

Review: Typical pf-NCS Exam: In the cervical and lumbosacral tests the patient is face up on the examination table. In the cervical study the ground sponge (DRIPPING WET WITH TAP WATER) is placed under T6. In a lumbosacral study the ground sponge is placed under L4. Place a towel under the ground to capture the excess water. It is important to anchor the hand holding the test electrode so it cannot move during the test and the examiner can focus on the dial. Notice in the picture how the examiner is resting his hand

on the pillow. Press enough to dimple the skin, but not so hard that it leaves an impression in the skin. The main thing is **DO NOT ALLOW THE PROBE (TEST ELECTRODE) TO MOVE**. Hold the test probe steady. If the probe moves the patient may mistake this movement as that of the stimulus sensation.

Cervical Plexus
Ground and potentiometer electrode placement The examination forms in the **Delta NCS System** manual show the exact placement for the potentiometer electrodes. Note that some nerves are considered to be controls, however, all nerves and branches must be tested to allow for accuracy detecting pathology.

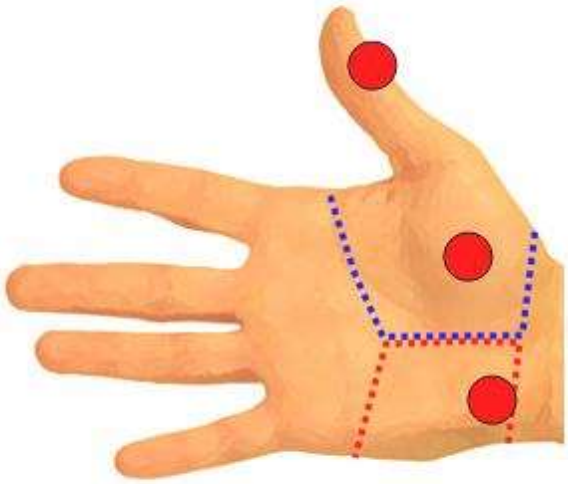
Waveform Potentiometer Electrode Placement

The **Delta NCS System** manual shows the exact placement for the potentiometer electrodes on the examination charts.

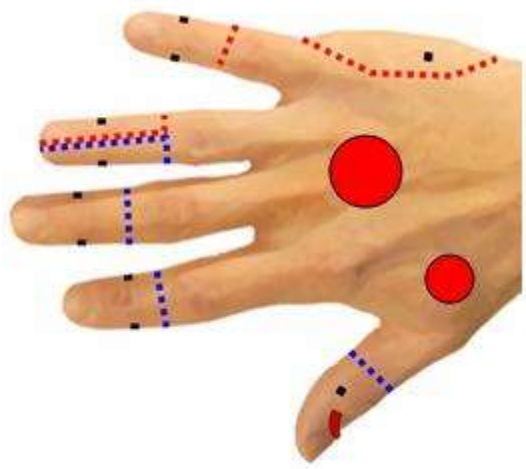
Alternative Ground Sponge Placement: When testing the lower extremities, especially the bottom of the foot, place the ground sponge under the calf and test that side. After testing one side, rewet the sponge (Dripping Wet), place it under the other calf and test that side. **The potentiometer electrode placement:** The potentiometer electrode can be moved as required to obtain clear measurements. If you cannot obtain what seems a reliable reading, the results may be recorded as EQ (equivocal). This is well within the parameters of EDX studies. In fact many times an entire EMG-type study is equivocal.

Upper Extremity Study (Graph Analysis): Place the ground sponge (Dripping Wet) under

T6 (same as the cervical study). If the patient has a pacemaker, place the ground under one scapula and test that side. Then rewet the sponge (Dripping Wet) and place it under the opposite side and test that side.



To test the palm and lateral thumb, have the patient open the hand at his side, with the palm up. After testing the palm sites and lateral thumb site, place a towel on the patient's abdomen and put his hand palm side down on the towel, then test the remaining digital sites. Dip the tip in the saline solution between each test site and briefly touch the tip to a towel to catch any drip. If the drip is left it may run across the skin and the patient may mistake this as the sensation. Repeat the above procedure on the opposite side.

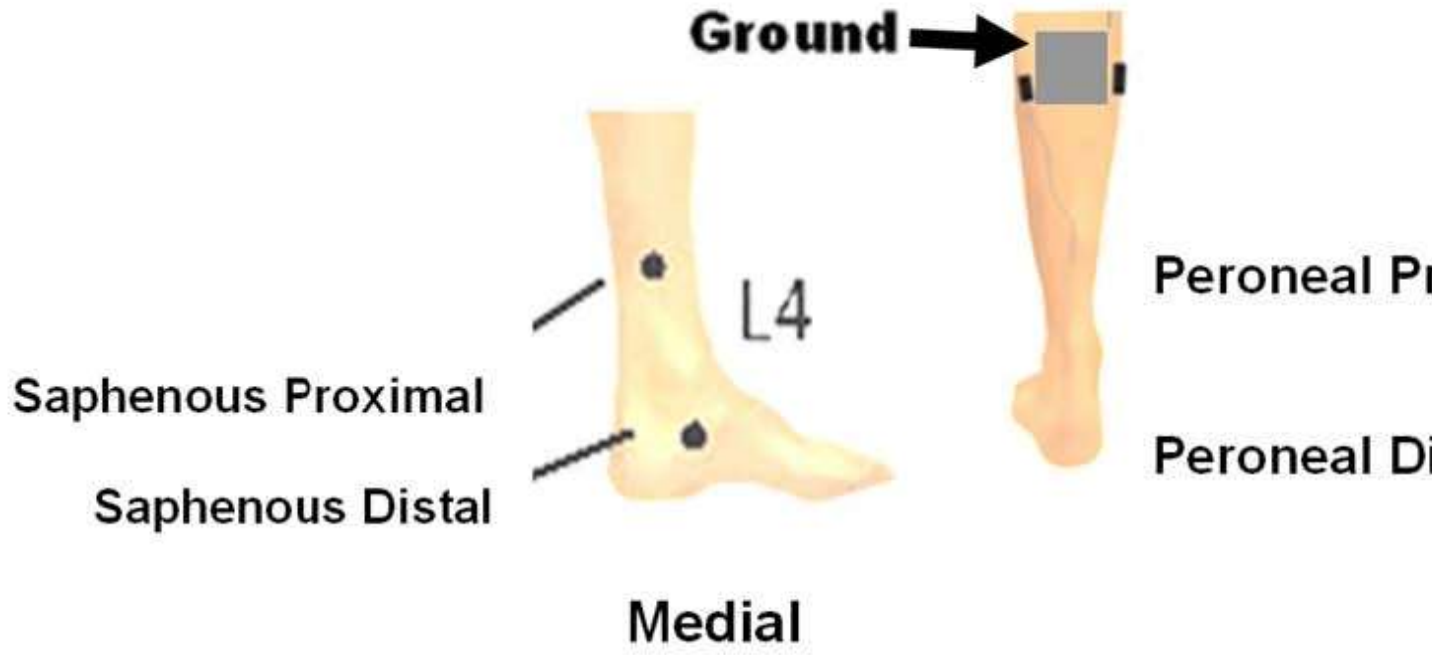


Anatomical Considerations The C6-7 nerve-roots are the origin of the median and radial nerves. They separate in the brachial plexus and travel to their respective areas in the upper extremity.

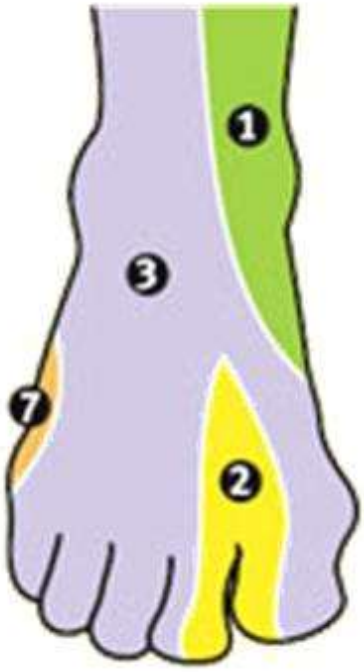
The motor branch of the median nerve passes through the carpal tunnel. However, the sensory palmar branch of the median nerve does not pass through the carpal tunnel, nor does the sensory palmar branch of the ulnar nerve pass through Guyon's canal. Therefore, sensory palmar branch dysfunction suggests possible proximal pathology.

If the digital branches and palmar branches both demonstrate dysfunction, the problem could be a double crush - the wrist and proximal. The radial branches, with sites on the back of hand, have the same origin as the median nerve (C6-7), so the radial nerve rules in or out cervical radiculopathy. Testing above and below the medial elbow detects cubital tunnel entrapment. The non-symptomatic side is used as a control.

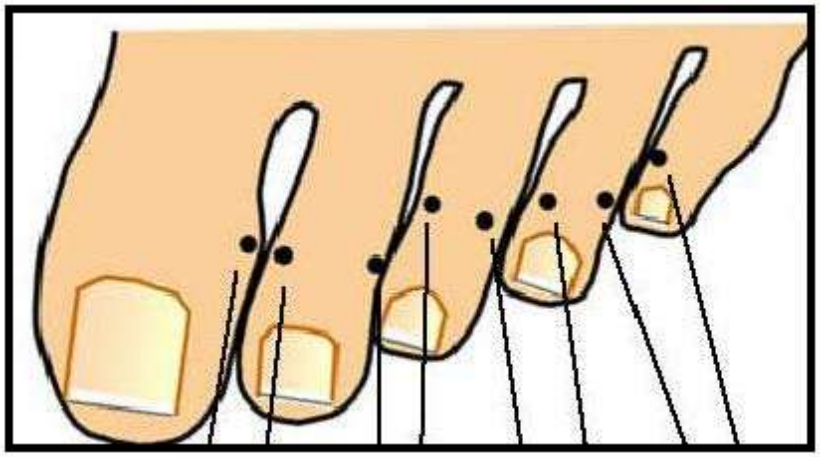
Lower Extremity Exam (No Graph - Numerical Analysis): The physician chooses which of the nerves to test; sites are tested above and below the location of a suspected entrapment. Sites on the non-symptomatic side are tested for controls. Significant findings are usually noted by a 30% or higher measurement distal to the suspected entrapment, and verified by comparing the measurement to the opposite side. If the proximal site also tests high then the problem is likely proximal, such as radiculopathy.



Saphenous nerve ❶
Deep peroneal nerve ❷
Superficial peroneal nerve ❸
Medial plantar nerve ❹
Lateral plantar nerve ❺
Calcaneal branch (tibial nerve) ❻
Sural nerve ❼

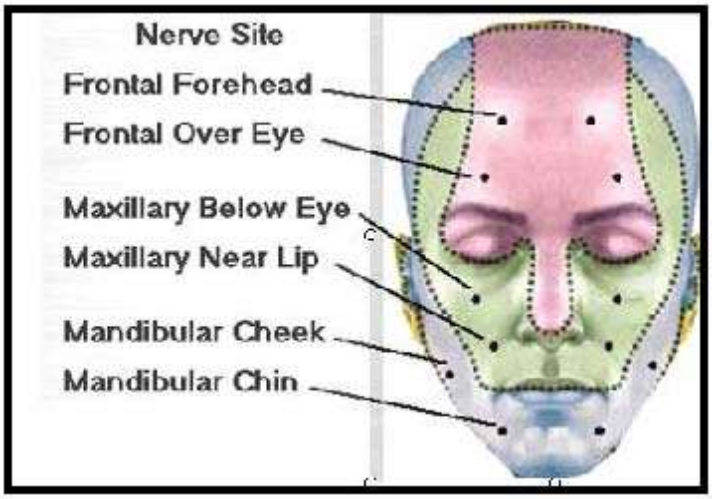


Neuroma (No Graph - Numerical Analysis): The lumbosacral or lower extremity test may be performed before to rule in or out proximal entrapment or radiculopathy.



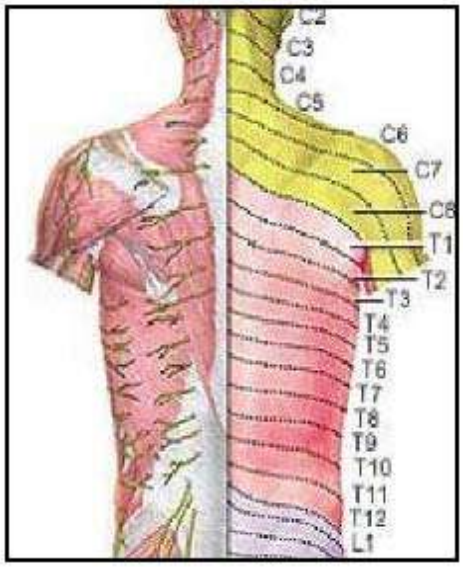
Only branches suspected of pathology need to be tested. Ground placement is under the calf of the side being tested. Remember, rewet the ground and move it to the opposite side before testing the opposite side. Neuromas are detectable by testing the digital branches associated with the suspected neuroma and comparing the measurements with the same digital branches on the opposite (i.e., normal) side. **Note: A Neuroma may cause HYPER-function.** As with all studies, the measurements supply a baseline for future comparison.

Trigeminal (No Graph - Numerical Analysis):



Consider first performing a cervical study to rule in or out pain referred from the cervical spine. Ground placement is the same as cervical study. Mark sites by drawing a circle around each. Be careful not to over stimulate the area. The sites do not need to be the same as shown on the chart. Pick the sites and mark them on the examination sheet. Test the exact same sites right and left for comparison. A 20% or more difference suggests pathology.

Thoracic Exam (Graphic Analysis):

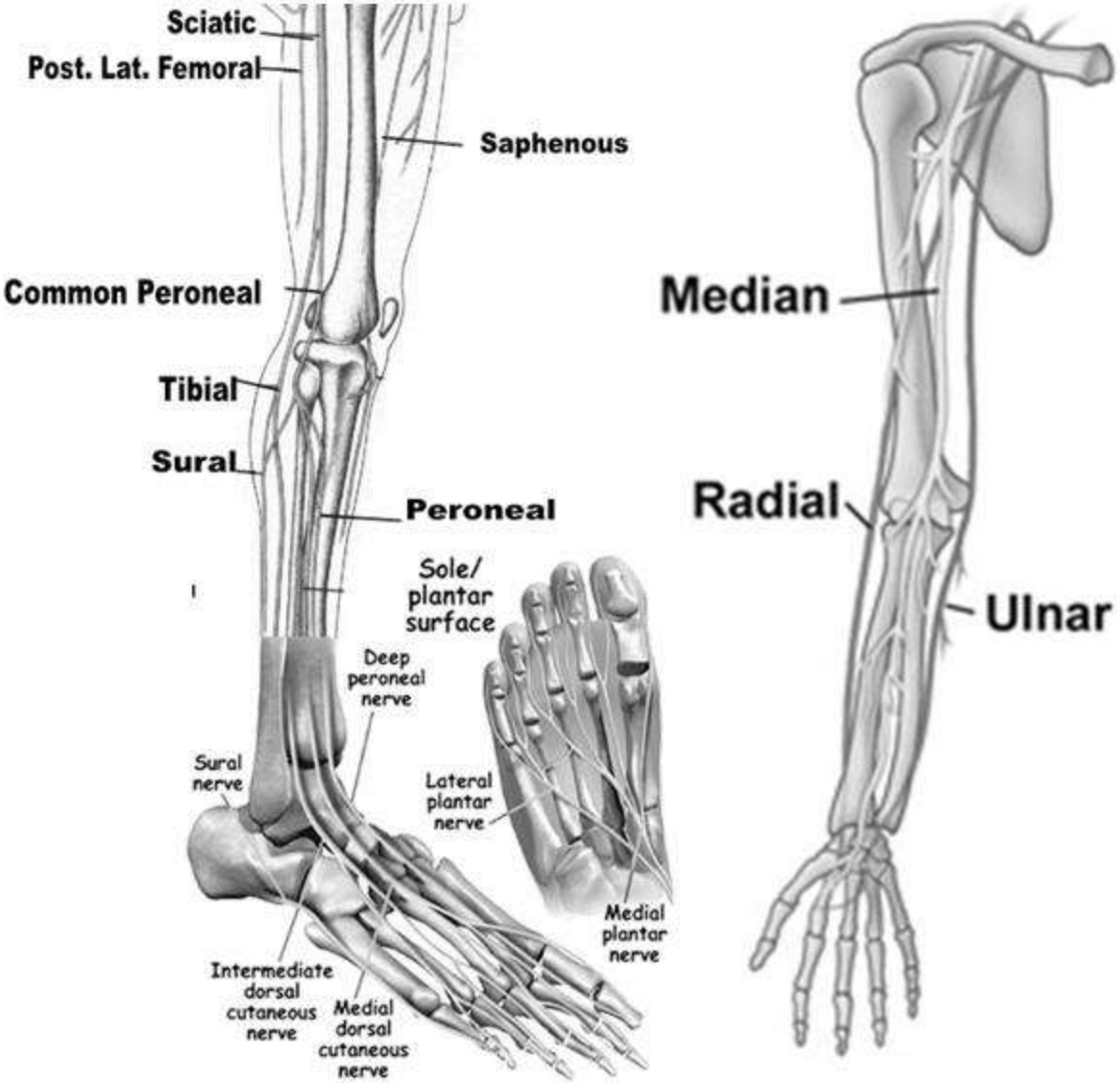


With upper thoracic problems consider a cervical study first to rule in or out referred

symptoms. If the symptoms are in the lower thoracic region consider performing a lumbosacral study. Ground placement is under either thigh with the patient sitting.

Have the patient forward flex and extend the neck while your fingers are on the tips of the lower spinous processes of the cervicothoracic junction. The last spinous process moving forward and backward is C6. Count down to T4 and place a circle 1 inch to each side of T4 and 1 inch to the side of the next 11 spinous processes. Be careful not to over stimulate the area while drawing circles around the sites. Due to anatomical variances of the thoracic cutaneous nerves measurements give a general idea of the level of pathology. (See thoracic radiographic correlation page 148).

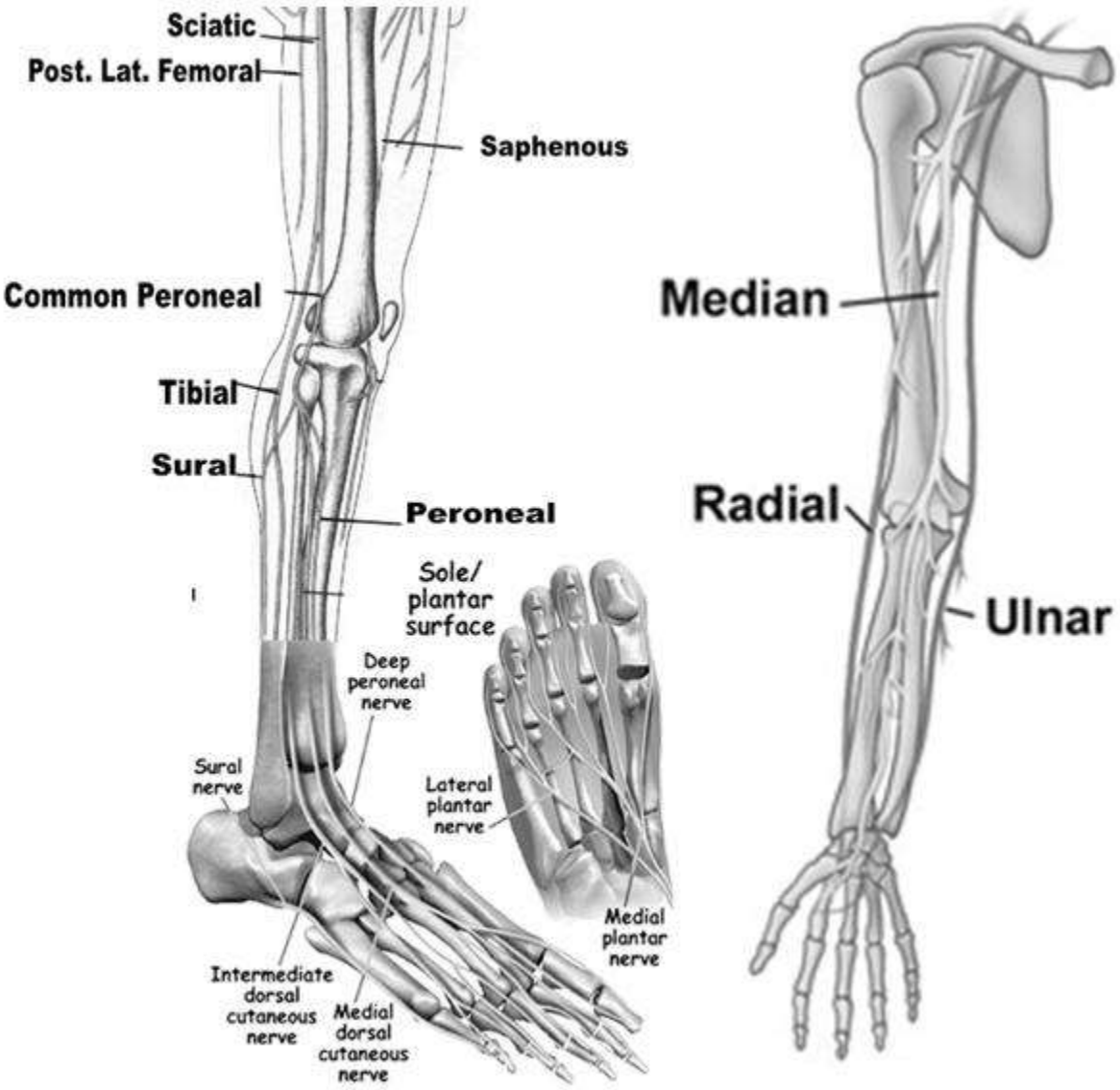
Custom Exam (No Graph - Numerical Analysis):



Always consider a study of the region proximal to the area of interest. For example, if cubital tunnel entrapment is suspected first perform a cervical study. If the problem is suspected in the area near the knee, perhaps a lumbar study should be performed first. Mark the sites to be tested by drawing a circle around the sites, being careful not to over stimulate the area. The anatomical site is typed into the software in brief terms. For example, typing, "knee femoral nerve", results in the report reading, "Above the right knee femoral nerve 45, below the right knee femoral nerve 83."

WARNING DO NOT ALLOW THE CURRENT TO PASS THROUGH THE CHEST

CONTROLS:
Controls ensure accuracy, so test the exact same sites on the opposite side for comparison. Deviations of 20% or more are significant above the navel and 30% or more is significant below the navel. Always take the history into consideration because this rule of 20% and 30% is the average deviation. In some cases lesser deviations may be significant. In any case the measures become a baseline for future comparison to evaluate change. Find the raw data at the bottom of the report.



PART VI - TROUBLE SHOOTING

Calibration

The device manuals show the steps for calibration. It should be kept in mind that a lower or high calibration will not affect accuracy in detecting pathology. All of the measurements are on the same scale, making the measurements relative. In other words, the measurements are all plus or minus by the same percentage so they are relative and, therefore, comparable. **Ground Sponge**

Many problems can be traced back to the sponge being too dry. Be sure the sponge is

DRIPPING WET with **TAP WATER**. If the sponge is too dry, the current may **not** flow effectively. For example, the dial could be turned to 100 and still the patient feels nothing. Also, a too dry sponge can cause the ion pathway to collapse at a fairly high setting as the dial is being turned down. If a reading is high and shifts then suspect the sponge is too dry. Another possible problem with a dry sponge is the patient may feel the sensation **at** the sponge.

Do not soak the sponge in saline.
Tap water works quite well; saline can cause itching and possibly cause the patient to develop a mild rash.

Pacemakers
In a cervical study where the patient has a pacemaker or in any situation where the examiner is not sure if it is safe to place the sponge between the shoulders, place the ground (Dripping Wet) under the scapula and test that side. Then rewetted sponge and move it under the other scapula and test that side. **DO NOT ALLOW THE CURRENT TO PASS THROUGH THE CHEST.** Pacemakers are a concern only in upper body tests, i.e., cervical, trigeminal and upper extremities.

Potentiometer:
1) Do not remove the sticky gel over the three black contacts.
2) Wipe the skin with alcohol. **LET IT DRY** before placing the electrode.
3) If the potentiometer screen reads **MEMORY FULL**, don't turn it off. Leave it turned **ON** and in about 15 seconds it will return to normal operation. It does this because the potentiometer has a memory which allows it to interface with a computer to generate a graph; this is not needed in the pf-NCS.
4) If the potentiometer does not seem to be working it is often the cable. Look in the manual for the page regarding ordering accessories. This page includes information related to the cables and electrodes, which are not covered by the limited warranty.

Test Check List:
Avoid Patient Confusion
It only confuses the patient to provide a list of what he may feel. Instead explain to the patient that s/he may expect to experience a slight tickle, and that it will feel much weaker after the first sensation at each test site. After testing about four sites patients usually remark that it feels different at each site and even different each time at the same site. Confirm this experience by saying, **"Yes, it may feel different, but remember to say NOW the INSTANT you feel any sensation."**
Do **not** talk while turning the dial; this is a distraction.
Do **not** ask questions, such as; **"Do you feel it?"** If you think the patient missed the threshold sensation because the measurement is too high, turn down the dial to 10 and say, **"Tell me, again, the INSTANT you feel the tickle."** Wait 3 seconds and start turning the dial up. The patient will often feel the sensation at a distant point along the nerve being

tested. For example, testing T1 (at the point above the medial elbow) the patient often reports feeling a sensation in the little finger (ulnar nerve). Assure the patient that this is normal. The brain is feeling the sensation from the nerve that comes from the little finger.

PART VII - Sympathetically Mediated/Maintained Pain, Allodynia

General Considerations: Usually an injured nerve's threshold sensation is not different from that of a normal nerve - both are a tickling sensation. However, the A-delta fibers transmit hot, cold, vibration and pressure. Therefore, a patient may report the threshold sensation as any of these. In patients diagnosed with one of the several types of pain syndromes, referred to as allodynia, it has been observed that the threshold sensation is not a tickle but a painful sensation, which causes a withdrawal reflex away from the electrode.

Testing a patient suspected of having allodynia is a challenge. If the skin's impedance is much higher than the threshold the breakthrough sensation experienced when overcoming the impedance may be uncomfortable. For this reason it is necessary to tell the patient to pay attention before the second test because the sensation **may feel much weaker** than the sensation associated with breaking the impedance. Note that if a patient reports an uncomfortable sensation on the first breakthrough, this does not ultimately suggest allodynia. However, if electrode contact is not broken and the dial not turned to zero and then the next test causes pain, this may suggest some type of allodynia.

ll To prevent the patient from pulling away from the electrode, it may be necessary to both assure the patient that the stimulus will be turned down immediately when he feels anything and to hold the test electrode in such a way that the patient cannot pull away. Only then can the second test determine if the nerve's threshold sensation is truly painful.

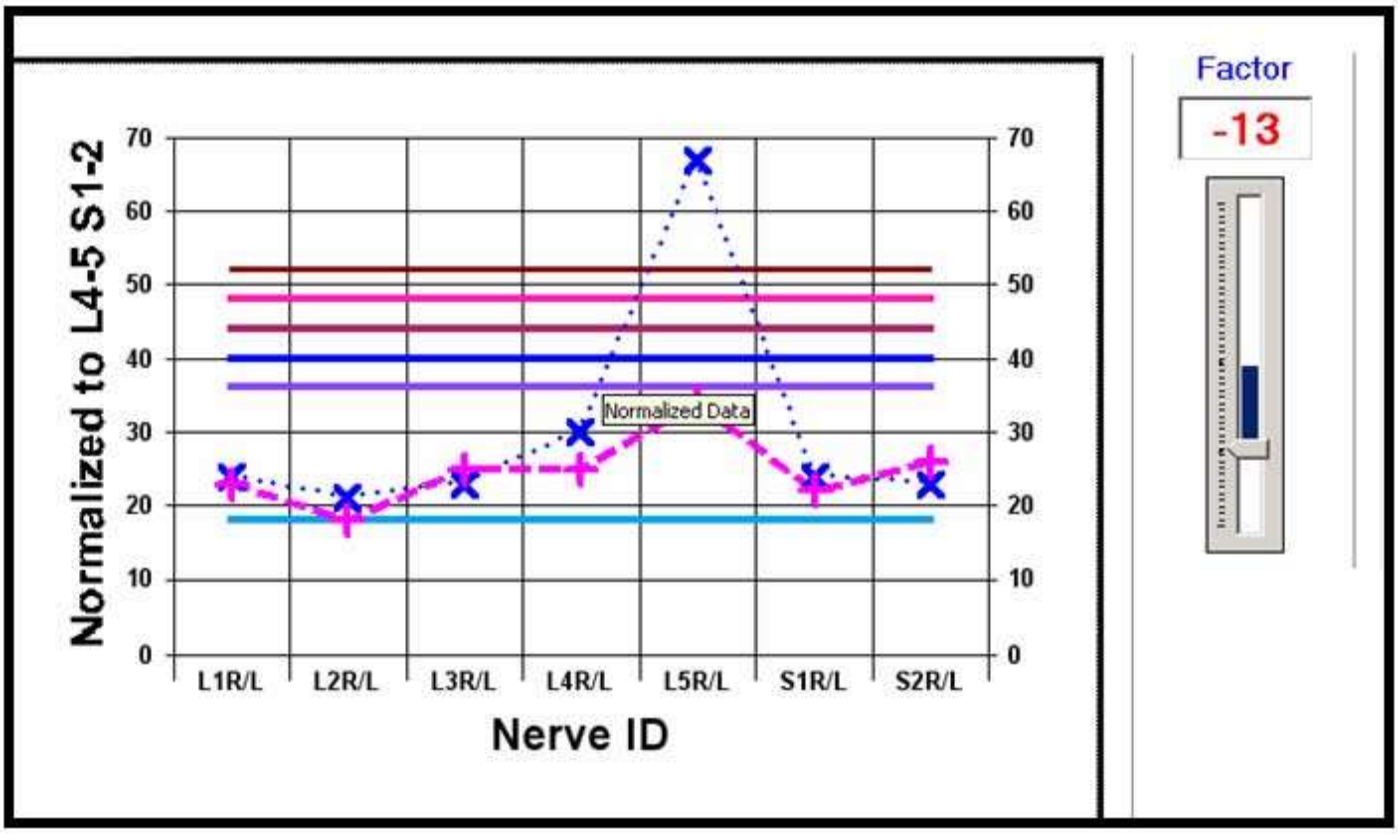
Baseline Data: If allodynia is suspected, it is recommended that the nerve-root levels above and below the suspected nerve be tested for threshold and pain tolerance threshold using all three frequency settings: 2000 Hz (A-beta), 250 Hz (A-delta) and, 5 Hz (C-Type) fibers. Threshold can be quickly estimated without using the potentiometer. Pain tolerance is measured by asking the patient, **"Tell me what this feels like as I turn it up."** Then, turn up the dial at a steady rate and the instant the patient pulls away from the electrode immediately remove your fingers from the dial. The dial setting is his pain tolerance threshold. These tests are invaluable as baselines to monitor these disorders.

When testing all three fibers, it is recommended that the 2000 Hz sensitive (A-beta) fibers be tested first, since they transmit painless light touch sensations. Next the 250 Hz (A-delta) fibers are tested for pain tolerance since they have already been tested for threshold. The last should be the 5 Hz (C-Type) fibers. Before each test it might be effective to say, **"Let's see if you even feel this."** If you tell the patient to tell you when it hurts, he may, due to the power of suggestion, say the first sensation hurts.

Polyneuropathies: Diabetic polyneuropathies advance from the lower extremities upwards. The earliest functional change occurs in the lower extremities and is detectable by hypo-function in the A-delta fibers. Though a comparison can be made between the general difference of the feet, hands and neck, looking for what is termed "stair-stepping", wherein the feet are more hypoesthetic than the hands, while the hands are more hypoesthetic than the neck, it is reported that simply performing a lumbosacral study looking at the general change in the long nerve: Saphenous (L4), peroneal (L5) and sural (S1), allows detection of early onset polyneuropathies. Indeed, it has been reported by many endocrinologists that early detection of A-delta fiber hypo- function has greatly reduced the number of diabetic patients undergoing lower extremity amputations. In these types of cases, if may be desirable to test with all three frequencies to supply more data to follow the progress of the disease.

Chapter V

Analysis



PART I - Software Assisted Analysis

The analysis is assisted by software that employs three methods dependent on the anatomical region and the suspected type of pathology. All three methods are based on detecting diminished A- delta fiber sensitivity (hypo-function).

Analysis Methods

1. Graphic Assisted Analysis
Cervicothoracic, lumbosacral, thoracic and upper extremity studies employ a graphic assisted analysis, which use the patient as his own control.

2. Side to Side Comparison

The trigeminal study is a side to side comparison. The A-delta measurements are compared with the contralateral matching sites. Sites are chosen within any or all of the three regions of the trigeminal distribution: frontal, maxillary or mandibular.

3. Proximal & Distal Comparison & Contralateral Control
The third method involves testing proximal and distal (above and below) a suspected entrapment or site of injury and comparing measurements with the exact same sites on the contralateral (asymptomatic) side. The cubital tunnel study is an example of this, in which the ulnar nerve is tested distal and proximal to the medial elbow with the contralateral (asymptomatic) side used as a control. Note: The terms 'distal' and 'proximal' are in relationship to the central nervous system (CNS); the body part furthest away from the CNS is distal, while the part closest is proximal.

PART II - Radiculopathy Analysis

Over 50% of patients incorrectly localize the source of neck and back pain, and 20% localize pain so poorly that they identify pain as coming from the side opposite from the injury. Hence, there is no diagnostic value in testing only those nerves suspected of pathology. The software for the cervicothoracic, lumbosacral, thoracic and upper extremities studies is programmed to process data only when all the test site data is inputted. This insures sensitivity approaching 100%. The cervicothoracic study requires data from 18 sites (9 on each side); the lumbosacral study requires data from 14 sites (7 on each side). In most cases the patient has pathology of a single peripheral nerve or one to three nerve-roots, so the other sites act as controls. The data literally reveals the patient's unique bell-shaped curve. Pathology is identified as the high number, while the lowest measurements suggest possible irritation (hyper-function) at the other end of the curve.

Software Averaging: Averaging is exactly that: all the patient's measurements are averaged and the software program places the average in the center of the **NORMAL ZONE**. The measurements are arranged relative to the average. Those higher on the graph required more than average voltage to cause conduction. On a bell-shaped curve these would be at one end while the lower measurements are at the other end of the curve. The nerve with the greatest loss of A-delta function is at the highest end of the curve (highest on the graph).

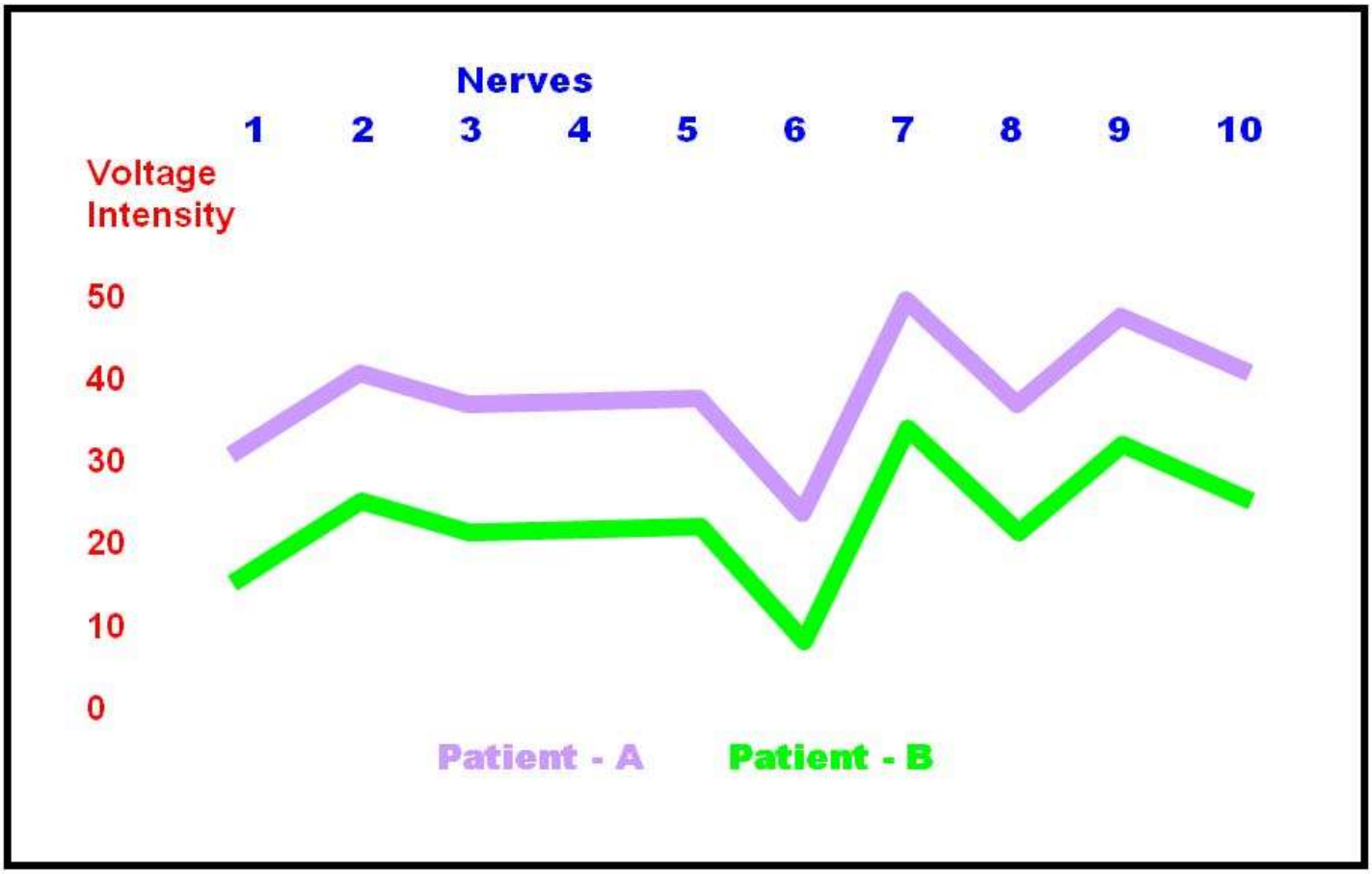
Hypo-Function: The extreme of hypo-function is no function, i.e., death. Hyper-function is the opposite, in that it is probably a normal nerve reacting to irritation from adjacent tissue activity or inflammation.

Variations - Normal Zone: The skin's thickness and nerve depth cause natural variations in the strength of voltage intensity required to cause an action potential. Although each

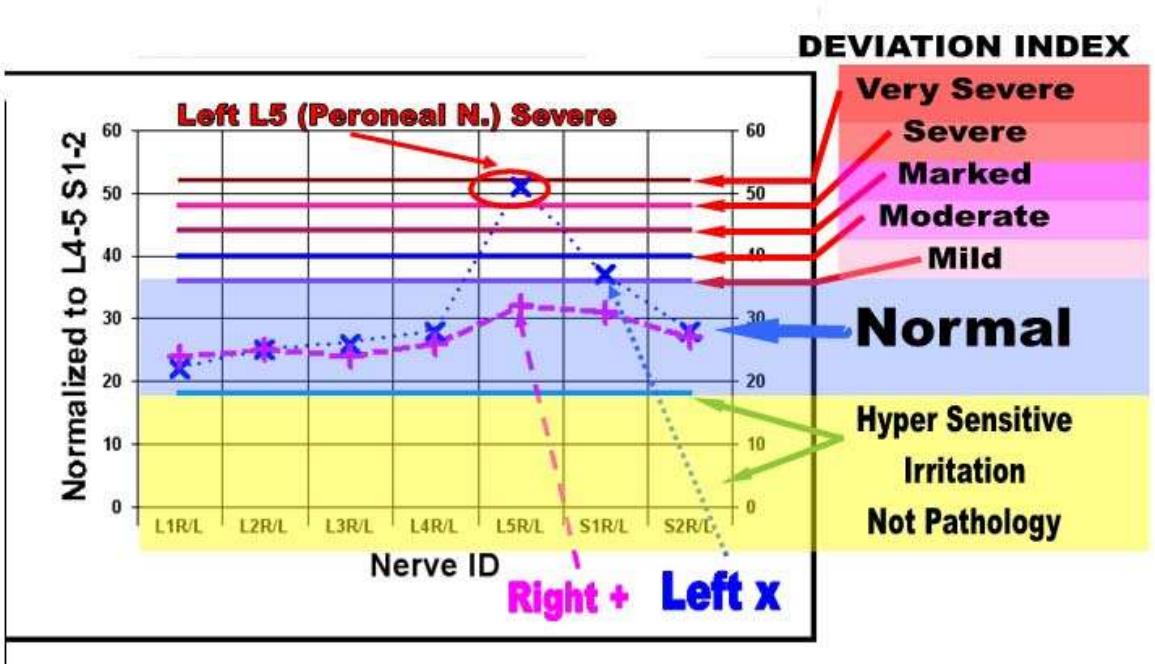
individual varies in overall sensitivity, the pattern of variation between nerves is similar.

Graph Alignment: To facilitate the analysis, the software is programmed with the top of each nerve's bell-shaped curve aligned, so that the graph has symmetrical zones rather than undulating zones. To accomplish this, the software assigns the same value to the peaks of each curve. For example, if the average voltage causing conduction of the A-delta fibers in the ulnar nerve is a dial setting of 18 (9 volts) while it is 28 (24 volts) for the radial nerve, the software is programmed to adjust for this 10 point difference. In this way a measure of 18 for the ulnar nerve is on the same line as is 28 for the radial nerve. The graph below shows the variations of two normal subjects; Patient A and Patient B. It is seen that Patient A is naturally less sensitive (more voltage is required) than Patient B (i.e., Patient A's threshold is higher), but notice that both have the same relative sensitivity variations. They have the same or very similar patterns.

The analysis rules take into consideration that the zone system is imperfect. Therefore, the **Normal zone** is wide enough to take into account natural variations but still narrow enough to detect mild rated pathology. The analysis rules are explained in Part III of this chapter.



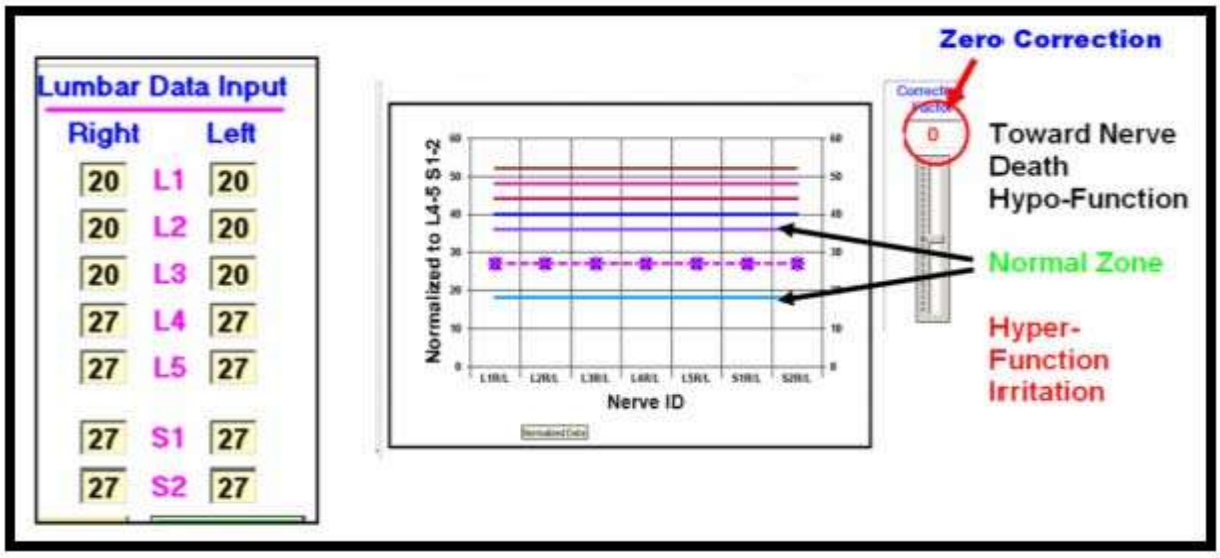
Graph Layout: The graph is a grid upon which measures are compared. The numbers along the side of the graph provide a scale for comparison. Do not expect a patient's measurements to match them because they will not match.



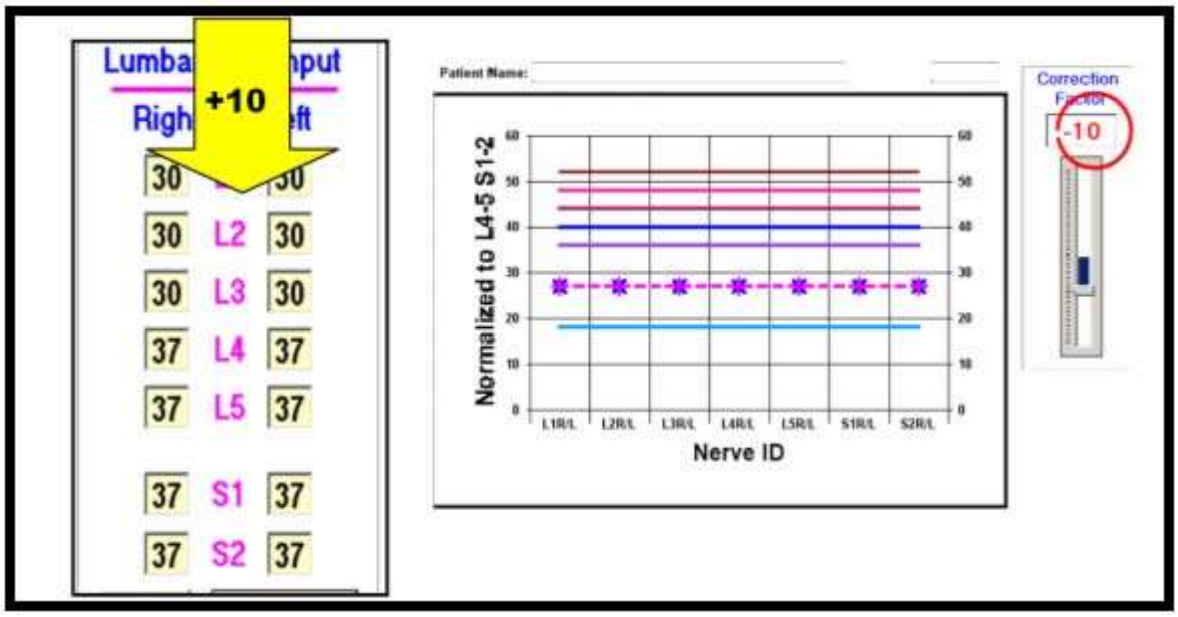
Deviation Index: The Normal Zone is located between the lowest (blue) line aligned to 18, and the above (purple) line aligned to 37. Imagine a nerve is being irritated by some adjacent process, such as a mild inflammation. If this nerve normally fires at a dial setting of 25 and it is irritated at a intensity equal to a dial setting of 15 points, then the result is that it will only require a dial setting of 10 to cause an action potential. Since 10 is lower than that patient's average, the software would place 10 in the Hyper-Function zone (below the lower blue line).

Above the **Normal Zone** are the **Hypo-Function** ratings. These ratings are divided into a **Deviation Index**, which consists of (+1) mild, (+2) moderate, (+3) marked, (+4) severe and (+5) very severe.

Averaging Demonstration: The following and next graphs demonstrate the software averaging process. This hypothetical graph is represents a perfectly normal subject. Every measurement is in the middle of each bell-shaped curve. Note: the Correction Factor is zero (0) and that all of the measurements line up in a horizontal line in the center of the **Normal Zone**.

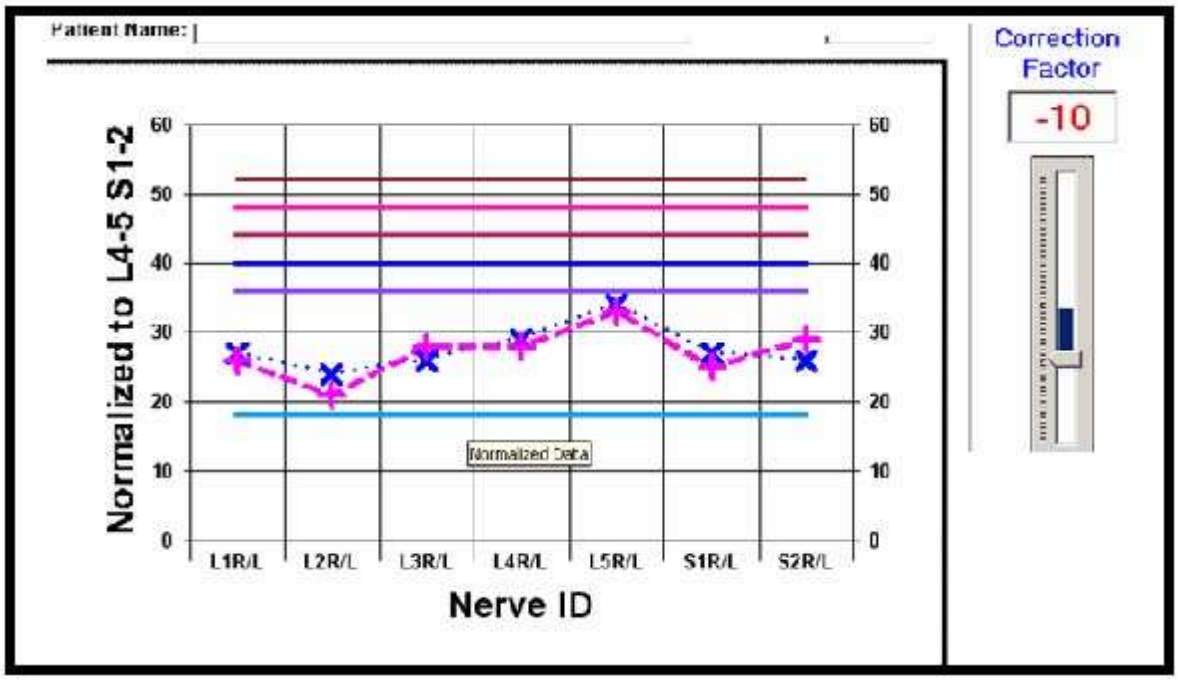


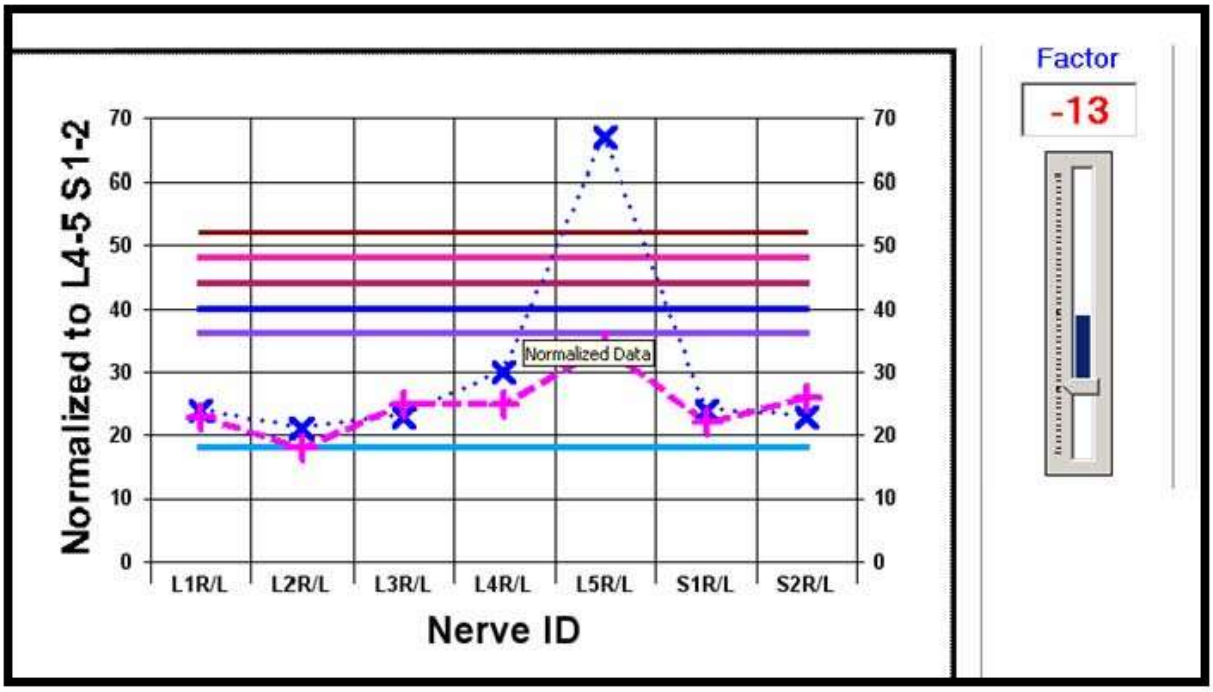
The graph below has 10 added to all of the measurements of the previous graph. Note how the software has the **Correction Factor** at minus (-10) to bring the measurements down 10 and into the **Normal Zone**. If 10 had been subtracted instead of added from the original numbers the Correction Factor would be +10 to bring them up to the Normal Zone.



The advantage of the **Nomogram** is that the patient is his own control, so it is his own bell-shaped curve and the highest measurement is his greatest pathology because it is the nerve requiring the most voltage to fire. This is why the pf-NCS sensitivity approaches 100%, while comparing measurements to population at best has 67% sensitivity.

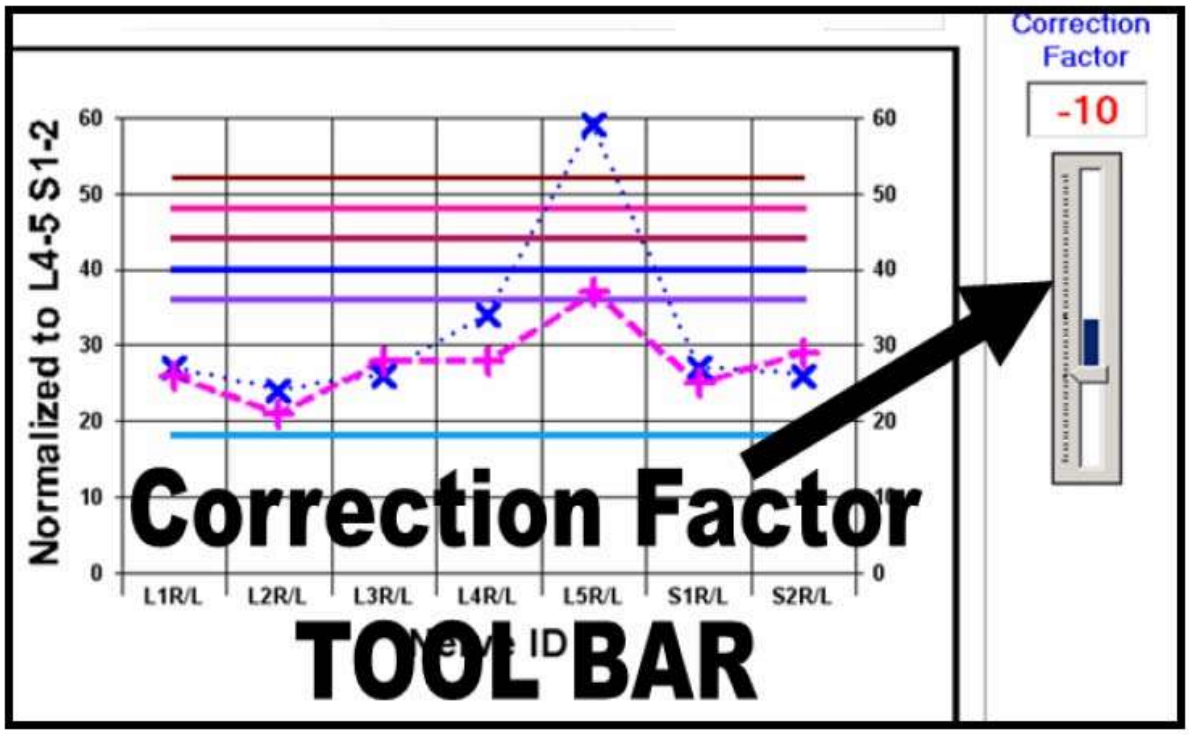
TYPICAL NORMAL: In real life, no patient's measurements line up in a straight line. However, they will be within a pattern and each pair of nerves will have very little right to left deviation. Note that the program does not pick a particular measure to place in the center of the **Normal Zone**. Inthis graph the average happens to match the right L4 (pink). The **Correction Factor** has no diagnostic meaning, but it is useful to detect an overall shift on tests at a later time. Additionally, the raw measurements are recorded at the bottom of the report, which simplifies later comparisons.





Importance of understanding averaging: This graph will help to demonstrate why it is important to understand averaging. Here, 11 of the 14 measurements are identical to the previous normal graph, but one is an exception: left L5. Notice how averaging in L5 has pushed the normal ones down. In fact, the normal right L2 is pushed onto Hyper- Function line. If left unchanged the right L2 would be named in the report as being hyper-functional, but obviously it is not. To prevent this false finding the Correct Factor tool is used to shift the measures up. This does not change the highest - the pathology. The challenge is for the physician to be able to identify the normal pattern, which the software cannot identify. With a little practice the physician will find this is quite simple.

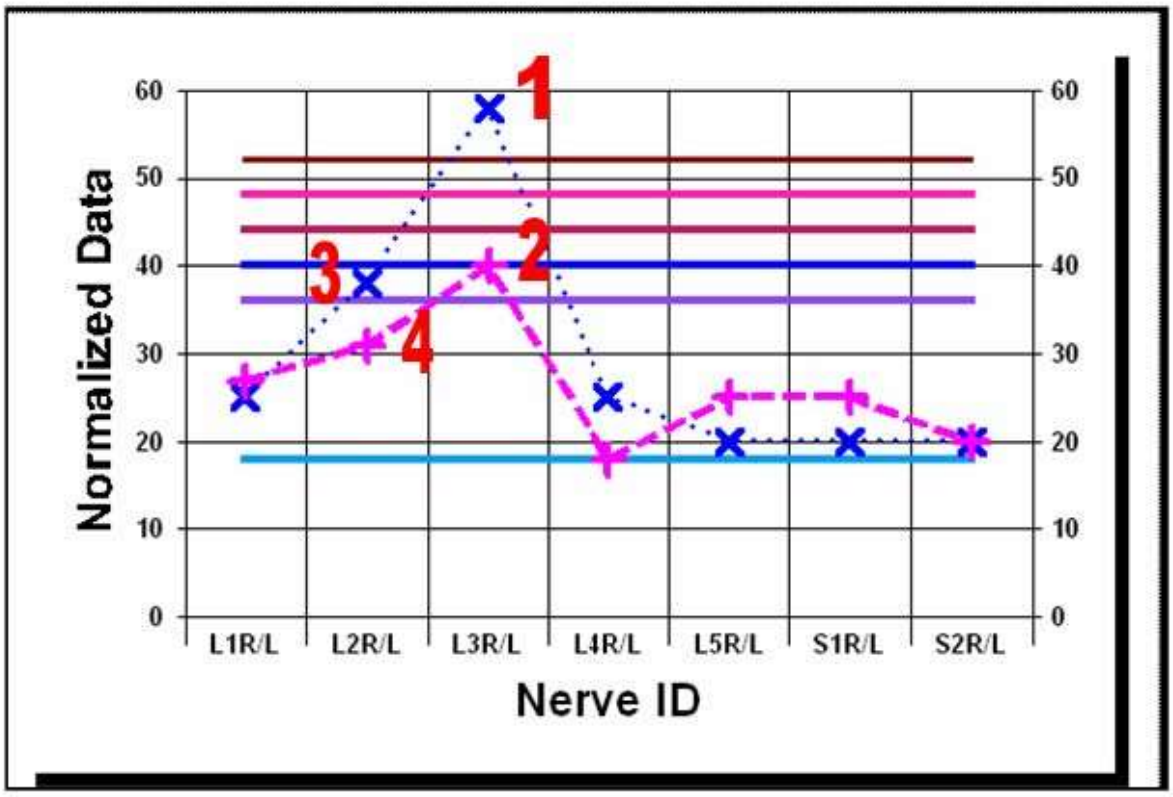
In the next graph the **Correction Factor** tool has been used to shift the measure- ments up 3 points. Now, the right L2 is no longer rated as being hyper- functional and will not be reported as such in the report. Note that this does not change the left L5 from being pathology - the highest remains the highest regardless of how the software or how the physician adjusts the **Correction Factor**. It may change the rating of the primary problem, but it remains the highest measure on the graph.



Keep in mind that the reverse can also take place: a hyper-function can push a normal measurement up, causing a normal measurement to be pushed into a Hypo-Function rating.

Can hypersensitivity suggest pathology? In some cases, **hyper-function** combined with frank pain at threshold may suggest a sympathetically medicated pain syndrome. Following injury A-delta fibers diminish in sensitivity during the **Protopathic Phase**, but the presence of hyper- function in conjunction with strong pain at threshold means continuing synaptic activity into the motor pathways. This may help initiate reflex central changes playing a part in the development of a sympathetically mediated pain syndromes.

Deviation Index Ratings: There are several reasons why the ratings do not necessarily correlate with the degree of pathology or the intensity of pain experienced by the patient. For example, chronic pathology will often be rated in a mild (+1) to moderate (+2) zone because usually over the course of two or more years, the spinal cord down-regulates the filtering of signals and allows more signals to pass to the brain. This mechanism is termed **disinhibition** and is discussed later in this chapter. Likewise, patients may have severe loss of A-delta function with measures in the +5 (very severe) rating and have little or no symptoms because diminished function equals numbness, and not necessarily pain. Chronic pain is dependent on C-fiber activity and if the C fibers quite down, then there may be little pain, in spite of quite severe pathology. Few patients notice numbness, especially if there is concomitant pain at an adjacent spinal level.



Mirroring: The side opposite pathology often displays a similar pattern. This graph shows typical mirroring. The left and right L3 is the highest. Left L3 is labeled #1 in the graph and the right L3 is #2. Left L2 is third highest, #3, and right L2 is the fourth highest, #4. The cause of this arrangement is based on the way the neurons interconnect in the spinal cord. Here, the left L3 is injured, so the interconnectivity of the primary neurons is as follows: Left L3, labeled #1 on the graph, is the highest because the greatest number primary neurons synapse with secondary neurons on the side where they enter the spinal cord. The second greatest number labeled #2, right L5, crossover to the opposite side of the spinal cord and synapse. The third greatest number, labeled #3, left L3, has primary neurons which ascend one level and synapse. Lastly, the fourth greatest number labeled #4, right L2, which ascend one level, crossover and synapse. Therefore, it can be seen that this natural anatomical configuration between primary and secondary neurons causes the opposite side to mirror the pattern of the pathological side.

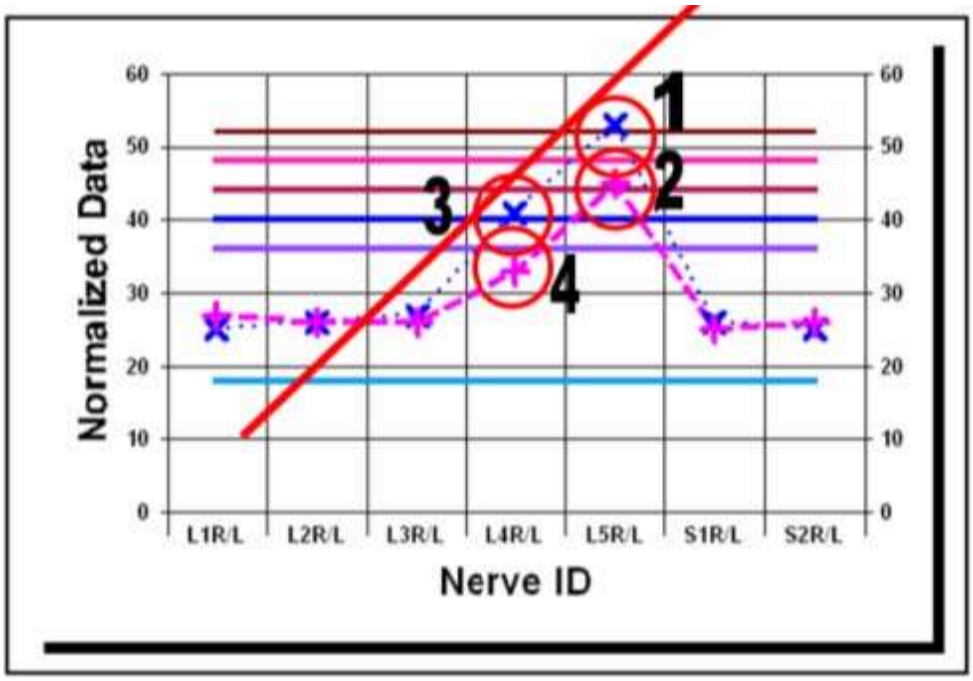
PART III - Three Analysis Rules in Radiculopathy

1: The Highest Measurement Identifies Pathology

2: Absent Rated Dysfunction Deviation May Suggest Pathology

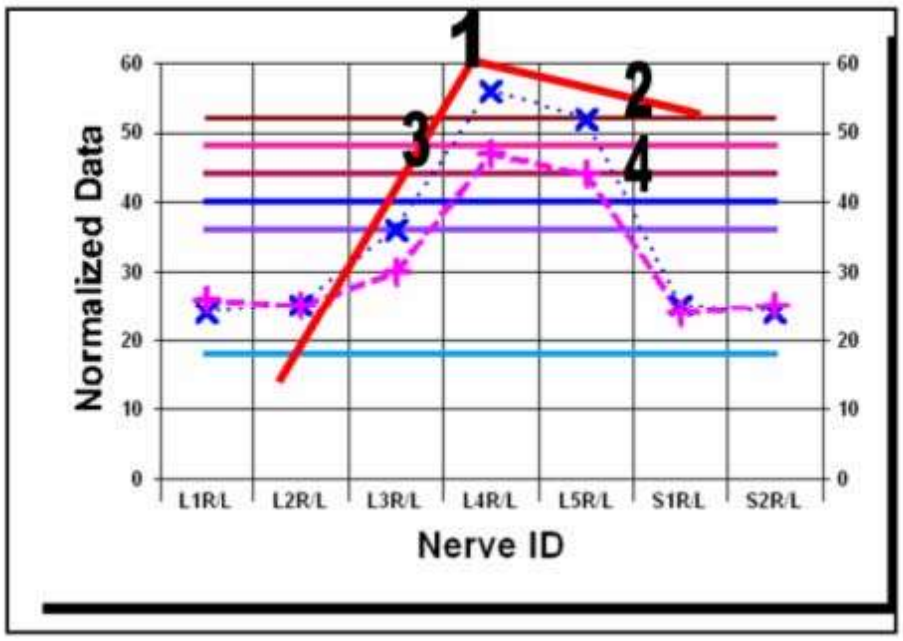
3: Pathological Pattern Recognition

Rule #1: The Highest Measurement Identifies Pathology: Nerve-root pathology causes diminished A-delta function, so the highest measurement identifies pathology. As seen in the previous section, the primary nerve affects adjacent normal nerves and those on the opposite side. It is quite easy to see which nerve is the highest on the graph, but the challenge is to differentiate mirroring from secondary pathology.



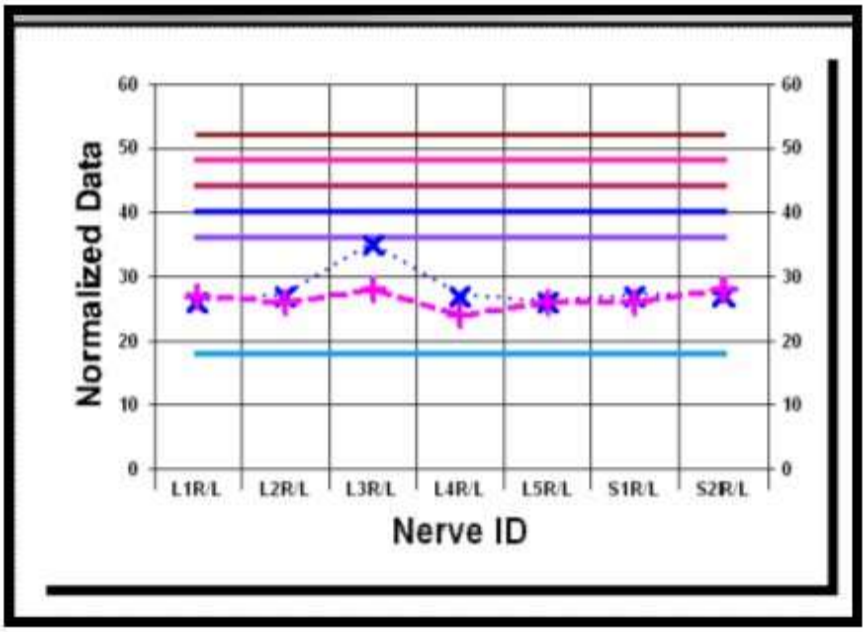
Mirroring: In this graph, only the Left L5 (labeled #1) is pathological. Mirroring is noted because the next highest is the opposite side, Right L5 (labeled #2) because it receives more fibers from #1 than the #3. L4 (labeled #4) receives less than #3. Keep this pattern in mind while looking at the next graph, which has two pathological nerve-roots.

Two Radiculopathies: The bottom graph is the same as the previous one with the exception that the Left L4 is also pathological. It is no longer third in order, now it is the highest and the Left L5 is second highest. Left L4 may not be as severe as the Left L5, but the mirroring affect from the Left L5 is added to the hypo- function of the Left L4.



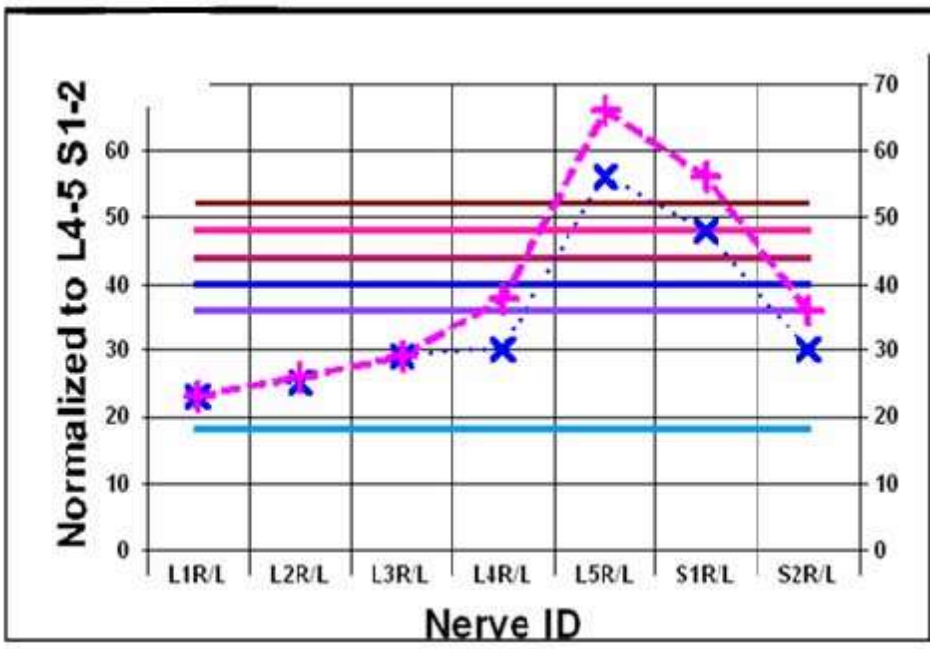
Generally measurements of the nerves entering the spine below a pathological nerve-root will drop down on the graph (toward normal), as is seen in the two previous graphs.

Baseline Data: There are many cases of multiple radiculopathies, especially when the patient has been involved in multiple traumatic episodes, failed surgeries, etc. In any case the graph serves as a baseline that can be used for comparison following treatment to evaluate change. The entire test need not be repeated. The pf-NCS gives the pathology a number that allows specific comparison.

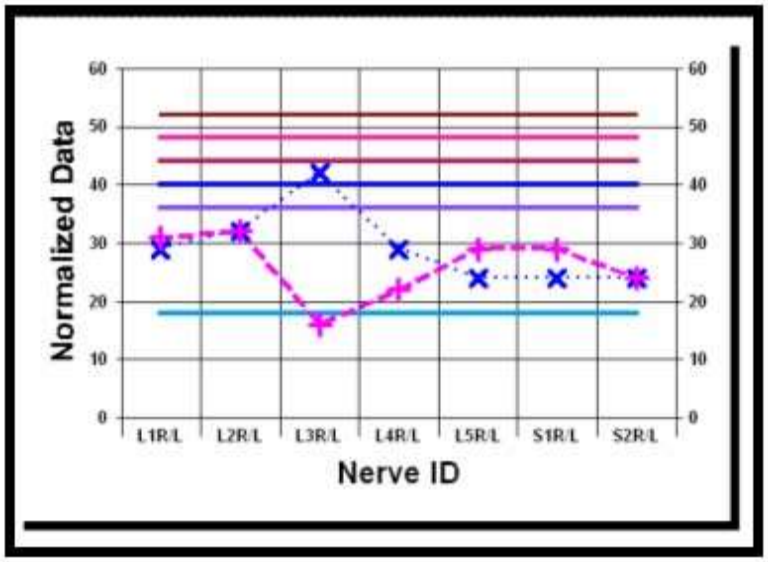


Rule #2: Absent Rated Dysfunction, Significant Right/Left Deviation Suggests Pathology: In this graph all the measurements are within the **Normal Zone**, none are hypo or hyper-function. However, the Right to Left deviation is 30% at L3. Calculating deviation is accomplished by dividing the smaller number into the larger, subtracting 1 from the Left side of the decimal and moving the decimal 2 places to the Right. For example: 23 into 30 = 1.30 = 30%. In this graph it is seen that the Right L3 "fits" the normal pattern, while the Left L3 does not. Note too that the Left L3 is the highest measurement, which follows Rule #1. Therefore, even though the Left L3 is within the **Normal Zone**, Right to Left deviation "Rule #2" suggests that if pathology is present it is most likely the Left L3. What is required now is a careful review of the patient's history and correlation with other findings.

Rule #3: Pathological Patterns: Piriformis Entrapment: The graph below demonstrates a pattern first identified by Randall Cork, the Chairman of the Department of Anesthesiology and Director of Pain Management at LSU. Dr. Cork and his colleagues found that when the L5 and S1 are both hypo-functional on the same side, there is an 80% probability of piriformis entrapment of the sciatic nerve.



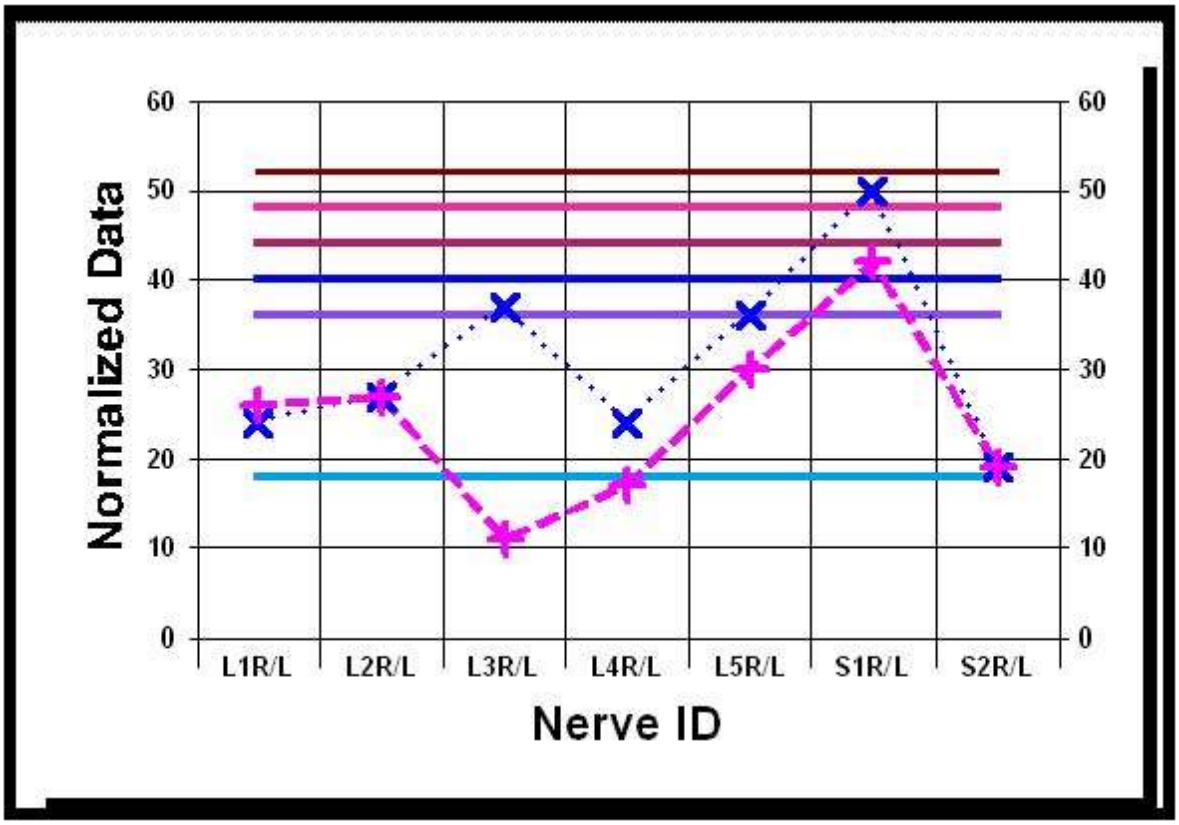
The last graph of this chapter's third part demonstrates another recognizable pattern. At first glance it seems to show moderate Left L3 hypo-function with hyper- function on the opposite side, Right L3. Actually, this is a typical chronic radiculopathy (Left L3). Next, Part IV, will explain chronicity.



PART IV - Pain Fiber Disinhibition

Hedgecock's Radiculopathic Disinhibition: Much as been reported concerning efferent (motor) disinhibition.xliv Nothing of significance had been reported concerning sensory disinhibition. The author was the first recognize that in chronic sensory (A-delta) radiculopathy, the function of the opposite nerve-root shifts toward hyper-function.

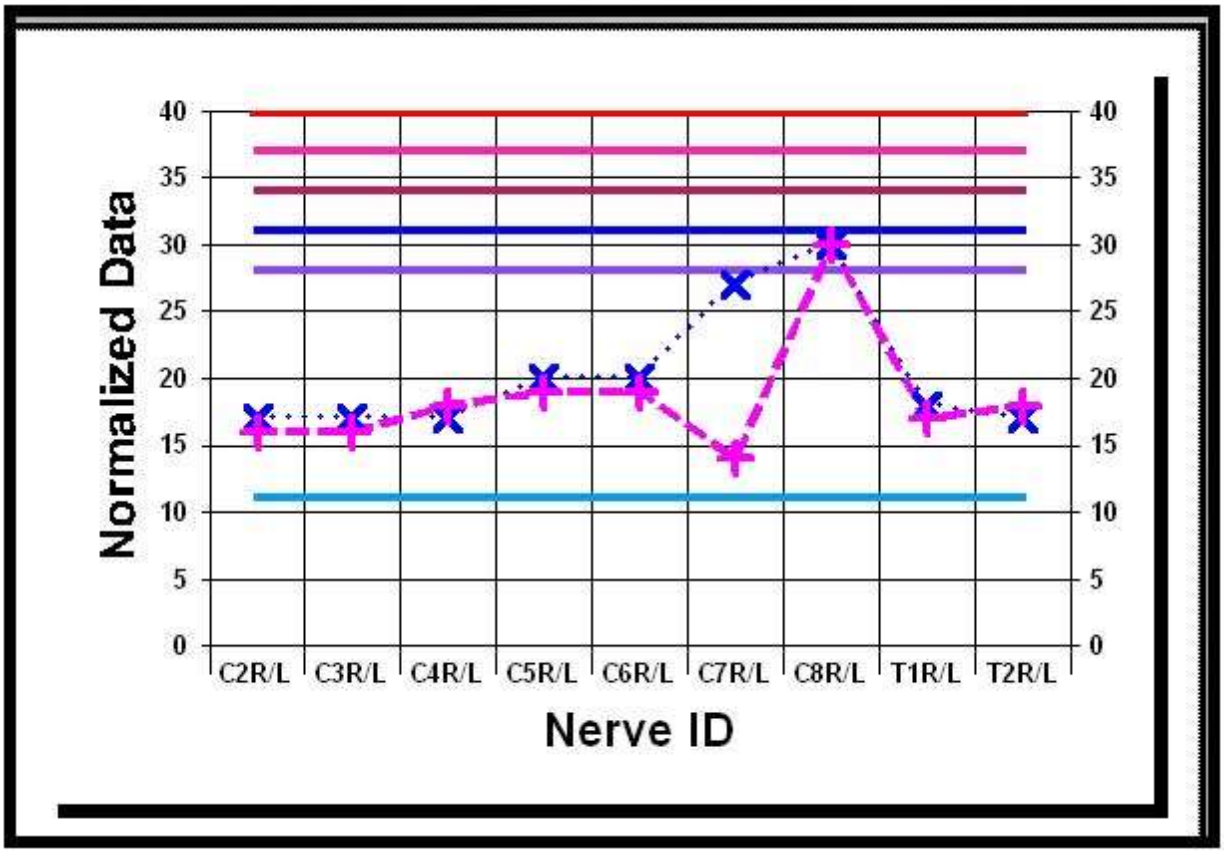
Disinhibition is an adaptation of the spinal cord's filtering process. Secondary neurons and interneurons act as a filtering system to lower the volume of signals reaching the brain. If an injury causes unrelenting dysfunction, the **Protopathic Phase** continues and the A-delta fibers remain down-regulated. Over time, the filtering mechanism adjusts to allow more A-delta signals to pass. However, disinhibition cannot selectively allow more signals to pass from one side only. More signals also pass from the opposite side normal nerve-root. It was also found that, in order to display this type of deviation, the lesion had to be unrelenting for over two-years, before the primary lesion tests within a lower rating than it actual degree of dysfunction, while the normal side tests toward hyper-function.[xlv]



Keep in mind that in bilateral chronicity, both severe hypo- functioning nerve-roots will be lower on the graph, and appear to be a mild (+1) to moderate (+2) bilateral pathology. This points to the need of a comprehensive history and correlation with all findings.

This graph shows a typical chronic pattern with a more acute radiculopathy. As reported, chronic radiculopathy must usually be present for two or more years before the pattern of disinhibition is noticed. If the examiner were unaware of the pattern associated with disinhibition, the Left L3 could be thought to be unimportant or a mild problem compared to the more severe S1 pathology. However, this patient has two pathologic nerve-roots: a chronic Left L3 and a more acute Left S1. To estimate the severity of the Left L3, measure the distance the Right L3 has dropped from the center of the **Normal Zone** and add this to the Left L3. In this case, it is likely that the Left L3 could be rated as +5 (very severe). Note, too, that the primary pathology is not always in the mild to moderate rating. If the injury is sufficiently severe, hypo-function may ***not*** improve as the normal (opposite) side moves toward hyper-function.

It seems likely that **Hedgecock's Radiculopathic Disinhibition** is at least partially due to the need to allow more proprioceptive signals to pass to the CNS, which is necessary for effective spinal locomotion.

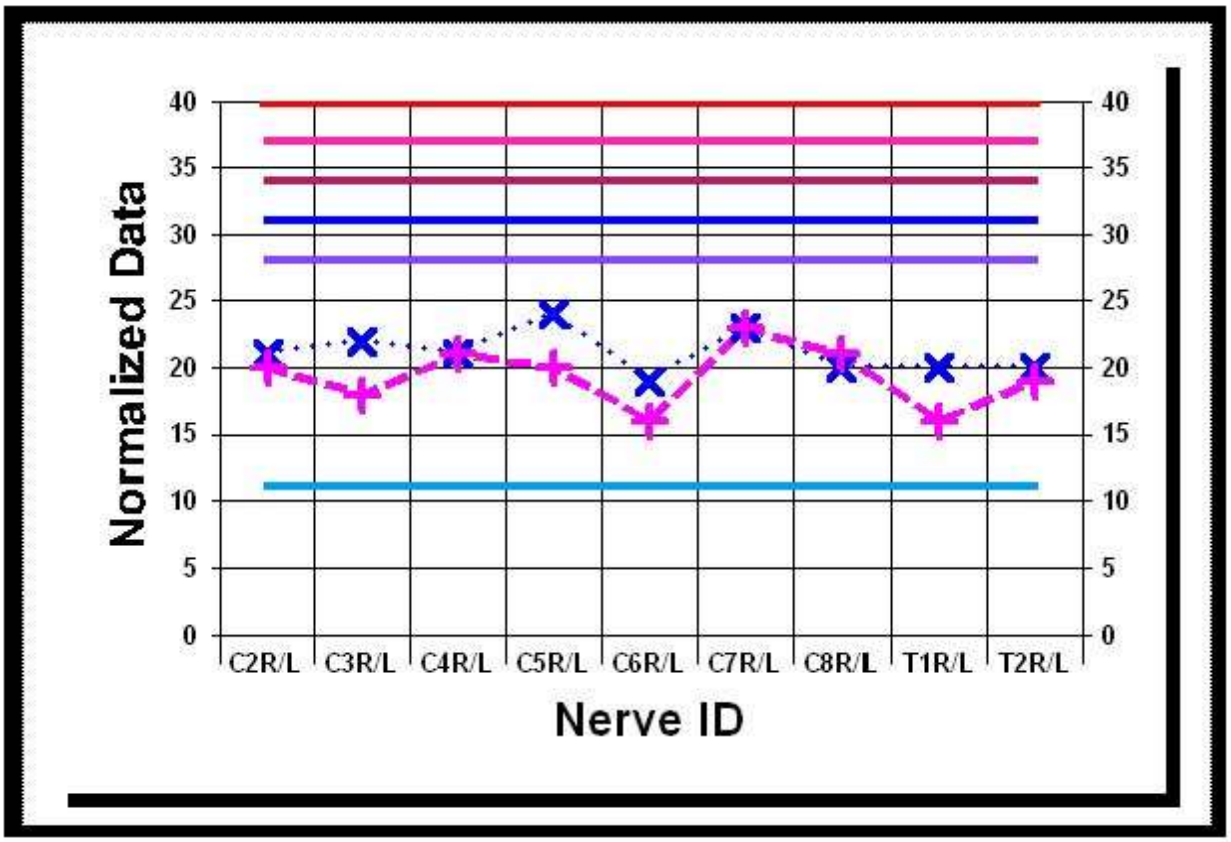


Bilateral Disinhibition: This graph points out the importance of taking a comprehensive history. In this case the patient suffered a whiplash injury 7 years previous to her pf-NCS exam. At the time her symptoms included severe disabling headache, vertigo and short-term memory impairment, which caused her to drop out of school.

The initial impression is that of C8 bilateral Mild (+1) Hypo-Function. However, taking into account the duration of 7 years and severity of the symptoms along with the fact that she is a citizen of Holland where no monetary gain is involved with such injuries, it becomes clear that the bilateral C8 is not mild, but chronic. The Left C7 is also chronic. Note the opposite side Right C7, is the lowest measurement on the graph.

The nerve-roots involved were confirmed by identifying reverse rotation of the spinous processes of the vertebrae supplied by these nerve-roots, C7 and T1. It can be seen in the right X-ray that the C7and T1 spinous processes rotate toward the side of lateral head tilt, which is the reverse of normal coupled motion.xlvi In the radiographs the lateral boundaries of the vertebral bodies are marked by the tips of the dark pointers and the central pointers identify the tips of the spinous processes. This reverse motion is likely caused by weak proprioceptive signals from the vertebral joints, capsules, ligaments and tendons passing through the affected nerve-roots. In the cervical spine the spinous

processes normally rotate away from the side of head tilt, while in the lumbar spine the spinous processes normally rotate toward the side of lateral bending. An interesting finding in the film on the right is that the spinous processes of C4-5-6, above C7, demonstrate exaggerated normal rotation. This exaggerated normal rotation is a compensation for the reverse rotation at C7 and T1, and allows the overall lateral bending to be closer to normal.



Five days after manipulation the left film was taken and the pf-NCS repeated. The radiograph shows normal rotation of C7 and T1 and the previous exaggerated rotation of C4-5-6 is now symmetrical. The graph shows normalization of A- delta function. Symptomatically, the patient's 7-year old constant headache stopped within two hours of manipulation, and within a few weeks, her short-term memory and vertigo abated. She was able to return to school and after three months, she returned to her favorite pastime - horseback riding. Some three years have past and during this time she required only one additional manipulation, which was after a fall from her horse.

Manipulation: Leading Conservative Treatment: The preceding case is not unique. Based on our observations, osteopathic and chiropractic physicians must revise their long held theories concerning vertebral displacements and subluxations.

Radicular Proprioceptive Vertebral Dyskinesia: Another discovery of was the recognition of a consistent correlation between sensory A-delta radiculopathy and abnormal coupled motion of the vertebra above and below a pathological nerve-root. This discovery has come to be termed Hedgecock's Vertebral Dyskinesia (HVD).[xlvii]

Definition: ***HVD is a condition in which concomitant with diminished A-delta fiber sensitivity there is a proprioceptive disruption causing aberrant vertebral rotation during lateral bending of the segment(s) above and/or below the involved nerve-root.***

Evidence Based Medicine: The correlation between the pf-NCS and lateral bending radiographs is the strongest of objective evidence in support of nerve-root pathology. This also supports the osteopathic and chiropractic theories that spinal nerve lesions can cause more than spinal pain. Already, the pf-NCS has shown that physiologic processes are affected. An example was mentioned earlier: a study lead by Irving M. Bush, MD, Professor of Urology at **The Rosalind Franklin University of Medicine and Science** and **The Chicago Medical School**, and former head of the **Food and Drug Administration (FDA) National Scientific Advisory Committee on Gastroenterology**, along with his colleague, Mohamed Baddruddoja, MD, presented findings at the **International Pelvic Pain Society's 15th Annual Scientific Meeting** held in San Diego California, on October 27th, 2007. Simultaneously, while Dr. Bush's associates presented the study, Dr. Baddruddoja was presenting the study to the **American Association of Sensory Electrodiagnostic Medicine (AASEM)** at its annual conference held in Newport Beach, California. Using the pf-NCS Dr. Bush and Dr. Baddruddoja discovered a correlation between chronic prostate inflammation and lumbosacral radiculopathy. They also found this correlation in vulvadynia sufferers.

It is important to note that many of these patients had little or no concomitant lumbosacral symptoms. The tentative conclusion is that nerve-root pathology may predispose patients to these disorders.[xlviii] Based on these findings and preliminary results of a pilot study, **The UCLA David Geffen Medical School OB/GYN Department** is now carrying out a study using the pf-NCS to investigate the correlation between lumbosacral radiculopathy and vulvadynia.

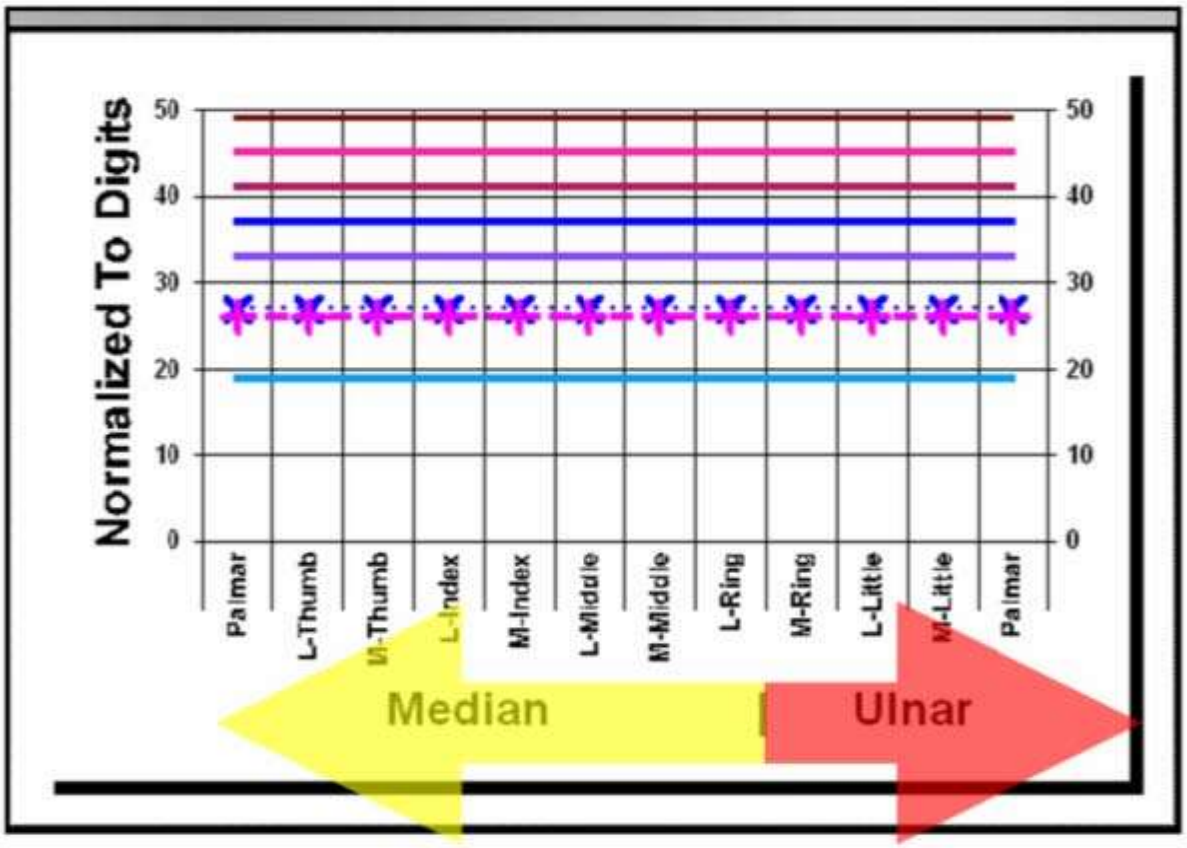
Manipulation has been found to "unlock" the vertebral segments. Depending on the presence, degree and type of degenerative changes manipulation has proven to be safe and effective. Several modalities have been noted to be useful for aiding in re-establishing long-term return to normal motoricity of the vertebral segments. This will be discussed in more depth in Chapter VI.

New Perspective: In the past, the osteopathic and chiropractic professions have held out theories that vertebral lesions caused far ranging health problems. Osteopaths attributed many disorders to poor circulation concomitant with vertebral displacements, while the chiropractor theorized a neurological etiology. Let's be historically honest. Not too very

long ago physicians believed in bloodletting as a cure for many diseases. We may think of these ideas as bizarre, if not outright ridiculous, but based on the objective evidence it is reasonable to put forward the proposition that vertebra lesions can affect seemingly unrelated systems. Not so far fetched is the concept that, based on objective evidence, when abnormal vertebral motion is found concomitant with nerve-root pathology manipulation is the conservative treatment of choice in conjunction with adjunctive therapies, such as trigger point injections, analgesics and physical therapy. Certainly, surgical intervention should be at the bottom of the list.

Part V - Upper Extremity Graphic Analysis

As in the radiculopathy studies, the upper extremity study averages the measures to the center of a **Normal Zone**.

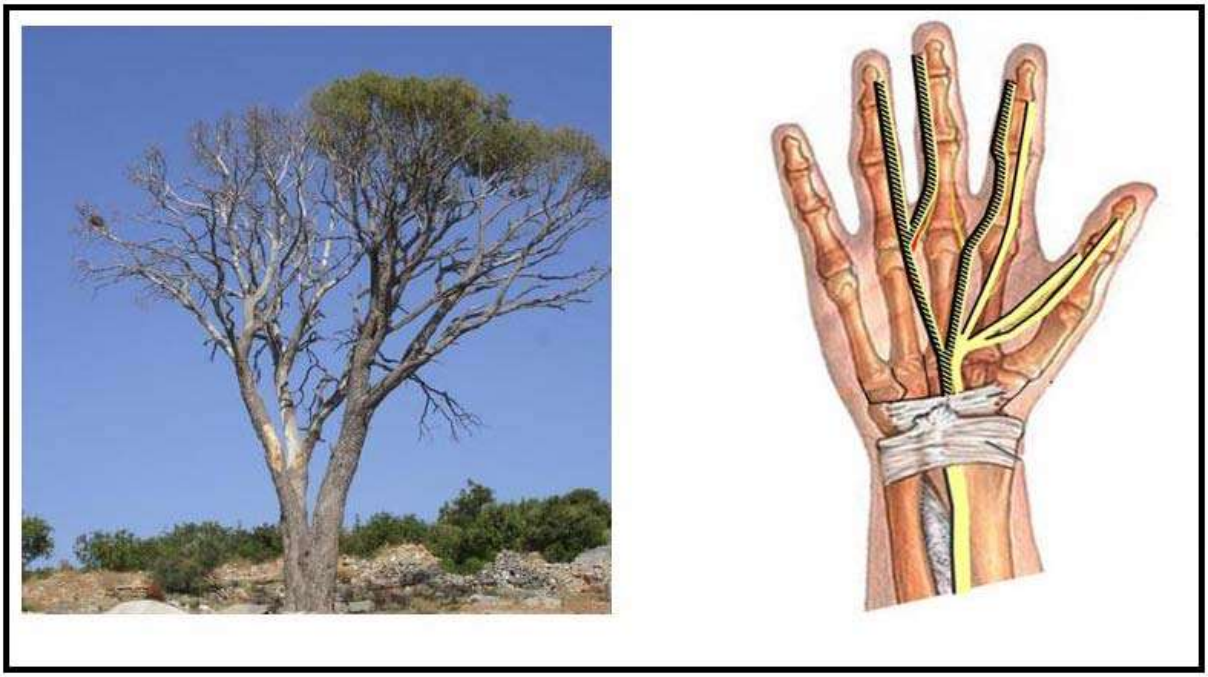


Graph Layout: There are seven (7) median nerve branches (yellow arrow) and 4 ulnar nerve branches (red arrow). The respective palmar branches are located at each end of the graph. Unlike the motor branches, the sensory palmar branches do not pass under the carpal tunnel (median nerve) or through Guyon's canal (ulnar nerve), but pass over them. Therefore, sensory dysfunction of the palmar branch suggests possible proximal pathology.

Rule #1: Median Nerve Pathology is suggested if <u>two or more branches</u> are rated with Hypo- Function. The more branches and higher the ratings the more likely pathology is present. Why are 2 or more branches needed to suggest pathology? The answer is that it is quite common to injure a finger at sometime during ones life by closing a door, drawer, or hitting a finger with a hammer. Most people are unable to recall such minor mishaps, therefore, an isolated single digital branch with hypo-function should not lead the physician to suspect or confirm pathology.

Repeat: The palmar sensory branches of the median and ulnar nerves do not pass through the carpal tunnel and Guyon's canal respectively. Therefore, dysfunction of either palmar branch suggests damage proximal to the wrist.

African trees offer a good example of why not to expect all of the digital nerve branches to be pathological. Elephants love the bark of some species of trees. It is observed that when the elephant eats around only part of the trunk, the uneaten bark feeds those branches



and they live while the other branches die. In a similar way symmetrical median nerve entrapment is not seen. Many digital branches are spared. Therefore, the rule is that 2 or

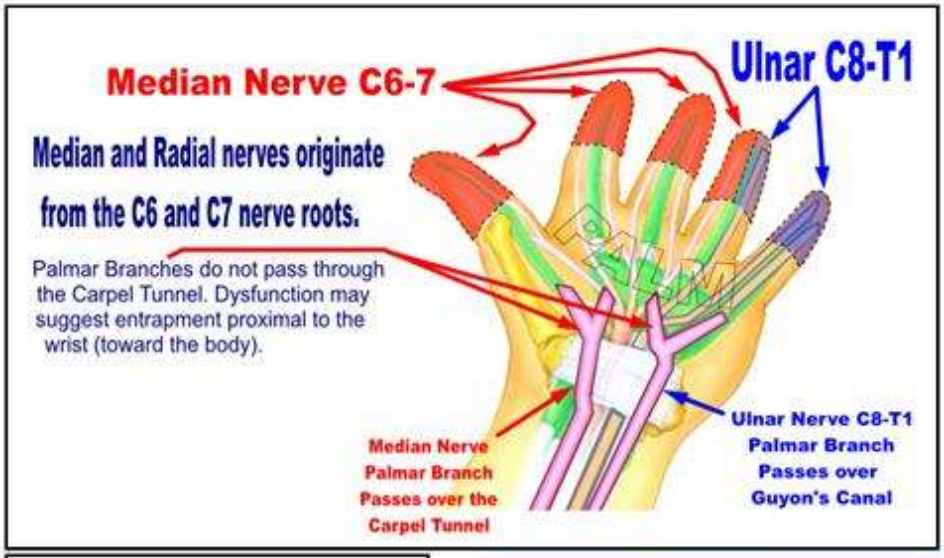
more branches must be involved to suggest pathology. The more branches involved and the greater the hypo-function, the more positive the diagnosis.

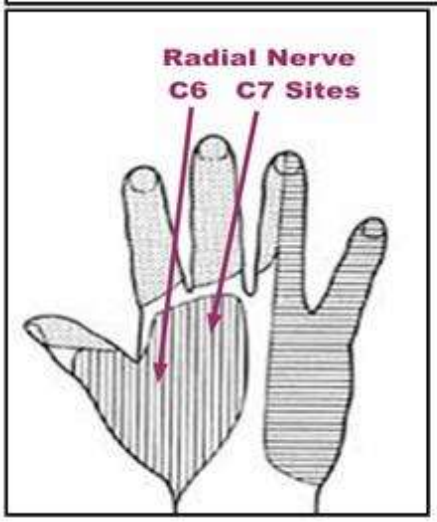
Testing the radical nerve branches on the back of the hand can easily differentiate between radiculopathy and median nerve pathology since both have the same nerve-root origin (C6-7).

Diagnostic Anatomical Considerations

Median and radial nerves originate from nerve-roots C6-7. Median and radial nerves separate in the brachial plexus. The median and ulnar **sensory** palmar branches do not pass through the carpal tunnel (median), Guyon's Canal (ulnar). The **sensory** branches pass over the transverse carpal ligament and only the motor branches pass under the ligament. Therefore, palmar branch dysfunction suggests possible proximal sensory pathology. If the palmar and digital branches are affected the problem may be double-crush (wrist and proximal).

The cervical sites (radial nerve - C6-7) on the back of the hand rule in or out proximal nerve-root pathology. Testing above and below the medial elbow detects cubital tunnel entrapment. Always compare measurements to the opposite (asymptomatic) side. Any cutaneous site can be tested and compared to the opposite (asymptomatic) side. The higher the A-delta fiber measurement, the more likely the presence of pathology. Always correlate the pf-NCS findings with the patient's history and other finding before making a final diagnosis.

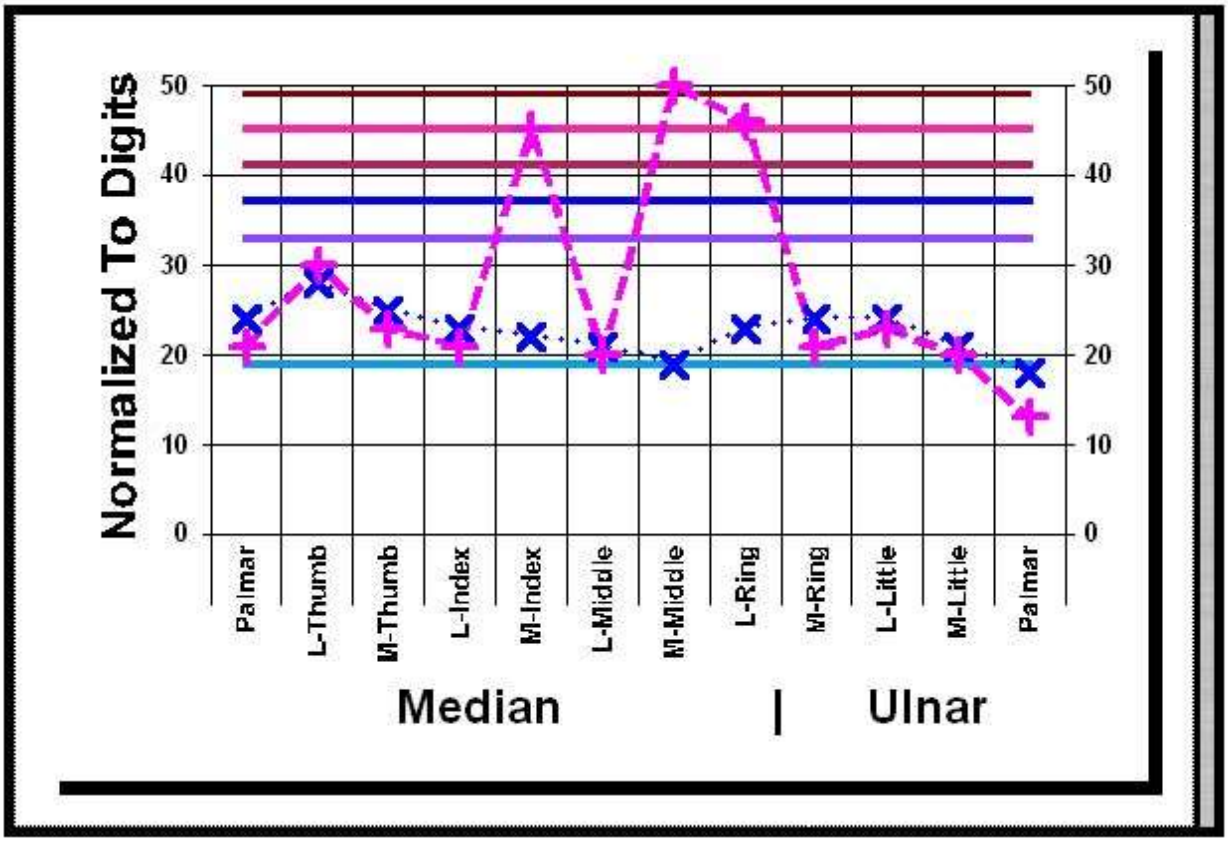




Carpal Tunnel Syndrome - Median Nerve Entrapment

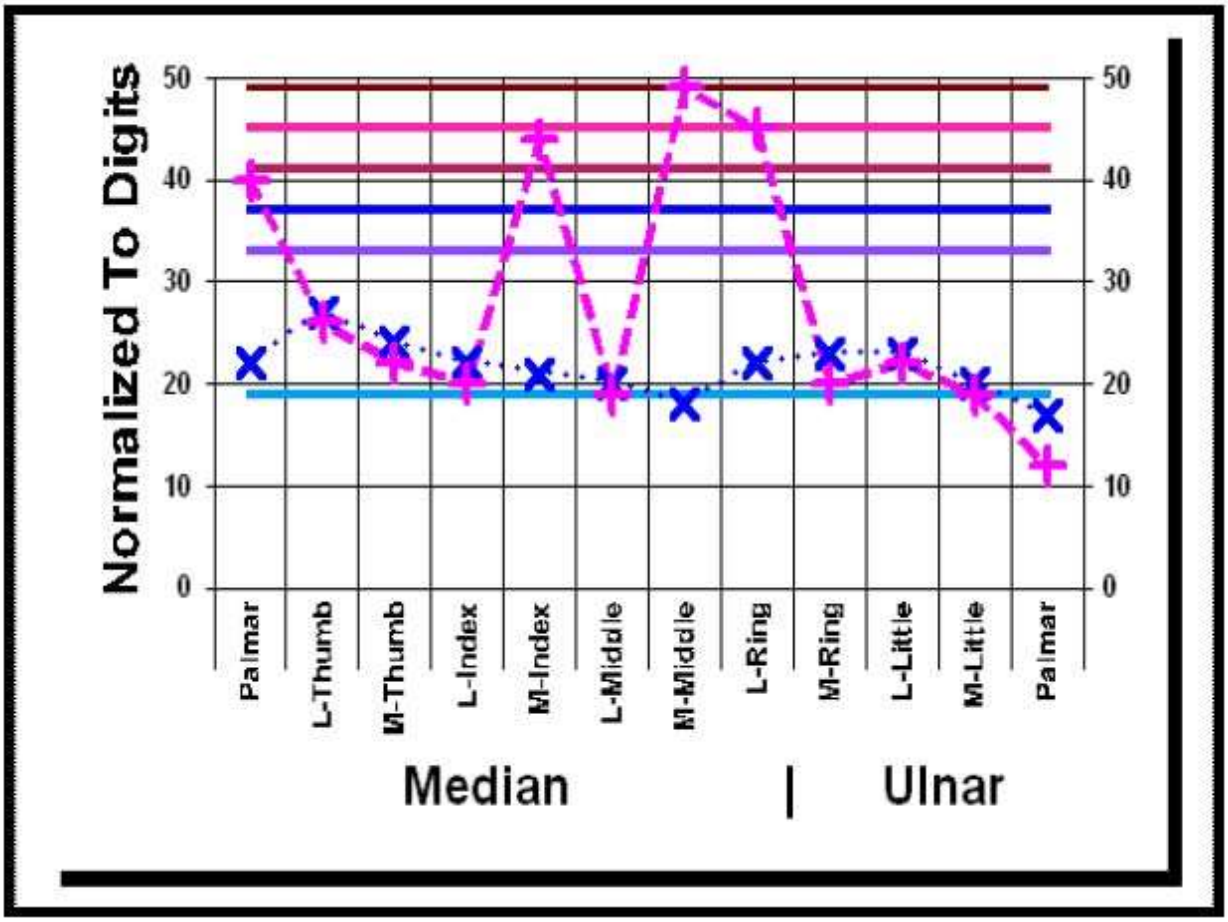
Right - side is represented by pink dashes and +.

Left - side is represented by blue dots and X.



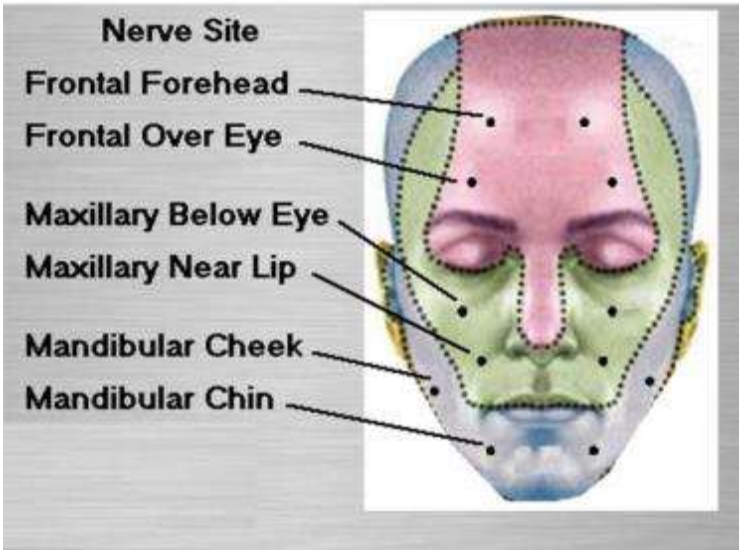
This graph shows three rated median nerve digital branches, which meets the requirement that two or more branches must be rated to suggest pathology. The palmar branch (at the far left of the graph) is within the **Normal Zone**, which rules out proximal pathology. Note how the high hypo-function measures have pushed the normal measures down in the averaging process. A middle finger branch has been pushed into the **Hyper-Function** rating. A click up on the **Correction Factor** lifts false hyper-function into the **Normal Zone**.

Median Nerve - Proximal Problem: In the graph below, the right median nerve has three **Hypo- Function** rated branches, but note that the right palmar branch (far left on graph) is also hypo- functional, which suggests proximal pathology. Keep in mind that this does not rule out the possibility of a double-crush, meaning possible median nerve entrapment at wrist and proximal damage. In such cases, a cervical study which includes the radial nerve branches on the back of the hand (C6-7 same origin as the median nerve) can rule in or out cervical radiculopathy as the cause of the high palmar measurement.



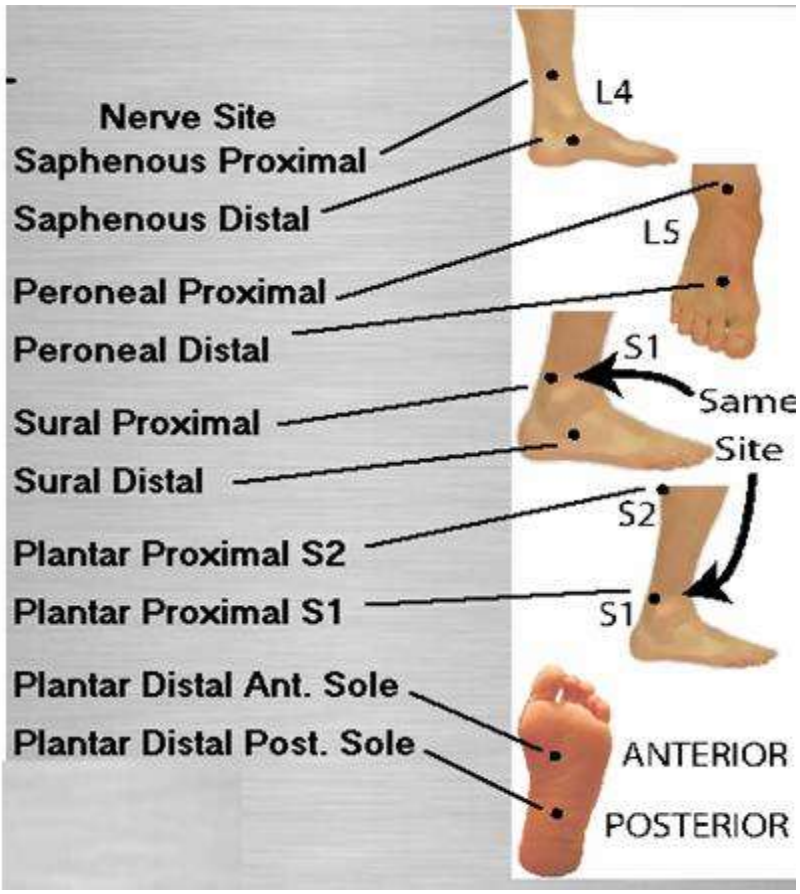
Part VI - Peripheral Nerve Studies

General Considerations: Peripheral nerve studies do not use a graphic analysis, as in the radiculopathy and upper extremity studies. The peripheral analysis consists of comparing the difference between the symptomatic side and the asymptomatic side. Calculations are made to give the percentage of right to left difference, and above and below the sites of possible entrapment or injury. The trigeminal study is an exception, so it will be discussed first.

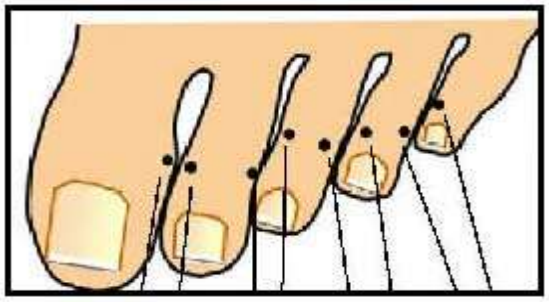


Trigeminal Nerves - Right to Left Comparison - Normal < 20%: Not all branches need to be tested. The examination can be confined to those branches of interest. Test and compare each site with the exact site on the opposite side. The sites shown in the software data **Input Screen** and charts are general sites. Usually the exact site of most intense pain or numbness is tested and the same exact site on the opposite side. The software calculates the deviation percentage, placing it in the report. A generic explanation is given and the physician edits and includes his diagnostic impression.

Lower Extremity Studies: Saphenous - Peroneal - Sural - Plantar Proximal and Distal: It may be desirable to perform a **Lumbosacral Study** to rule in or out radiculopathy before proceeding with a lower extremity study. The **Lower Extremity** study is accomplished by testing above and below the suspected site of pathology or entrapment, as well as the exact opposite sites. The software places the deviation percentage in the report. A distal high measure suggests pathology. However, high proximal and distal hypo-function suggests radiculopathy or proximal pathology. In editing reports, keep in mind that an EDX reports are not comprehensive report. It should give the results and, at most, some idea of the diagnostic impression.

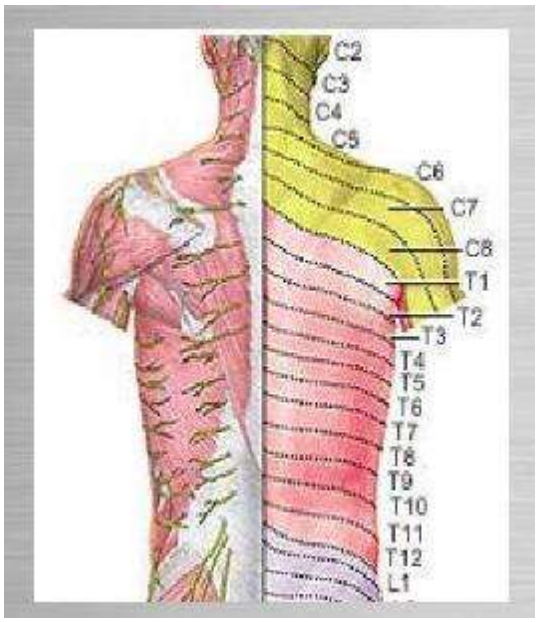


Neuroma - Normal < 30% right to left deviation: Not all sites need be tested; test the sites of interest and the exact opposite side sites. The software calculates the percentage of difference between the right and left. In this study, as in all studies, the report is edited by the physician.



Custom Study - Right to Left < 20% above the navel and <30% below: The **Custom Study** allows the examiner to choose any anatomical site and test above and below and to compare these to measurements on the opposite (asymptomatic) healthy side. The anatomical sites and the nerve(s) being tested are typed into the software; the software

calculates the percentage of deviation and places the name of the anatomical site and the deviation in the report, which is edited to explain the relevance findings.



Thoracic - Right to left deviation < 20% above navel and < 30% above: The thoracic spine's cutaneous branches have wide variations. Therefore, the study gives an approximation of the level of involvement. The level can easily be off by up to two levels, therefore, it is important to correlate the findings with the patient's history: physical findings and imaging. The test sites are 1 inch lateral to the spinous process tip. Locating the correct nerve requires counting down from C7 down to T4. The T1 cutaneous branch is lateral to T4. To find C7 have the patient flex his neck forward and backward while feeling the spinous processes. The C6 is the last movable spinous: C6 moves forward and backward on C7 with forward flexion and backward extension.

Chapter VI

Evidence Based Medicine

PART I - Early Diagnosis = Timely Intervention

With 43% of pain patients developing chronic symptoms and up to 80% of back surgeries failing, it is certainly obvious that conventional diagnostic methods are not working. Treatments will improve, but the best treatment cannot possibly be effective when it is directed to the wrong peripheral nerve or wrong nerve-root.

Previous chapters explained how pf-NCS can prevent misdirection by accurately diagnosing the presence and location of sensory pain fiber pathology. This chapter will address treatment options based on early and accurate localization of nerve-root pathology.

Third-party payers often argue that approximately 90% of neck and back patients recover without treatment, therefore, they question the cost and time spent testing patients during early episode of neck or back pain. It is felt that, based on the present failed system, it makes more sense to wait and see. In light of the pf-NCS, this position is no tenable. Now that there is capability of diagnosing pain pathology in its earliest stage, it makes sense to rule in or out the presence of a neurological deficit as soon as possible.

Additionally, with 25% of the population suffering from spinal pain at any given time, the odds are overwhelming that a patient will suffer a recurrence. Studies have shown that 90% of pain patients suffer recurrences that are progressively more painful and with increasing disability. Therefore, detecting neurological deficit in as early a stage as possible, when conservative treatment is more likely to be effective, is the better course to avoid the catastrophic costs associated with chronic neck and back pain. The patient who "pulls his back" putting on his socks may sound like a simple muscle strain, however, if the pf-NCS reveals nerve pathology then action should be taken to avoid serious complications.

Cure and Prevention: Taking advantage of the early diagnostic capability of pf-NCS requires physicians to think beyond treating with a prescription pad. The physician must learn what predisposes a patient to spinal problems. However, let's first look at what usually takes place in neck and back pain cases:

1. Most patients wait before seeking medical attention in the hope that their pain will go away. This delay carries with it the high probability that by the time the patient seeks help he is dependent on C-pain fibers to locate the source pain and, as a result, there is over a 50% probability that the patient will incorrectly localizes the source of his pain.
2. When the patient decides to seek help, most probably s/he will likely consult a primary-

care physician (PCP), whose treatment of acute spine pain is usually limited to analgesics and rest. If this works, there is a 90% probability that the patient will suffer progressively more painful recurrences and experience increasing disability.
3. The next step is referral to a specialist.

Early Intervention Algorithm:
Even if a physician has no intention of treating his pain patients, accurately diagnosing the specific nerve-root causing symptoms can prevent misdirection by the specialist to whom he refers his patient. Even better, if the primary-care physician takes time, s/he may learn the cause of the neck and back disorders, and may be able to provide a more positive outcome than the specialist can offers.

Choosing the right specialist: To the man with a hammer everything looks like a nail.
Surgeons are bias to surgery, while pain specialists see epidural blocks or radiofrequency as the final solution, and the chiropractor sees realigning vertebrae as the most effective cure. These specialists may all be correct in a given case, but if any one were correct all of the time, then 43% of patients would not be developing chronic symptoms.

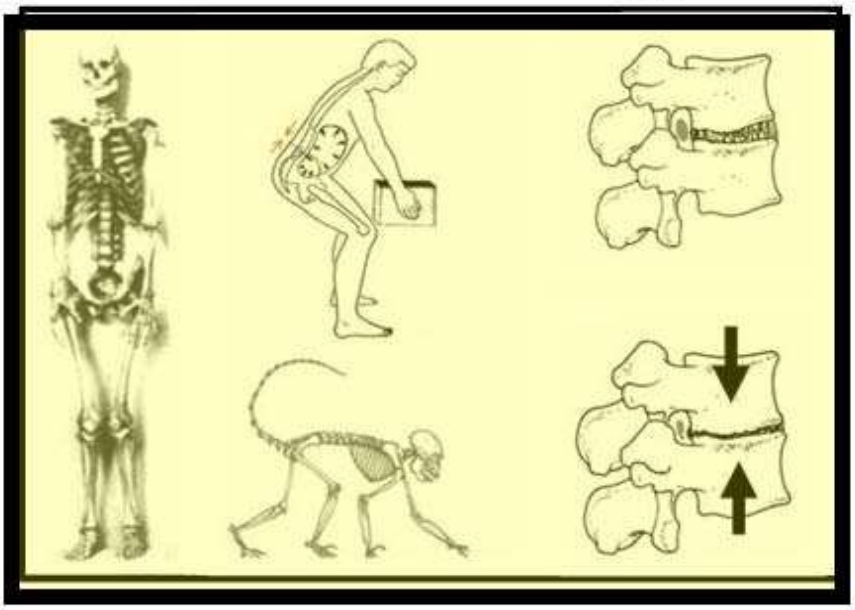
Certainly, having the capability to more accurately localize pathology to the correct nerve-root will improve the outcomes regardless of the specialist selected, but selection of the most appropriate specialist makes the difference between moderate and significantly improved outcomes.

The first step in choosing the most appropriate specialist is to understand why spinal problems are endemic to humans. So, let's start there...

PART II - Proving Nerve-Root Pathology

Neurological & Anatomical: The preceding has hopefully helped the reader better understand the neurological; now, the anatomical should be addressed. In most radiculopathy cases, the neurological component is intimately related to anatomical changes.

There can be no doubt that medicine has come a long way in its understanding of the physiology of pain. Science has developed interventions that rival civilization's greatest engineering feats, but in dealing with pain, especially neck and back pain, the data shows we have a long way to go. Surely, being capable of more effectively detecting the nerve tract causing a problem is a huge step forward, but its time to take a step back and look at the underlying factors that make humans prone to spinal disorders. In doing so, we may learn more effective conservative methods to improve outcomes and prevent pathology.



Anatomical Considerations: The reason 40% of patients seek medical help for neck and back pain is largely ignored, but the basis of the problem comes down to one simple fact: humans are the only great ape walking upright with locking knees. Medicine in general ignores this reality in favor of pain medicine, therapy, and surgery, which generally yield poor outcomes and too many side effects. We can do better, when we understand the involvement of structural and neurological factors.

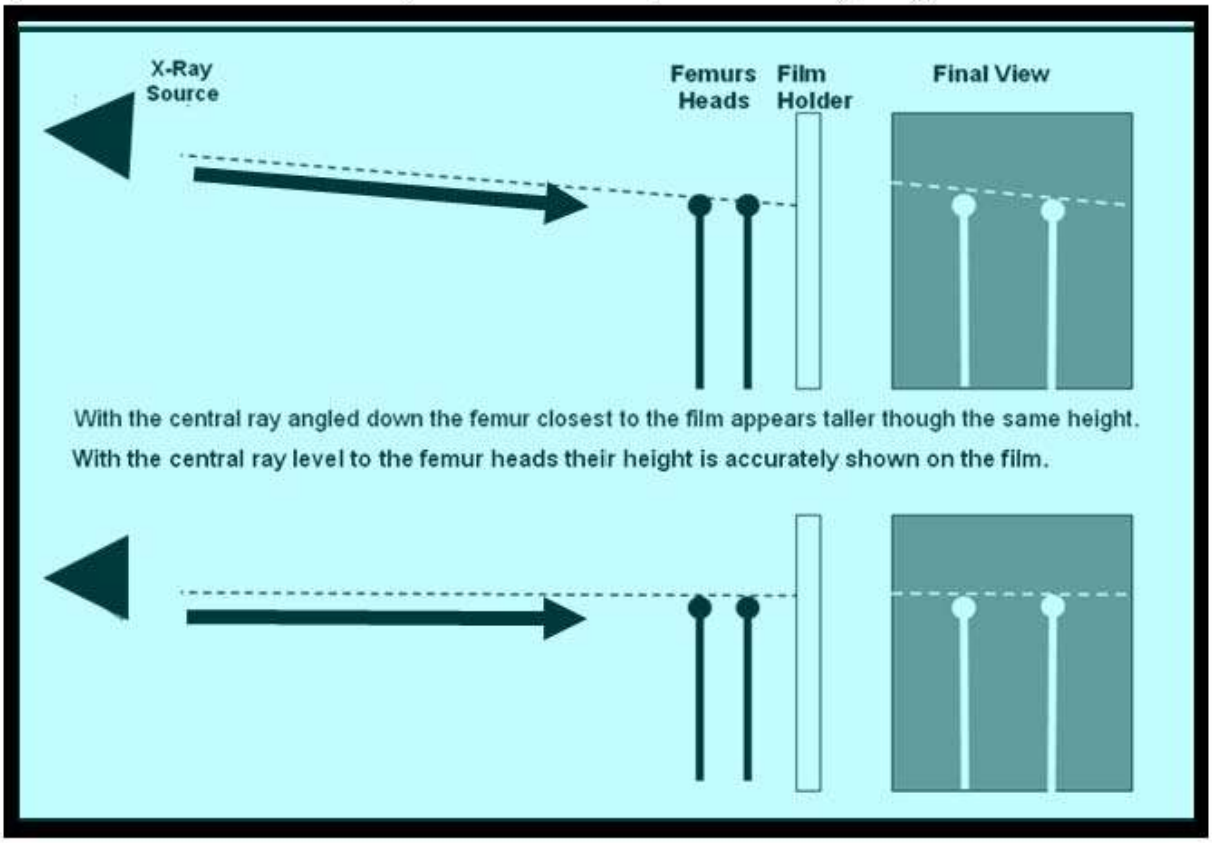
Standing and sitting in vertical posture with a spine that did not evolve to handle constant compression is the main contributing factor to spinal degenerative diseases. Ultimately, evolving more as a suspension bridge, the spine cannot effectively accommodate to vertically posture. Throughout life the spine is under constant compression. As a result, humans loose an average of four to six inches in height by the age of 70. The vertebral bodies and the disks compress, reducing the size of the intervertebral foramen, which compromises the nerve-roots and predispose disks to herniation and facet degeneration.

How do we stop the compression? The best advice to help prevent compression is to hang from a chip-up bar as often as possible. Hanging a few times a day for several seconds is a good start and has been shown to significantly slow disk compression.

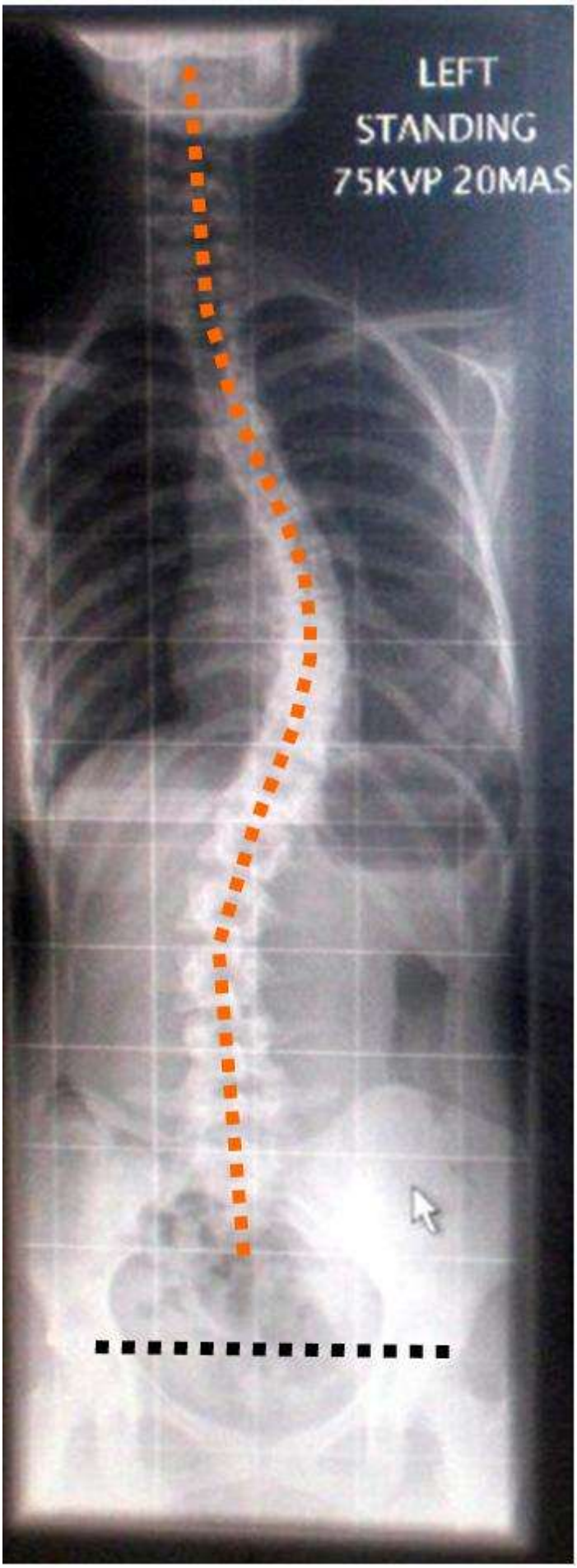
Balance: Besides compression, vertical posture makes us vulnerable to imbalances. The stress placed on the disks and spinal joints from an imbalanced foundation is often a factor in slowing and hindering recovery of low back and cervical injuries. In fact, a short leg can be the single cause of lower back pain. A short leg on one side shifts the weight distribution throughout the entire spine. However, a simple set of radiographic views taken in the weight bearing position can determine if an imbalance exists.

Short Leg Syndrome Measurement: The spine must be looked at as a whole functional unit and not as separate regions. The legs and pelvis form the body's foundation. If that foundation is imbalanced then every structure above is imbalanced. So even if the problem is in the cervical spine, it may be necessary to study the lower spine. This is especially true if the cervical anteroposterior (AP) radiographic view reveals a lateral inclination, which suggests the cervical spine is sitting on an uneven foundation. In 1969 the author devised a radiographic procedure to accurately measure comparative leg height. In the standing (weight bearing) AP view, the central ray is directed level with the tops of the femur heads. The top diagram shows the standard method, which results in femur head height distortion. The lower diagram shows **Hedgecock's Standing View**.[xlix]

procedure to accurately measure comparative leg height. In this



Hedgecock's Standing View eliminates distortion in femur head height. In the standard view the femur head closest to the film will appear taller than it actually is while the one farther way from the film will appear to be shorter than its true height. Lowering the X-ray tube (source) so that the central ray is level with the femur heads removes height distortion. The view is taken by lowering the X-ray tube (source) so the central ray is level with a point 3 inches below the anterior iliac spine. Angling the tube up 15 degrees (based on 60 inch distance) produces the usually full view of the lumbosacral region.


LEFT
STANDING
75KVP 20MAS

AP Lumbosacral Evaluation: If the AP film was made with the central ray level to the tops of the femur heads, as described above, there will be no vertical distortion in the height of the femur heads. If there is an imbalance in leg length, it will be noted that the spine leans from side to side, which places increased pressure on disk and facet joints on each concave side.

The example at right is an old-style, chiropractic full-spine radiograph. There is no possible way to accurately determine relative femur head height because the central ray in the lower half was level with L4. This prevents determining leg length and precludes comparing leg length to the angle of the sacral base. It cannot be determined if a curve is due to a short leg or is idiopathic. Two factors determine how soon a patient develops symptoms due to a short leg:
1. How much time the patient spends standing or walking.
2. How great the difference in leg length.

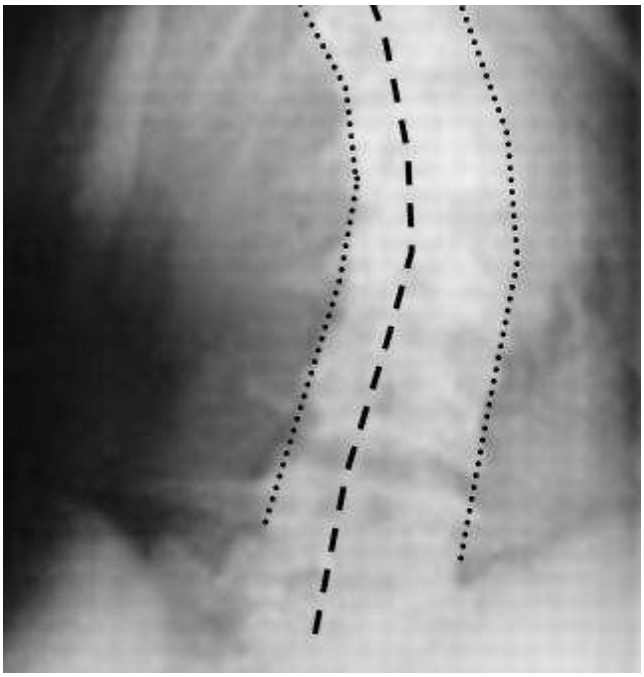
A typical example of how a short leg can cause problems is shown in the case of an engineer who came to the author for help with his low back pain. At 45 years of age, he experienced his first episode of low back pain. He was found to have a 1/2 inch difference in leg length. Why did it take so long for such a major discrepancy in leg length to cause problems? The only sport he participated in was swimming, and he had always worked at a desk job. Two weeks prior to the onset of severe back pain, he decided to get into shape and started jogging. A heel lift ended his pain within hours without any further treatment.

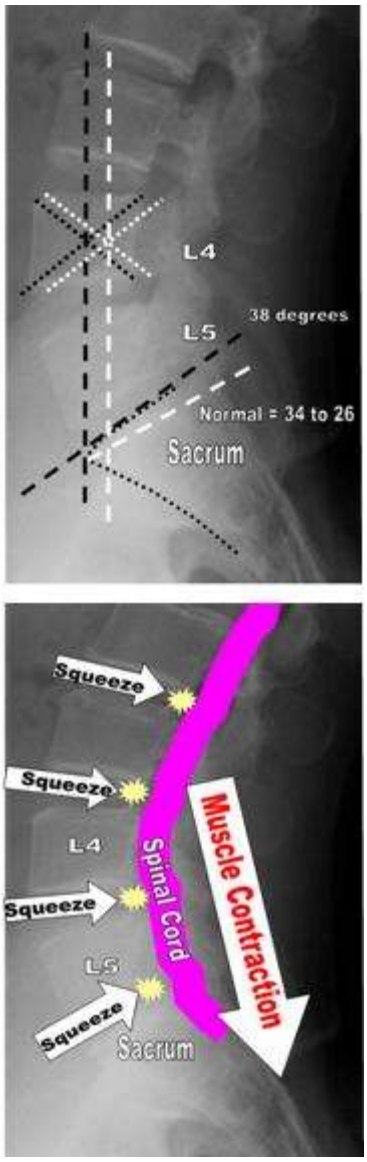
Another example is that of a 50 year old male who attributed his back pain to the fact that he had polio as a child, which caused one leg to be shorter. An AP, standing radiograph, taken with the central ray at femur head height, revealed that his prosthetic shoe was 1/2 inch too high. No one had bothered to measure is leg length since he was 17, and the short leg had almost caught up to the normal leg. Decreasing the height of the prosthesis resulted in his chronic pain, with which he suffered for 10 years, completely resolving in a few days.

Don't be surprised to hear patients ask why they just began having back pain if they had a short leg all their life; a little thought makes it obvious: Over time, the mechanical difference causes accumulative damage until a nerve-root is compromised.

Lateral Bending Views: Included with the standard AP, lateral, right/left oblique views, the patient should be X-rayed in lateral bending. The lateral bending views rule in or out that pathology is in a nerve-root. In the film to the right, the small dots follow the lateral border of the vertebral bodies; the dash marks follow the spinous processes. This shows normal spinous rotation. The lumbosacral lateral bending views are taken after the patient runs his hand down his lateral thigh to the limit of motion. It is essential that there be no rotation of either the pelvis or thorax. The normal spinous rotation in the lumbar spine is

toward the side of lateral bending, while in the cervical spine the normal spinous rotation is away from the side of lateral bending. Remember, too, that in the cervical lateral bending views, the head should not be allowed to rotate; the patient simply leans his head to the side as if dropping his ear toward his shoulder without lifting his shoulder.

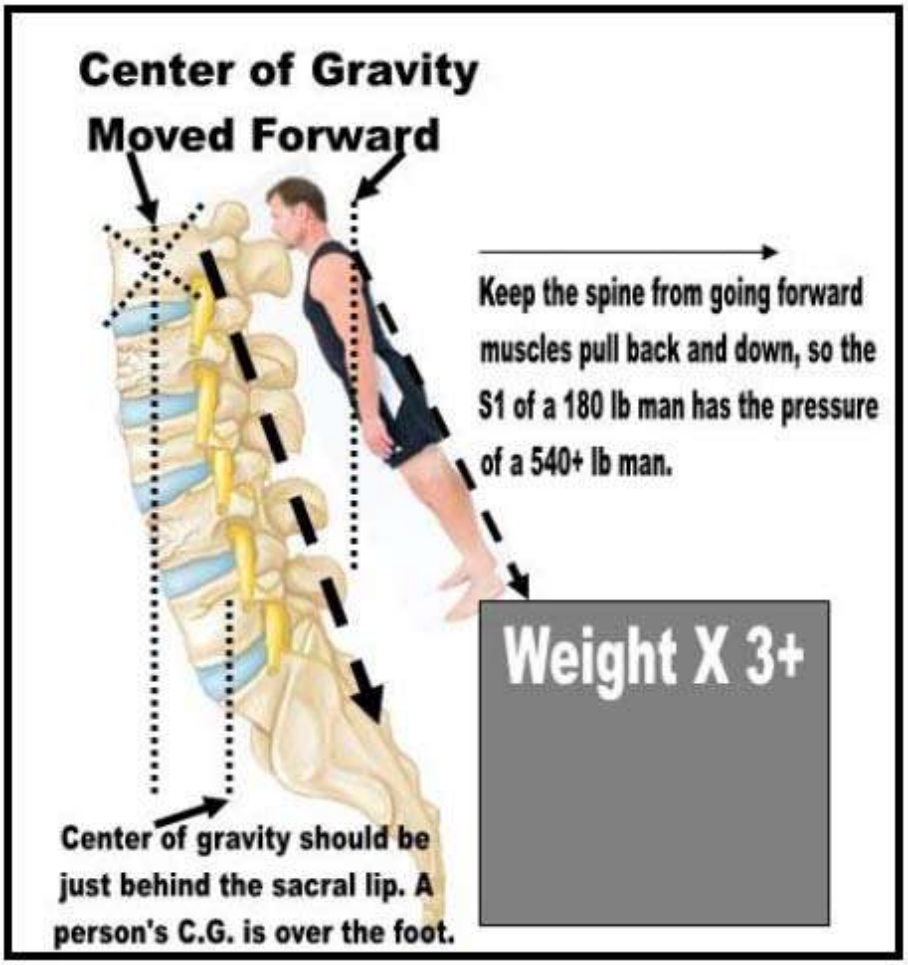




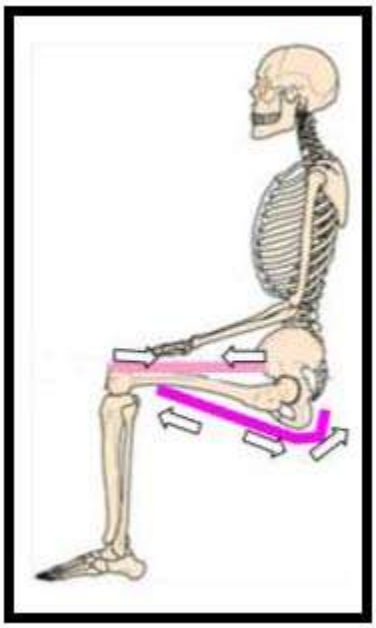
Lateral Lumbosacral View: Lateral views are weight bearing (standing). Most patients will be found to have hyper- lordosis. In perfect balance, a vertical line from the center of the most forward vertebral body should pass 1/4 inch posterior of the anterior lip of the sacrum. Absolute normal finds the L4 vertebra the most anterior segment. In the figure on the right, the white dashes show where the normal positioned of the structures. The black dash lines show the position of this patient. Note: L4 is the farthest forward segment, as it should be, but its center is anterior of the sacral lip. This means that the center of gravity is in space and not over a structure. The lower black dash line is the angle of the sacral base (38 degrees); the angle should be between 34 and 26 degrees. Anterior displacement of the center of gravity means the lumbar spine is being held in position by constant contraction of the posterior muscles. These muscles cannot pull straight back because the attachments are almost vertical. In pulling back, the posterior spinal muscles compress the spine even more. Even worse, the compression is greatest in the posterior disk compartment directly

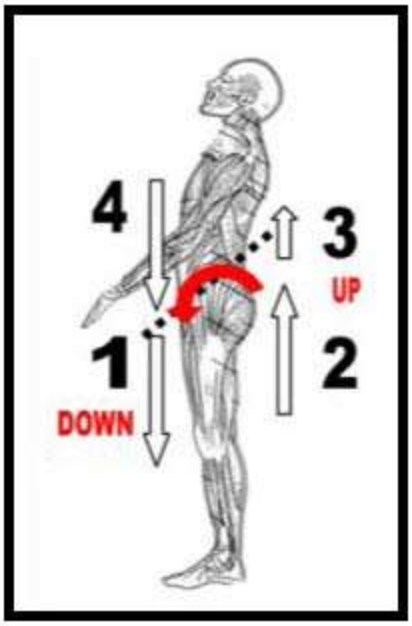
adjacent to the spinal cord and nerve-roots. As the posterior disk compartment squeezes, the facets jam together while the disk compresses and is predisposed to herniation.

In the picture to the below, it appears the man is falling off a cliff. The next picture is a close-up view that shows what is really happening. The close- up reveals that the man is being held back by a cable. The man represents the lumbar vertebrae and the cable represents the posterior spinal muscles. His center of gravity is in space, so like a hammer pulling a nail, the pressure is several times greater than the backwards pull. The farther the center of gravity moves forward, the greater the compression on the posterior disk compartment. Let's consider that the average length of a hammer handle is about the same as the distance between L1 to S1 and an average hammer has a leverage of 8 to 1. Therefore, if the force required to pulling the spine back is between 10 and 20 pounds, the added pressure on the disk is equal to the weight of the upper body, which for a 120 lbs person would be about 80 pounds and for a 220 pound person about 165 pounds. Additionally, the 8 to 1 leverage factor means the 120 pound person has 160 pounds of pressure on the posterior compartment of the lumbar discs, while the 220 pound person has a pressure of 320 pounds of pressure on the posterior compartment of his lumbar discs. Because the discs are void of pressure receptors, neither the small or larger person feels that anything is amiss until something gives way. No wonder vertebral bodies become wedge shaped and low back disorders are our most common ailment.



Understanding the cause helps to understand the cure: Like all creatures, humans are not exactly symmetrical. Gorillas and chimpanzees are not symmetrical either. However, if they have a short femur on one side it would not cause an imbalance because their knees do not lock and they knuckle walk, which reduces the weight on the spine. Having non-locking knees means a gorilla or chimp unconsciously bends the longer leg a slight bit more than the shorter leg.

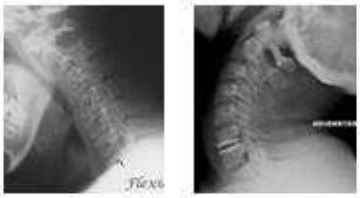




Anterior Sacral Tilt: Humans, especially in this time, sit most of the time. When seated the attachments of the anterior thigh muscles are closer together than when standing. Simultaneously, the attachments of the posterior muscles are separated while sitting. The result is the anterior muscles shorten while the posterior lengthen and when we stand, the pelvis is pulled down in the front and rises in the back. Combine this with weak abdominal muscles, which let the pelvis drop in the front, and we have what is commonly called sway-back or sitter's disease. This anterior tilting of the pelvis is literally endemic in all industrialized nations. How the structural weaknesses of the spine are effectively treated

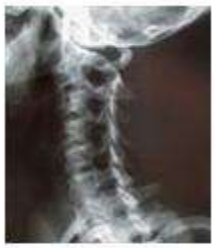
with conservative method will be discussed in Part III of this chapter. For now, don't skip ahead because the next subject is also an important piece of the puzzle.

Cervical Evaluation: The cervical and lumbosacral radiographic studies include the same basic scout views, with the exception that no oblique views are necessary in the lumbosacaral study since the intervertebral foramen are visualized in the lateral views. In both the cervical and lumbosacral studies, the lateral bending views should be included. It is essential that there be no head rotation in the cervical and no pelvic or thorax rotation in the lumbosacaral lateral bending studies.

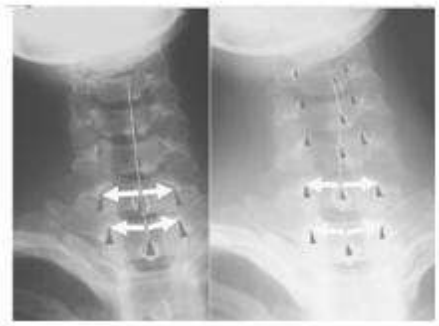


AP View Evaluation: If the AP cervical view shows poor vertical alignment, then a standing AP lumbosacral view is in order to evaluate alignment of the lower spine. Pelvic asymmetry or a short leg can imbalance the entire spine. Imagine the cervical continually tilted to one side in order to hold the head straight; this constant stress initiates problems over time.

Oblique Views: These views allow evaluation of the intervertebral foramina through which the nerve-roots exit the spine.

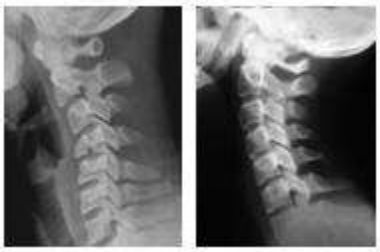


Flexion and Extension: These views allow evaluation of motion between vertebral segments. Look for both restricted motion, which can be caused by adhesions and facet pathology, and hyper-mobility, which is caused by ligament laxity. In the presence of C2 pf-NCS findings one can suspect a suboccipital problem. Since there is no C1 test site, flexion/extension lateral cervical views allow evaluation of movement between the occiput, Atlas and axis. In flexion, the spaces between the posterior arch of the Atlas, the occiput and axis should separate while extension normally narrows these spaces.

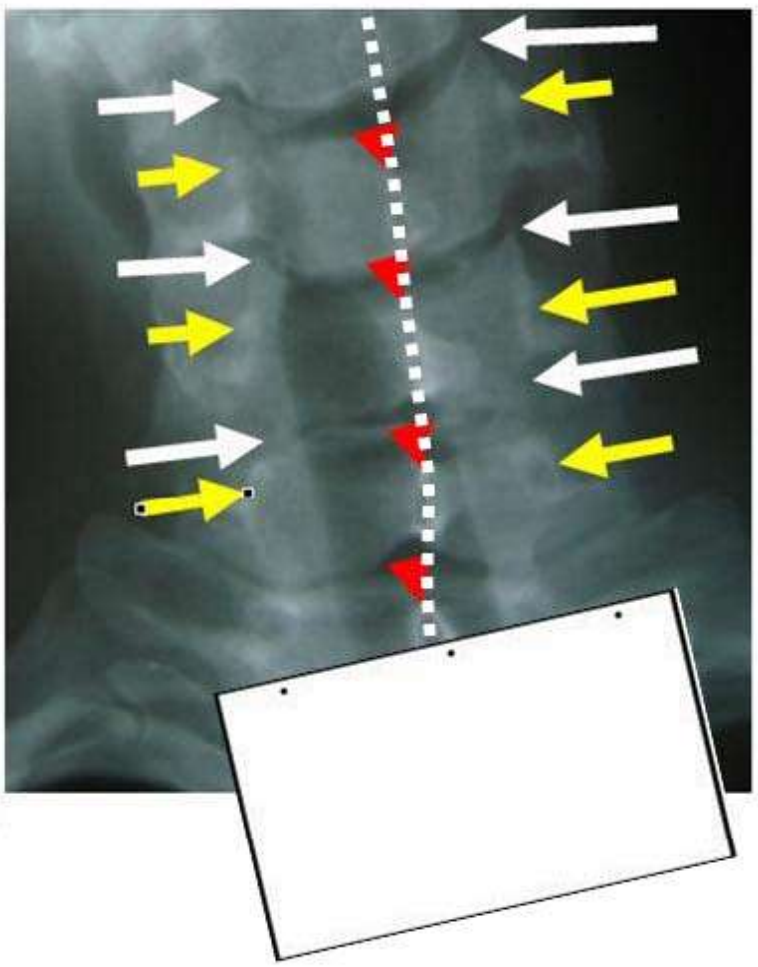


AP Lateral Bending Views: These views evaluate coupled motion. Normally the cervical spinous processes rotate away from the side of lateral bending. Abnormal vertebral rotation of the segment above and/or below a nerve-root suspected of pathology, based on the pf-NCS findings, verifies radiculopathy. If the coupled motion is normal this would rules out radiculopathy and suggest peripheral neuropathy.

Lateral View: Lordosis, the forward curve of the spine, opens the disk spaces. Kyphosis, the backward curving of the cervical spine, compresses the disk spaces leading to degenerative disk disease. Patients with reverse curvature (kyphosis) should avoid working with the chin down and use a telephone headset.

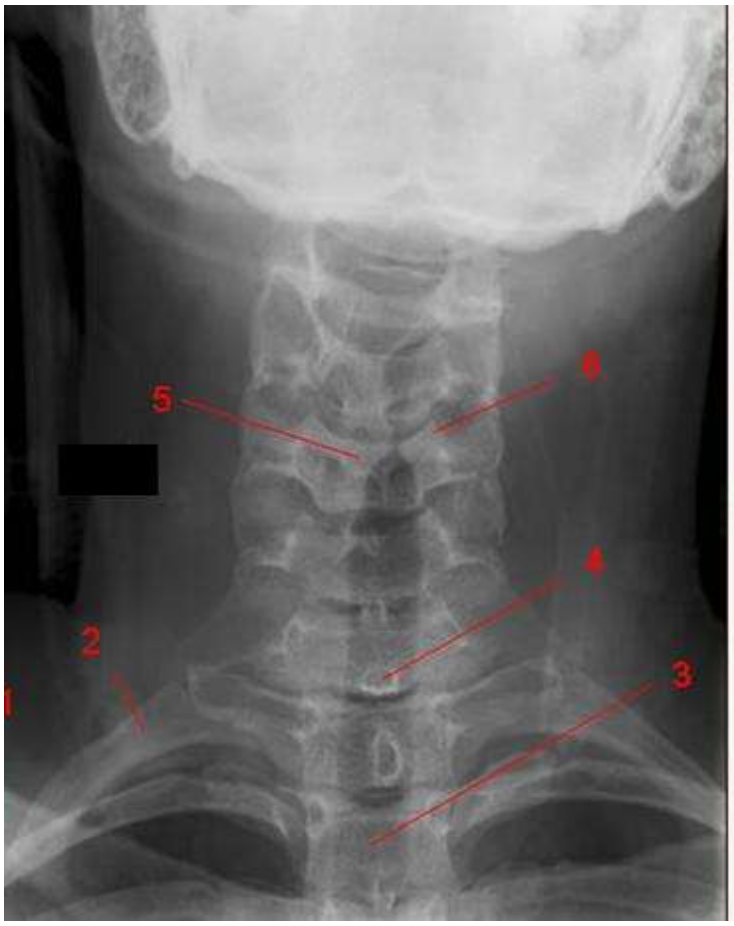


Thoracic Radiculopathy: Lateral bending views of the thoracic spine will, in the presence of nerve-root pathology, demonstrate reverse wedging of the disk space associated with the involved nerve-root. In other words, normally the disk spaces compress on the concave side while opening on the convex side and look like a narrow slice of pie; at the involved level, the slice is backwards.



Taking Lateral Bending Views: The main point in taking the lateral bending views is to have no element of rotation in either the cervical or lumber views: No head rotation in the cervical and no pelvic or thoracic rotation in the lumber.

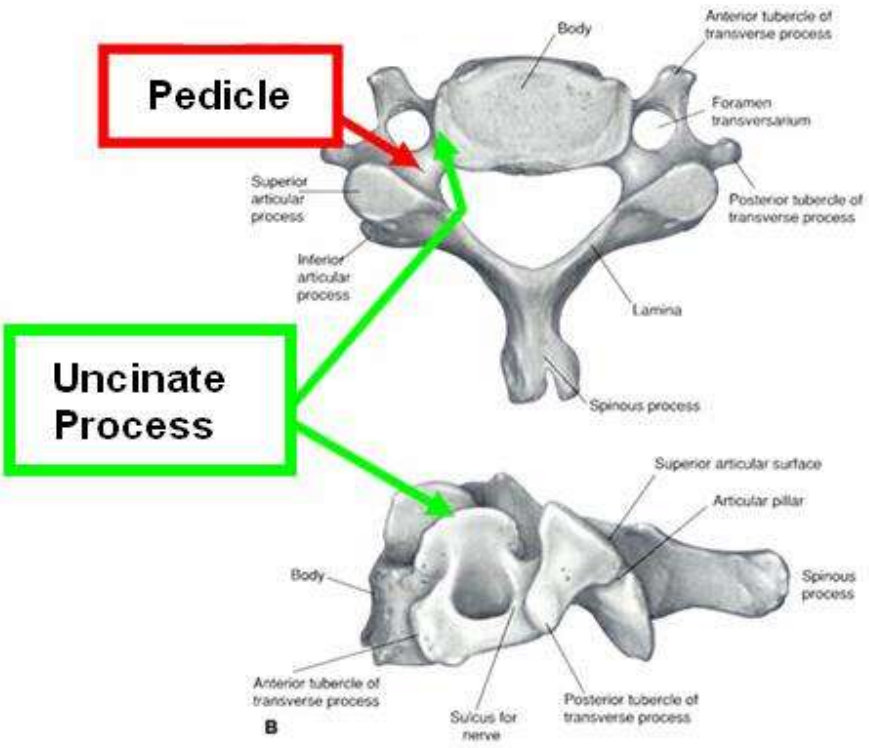
Analysis: Identify the lateral boarder of the vertebral body by finding the tip of the uncinate (joint of Luschka) processes (white arrow), or the lateral pedicle (yellow). The upper tip of the spinous process is marked red. In the lumbar spine, the lateral body is easily seen. The back of a business card works as a tool. Place dots as shown and line up outer dots with the outer boarder. Then note the position of the spinous. **1. Clavicle**
2. 1st Rib
3. Trachea
4. Spinous Process
5. Vertebral Body
6. Uncinate Process (Joint of Luschka)



Follow-Up X-ray: To be sure the treatment is effective it may be desirable to take a follow-up view. Only the view showing the greatest abnormal motion is usually necessary. Also, it is not usually necessary to perform the entire pf-NCS again, since the major nerve-root(s) involved can be tested and a comparison made between the number from the first test and the second.

Motion Radiography: Over the last few years, dynamic motion radiography has been popularized for viewing regions of the spine as they move through a range or motion. However, since the greatest abnormal motion is at the limit of motion, there is no need to watch the spine move through its range of motion. Remember, as Guyton explains, locomotion is not under the direct control of the brain. Along with postural coordination locomotion is controlled by spinalcord centers. Humans are incapable of voluntarily control of the rotation of a vertebra. If it rotates in the wrong direction, this indicates a neurological pathology.

Ether the uncinate process or the pedicle can be used to mark the lateral boundary of the vertebral body.



PART III - Conservative Treatment Algorithm

Conservative Treatment Algorithm:

1. In a case where abnormal coupled motion is noted above and/or below the involved nerve-root, the most conservative treatment is specifically targeted manipulation, along with addressing any anterior to posterior or right to left imbalances.

2. Adjunctive conservative therapies should be considered, such as analgesics, ice packs, and traction. Bed rest is recommended in severe pain, especially when pain is aggravated by activities.

3. Absent a positive response, the next option would be to add trigger point injects with facet or epidural blocks in conjunction with the above.

4. The last option is surgical consultation.

Ice Packs: Most patient dislike ice, but the truth is that ice is much more effective than heat

because it increases blood flow, like heat, but unlike a hot pack, ice reduces swelling. Motion with ice is recommended regardless of the region being treated.

Cervical Traction: Traction is quite effective in conjunction with ice and after specific manipulation as correlated with pf-NCS and lateral bending radiographic views. Traction is to patient tolerance, but should be strong enough to open the joints - usually 30 to 40 pounds or more. Intermittent traction may be more effective than constant traction. This really depends most on what is acceptable for the patient.

Lumbar Traction: Lumbar traction should be with the pull from the posterior with the patient face up and the knees flexed. The theory that a specific angle is required to pull a specific disk space apart has been used to market traction units, but it is highly unlikely that this makes much, if any, difference. This seems to be more of a sales gimmick to justify the high price of some traction units. What is effective is using muscle stimulation on the abdominal muscles while the patient is in traction. Weak abdominal muscles predispose the patient to low back problems, so strengthening the abdominal muscles while weight is off the spine is logical and quite effective. The pull on the lumbar spine should be constant. Computerized devices that monitor muscle fasciculation are not logical. A muscle going into spasm is not stopped by releasing the pull on the muscle; quite the opposite is true. Stretching a muscle stops a spasm. At any rate, the traction pull should be close to half the patient's body weight.

At the end of a 20 minute (maximum time) session, the pull should be reduced very slowly. As the pull is reduced the patient should slowly lateral flex the lower back. This helps prevent a sagging disk from being acutely pinched as the disk space closes at the end of the traction session. Patients who experience an acute pinch should have subsequent traction in the prone (facedown) position, so the sagging disk falls away from the spinal canal and nerve-roots.

Water Exercises: An effective adjunctive treatment in combination with traction is water exercises. The water exercise that is the simplest, and very effective, is to have the patient use a floatation vest while moving his legs as if pedaling a bicycle. Additionally, the patient can wear an ice pack over the affected region while doing the water exercise. Disk herniations of as much as 12 mm have been reported to respond well to this method.

Healing Disks: Disk tissue is known to have almost no way to heal, but generally disk tissue can heal if given enough time. In 9 to 12 months disks have been known to heal if the patient avoids any activity that caused pain during the acute phase of his symptoms. In other words, he cannot do more and more as he feels better. For 9 to 12 months he must act as if he still is in the acute phase. Whatever aggravated his symptoms during the acute phase is sure to bring on a recurrence, which means the 9 to 12 month clock starts again.

Acupuncture: Acupuncture is an effective treatment that can add to any program of care. It

is reported that one off label use of the pf-NCS device is the stimulation of acupuncture points. The pf-NCS device selectively stimulates A-delta (fast pain) fibers ("pin prick" fibers). These devices have not been given FDA clearance for such use, and no claim of effectiveness is implied by the manufacturer. However, it is obvious that the 250 Hz frequency would stimulate A-delta fibers, and probably be more specific in doing this than a needle. As explained earlier in the text, the A-delta stimulation causes a release of histamine, which is what Dr. Nakatani found needling causes. However, it will be recalled that Nakatani found an electrical stimulation of 10 to 20 seconds caused a release of histamine equal to over 2 hours of standard needling. All that is needed is to bring the A-delta stimulation high enough to cause a mild stinging sensation for several seconds. Remember to warn the patient not to scratch the areas treated, which will disperse the histamine and stop the reflex neurological benefits of the treatment, such as increased circulation.

Whatever the device used for treating acupuncture points, the low back sites are those seen on the photo of the "Iceman" in chapter II. The stimulus should be strong enough to cause pain and the duration at each site is from 15 to 20 seconds. Once the sites are stimulated the patient must be warned to not scratch the area. It will become itchy due to histamine release from the cutaneous cells. This itchiness continues stimulating the points and the physiological response, due to histamine release from the cutaneous cells. Be very observant, because even while explaining to a patient that he should avoid scratching the sites, he may start scratching.

Manipulation: Before performing a manipulation, the examiner should carefully assess the range of motion of the region. Limitations noted previous to manipulation are subsequently useful in determining the reoccurrence of abnormal coupled motion and need for repeating manipulation. This helps avoid exposing the patient to unnecessary repeat radiographic studies.

Cervical Pre-Testing: Have the patient face up on a table and ask him to roll his head as if it were a ball on the surface of the table or floor. Have the patient roll the head slowly to the right and left. Do not let the patient slide the back of the head on the table surface, but roll it so that the entire head moves to the side to which he is turning. This creates normal movement between the facet joints. Turning the head on an axis, where the head stays in the center, is not a natural movement. The cervical facets (zygapophyseal joints) are true diarthroidial (freely movable gliding) joints. However, when the head is turned without any element of lateral bending, these joints do not glide but, instead, jam against each other on the side toward rotation and separate on the side away from rotation. This is an example of how human vertical posture has changed the stresses placed on the spinal structures.

It is important to understand how the cervical spine should move because once the manipulation re-establishes correct coupled motion, non-weight bearing head rolls will help prevent recurrence and establish neurological coordination. After manipulation the

range of motion is checked to see if the previous limitations have improved. If the motion is improved, then generally the patient should be instructed to slowly perform the non-weight bearing head rolls for about 60 seconds every waking hour, followed by a minute of two of rest. The patient should also perform the head rolls just before falling asleep and upon waking. If at anytime between the hourly head rolls the patient feels neck stiffness, he should repeat the rolls. S/He should strictly avoid twisting the neck around while seated or standing to relieve stiffness. Patients who closely follow this regime recover quickly, while those who do not suffer recurrences until they do.

Observation: The examiner must closely observe how far the patient can roll the head from side-to-side and should especially watch the shoulders. By placing a hand on each shoulder and holding out the index fingers, the examiner can compare the distance from the chin of the patient to the index fingers as the patient rolls his head. Often, it appears that the patient is rolling his head equally, but closer inspection reveals that the patient is slightly lifting his contralateral shoulder in order to complete the full range. After noting the range of motion, ask the patient to roll the head again and point to the exact location where he feels stiffness, restriction; ask if the neck feels stuck. Most patients will give an indication of the general area, but it is important to make the patient repeat the roll until he can exactly identify the location of the restriction. The examiner should make careful notes and a diagram of his observations to use during follow-up evaluation — careful notes avoid taking repeat X-rays.

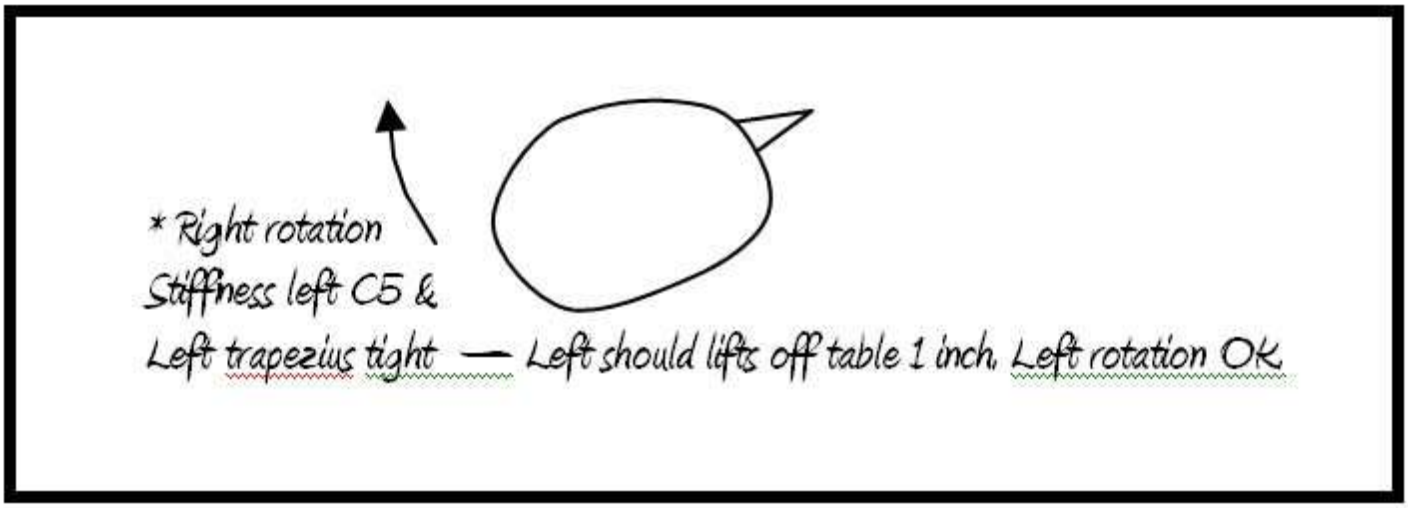
Post Manipulation Testing: The head roll allows the examiner to evaluate the effectiveness of the manipulation. The patient should, as explained above, perform the non-weight bearing head roll every waking hour during the first 24 hours following the manipulation. The next day the patient should perform the roll every 2 hours, and the next day, every 3 hours. Eventually, the patient should perform the roll before falling asleep and upon waking, and a few times each day or when experiencing any stiffness. Usually patients learn how it feels to have normal neck motion. Remind the patient to make an appointment if any restriction does not abate after performing the head rolls a few times.

Wrong Head Roll: Note the point where the head touches the surface does not change as the head rolls. This means the head is rubbing across the surface, and the facet joints are not going through normal diarthroidal motion.

Correct Head Roll: The point where the head touches the surface at rest (middle) is not the point where it rests after rolling to the side. The head rolls off the center starting point to the side and does not rub the table, or surface of the bed. This gives an element of lateral bending as the head rolls to the side and the diarthroidial joints glide over each other rather than jamming together on one side and pulling apart of the other side.

Asterisk (*) denotes the center starting point.

Detecting Abnormal Motion: In the illustration, notice the left shoulder lifts off the table as the head rolls to the right (red arrow). The patient may have limited right or left rotation or both and may or may not lift the shoulder. Remember to ask how it feels and ask the patient to pin-point the exact location of stiffness. Notes generally look like this example:



Lumbosacral Pre Manipulation Test: The simplest and most effective test for the pelvis and lumbar spine is the prone leg raise. Regardless of the type of mechanical problem, the patient will be unable to lift one leg as high as the other. Place the patient prone on an examining table and have him lift each leg as high as he can without lifting the pelvis off the table. The examiner can place his hand over the sacrum to steady the pelvis. The patient must not bend his knee (see the illustration). Have the patient try this a few time and gauge the difference in height. When the mechanical problem is "unlocked", the leg will lift higher and be approximately equal. Often times both legs can be lifted higher after correction of the mechanical problem. This prone leg raise is also a good exercise to keep the joints mobile. Like the head roll, the prone leg raise can be performed hourly to maintain mobility. Obviously, the patient should avoid sitting.

Note: Immediately following manipulation there may be no improvement in the height the patient can lift the leg, but on the second or third try the leg will raise higher if the manipulation was successful in unlocking the joints. This is probably due to the proprioceptive feedback that must reset. Walking after manipulation is recommended.

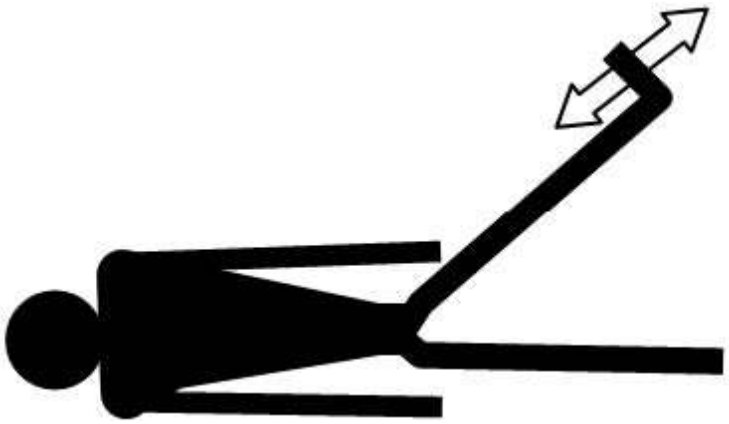
Stretching the anterior femoral muscles: The anterior femoral muscles shorten with sitting. The best way to stretch these muscles is a standing stretch. Have the patient stand in a doorway and hold the door jam with one hand while he bends the knee away from the jam, holding the ankle with the hand on that side. He pulls the foot up and back so the knee points straight down. If performed correctly, the patient will not be leaning forward and his anterior femoral muscle will feel a strong burning sensation. It is important to hold this stretch for at least 60 seconds. Holding for less time will only cause the muscle to contract. After 60 seconds the proprioceptive receptors begin resetting and elongate the muscle. This should be followed by walking or stair climbing to shorten the posterior femoral and gluteal muscles and increase joint mobility. This stretch is reported by many patients to relieve acute episodes of low back pain.

Full-Spine Motion: A simple rehabilitation exercise is the use of non- weight bearing full-spine lateral flexion. The patient is face-up on a smooth surface, such as a Linoleum floor or

a Formica table, and simply lateral bends back and forth without any rotation. This technique can also be used to detect restrictions before and after manipulation.

Abdominal Strengthening: To strengthen the abdominal muscles, lift the knees while hanging from a chin-up bar. This is done until a slight burning sensation is felt. Two such sessions per day is sufficient to strengthen the abdominal muscles.

Prevent Back Pain & More: The above exercises may help to prevent back problems, especially when combined with hanging from a chin-up bar a few times daily. Long term benefits have been noted by the author, who at 15 years of age fractured the L5-S1 facets, resulting in a grade 2 spondylolisthesis. Hanging from a chip-up bar prevents and relieve pain, plus there are side benefits. For example, at the age of 66 I have lost less than an inch in height and maintained the vital capacity I had at age 25. This has also helped maintaining upper body flexibility, evidenced by the fact that I can bring my up-raised arms well behind my ears, while few 50 year olds can bring their arms high enough to covering their ears. Remember to flex the knees while hanging to strengthen the abdomen muscles, without irritating or compressing the lower back.



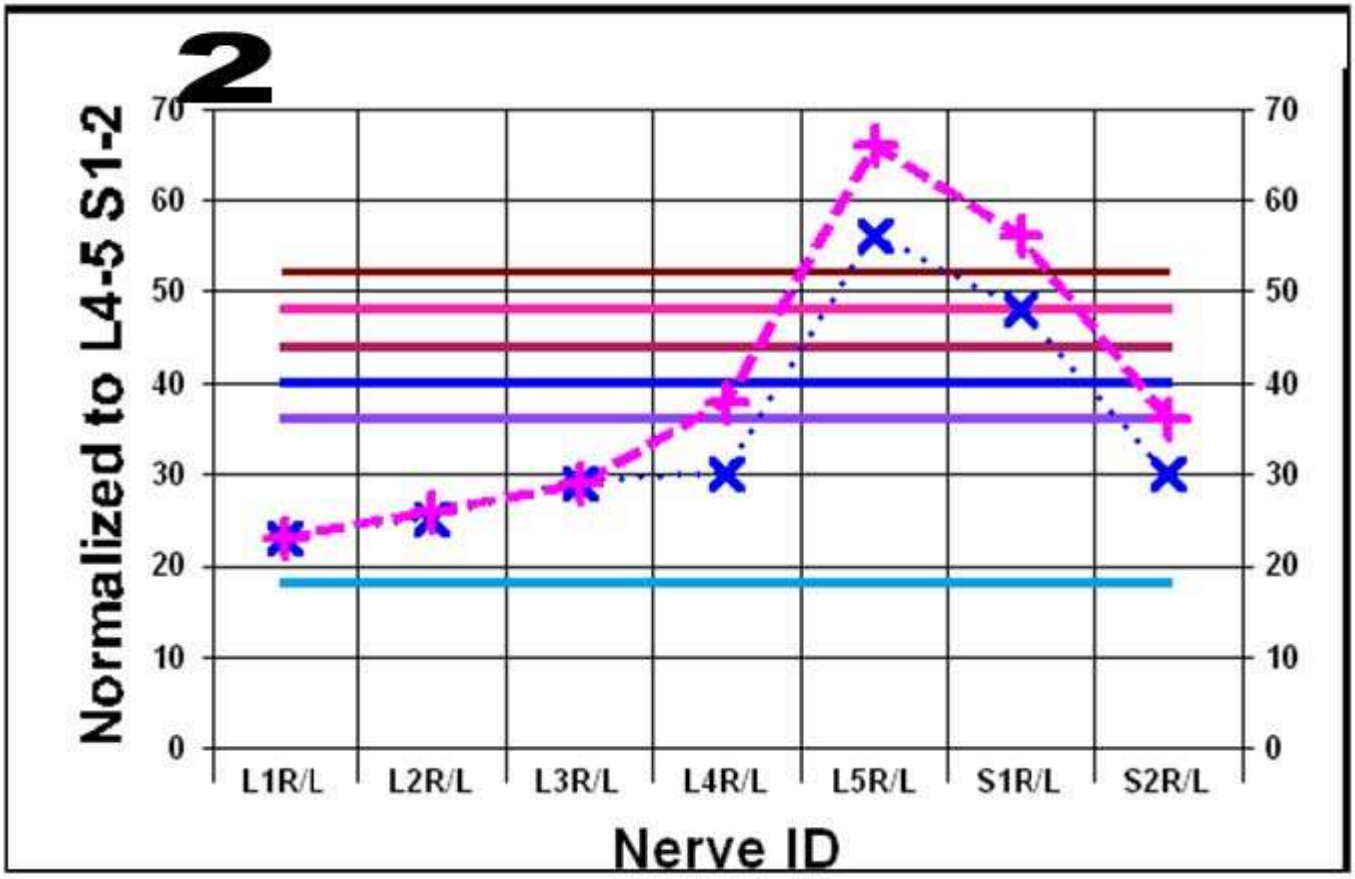
Piriformis Syndrome: Botox® injects have recently been touted as the new way to relieve piriformis spasm. However, there is a much simpler low-tech method that should be tried first. Similar to spinal compression, the piriformis muscle is a victim of vertical posture. We are all familiar with how a calf muscle spasm can be "walked-out". Likewise, the piriformis can be stretched to relieve spasm, but it requires understanding the simple mechanics of this muscle. The piriformis only stretches when the foot is externally rotated and the leg abducted. To accomplish this, place the patient prone. Point the toes laterally and, while stabilizing the knee to keep the leg straight, abduct the leg until the pelvis on that side slightly lifts off the table. Now pump the foot up and down (extending and flexing) about a dozen times. This action stretches the piriformis. Repeat this maneuver a few times and then have the patient walk. It is often found that in males the prevention of piriformis syndrome is to simply have him stop carrying his wallet in his rear pocket. Apparently the pressure form the wallet can cause the piriformis to go into spasm.

Neurological Coordination: A fertile area for enquiry is the correlation between motion and neurological symptoms. Feldenkris, an Israeli physicist, developed a system in which various body parts are moved through ranges until synchronistic motion is achieved. For example, the jaw is opened and closed while the eyes look from side to side. When capable of smooth synchronous motion opening the mouth while looking to the right and closing while looking left, the subject then switches to opposite: mouth open, eyes left, etc. This seemingly simple process can have profound effects. This seemingly ridiculous process has been observed by the author to produce remarkable changes, not only relieved pain, but restoring motor function even in serious cases, such as stroke victims. Most fascinating is that any body parts can be chosen. There are no hard fast rules, just pick a leg or neck motion and start trying to coordinate the motions.

Carpal Tunnel - An Effective Conservative Treatment: The C-Trac is a simple and effective way to relieve and help heal early stage carpal tunnel entrapment. The C-Trac® only needs to be used a few minutes per day to open the tunnel and allow circulation to be reestablished.

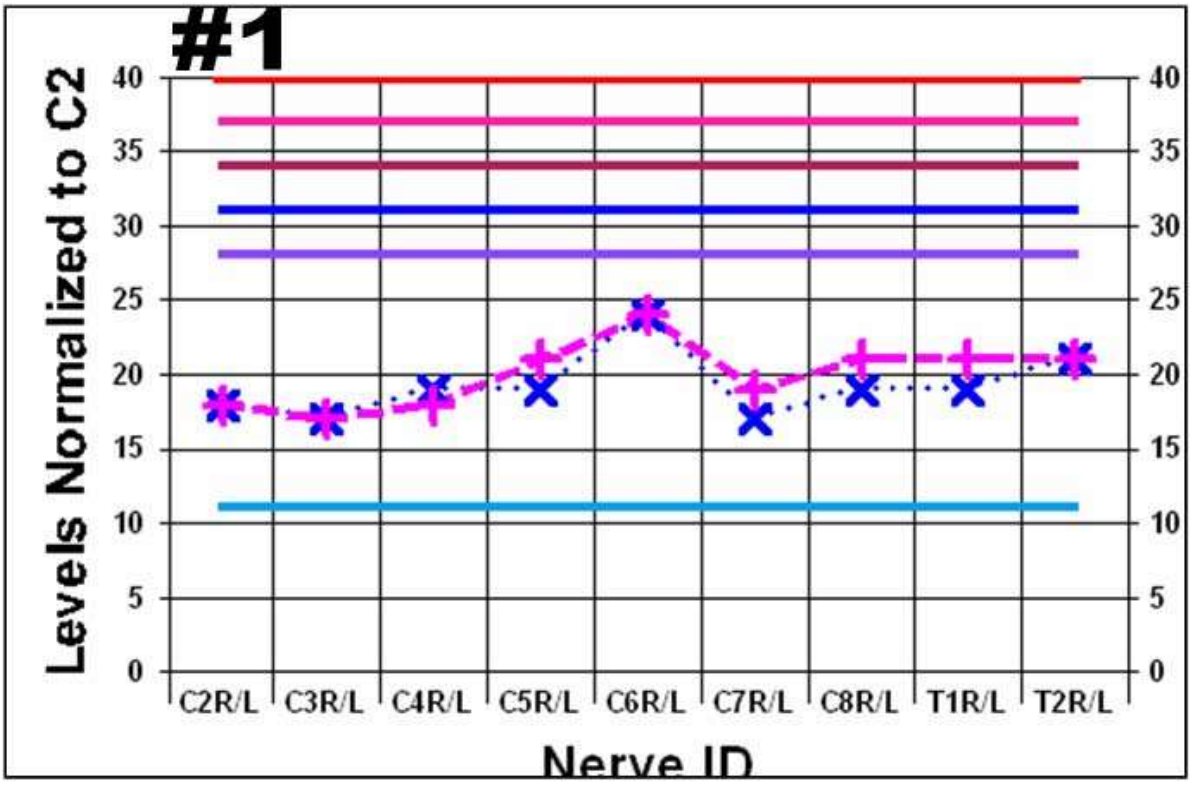
Chapter VII

Analysis Review



THE THREE ANALYSIS RULES
I: The highest rating (hypo-function) identifies pathology.
II: Absent a rating, right to left deviation (20% above and 30% below the navel) suggests pathology.
III: Look for the average normal pattern and notice pathological patterns.

Follow the rules and on a separate sheet of paper write your impression for each graph. Following this section you will find a discussion on each graph. After reviewing your answers, get a new sheet of paper and do it again. Practice - Practice - Practice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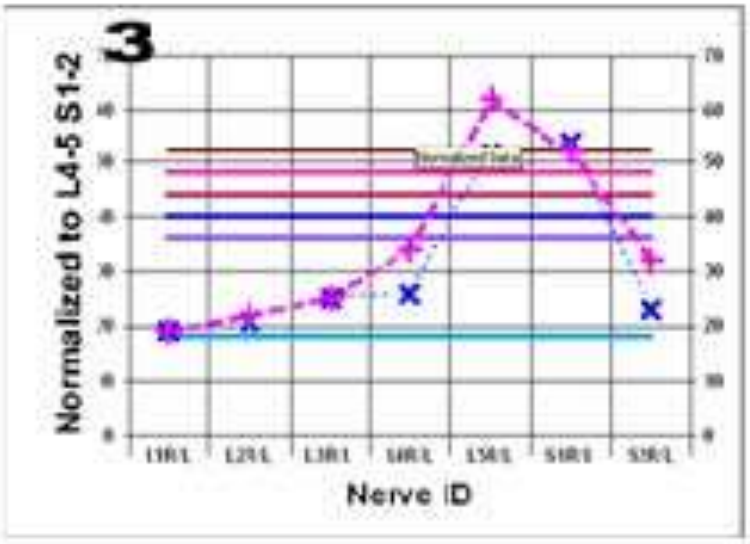
Levels Normalized to C2
40
35
30
25
20
15
10
5
0
C2R/L
C3R/L
C4R/L
C5R/L
C6R/L
C7R/L
C8R/L
T1R/L
T2R/L
Nerve ID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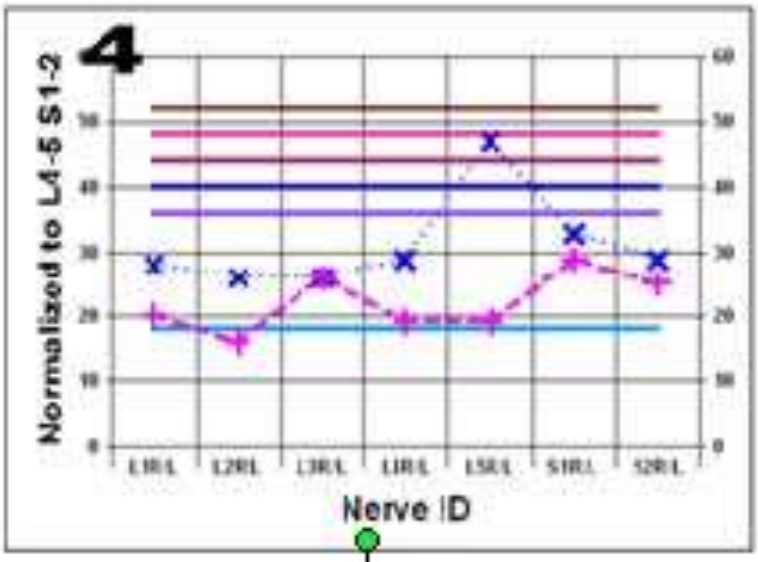
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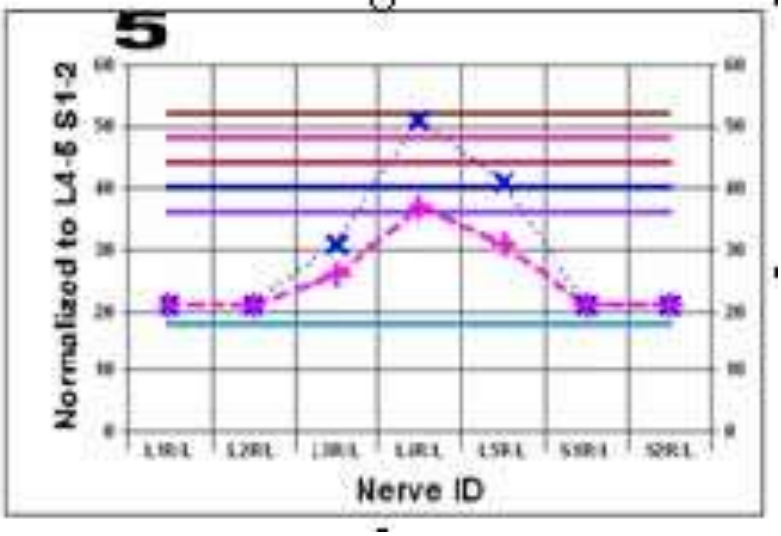
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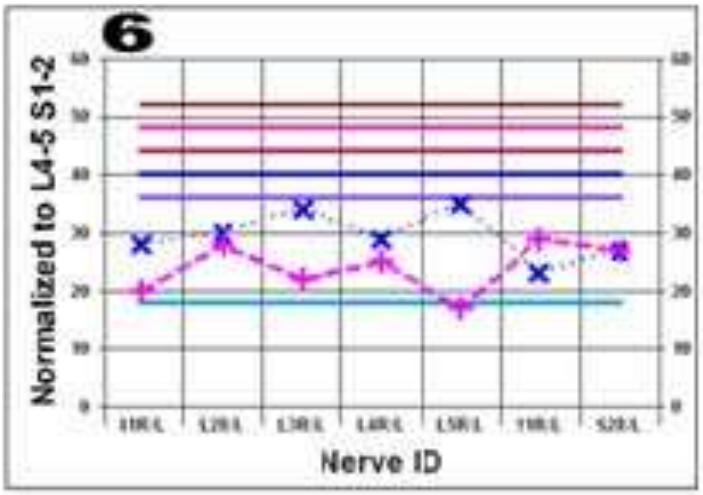
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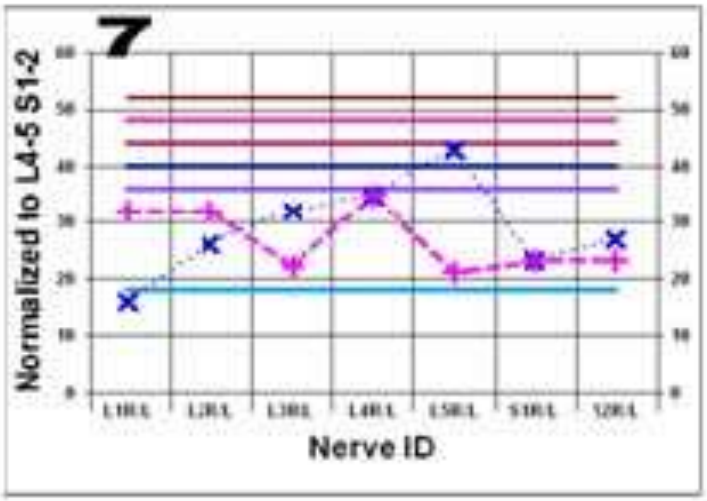
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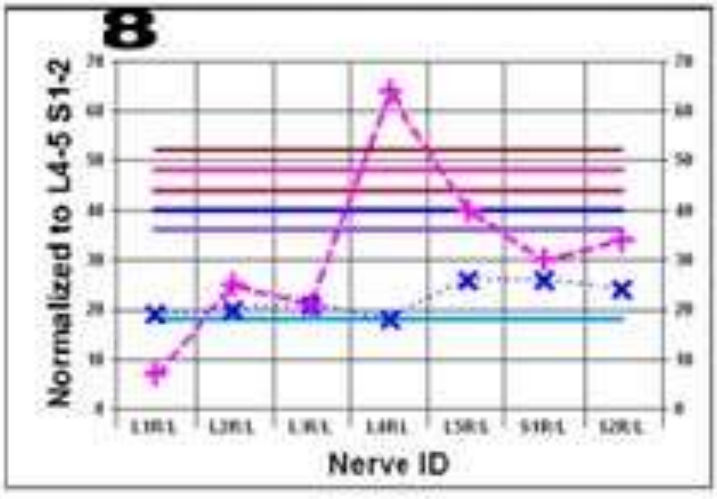
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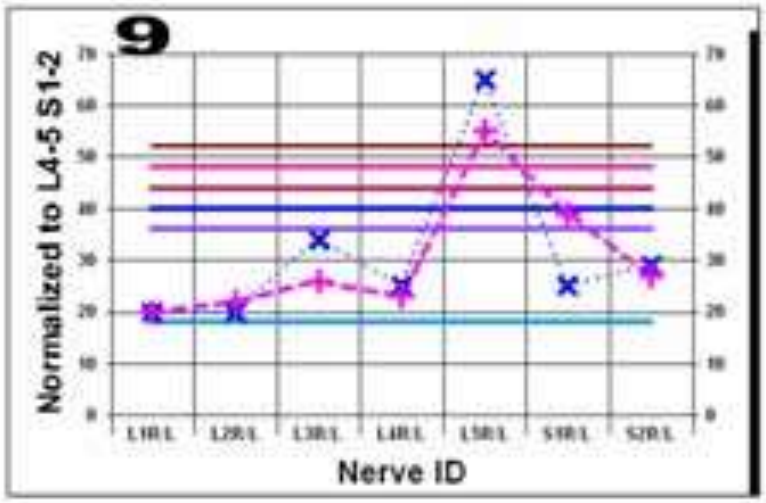
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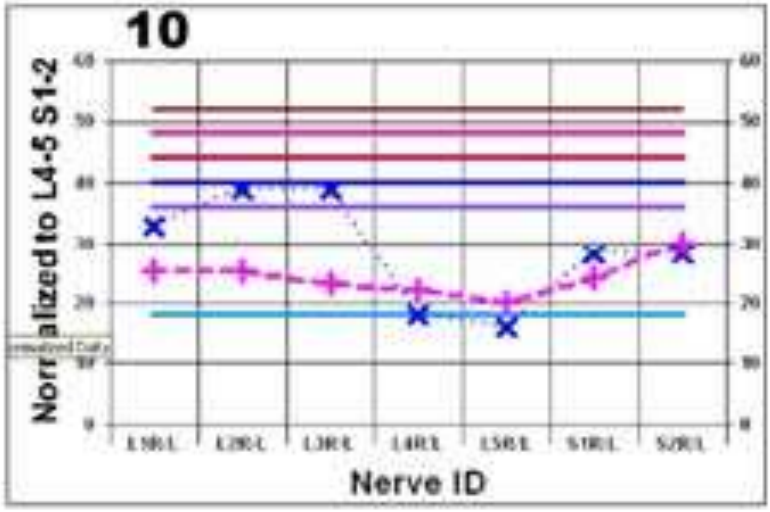
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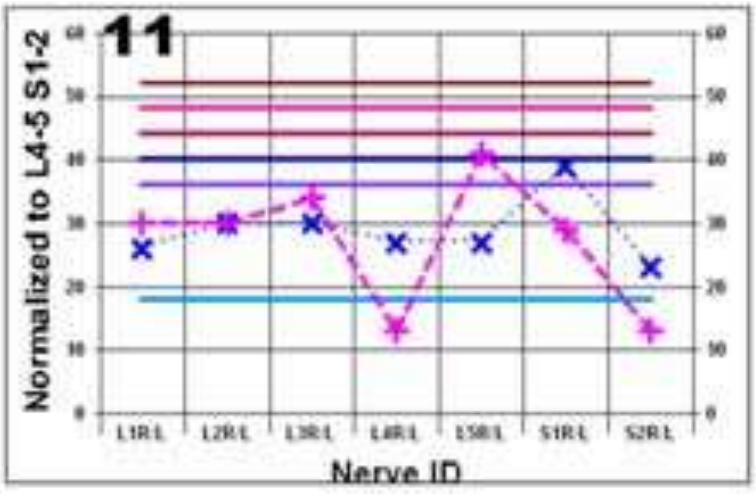
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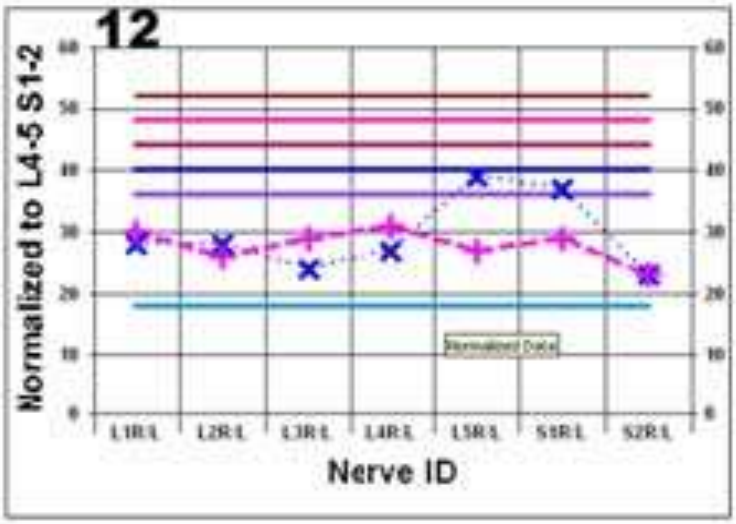
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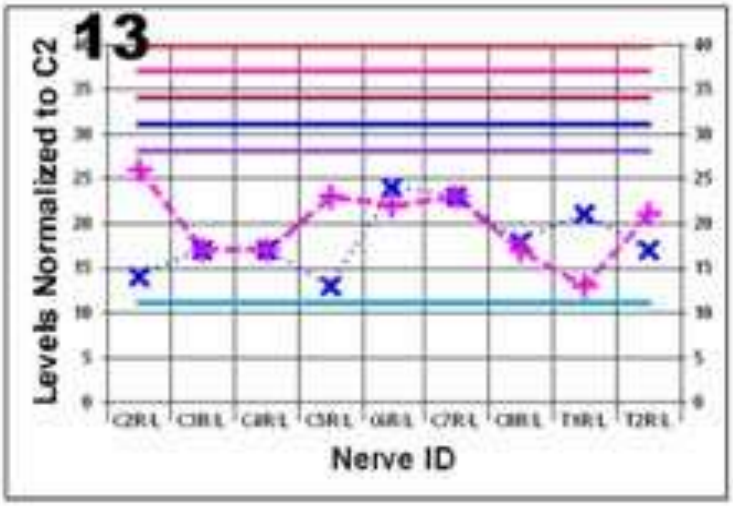
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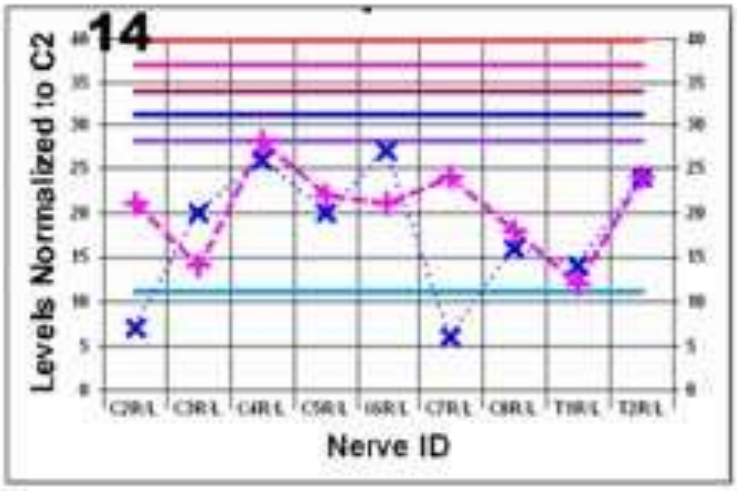
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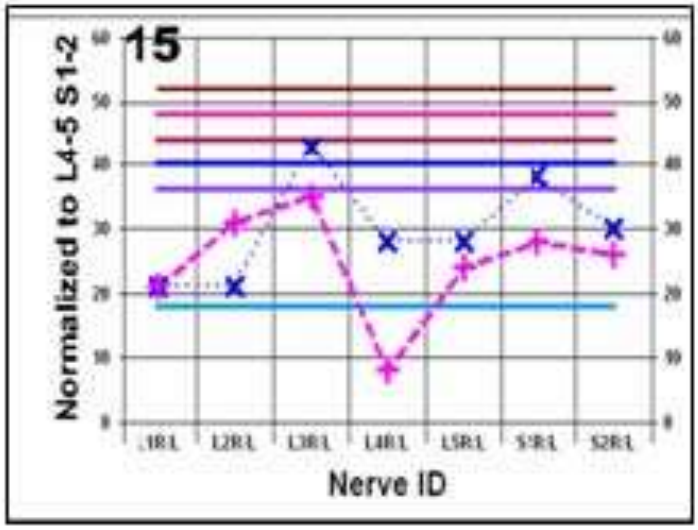
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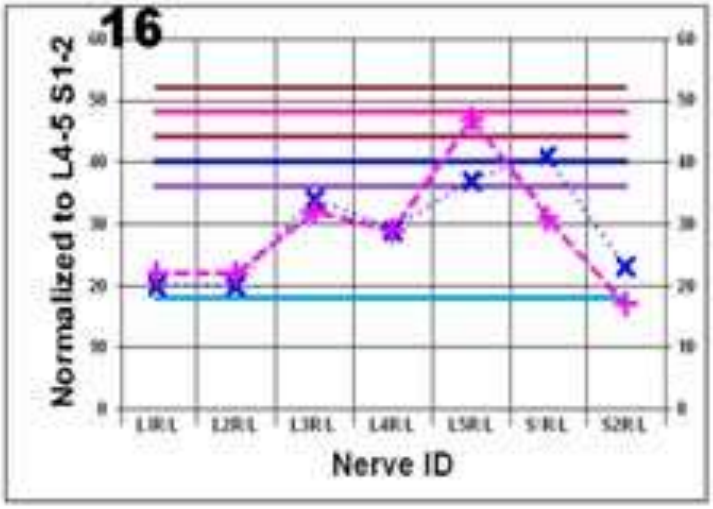
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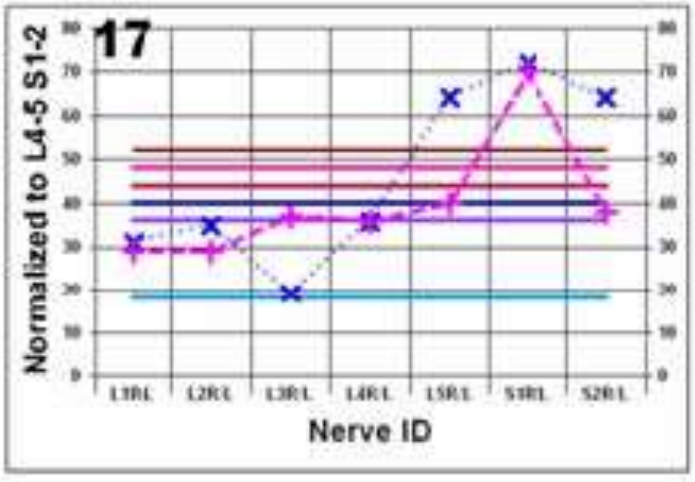
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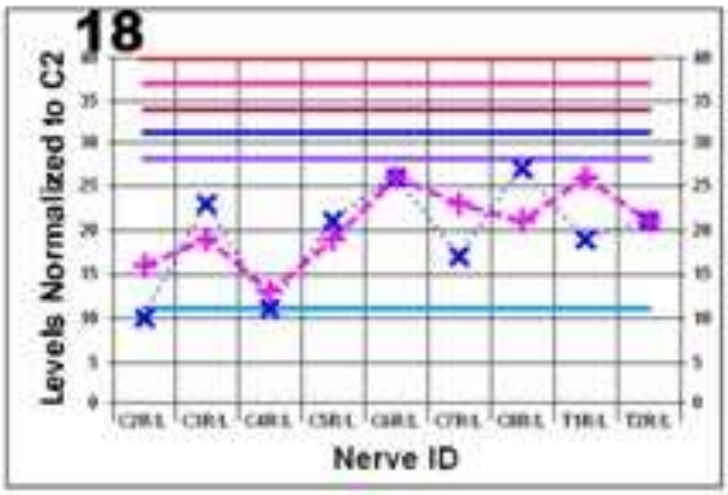
Nerve ID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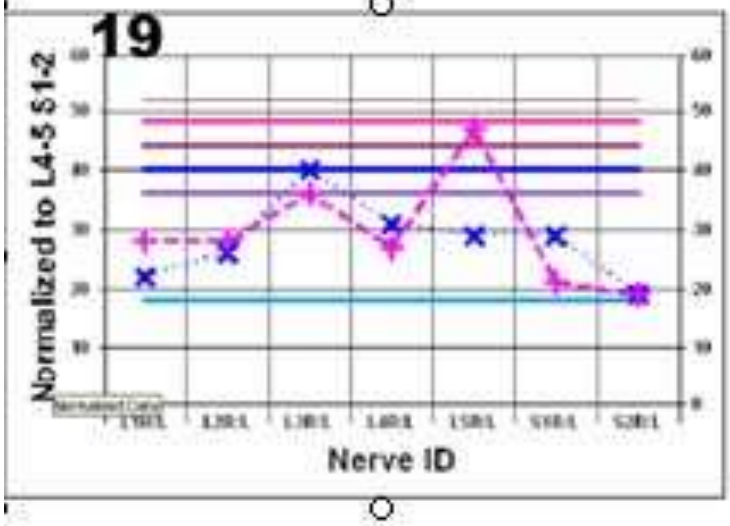
Nerve ID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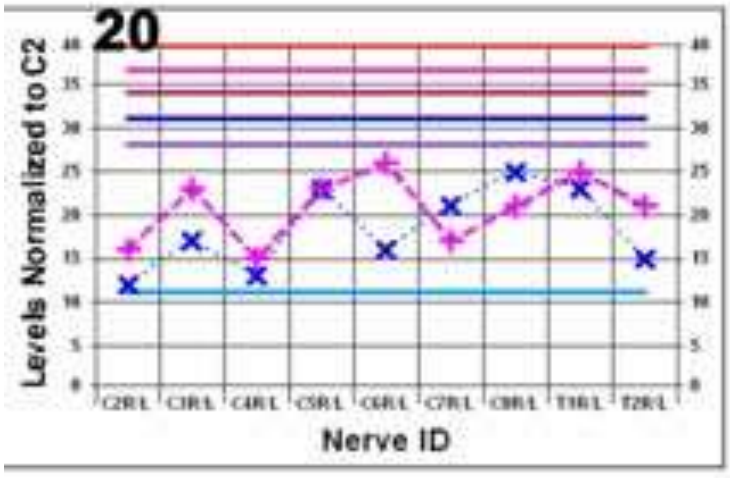
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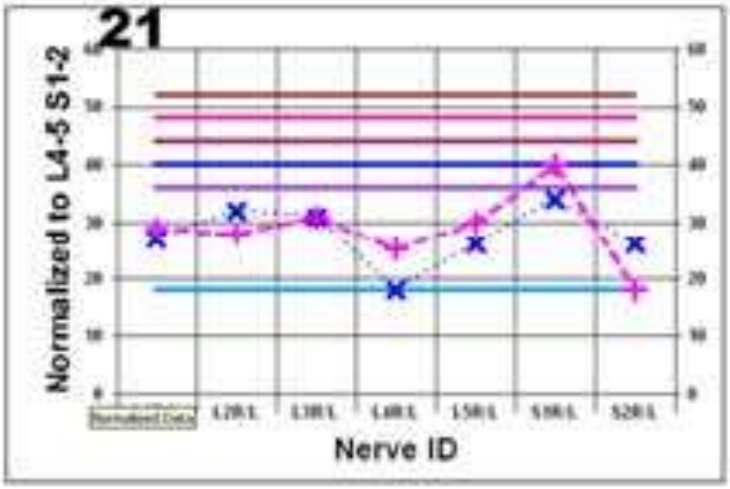
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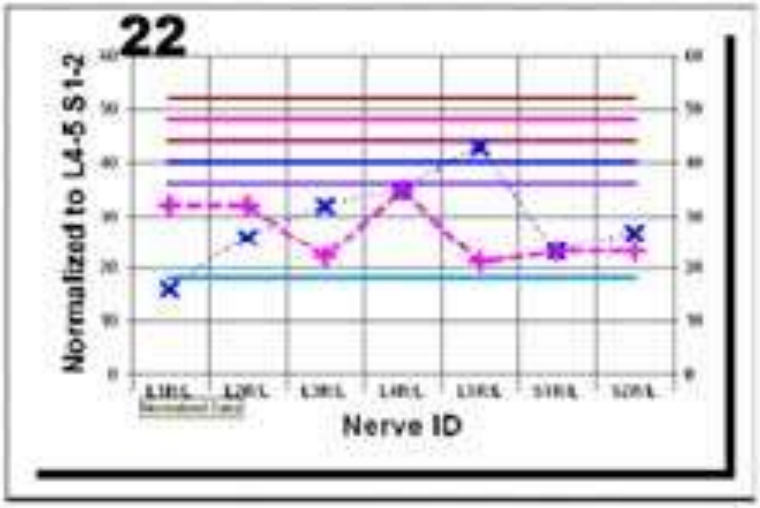
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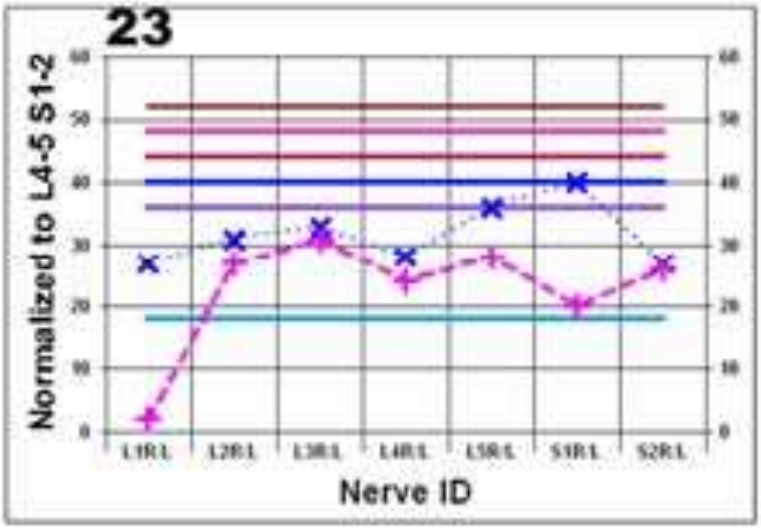
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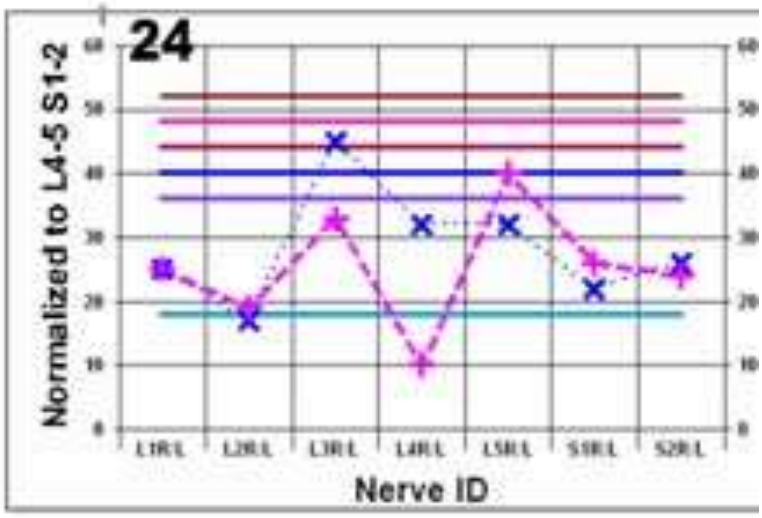
L3R L
L4R L
L5R L
S1R L
S2R L
Nerve ID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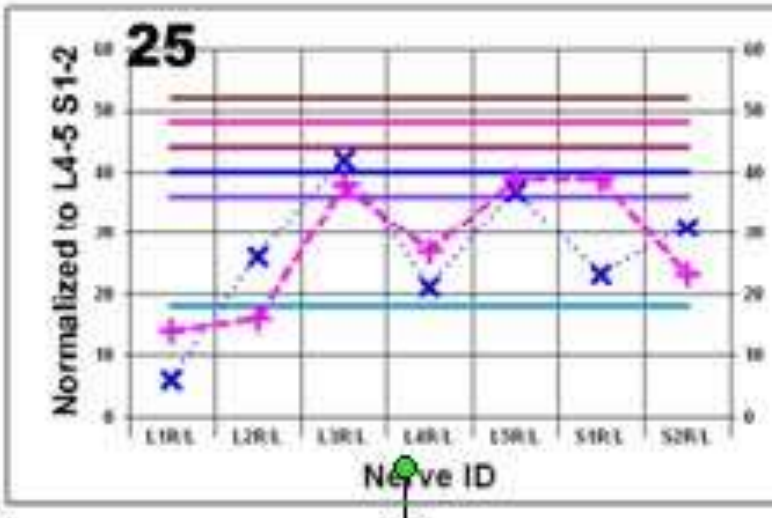
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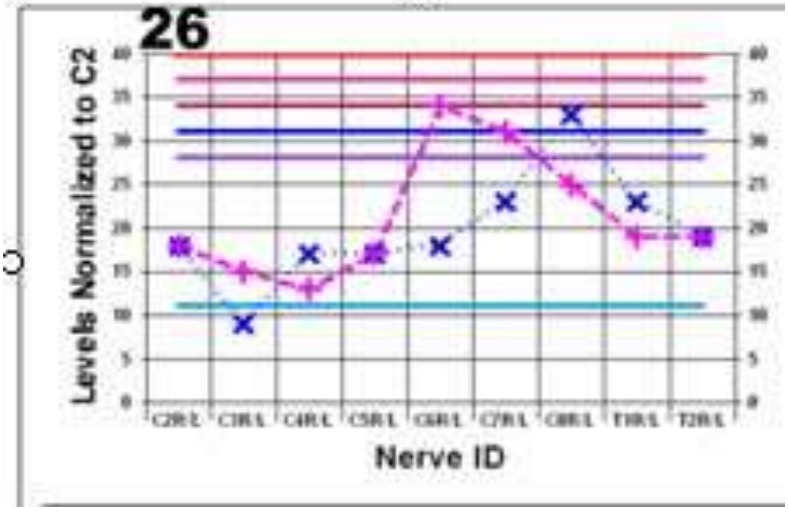
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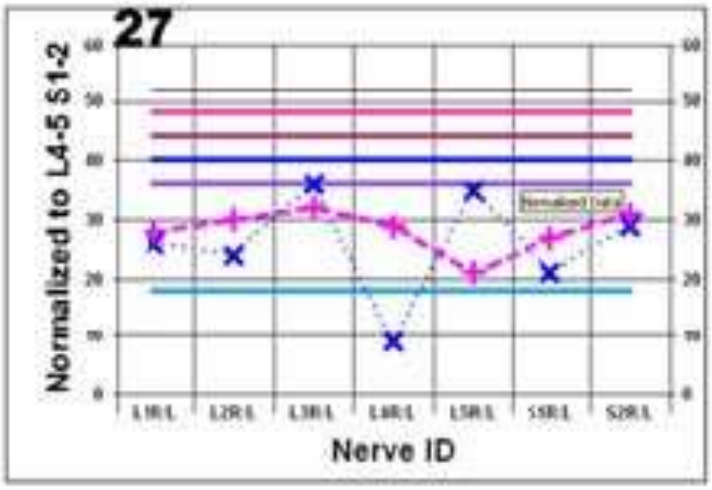
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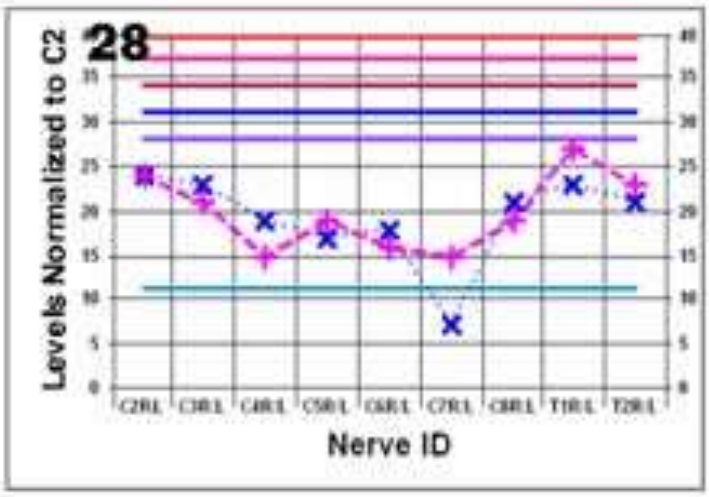
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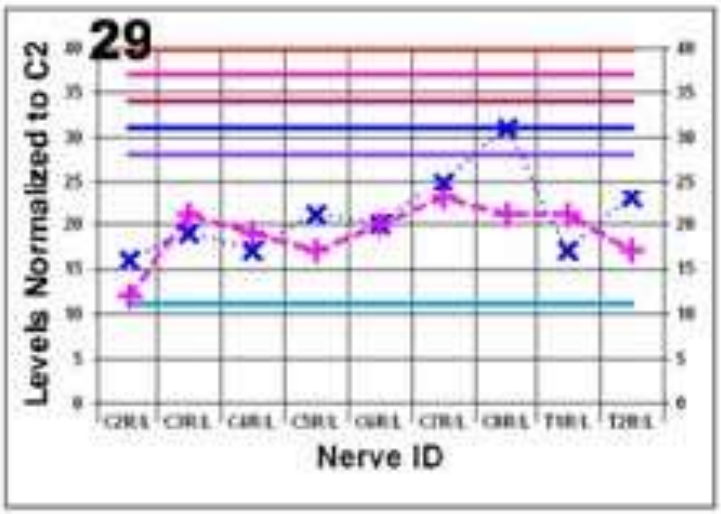
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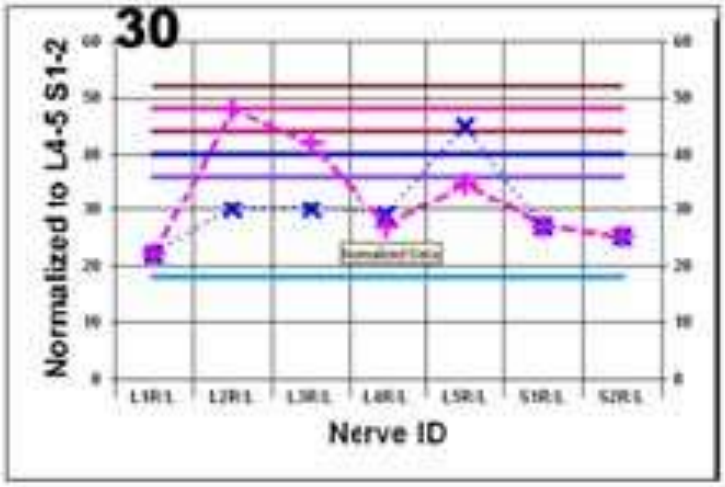
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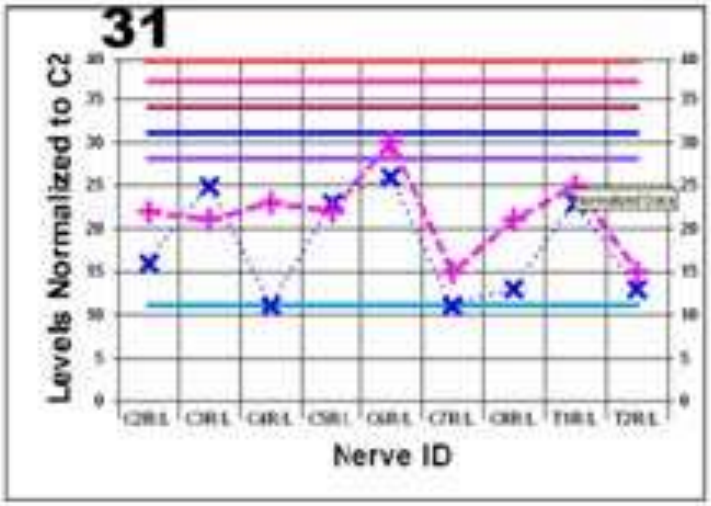
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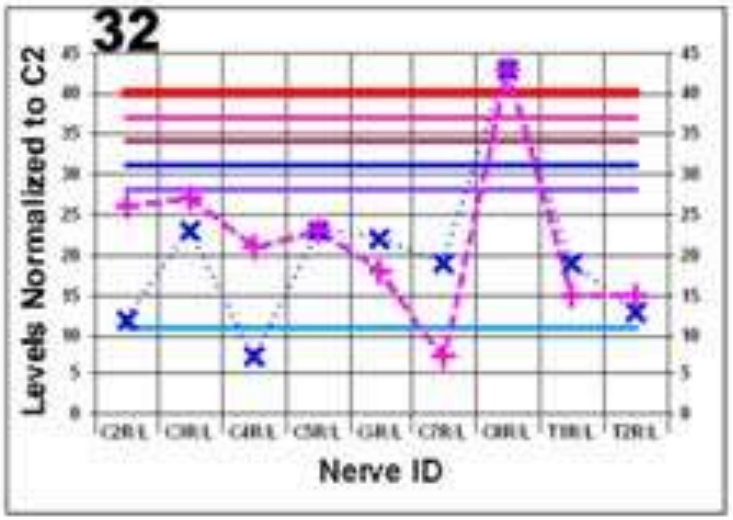
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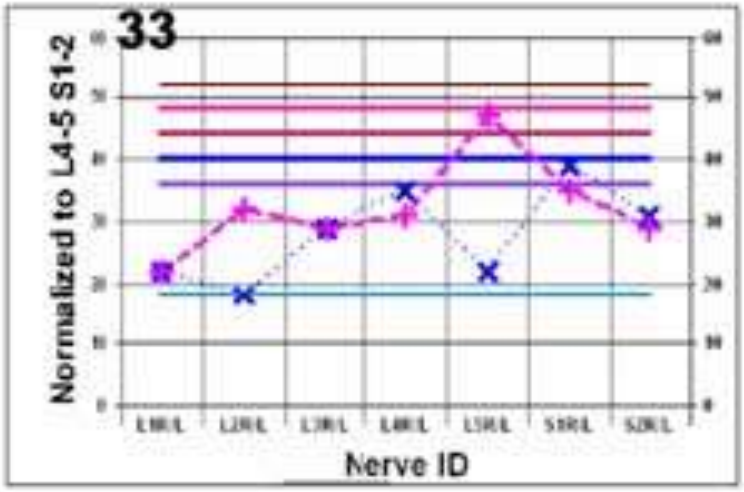
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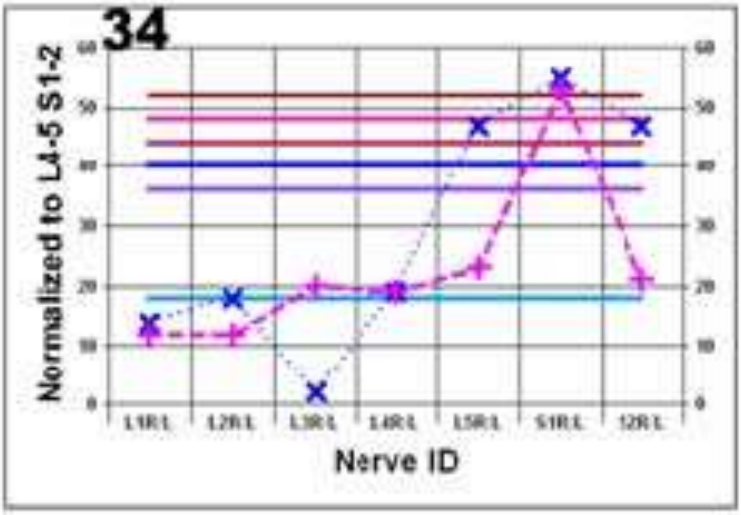
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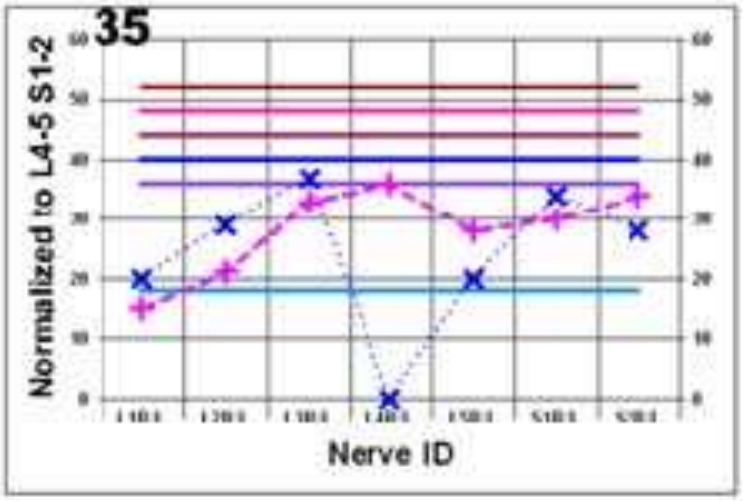
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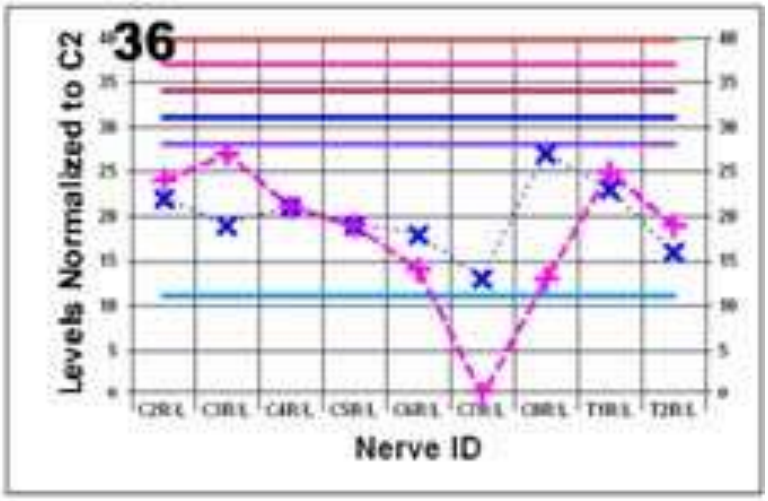
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C4R L
C5R L
C6R L
C7R L
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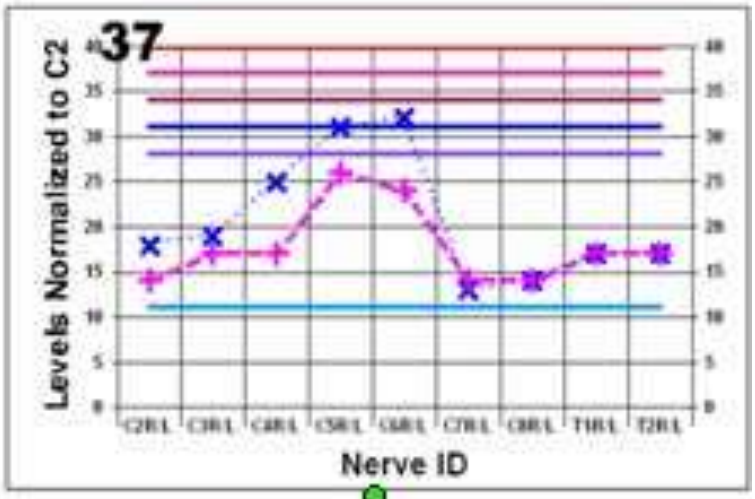
T2R L
Nerve ID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Levels Normalized to C2
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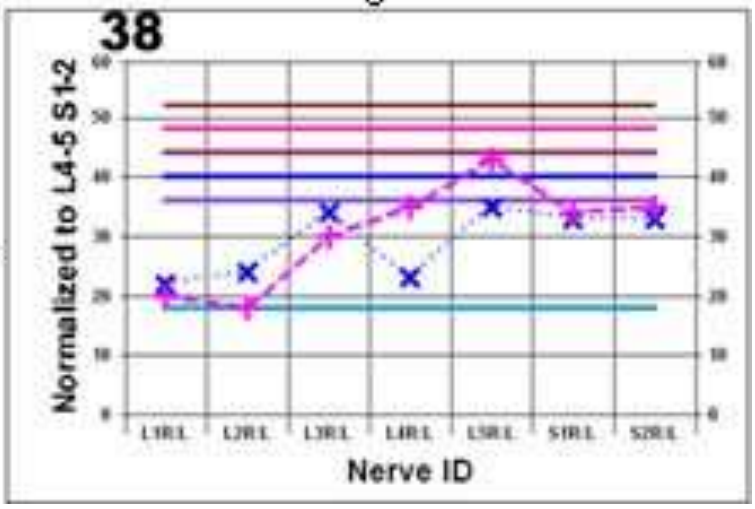
C4R L
C5R L
C6R L
C7R L
C8R L
T1R L
T2R L
Nerve ID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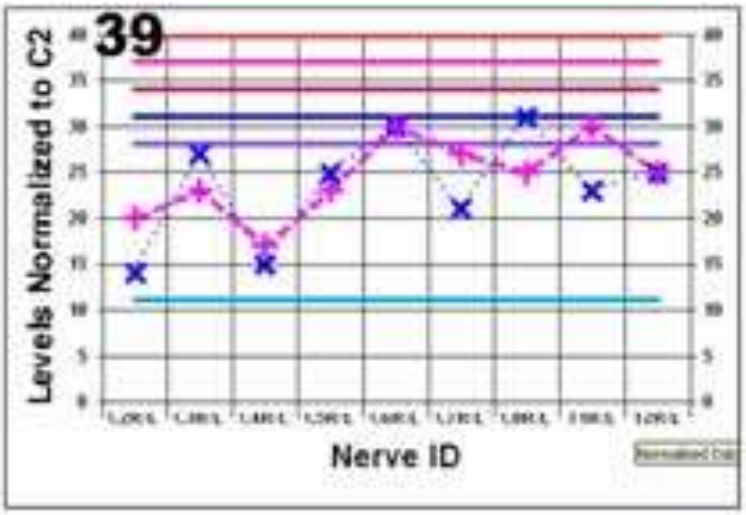
L2R L
L3R L
L4R L
L5R L
S1R L
S2R L
Nerve ID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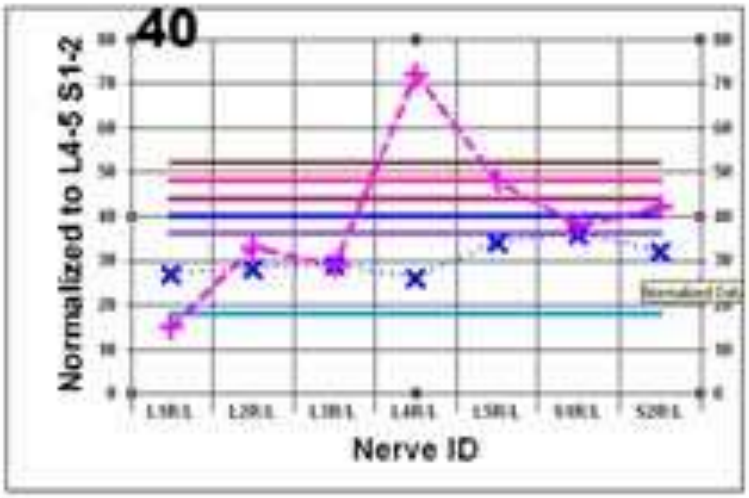
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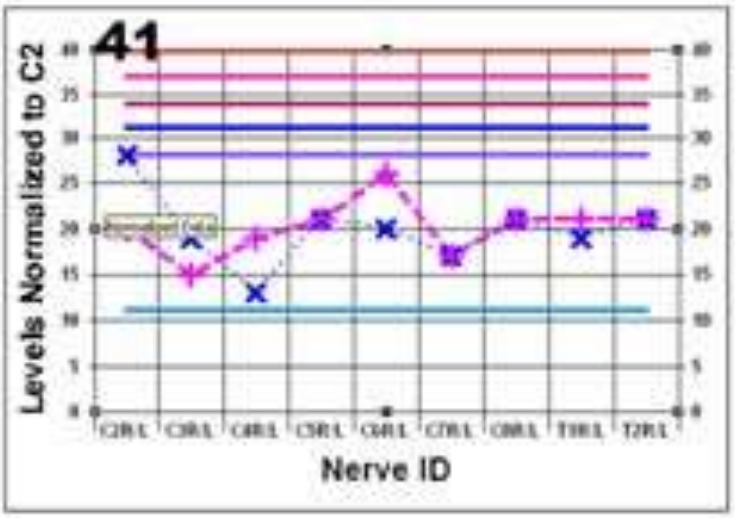
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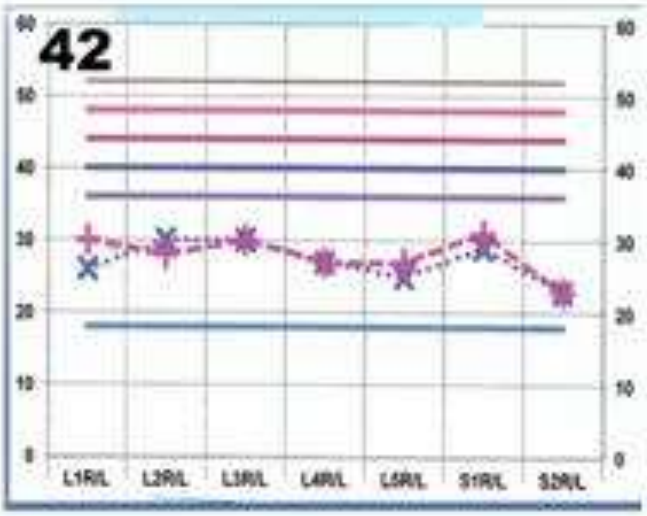
Nerve ID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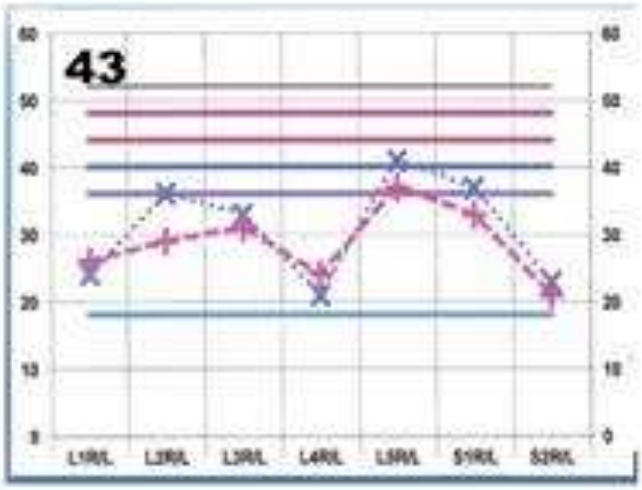
S2R/L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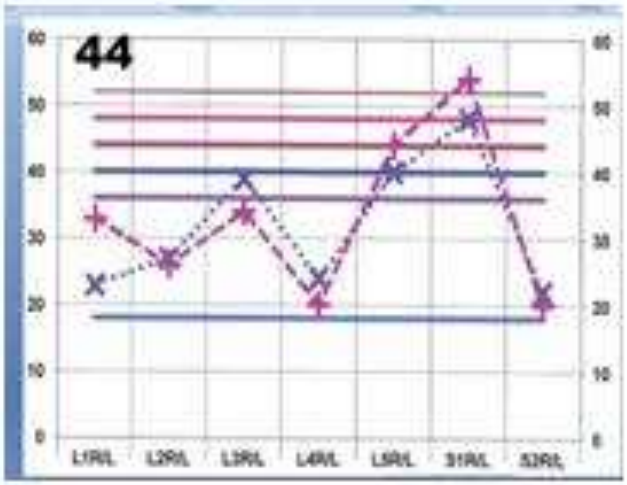
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60
50
40
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20
10
0

1.
Rule #1: No measurement is in a rating.
Rule #2: No significant left/right deviation. Conclusion: No suggestion of sensory pathology.

2.
Rule #1: Two rated hypo-functions - Right L5-S1 (+5 Very Severe).
Rule #2: Does not apply if there is rated hypo-function Usually the level below pathology drops back toward normal. Here the Right S1 is also pathological and S2 drops back to normal. L5/S1 high on the same side means there is an 80% probability of piriformis entrapment. If only one is a radiculopathy and other due only to piriformis entrapment, the worst is likely the radiculopathy since it has two entrapments (nerve-root and piriformis). Note the ramping up on the same side and mirroring of the opposite side (due to crossover).

3.
Rule #1: Right L5 is the primary.
Rule #2: Does not apply. Usually the level below drops toward normal. Here the S1 shows bilateral pathology. The space between the Right and Left L5 is more than a deviation index space. Therefore, the high Left L5 is likely mirroring due to fiber crossover in the spinal cord. Could there be piriformis entrapment? Yes - on the Right. Correlate history and symptoms. Palpate the piriformis to detect spasm. Lateral bending radio-graphic studies can confirm nerve-root pathology.

4.
Rule #1: Left L5 is the primary.
Rule #2: Does not apply. However, the wide separation between the primary pathology (Left L5) and Right L5 is strongly suggestive of Left L5 chronicity. If more acute the Right L5 would be mirroring. This graph could be adjusted up slightly since the Right L1-2 are probably normal and being pushed down by software averaging.

5.
Rule #1: Left L4 is the primary.
Rule #2: Does not apply. Could the Left L5 be a problem? Possibly, because usually the level below the primary pathology drops back toward normal. Here the Right L5 is high as it mirrors the problem at Left L5. The drop is at S1. Note the typical mirroring. Could the Correction tool be used to bring up the measures? Yes, but the highest is still the highest regardless of how the software or examiner adjust the graph. The raw data at the bottom of the report can be used for comparison.

6.
Rule #1: Does not apply.
Rule #2: Right/Left Deviation - L3 and L5. Rule
#3: Pattern - The pattern - highest Left L5, lowest is Right L5. This pattern suggests chronicity of Left L5. The same pattern exists at L3, so it too could be chronic.

7.
Rule #1: Left L5 is the primary.
Rule #2: Does not apply. Left L5 is likely chronic. Note the clear drop off below L5. Left L1 looks like irritation. Generally, L1 can be discounted because this nerve wraps over the iliac crest and is subject to pressure from belts. History and other findings help rule L1 in or out.

8.
Rule #1: Right L4 is the primary.
Rule #2: Does not apply. Right L4 is most likely chronic. Note that disinhibition has failed to bring the Right L4 down. This shows it has severe damage. A positive EMG may be expected. However, would this add any diagnostic value?

9.
Rule #1: Left L5 is the primary.
Rule #2: Does not apply. It is likely L5 is bilateral pathology with the Right also so high. The Right is not within a deviation index space, but it is so high that one should suspect a bilateral lesion, such as central annular disk. There is possibly a Mild +1 secondary problem at Left L3. The examiner did not sufficiently raise the Correction Factor to bring it within a mild rating. History and symptom correlation is needed with lateral bending X-rays.

10.
Rule #1: Left L2-3 primary.
Rule #2: Does not apply. Note the symmetry of the Right side. The right should have been adjusted to the center of the Normal Zone, then the L1-2-3 would be rated in the moderate to marked Hypo-Function rating. The left L4-5 is not hyper-function but being pushed down by the averaging.

11.
Rule #1: Right L5 - Left S1 primary.
Rule #2: Does not apply. Left S1 is bringing the Left L4 up due to mirroring. If the Left S1 was not pathological then the Left L5 would be lower and the chronic pattern of disinhibition more obvious.

12.
Rule #1: Left L5 - S1 primary.
Rule #2: Does not apply. Right measurements are symmetrical - the right is normal. Left L5 - S1 without mirroring suggests a weak possibility of chronicity. History may reveal two or more years of symptoms. If the history does not suggest chronicity then the left L5-S1 is likely mild.

13.
Rule #1: Does not apply.
Rule #2: Significant Right/Left Deviation at C2 - 5 - T1. There is no C1 test site, so a C2 being the highest (Rule #1) justifies looking closely at the suboccipital area. Lateral x-rays views in flexion and extension may reveal poor motion between the occiput, posterior arch of the Atlas and C2 (axis). The space between these structures should open in flexion and close in extension. The C5 and T1 levels show either irritation or chronicity. History and other findings will clarify, along with AP lateral bending views.

14.
Rule #1: Bilateral C4 - Left C6 - Chronic Right C7.
Rule #2: Does not apply. This shows the importance of a complete history. Remember that the data is a baseline for later comparison to determine if conservative treatment is being effective. Lateral bending X-rays are essential in this type of case. Lateral view flexion & extension may be helpful too.

15.
Rule #1: Left L3 - Left S1 primary. Irritation Right L4.
Rule #2: Does not apply. Mirroring is noted. History? Radiographic studies?

16.
Rule #1: Right L5 - Left S1 primary.
Rule #2: Does not apply. Could be adjusted up a little more because it is doubtful that the Right S2 is irritated, and the L1-2 look so symmetrical they belong much higher.

17.
Rule #1: ?????
Rule #2: ????? The pattern strongly suggests L4 spinal stenosis. Nerves leave the spinal cord one level above their exit. Often the S2 is spared.

18.
Rule #1: ?????
Rule #2: ????? The pattern is scattered. There is no specific high or low that suggests

pathology. Following Rule #1 left C8 is the highest. Rule #2: C8 right/left deviation is 50% (normal deviation <20%). Deviation at C7 is 41% and T1 deviation is 37%. Note that above and below Left C8 the measures are low. This strongly suggests that C8 is a primary problem with irritation on the same side above and below. History - X-rays!

19.
Rule #1: Right L5 primary - Probably chronic.
Rule #2: Does not apply. Probably bilateral L3.

20.
Rule #1: Right L5 is the primary.
Rule #2: Does not apply.
Normal? Rule #1: Right C6 is highest and Rule #2 the deviation is 63%. History: Is the right C6 chronic? Did it start over 3 years age? Has the patient had a problem with occipital headaches? AP radiographs in lateral bending - look for reverse rotation at C5 and/or C6.

21.
Right S1. Bilateral?

22.
Chronic Left L5. L1? Belt? History?

23.
Chronic Left S1. Irritation Right L1. Seatbelt?

24.
Left L3 & Right L5 Irritation Right L4.

25.
Irritation L1- belt? Highest left L3 - bilateral? History will show if this is bilateral mild to moderate or chronic bilateral. Chronic Right S1? L5 could be chronic bilaterally. History - History - History!

26.
Chronic Right C6. C7 is also a problem. Left C8! Headaches? Arm right or left pain? In 20% of cases the pain is opposite the side of pathology.

27.
Left L3. Next highest Left L5. Irritation between (Left L4). Could Left L5 be chronic? History!

28.
Irritation of the Left C7. Note the reversed mirroring. History? Any problems with right shoulder?
Rule #1: Right T1 suspicious? History!

29.
Chronic Left C8? Note the usual drop off below.

30.
Highest is Right L2. No drop off - Right L3 is secondary pathology. These could be chronic? More acute Left L5 with mirroring, no disinhibition.

31.
Right C6. Could be bilateral? Noted drop off. Irritation left C4-7-8. Headaches? History? Flexion/extension and AP lateral bending X-rays.

32.
Bilateral C8. Irritation above at C7, especially on the right. Remember, there is no C1 site so the C2 (greater occipital nerve) problem justifies lateral X-rays in flexion & extension. Look for poor motion between occiput, posterior arch of the Atlas and C2 (axis). Space between these structures should open in flexion and close in extension.

33.
Chronic L5. The right to left deviation is usually ignored when there is a rated Hypo-Function, however, the separation at L2 is suspicious because the Left L2 is the lowest on the graph. This suggests probably irritation. But the Right L2 could be chronic - History! Lateral bending X-rays looking for reverse rotation of the L5 and L2.

34.
This is graph #17 before Correction Factor tool was used.

35.
Either chronic Right L4 or irritation Left L4. Note the ramping up to the right L4. This means that it is sure to be Right L4. History and lateral bending X-rays!

36.
Highest Right C3. Next, Left C8. Irritation C7. Chronic C3 - 8? History? Other findings? Lateral bending X-rays?

37.
Left C5 - 6. Lateral bending views!

38.
Right L5. Irritation Left L4 or chronic Right L5? History and lateral bending views.

39.
This is an example of an over adjusted Correction Factor. The examiner is trying to remove all hyper-functions. Note the pattern. If this graph were down everything would fit in the normal zone, except the Left C2 (irritation). History!

40.
Easy - Chronic Right L4. L1 - belt or seatbelt?

41.
Check C1? Right C6? Headaches? History?

42.
This is a 64 year old male with leg pain proximal to right ankle. No sign of radiculopathy. All the lumbosacral test sites are proximal to entrapment cites in the ankle. Lower extremity study is warranted.

43.
This 46 year old male suffered a head-on collision. He has bilateral low back and leg pain. Seatbelt may have injured the L2 - 3. Graph could be adjusted a bit higher - L1-4 and S2 are likely normal and controls (S2 has no disk or facet joints).

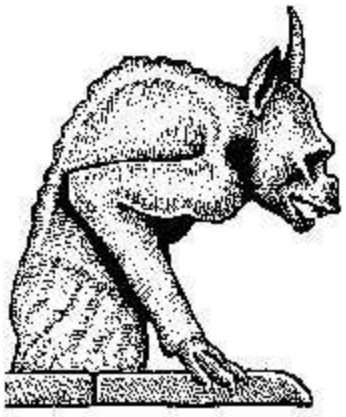
44.
58 Year old male: Twisting fall to floor. Bilateral low back and leg pain. L5 grade 1 spondylolisthesis

Last Word: The pf-NCS is a snapshot of A-delta fiber function at the time of the examination. Therefore, it is important to take into consideration the patient's symptoms at onset, including location, etc. It is especially important to know what the symptoms were at the time of the exam. A patient having a "good day" may test as if he were nearly normal. The pf-NCS is a baseline for comparison. High A-delta measurement (hypoesthesia) without C fiber up-regulation may result in mainly numbness. This is especially important because most patients will not notice numbness unless it is located in the face, or hands. Listen to the patient, but do not trust that he can tell you where his pain is coming from. Remember that motor take much long to heal than small pain fibers. This means you can

have a patient with a positive EMG and a negative A-delta test. His small pain fibers have healed but the motor fiber have not yet healed.

Recent Studies

Updated anthology is available on the website www.paindx.net



PPM
PRACTICAL PAIN MANAGEMENT

A-Delta Pain Fiber Nerve Conduction Study Benefits Patients With Spinal Pain

Of 151 pain fiber nerve conduction studies administered to patients with cervical and lumbar spinal pain, 56% changed treatment plans for the patient and 35% confirmed the planned treatment. Additionally, in 8% of patients, the studies changed the side of the patient receiving treatment.

By Peter M. Carney, MD, FAANS

Because spinal pain has many causes, any test that can accurately diagnose the source of pain serves as an invaluable tool for making sure the patient receives the correct treatment. A 2002 study by Cork et al studied the nerve conduction of pain fibers with a device that had 94.6% sensitivity in detecting lumbar nerve-root function pathology as confirmed by epidurograms.[1] The basis for this type of testing is the pain fiber nerve conduction study (pf-NCS). The pf-NCS uses electrical voltage applied at predetermined points that correspond to areas innervated by a specific nerve root [2] to determine whether that nerve root has a normal response to the current. A hyperresponse indicates increased sensitivity to the current, while a hyporesponse indicates impaired sensitivity of that nerve root.

Despite the sensitivity of the pf-NCS, no prospective clinical studies have been able to demonstrate that the pf-NCS improves the management and clinical outcomes of patients with spinal pain any better than other methods. Indeed, the Center for Medicare and Medicaid Services in 2004 concluded that prototype devices (like that used by Cork), which relied on the patient's psychophysiological assessment (perception of a sensation), were unacceptable for Medicare coverage because, "there continues to be insufficient scientific and clinical evidence to consider the pf-NCS test and device used in performing this test as reasonable and necessary."[3]

More recently, however, the use of a potentiometer, in conjunction with pf-NCS, precisely recorded an objective increase of 20 millivolts or more a second or two before the patient felt a sensation generated by the pf-NCS.[4] Thus, theoretically, the pf-NCS gives more than just a "psychophysiologic assessment" as to whether a given patient perceives pain.

Objective
This study was undertaken to determine the effectiveness of the pf-NCS in improving outcomes of patients suffering from cervical and lumbar pain. The pf-NCS (performed with a Neural-Scan, PainDX, Inc., Laguna Beach, California) employs a voltage-regulated stimulus in conjunction with a potentiometer, an objective measurement of the amplitude of the action potential.

Evaluating the sensitivity of the test in determining which nerve generates a given patient's pain, as well as the specificity of that test in reducing the patient's pain while improving the patient's function, will demonstrate whether the use of the pf-NCS is "reasonable and necessary."

Methods
From August 1, 2008, through July 31, 2009, 151 individual pf-NCS electrodiagnostic examinations were performed on 124 patients, who were then followed for at least 1 month after receiving treatment. The patients' ages, sexes, clinical diagnoses as determined by history, physical findings, x-rays/magnetic resonance imaging/computed tomography scans, and results of the pf-NCS were recorded. The pf-NCS results demonstrated that a

given nerve root had one of six responses: a normal, mild, moderate, marked, severe, or very severe reaction (see box). All patients had their visual analog scale (VAS) and Oswestry Disability Index (ODI) measured and recorded before and after they received treatment. Patient treatment was dependent upon what the pf-NCS showed to be causing the patient's pain.

If the results were normal, the patient underwent conservative treatments including physical therapy, medication, and counseling where indicated. If the results showed mild, moderate, or marked nerve root abnormalities, then diagnostic medial branch facet joint blocks (MBB) were performed at the appropriate level according to International Spine Intervention Society Guidelines,[5,6] and medial branch facet rhizotomies were performed when indicated.[7,8] If the results showed severe or very severe nerve root abnormalities, then transforaminal lumbar epidural steroid injections (TF/LESI), LESI, or cervical epidural steroid injections (CESI) were performed at the appropriate level. Some patients received other interventional techniques such as sacroiliac (S/I) joint injections, piriformis injections, percutaneous disc decompression (Disc Dekompressors, Stryker, Kalamazoo, Michigan), or vertebroplasties.

Classification of pfNCS Results

pfNCS Results	Diagnosis
Normal	Myofascial or other origin
Mild, moderate, marked	Facet origin
Severe, very severe	Discogenic pain generator

pfNCS, pain fiber nerve conduction study

The test results were then divided into three categories (Table 1). The treatment selected for a given patient was considered to have "helped" if the patient's VAS was reduced by at least 2 points or 25% and/or the ODI was less than 40 and improved by at least 25%.[9] Overall average changes in the VAS and ODI were evaluated using several different parameters. In addition, the patients were divided into those who had pf-NCS of either the lumbar or cervical spinal regions and evaluated according to the treatment given to them and their response to treatment.

Results

Data from 151 pf-NCS were analyzed. The average patient age was 56.9 years, with a range of 14 to 94 years; 59 men and 92 women were tested. Of the patients tested, the average decrease in VAS score was 49% and an average functional improvement in the ODI was 44%. When analyzed by category, results of the pf-NCS had one of three influences on the treatment patients received: change in the treatment given (56%, n=84); confirmation of what clinical findings recommended as appropriate treatment (35%, n=53); no influence on treatment (9%, n=14).

Of those 84 patients where the pf-NCS helped change treatment, 72 tests (86%) helped patients decrease their VAS scores by 75%, on average, and demonstrated approximately 42% improvement in functioning. Of special note are the 12 patients where the pf-NCS changed the side that received treatment (8%). None of these 12 patients would have had the correct side of their pain generator treated without the use of the pf-NCS. On average, these 12 patients demonstrated an 88% decrease in their VAS and a 54% improvement in function.

As noted, the pf-NCS also confirmed what the patient history, physical findings, and diagnostic tests such as plain x-ray films, CT scans, and MRIs suggested should be the appropriate treatment. Of these 53 patients, improvement was seen in 40 patients (75%), while 13 patients (25%) were not helped. The average patient in this group decreased their VAS by 55% and improved their ODI by 55%.

Fourteen of the pf-NCS did not influence the treatment a given patient received; 6 patients (43%) were helped anyway and 8 patients (57%) were not helped. On average, those treated in this group had a 42% decrease in VAS and a 27% improvement in ODI.

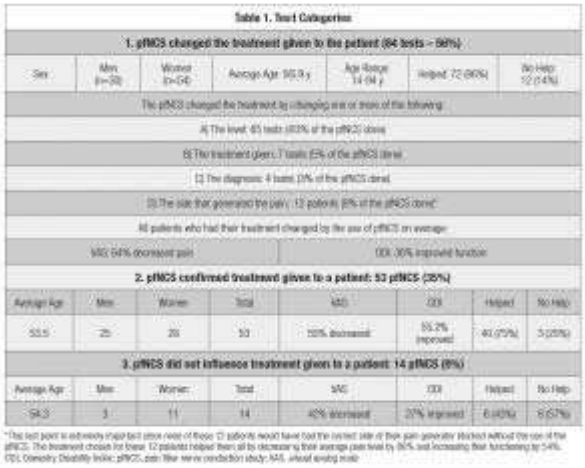


Table 1. Test Categories

1. pfNCS changed the treatment given to the patient (84 tests – 66%)							
Sex	Men (n=32)	Women (n=54)	Average Age: 55.9 y		Age Range: 14-94 y	Helped: 72 (86%)	No Help: 12 (14%)
The pfNCS changed the treatment by changing one or more of the following:							
A) The level: 45 tests (63% of the pfNCS done)							
B) The treatment given: 7 tests (8% of the pfNCS done)							
C) The diagnosis: 4 tests (5% of the pfNCS done)							
D) The side that generated the pain: 12 patients (8% of the pfNCS done)*							
All patients who had their treatment changed by the use of pfNCS on average:							
VAS: 64% decrement pain					ODI: 36% improved function		
2. pfNCS confirmed treatment given to a patient: 53 pfNCS (35%)							
Average Age	Men	Women	Total	VAS	ODI	Helped	No Help
53.5	25	28	53	55% decrement	55.2% improved	40 (75%)	5 (25%)
3. pfNCS did not influence treatment given to a patient: 14 pfNCS (9%)							
Average Age	Men	Women	Total	VAS	ODI	Helped	No Help
54.3	3	11	14	42% decrement	27% improved	6 (43%)	8 (57%)

*The last point is extremely important since most of these 12 patients would have had the correct side of their pain generator blocked without the use of the pfNCS. The treatment chosen for these 12 patients helped them all by decreasing their average pain level by 88% and increasing their functioning by 54%.
ODI: Oswestry Disability Index; pfNCS: pain fiber nerve conduction study; VAS: visual analog scale

Treatment Procedures

The pf-NCS resulted in 151 procedures or therapies being evaluated; 40% of all the pf-NCS (60) suggested that diagnostic MBBs should be done. These 60 MBBs resulted in 44 medial branch rhizotomies (MBR) being performed while results from 16 of the MBBs did not recommend that an MBR be performed (Table 2). Of the MBBs administered, 12 gave such long-term relief with just the use of local anesthetic that no MBR was needed; 4 of the MBBs failed to indicate that the patient would benefit from an MBR. Patients were helped by 50 (83%) of the diagnostic block procedures while 10 procedures (17%) did not help. The average patient in this group had a 63% decrease in VAS and a 32% improvement in ODI. TF/LESI was suggested by 34% of all pf-NCSs (51), which helped 49 patients (78%), but did not help 11 patients (22%). The average patient in this group had a 46% decrease

in VAS and a 34% improvement in ODI.

Data from 19 pf-NCS (13%) suggested that patients should receive medical therapy. These conservative therapies helped 10 patients (53%) who received them but did not help 9 patients (47%). The average patient in this group had a 38% decrease in VAS and a 26% improvement in ODI.

Table 2. Procedures

	Number of Procedures	Change in VAS	Change in ODI	Helped
TF/LESI	51	40% pain decreased	34% improved function	40 (78%)
Medical Therapy	19	38% pain decreased	26% improved function	10 (53%)
Other Interventional Pain Treatments	14	65% pain decreased	34% improved function	9 (64%)
CESI/LESI	6	65% pain decreased	14% improved function	3 (50%)
Medial Branch Block/ Rhizotomy*	60	63% pain decreased	32% improved function	50 (83%)

*Before diagnostic blocks were done to determine whether the patient was a candidate for a rhizotomy. They showed that 12 patients had such long lasting relief with just local anesthesia that they did not need a rhizotomy. The remaining 4 diagnostic blocks failed to help the patient, indicating that a rhizotomy would not help. CESI, cervical epidural steroid injections; LESI, lumbar epidural steroid injections; ODI, Oswestry Disability Index; TF, transforaminal; VAS, visual analog scale.

Only 9% of all pf-NCS (14) resulted in other treatments being given. These procedures included S/I joint injections, piriformis injections, percutaneous Disc Dekompressors, or vertebroplasties. Of these procedures, nine (64%) helped, but five (36%) did not. The average patient in this group had a 65% decrease in VAS and a 34% improvement in ODI. Interlaminar epidural steroid injections were done in 4% of all pf-NCS (6), three in the cervical spine and three in the lumbar spine. Three of these procedures (50%) helped and three (50%) did not. The average patient in this group had a 65% decrease in VAS and a 14% improvement in ODI.

Anatomic Location

Of the 151 pf-NCS performed, 40 were done for problems in the cervical spine (36%) and 111 for problems in the lumbar spine (64%) (Table 3). In 16 patients who received lumbar pf-NCS, various forms of medical therapy were recommended including oral steroids and physical therapy, which helped 10 patients (63%). Other forms of interventional treatments including S/I joint injections, piriformis injections, percutaneous Disc Dekompressor procedures, and vertebroplasties, were administered for 11 patients; seven of these procedures helped (64%).

Table 3. Cervical vs Lumbar pfNCS Tests

Location	Cumulative Results	By Procedure: MBB/ Rhizotomy	TF/LESI	Medical	Other Interventions	CESI/LESI
Cervical	Total: 40 Helped: 33 Not Helped: 7	32 29 (66%) 3 (10%)	— — —	5 3 (60%) 2 (40%)	— — —	3 1 (33%) 2 (67%)
Lumbar	Total: 111 Helped: 84 Not Helped: 25	30 21 (66%) 6 (20%)	51 40 (84%) 8 (16%)	10 10 (63%) 6 (37%)	11 7 (64%) 4 (36%)	3 2 (67%) 1 (33%)

CESI, cervical epidural steroid injections; LESI, lumbar epidural steroid injections; MBB, medial branch block; pfNCS, pain fiber nerve conduction study; TF, transforaminal

Conclusion
The use of pf-NCS in this series showed that more than half (56%) of the tests performed changed the treatment planned for the patient, approximately one third (35%) confirmed the planned treatment, and less than one tenth (9%) did not influence the treatment. The pf-NCS changed the planned therapy due to several factors. The test demonstrated which level generated the patient's pain when the MRI showed "multilevel degenerative disc and facet changes," and also changed the diagnosis of the pain generator from the disc to the facet or vice versa.

Additionally, the pf-NCS determined the best therapy for the patient, and confirmed in 12 patients that the generator of a patient's pain was located on the opposite side of the patient's body. This last finding was very significant.

The data in this study offer clear and convincing "scientific and clinical evidence to consider the pf-NCS electrodiagnostic examinations" as both a "reasonable and necessary" aid in helping all who wish to practice scientific and effective pain medicine.

If other studies confirm these findings, then an important diagnostic tool will be available to greatly improve the surgical, interventional, and medical treatment of spinal pain.

Paradoxical Relationship: A-Delta Function and VAS

Authors: Randall Cork, MD, PhD
Michael Bezel, MD

Abstract
The A-delta pain fiber nerve conduction study (A-delta pf-NCS) measures the sensitivity of fast pain fibers known to down-regulate soon after injury. This study compares changes in the sensitivity of the A-delta fibers in pathological nerve-roots with changes in the patient's subjective visual analog score (VAS). A significantly close relationship was found between the change in voltage required to cause an action potential (nerve impulse) in the A-delta fibers of the pathological nerve-root and changes in the subjective VAS rating. The findings support the utility of the A-delta pf-NCS for detecting the level and side of painful radiculopathy and measuring changes in pain.

Introduction
An objective marker associated with the patient's subjective pain perception has been an elusive challenge. A multitude of scales have been proposed in the attempt to quantify the severity of pain experienced by the patient and to quantify the response to treatment. The visual analog scale (VAS) has been widely used as a gauge of pain severity in acute and

chronic pain. [1,1,1,1] The A-delta small pain fiber nerve conduction study (pf-NCS) is used to locate pain generators. For several decades physiology texts have described that during the first epicritic phase of sensory nerve injury the A-delta fibers up-regulate causing a withdrawal reflex. This is shortly followed by the protopathic phase in which Guyton & Hall Textbook of Medical Physiology explains that the fibers capable of almost exact localizing, the A-delta fibers, down-regulate and the poor localizing C- Type fibers begin to up-regulate. Concerning this protopathic phase Guyton states; "It explains why patients often have serious difficulty in localizing the source of some types of chronic pain. "1 Cork, et al showed that the nerve-root adhesion causing radicular pain was associated with hypo-sensitivity of A-delta fibers in the associated peripheral nerve. 1 Other studies have used hypo-sensitivity of the A-delta fibers as detected by the pf-NCS as an objective marker for pain. [1,1,1]

The purpose of this study was to survey the clinical association between the patient's VAS pain rating and sensitivity of the A-delta fibers as measured by the pf-NCS.

Methods

A study group was formed from members of the American Association of Sensory Electrodiagnostic Medicine certified in A-delta pf-NCS electrodiagnostic examination (EDX). After IRB approval of the study by AASEM the study group members were asked to submit A- delta pf-NCS studies using the Neural-ScanTM (PainDX, Inc. of Laguna Beach, Ca.) for those patients who reported a change in VAS after treatment for pain. After one month, 83 pairs of VAS ratings and the associated graphs of the A-delta pf-NCS made on the same day were received. Data analyzed included the pre-treatment and post- treatment VAS pain scores, difference in the voltage required to cause an action potential of the A-delta fibers in the pathological nerve-root identified by the pre A-delta pf-NCS of the peripheral nerve associated with the nerve-root identified by the pre A-delta pf-NCS of the peripheral nerve associated with the nerve-root, sex and spinal region (cervical or lumbar). Data were analyzed using repeated-measures analysis of variance. Results were considered significant if $p<0.05$.

Results

Change in VAS and voltage inducing an action potential are shown in Figure 1. Both VAS and voltage decreased with treatment ($p<0.001$).

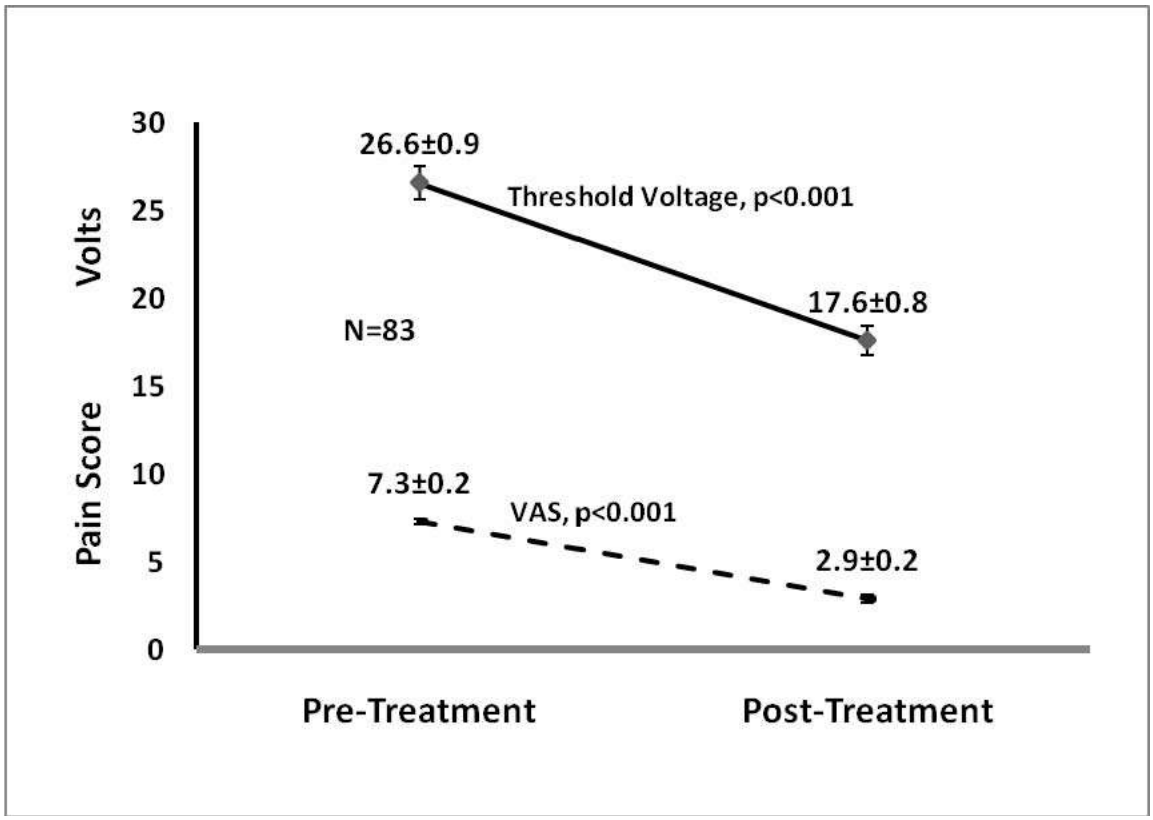


Figure 1. Change in VAS and threshold voltage with pain treatment.

Both sex and spinal level were significantly associated with threshold level (p<0. 05), but neither was significantly associated with VAS. A- Delta threshold voltage decreased from 28. 4±1. 4 v (SEM) (n=43) to 18. 7±1. 4 v for males (p<0. 001) and from 24. 6±1. 1 (n=40) to 16. 5±1. 0 v for females (p<0. 001). Figure 2 illustrates the different voltage thresholds for each sex and how threshold level changed with treatment for males and females.

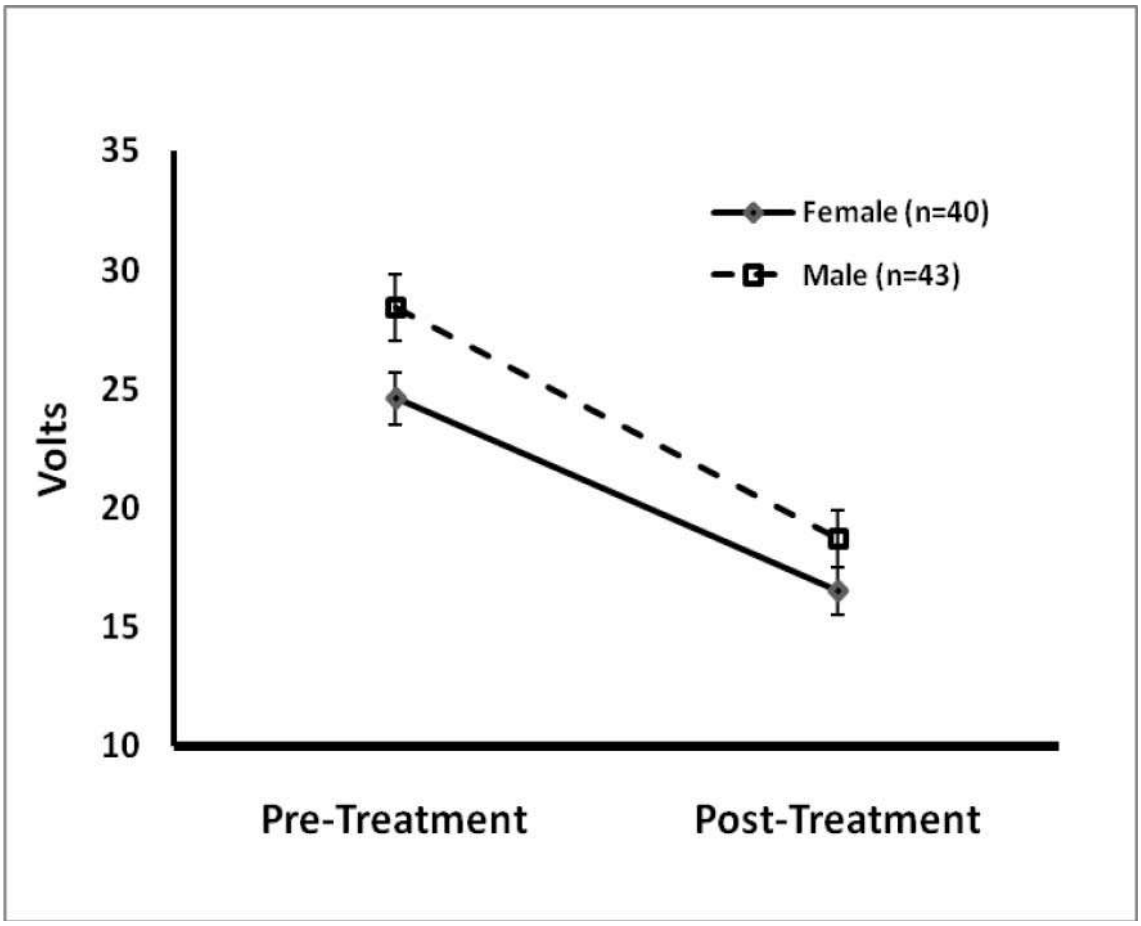


Figure 2. Decrease in threshold voltage for males and females with treatment. Males showed higher threshold levels than females ($p<0.05$), and threshold levels for both males and females decreased with treatment ($p<0.001$).

Threshold voltage for cervical dermatomes was significantly lower than threshold voltage for lumbar dermatomes ($p<0.05$). Threshold voltage decreased from 23.0±1.4 v (n = 36) to 14.6 ± 1.0 v ($p<0.001$) for cervical dermatomes and from 29.3±1. 1 v (n = 47) to 20 ± 1.0 v ($p<0.001$) for lumbar dermatomes. Figure 3 illustrates how lumbar voltage thresholds were significantly higher than cervical voltage thresholds ($p<0.05$) and how both lumbar and cervical thresholds decreased with treatment ($p<0.001$).

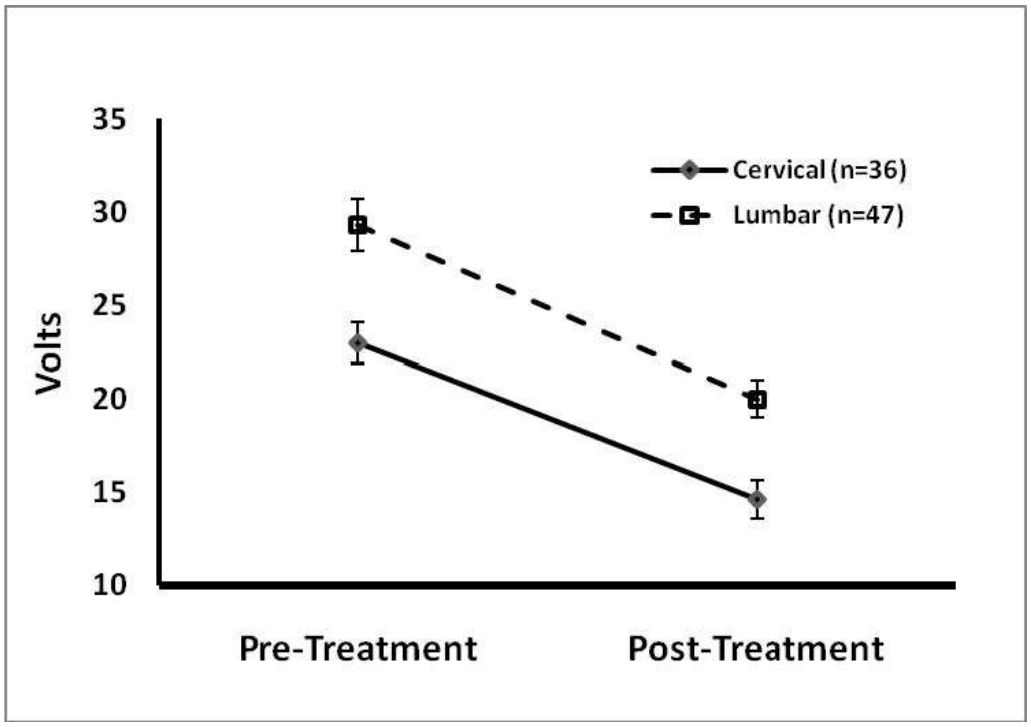


Figure 3. Decrease in threshold voltage for cervical and lumbar dermatomes with treatment. Lumbar dermatomes showed higher threshold levels than cervical dermatomes ($p<0.05$), and threshold levels for both lumbar and cervical dermatomes decreased with treatment ($p<0.001$).

In contrast to threshold voltage, VAS response was not different for males compared to females or for cervical dermatomes compared to lumbar dermatomes. Figure 4 shows the response to treatment for both males and females. Although there is a significant treatment effect ($p<0.001$), there is no difference in VAS response based on sex.

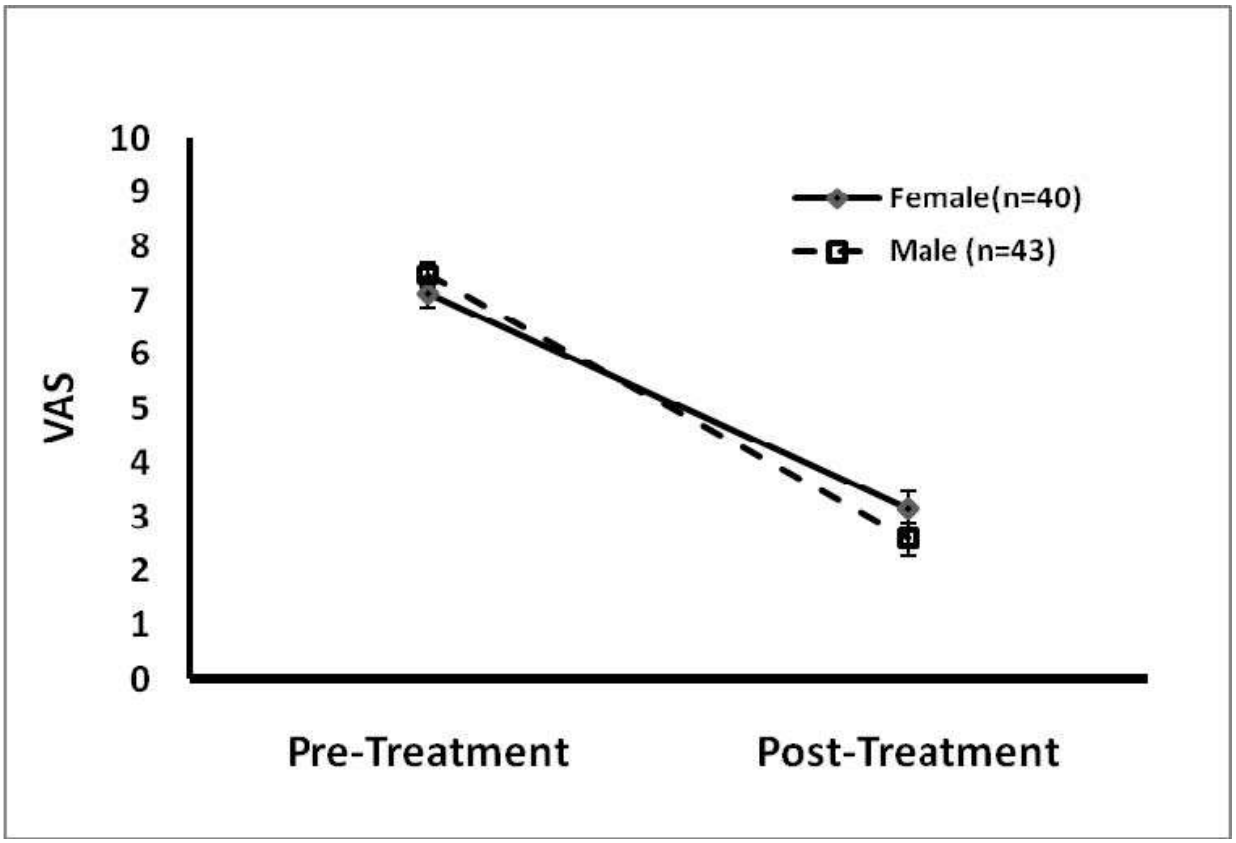


Figure 4. Change in VAS for both males and females in response to treatment. Although there is a significant treatment effect ($p<0.001$), there is no difference in treatment response based on sex.

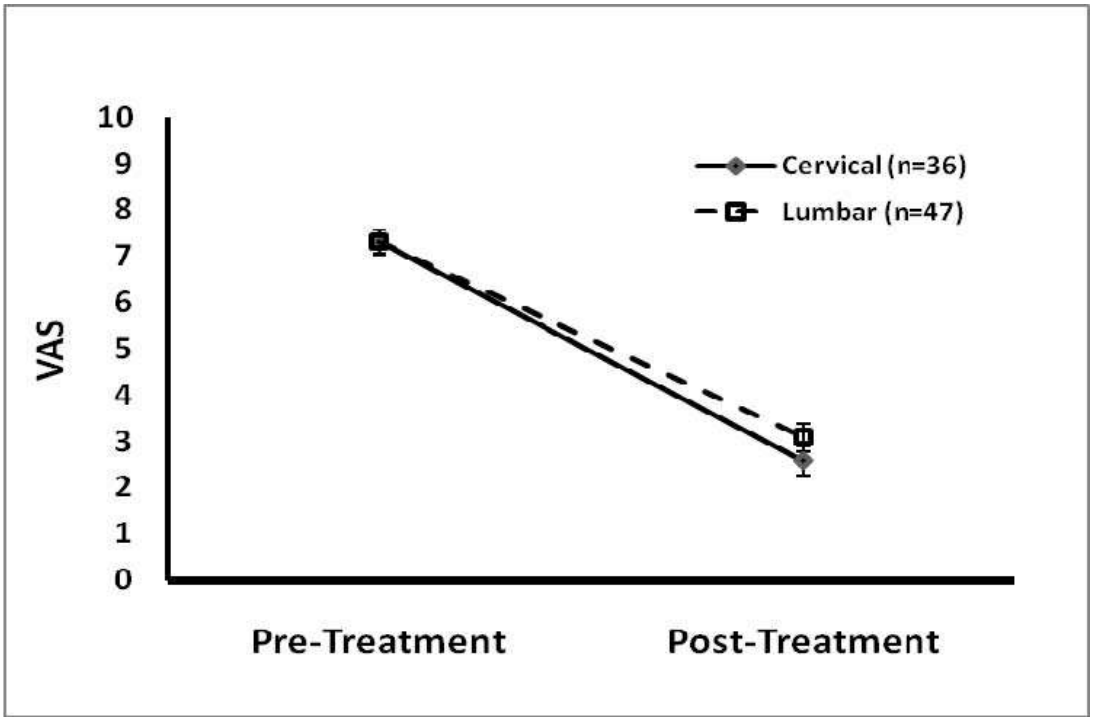
Figure 5 shows a similar response to treatment for both the cervical and lumbar region. Again, although there is a significant treatment effect ($p<0.001$), there is no difference between cervical and lumbar VAS responses.

Figure 5. Change in VAS for both cervical and lumbar regions in response to treatment. Although there is a significant treatment effect ($p<0.001$), there is no difference in treatment response based on spinal region or level.

Discussion

These data represent the results of a survey of members of the American Association of Sensory Electrodiagnostic Medicine. Thus, the responses to treatment reported may be biased by members submitting "typical" responses that make the treatment look good. Nonetheless, the focus of the survey was not on the effectiveness of treatment, but on how the response to treatment, as measured by VAS, was associated with the response to treatment as measured by the threshold voltage of the A-delta fibers. On the basis of these results, A- delta fiber threshold voltage is a good independent marker of pain decrease with

successful treatment, independent of the pain report given by the patient. This is a significant finding, as patient perceptions are very subjective and affected by many extraneous variables, including malingering.



Why females should have lower A-delta threshold voltages is an interesting question that certainly deserves more research. Similarly, why the cervical region would have lower A-delta fiber voltage thresholds than lumbar region is another potential area of investigation. However, the study data shows clearly that A-delta function as measured by voltage threshold is dependent on both sex and spinal region. With adjustments for sex and spinal region, A-delta hypo- sensitivy appears to be an objective marker for measuring the success of therapeutic interventions.

Conclusions

A-delta sensitivity/function as measured by voltage threshold using the pf-NCS is related to the patient's pain perception. As A-delta sensitivity improves from hyposensitivity to normal sensitivity, pain decreases; as A-delta sensitivity/function deteriorates, pain increases. Females have lower A-delta fiber voltage thresholds than males, and the cervical region exhibits lower A-delta fiber voltage thresholds than the lumbar region, but the A-delta voltage thresholds drop independently of sex and spinal level with treatment. In contrast, the VAS responses are not different based on either sex or spinal level, but VAS drops significantly in concert with the drop in A-delta fiber sensitivity/function. In general, A-delta sensitivity/function measured by A-delta small pain fiber nerve conduction study (pf-NCS) is an excellent objective measure of pain change following treatment, and a practical and painless electrodiagnostic procedure for detecting the side and level of

painful radiculopathic pain generators.

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Neural-Scan Gets to the (Nerve) Root of Fibromyalgia

Presented: 2002 American Society of Regional Anesthesiology and Pain Medicine (ASRA) Scientific Conference

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Summary: Using the Neural-Scan, LSU Pain Center found the majority of fibromyalgia patients have previously undiagnosed nerve-root pathology. Ten of these treated patients were available for a one-year follow-up. All ten were found free of all fibromyalgia symptoms.

Abstract:
Fibromyalgia is an obscure entity and a topic of on-going discussion between different medical specialties. It is a soft-tissue disorder characterized by diffuse musculoskeletal pain, and specific tender points on examination. We report a series of ten cases that presented with signs and symptoms consistent with the American College of Rheumatology (ACR) 1990 criteria for diagnosis of fibromyalgia (3). Neural-Scan, a sensory nerve conduction exams measuring the minimum voltage required to cause A-delta fiber firing was done on these patients and showed previously undetected nerve root involvement. Based on these findings, these patients were treated with medications and interventional pain procedures with resolution of symptoms and signs previously categorized as fibromyalgia. We therefore propose a neurological mechanism as the primary etiologic factor of fibromyalgia and emphasize Neural-Scan as an important tool in its diagnosis.

Methods:
Between May 2000 and September 2001, ten patients diagnosed with fibromyalgia, as defined by the1990 ACR criteria, were seen on our Pain Service at LSUHSC (Louisiana State University Health Sciences Center), Shreveport, Louisiana. As a part of initial workup, they all underwent V-sNCT tests of lumbar and cervical spine. Neural-Scan provides a reproducible (<0.2 mA) functional assessment of the peripheral sensory nervous system by measuring that voltage intensity which initiates membrane potential changes, to propagate a threshold level nerve impulse.

The Neural-Scan tests in nine of ten fibromyalgia patients showed both cervical and lumbar nerve root involvement. One of the ten only showed lumbar nerve root involvement. These patients were prescribed medications and also underwent interventional pain procedures. They were followed-up for six months to one year. They showed good response to treatment with resolution of symptoms, which were previously characterized as fibromyalgia.

To validate our observation that the cause of fibromyalgia is nerve root pathology in one or more than one body regions, we conducted lumbar and cervical tests in 40 patients, who met the 1990 ACR criteria of fibromyalgia. All of these patients showed either lumbar (10/40) or cervical (6/40) or lumbar and cervical (24/40) nerve roots involvement on Neural-Scan tests.

Conclusions:

1. The basis of fibromyalgia is nerve root involvement. Long-standing nerve root pathology can give rise to muscle spasms and tightness in the same area as well as tension bands particularly in neck region, which over time become tender points. Eventually abnormal synapses form between the somatic and sympathetic nervous system giving rise to CRPS Type-I symptoms and signs, including burning, swelling, temperature changes etc. HPA-(Hypothalamus-Pituitary-Adrenal) axis changes follow or occur concurrently with chronic pain.

2. Nerve root involvement as the basis of fibromyalgia can explain the correlation of fibromyalgia with trauma (5), whiplash neck injury (6) and in such diverse entities as inhalation of petroleum fumes (giving rise to toxic neuropathies) (7), and Lyme disease (8).

3. The good response of our fibromyalgia patients to interventional procedures, including epidural steroid injections, and/or epidural lyses of adhesions, botox injections, sympathetic blocks etc., further supports our theory that the cause of fibromyalgia is nerve root pathology and hence the associated pain can be decreased by appropriate pain procedures.

Neural-Scan Helps Identify Piriformis Syndrome

2002 - Presented: American Society of Regional Anesthesia and Pain Medicine (ASRA) Scientific Conference
2005 - American Association of Sensory Medicine (ASSM) Conference
Randall Cork, MD, PhD Chairman of the Department of Anesthesiology and Director of Pain Medicine, Louisiana State University Health Sciences Center, Shreveport, LA

Authors: Randal Cork MD, PhD, Sarosh Saleemi, MD, Lou Hernandez, MD, Susan Brandt, MD, Rakesh Chauhey, MD and Lori Alexander, MBA, CPC

Summary: This retrospective study of 50 patients found an 80% probability of concomitant piriformis syndrome when the Neural-Scan detected abnormal L5 and S1 dysfunction on the ipsilatyeral (same) side.

INTRODUCTION

Piriformis Syndrome has been documented as a primary and/or contributory cause for sciatica and low back pain (1, 2, 3, and 4). Botulinum toxin-A ™ is used by both medical and surgical specialists to successfully treat dystonic muscle disorders (6, 7, 8). During a seven-month period, 50 patients in our practice were diagnosed with piriformis syndrome and underwent intrapiriformis Botulinum toxin-A™ injection with remarkable pain relief at 6 to 8 weeks follow-up.

METHODS

A retrospective review of 50 patients of the Pain Management Service at Louisiana State University Health Sciences Center who received intrapiriformis (Botulinum toxin-A™) injection was performed. All patients were taking one of the following analgesics: NSAIDS, tramadol, or long acting opioids, or gabapentin. All patients gave informed consent for this procedure. The demographic characteristics and relevant past medical histories of our study group are given in Table I and Table II respectively. Our diagnostic criteria for piriformis syndrome include the following: Gluteal pain with or without pain radiating down the affected leg in the distribution of sciatic nerve, muscle spasms/cramps/pull in leg muscles, positive Beatty's Maneuver (9) with or without the presence of tenderness, and L5, S1 or both L5 and S1 sensory nerve root hypoesthesia, as measured with the Neural-Scan Small-Pain-Fiber Nerve Conduction Study (SPF-NCS) at 250 Hz (10)(26).
Botulinum-toxin A (Botox™, Allergan™) is a standardized preparation that comes in powder form. Botulinum toxin-A™ 100 units mixed in 5 cc of preservative-free normal (0.9 N) saline was used for each intrapiriformis injection. The fluoroscopic technique performed is as follows: In a prone patient, the greater trochanter of the femur and the lower part of sacrum or sacroiliac joint of the same side is visualized, and a marker (e.g., a large hemostat) is placed on an imaginary line connecting the two. This represents the anatomical location of the piriformis muscle. Injection can be performed anywhere on this line, but the selected site was closer to sacrum where the base of piriformis muscle lies. Injections were made over bone to avoid possible injury to the sciatic nerve and pelvic structures. A 22-gauge 3.5inch spinal needle was advanced until the characteristic loss of resistance was felt as the needle penetrates the piriformis facial sheath. Omnipaque 1ml was then injected to obtain a piriformis myogram. After x-ray analysis showed negative for aspiration of blood, Botulinum toxin-A™ was then injected. (See X-ray below).

RESULTS

The outcome measures of pain intensity were Visual Analog Scales (VAS) (11), and modified McGill (12) scores, and the outcome measures of disability were Oswestry (13), and Rolard-Morris Disability Scale (14) scores. The data below were obtained prior to treatment, and at 6-8 weeks follow-up after the procedure. VAS prior treatment was mean ±SEM 8.8>±0.151, compared to post treatment 4.53±0.242(p<0.05). Table III shows the change in McGill, Roland-Morris and Oswestry scores from before to after treatment. All patients reported a reduction in pain scores. VAS pain scores in the study population were 8.87± 0.15 prior to treatment and 4.5±0.2 after treatment (p<0.01). McGill scores were 40.6 ±3.04 before and 21.5 ± 2.51 after the injection (p<0.01). Oswestry scores changed from 25.9± 1.26 to 11.7 ± 1.02(p<0.01) and Roland-Morris scores decreased from 16.0 ± .935 to 20.6± 1.02 (p<0.01). Lumbar SPF-NCS showed hypoesthesia in nerve roots L5 in 7/48, S1 in 9/48 and both L5 and S1 in 32/48 patients.

DISCUSSION

Botulinum toxin-A™ is a 150 Kda protein produced by *Clostridium Botulinum*. It is a neurotoxin, which acts presynaptically by inhibiting the release of acetylcholine, thus leading to functional denervation of muscle (15). This effect lasts up to 6 months. In 1989, FDA approved its use for the treatment of strabismus, blepharospasm, and hemifacial spasm (ref). Botulinum toxin-A™ has been on the market for a while now, but its use in pain patients has gained popularity only recently (16, 17, 10, 19).

The piriformis muscle is a pyramidal muscle that arises as three digitations from the ventrolateral aspect of the sacrum from S1-S4, gluteal surface of ilium near the posterior inferior iliac spine and the anterior capsule of the sacroiliac joint. It passes through the greater sciatic foramen on its lateral trajectory to its tendonous insertion on anterior/medial aspect of the greater trochanter of the femur. Piriformis syndrome is a secondary cause of sciatica due to compression and/or irritation of sciatic nerve compressed by the contracted piriformis muscle. Its signs and symptoms can be explained by the proximity of the muscle to sciatic nerve at the sciatic notch. There are six possible relationships between the piriformis muscle and the sciatic nerve (23). Most commonly, the nerve is anterior and below piriformis muscle. The patient complains of pain, numbness and/or weakness in L4, L5 or S1 distributions. These may be associated with localized tenderness in piriformis muscle itself. Alternatively, pain due to piriformis spasm can also be felt as a deep, aching type of pelvic pain on the same side without signs and symptoms of sciatica.

As the piriformis muscle is a lateral rotator of hip flexion and assists in abduction, active muscle contraction can lead to pain reproduction (Beatty's maneuver (9). These physical signs if present are useful in differentiating piriformis syndrome, from sciatica due to other causes alone.

SPF-NCS provides a reproducible (<0.2mA) functional assessment of the peripheral sensory nervous system by measuring the voltage intensity which initiates membrane potential changes, to propagate a nerve impulse.

One problem with the diagnosis of the piriformis syndrome has been the lack of consistent objective diagnostic findings. We have found lumbar SPF-NCS reliable in detectng sciatica and, when correlated with signs and symptoms can confirm the diagnosis of piriformis syndrome.

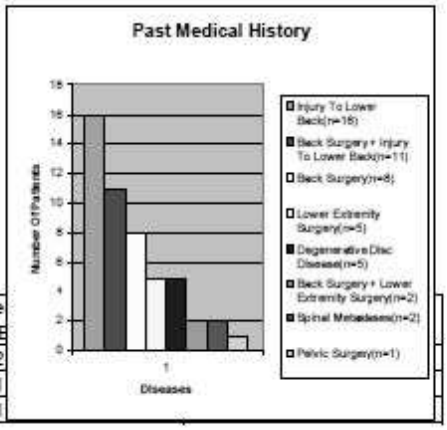
Our study shows an association of piriformis syndrome with low back injury and/or surgery, degenerative disc disease, total hip surgery, spinal metastases and pelvic surgery. Two of our patients had piriformis syndrome after hard falls to the floor. We speculate that piriformis muscles may go into spasm either secondary to irritation of its nerve supply, sciatic nerve irritation, as in disc disease, lumbosacral radiculitis, or surgery in its vicinity, such as in total hip replacement, pelvic surgery, etc.

A variety of therapeutic approaches have been suggested for the management of piriformis syndrome (20, 21, and 22). These include conservative measures such as analgesics, application of heat, osteopathic manipulation, stretching exercises and even surgical resection of the piriformis muscle (23). Except for the latter, none of these modalities offer significant pain relief, and surgery is associated with morbidity. Perisciatic injection of steroids (24) and caudal epidural steroid injection for piriformis syndrome (25) have been described, as well as injection of local anesthetics and steroids in the muscle belly, but at present there are no outcome data which show their efficacy. Our study shows that intrapiriformis Botulinum toxin-A™ injection significantly reduces pain and disability for at least 6 and up to 8 weeks. All of the patients who underwent Botulinum toxin-A ™ injection to piriformis muscle reported at least a 45% reduction in pain as well as improvement in their disability scores. Intrapiriformis Botulinum toxin-A™ injection can be performed easily and quickly (< 10 minutes) under fluoroscopic guidance, does not require EMG needle placement or the use of a nerve stimulator, and is less invasive than surgery. The technique for intrapiriformis injection described in this paper can be learned easily. After performing a few injections, one easily appreciates the characteristic feel of the needle entering the piriformis sheath. Intrapiriformis Botulinum toxin-A™ injection is an effective treatment for Piriformis Syndrome.

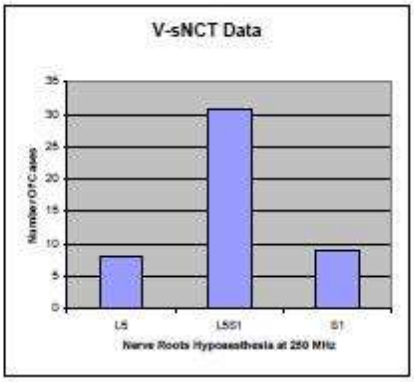
TABLE I: DEMOGRAPHIC CHARACTERISTICS OF 50 PATIENTS

Age (years)	51.76 +_ 1.7268
Weight (lbs)	180.82 +_ 5.15
Height (inches)	66.52 +_ 0.4959
Female	34
Male	16

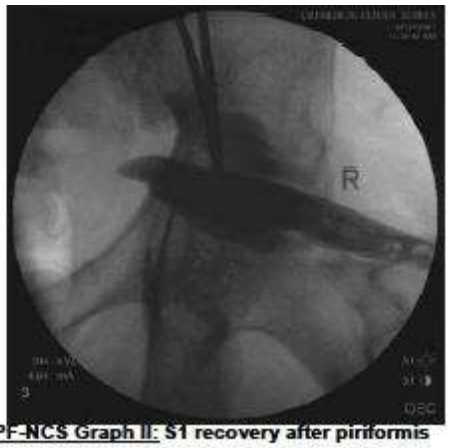
FIGURE 1: PAST MEDICAL HISTORY (N=50)



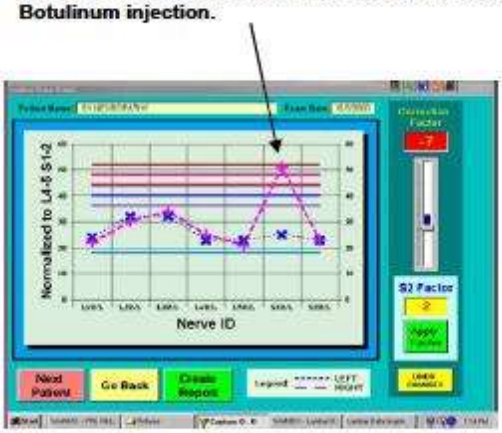
	Before Treatment Mean±SEM	After Tre Mean±SE
McGill	40.629±3.048	21.555±2
Roland Morris	16.074±0.935	11.740±1
Oswestry	25.963±1.260	20.666±1

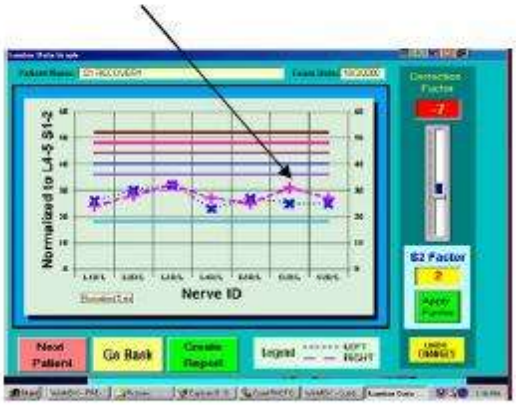


SPF-NCS Graph I: Right S1 severe rated dysfunction Botulinum injection.



SPF-NCS Graph II: S1 recovery after piriformis





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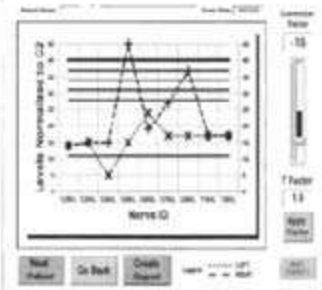
A-delta Fiber EDX Shows Correlation to VAS Scores

Alex Ambroz, MD Associate Clinical Professor John Marshall University Medical School

Amplitude of A-delta Fiber Action Potential Objectively Discriminates Severe Chronic Spine Pain Patients from Controls

Spine complaints are second only to upper respiratory problems as a reason for patient visits. Objective validation of the spine pain complaint is a major problem in the assessment and treatment of chronic spine pain. Is your treatment plan helping the patient get better? How do you know if someone is symptoms magnifying? Should you prescribe more opioids if the patient continues to complain of pain? Patient symptom description is purely subjective as is the VAS score. In the evaluation of acute coronary syndrome, a serum troponin has a sensitivity of 99%. By contrast, the sensitivity and specificity of the physical examination, imaging studies and needle EMG studies is low. As a result of this, litigation costs for spine pain are billions of dollars. Most chronic spine pain patients get labeled as symptom magnifying, drug seeking, by some health provider sooner or later. Clinical assessment comes down to clinical judgment which may be biased both philosophically and economically. There is a great need for a completely objective test of pain severity.

In evaluating the patients, the visual analogue scale (VAS) ratings of pain today and pain over the past month was assessed. Patients and controls underwent range of motion measurements of the lumbar spine and a neurological examination. In addition, the sum of the sensory nerve conduction measures (NS score) was measured for each patient and control.



Needle EMG studies are thought to be objective markers of nerve injury. Often, when a needle EMG study is negative, the patient's complaints of numbness, tingling, and pain are labeled as functional. The main problem with needle EMG is that they are measuring large caliber afferent fibers which are physiologically unrelated to pain which is mediated by pain fibers. It has been reported that up to 50% of electrodiagnostic studies evaluating possible radiculopathy reported as normal may have a compressive radiculopathy that is not being detected. Quantitative sensory testing has been suggested as an alternative. These procedures asses the small pain fibers which represent roughly 70% of the peripheral nervous system. The A delta fibers mediate the sensation of cold and the first components of pain and have a conduction velocity between 2 and 30 m/sec. However, since QST has used the patient response as the end point, it has been considered too subjective a test. To avoid this, in this study we took measurements from a potentiometer placed at the level of C6.

We have found that in chronic spine patients there is chronic hyperstimulation of A delta fibers and measuring this appears to be useful as an objective marker of severe spine pain.

Further research is needed to confirm these preliminary findings. Study of the sensory nerve conduction velocity of C fibers and A beta fibers would also be useful.

Transforamenal study - Presented at the 5th Annual AASM Conference - 2006

Pain Fiber Nerve Conduction Sensory Testing: Randall C. Cork, MD, PhD; Shweta Khedlekar, BS; Sanjay Madnani, MD Ahmad Elsharydah, MD; Paul Mayes, MD Department of Anesthesiology Louisiana State University Health Sciences Center, Shreveport, LA USA 7113

Introduction: Nerve conduction sensory (PF-NCS) test helps to diagnose severity, location & distribution of radiculopathy or neuropathy.

Non-invasive method.

Measures sensory threshold using neuroselective frequency to test Type A-delta fibers.

Abnormally high PF-NCS measures indicate significant nerve conduction loss.

Abnormally low PF-NCS indicate hyperesthetic state that corresponds with inflamed, irritated or regenerating nerves. Methods: PF-NCS tests are performed on patients with lumbar pain, before & after interventional pain procedures.
Time period between pre & post procedure PF-NCS test = 2 weeks.

Patients complete forms to communicate their intensity of pain, functional impairment, depression & anxiety.

Patient charts undergoing procedures at the LSUHSC Pain Clinic between Sept 2005 & Feb 2006 reviewed.
Data analyzed using Student's Paired t-test & Chi-square tests (significance defined as $p<0.05$).
Results:
Pain charts of 53 patients reviewed.

Significantly decreased PF-NCS scores after lumbar interventional procedures ($p<0.001$).

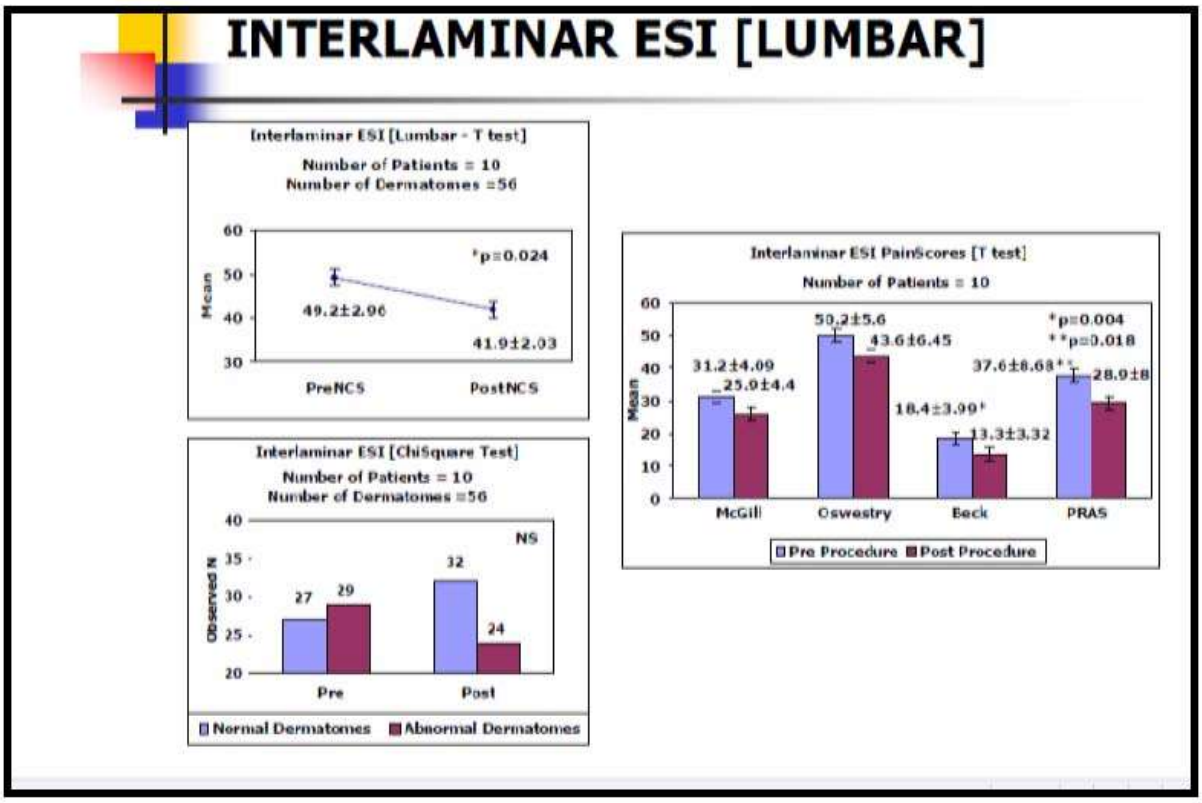
Lumbar Pain Patients - Paired T-test indicated significant changes in McGill Pain Questionnaire & Oswestry Disability Questionnaire with $p=0.012$ and $p<0.001$ respectively.

Transforaminal ESI & Interlaminar ESI resulted in decreased PF-NCS ($p<0.05$).

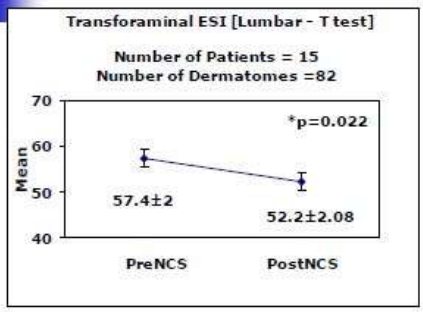
No significant change in number of abnormal dermatomes.

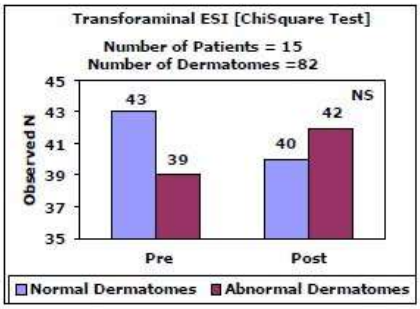
Pain intensity (McGill) & functional (Oswestry) pain scores significantly reduced following Transforaminal ESI, but not Interlaminar ESI.

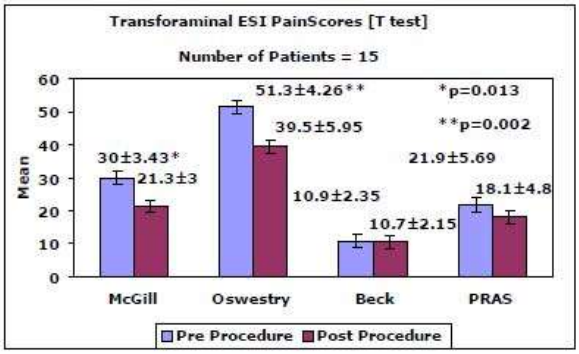
Depression (Beck) & anxiety (PRAS) significantly reduced following Interlaminar ESI, but not Transforaminal ESI.



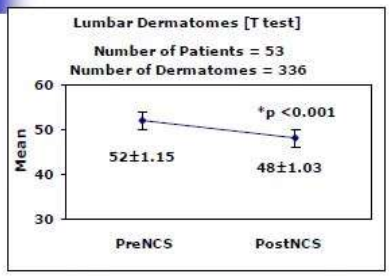
TRANSFORAMINAL ESI [LUMBAR]

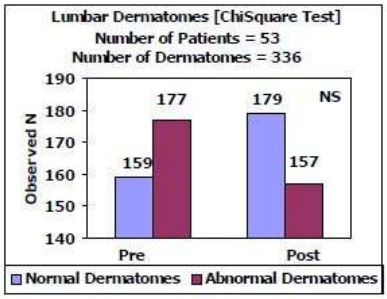


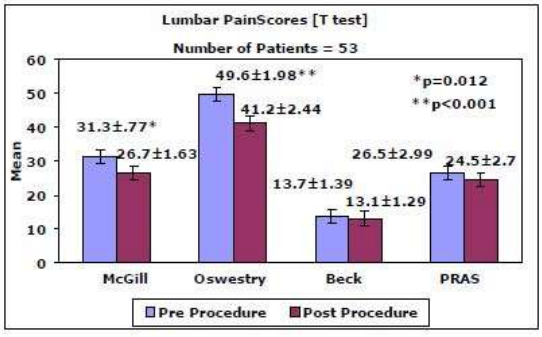




LUMBAR DERMATOMES







Conclusion: PF-NCS - direct sensory test.
Assesses peripheral sensory nervous system by measuring voltage intensity which initiates membrane potential changes to propagate nerve impulses.
Physician can identify a target & assess results of interventional pain procedures.
Reference
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Treating Piriformis Syndrome with Botulinum Toxin Using Pain Fiber NCS to Aid Diagnosis

Randall C. Cork, MD, PhD, Sarosh Saleemi, MD, Lou Hernandez, MD, Susan Brandt, MD, Rakesh Chaubey, MD and Lori Alexander, MBA, CPC.

Pain Management Clinic Dept of Anesthesiology, LSU
Medical School Health Sciences Ctr. Shreveport, LA

INTRODUCTION
Piriformis Syndrome has been documented as a primary and/or contributory cause for sciatica and low back pain (1, 2, 3, and 4). Botulinum toxin-A TM is used by both medical and surgical specialists to successfully treat dystonic muscle disorders (6, 7, 8). During a seven- month period, 50 patients in our practice were diagnosed with piriformis syndrome and underwent intrapiriformis Botulinum toxin- ATM injection with remarkable pain relief at 6 to 8 weeks follow-up.

METHODS
A retrospective review of 50 patients of the Pain Management Service at Louisiana State University Health Sciences Center who received intra-piriformis (Botulinumtoxin-ATM) injection was performed. All patients were taking one of the following analgesics: NSAIDS, Tramadol, or long acting opioids, or Gabapentin. All patients gave informed consent for this procedure. The demographic characteristics and relevant past medical histories of our study group are given in Table I and Table II respectively. Our diagnostic criteria for piriformis syndrome include the following: Gluteal pain with or without pain radiating down the affected leg in the distribution of sciatic nerve, muscle spasms/cramps/pull in leg muscles, positive Beatty's Maneuver (9) with or without the presence of tenderness, and L5, S1 or both L5 and S1 sensory nerve root hypoesthesia, as measured with the Neural-Scan voltage sensory nerve conduction device at 250 Hz (10) (26). Botulinum-toxin A (Botox, Allergan) is a preparation that comes in powder form. Botulinum toxin-A™ 100

units mixed in 5 cc of preservative-free normal (0.9 N) saline was used for each intrapiriformisinjection. Thefluoroscopictechniqueperformedisas follows: In a prone patient, the greater trochanter of the femur and the lower part of sacrum or sacroiliac joint of the same side is visualized, and a marker (e. g., a large hemostat) is placed on an imaginary line connecting the two. This represents the anatomical location of the piriformis muscle. Injection can be performed anywhere on this line, but the selected site was closer to sacrum where the base of piriformis muscle lies. Injections were made over bone to avoid possible injury to the sciatic nerve and pelvic structures. A 22-gauge 3.5inch spinal needle was advanced until the characteristic loss of resistance was felt as the needle penetrates the piriformis facial sheath. Omnipaque 1ml was then injected to obtain a piriformis myogram. After x-ray analysis showed negative for aspiration of blood, Botulinum toxin-ATM was then injected.

RESULTS

The outcome measures of pain intensity were Visual Analog Scales (VAS) (11), and modified McGill (12) scores, and the outcome measures of disability were Oswestry (13), and Roland-Morris Disability Scale (14) scores. The data below were obtained prior to treatment, and at 6-8 weeks follow-up after the procedure. VAS prior 0.151, compared to post treatment 0.242($p<0.05$). Table III shows the change in McGill, Roland- study population were 8. 87 treatment ($p<0.01$). McGill scores were 40.6 3.04 before and 21.5 2.51 after the injection ($p<0.01$). Oswestry scores changed from 25.9 1.26 to 11.7 1.02($p<0.01$) and Roland-Morris scores decreased from 16.0 0.935 to 20.6 1.02 ($p<0.01$). Lumbar Neural-Scan showed hypoesthesia in nerve roots L5 in 7/48, S1 in 9/48 and both L5 and S1 in 32/48 patients.

DISCUSSION

Botulinum toxin-ATM is a 150 Kda protein produced by Clostridium Botulinum. It is a neurotoxin, which acts presynaptically by inhibiting the release of acetylcholine, thus leading to functional denervation of muscle (15). This effect lasts up to 6 months. In 1989, FDA approved its use for the treatment of strabismus, blepharospasm, and hemifacial spasm (ref). Botulinum toxin-ATM has been on the market for a while now, but its use in pain patients has gained popularity only recently (16, 17, 18, 19). The piriformis muscle is a pyramidal muscle that arises as three digitations from the ventrolateral aspect of the sacrum from S1-S4, gluteal surface of ilium near the posterior inferior iliac spine and the anterior capsule of the sacroiliac joint. It passes through the greater sciatic foramen on its lateral trajectory to its tendonous insertion on anterior/medial aspect of the greater trochanter of the femur. Piriformis syndrome is a secondary cause of sciatica due to compression and/or irritation of sciatic nerve compressed by the contracted piriformis muscle. Its signs and symptoms can be explained by the proximity of the muscle to sciatic nerve at the sciatic notch. There are six possible relationships between the piriformis muscle and the sciatic nerve (23). Most commonly, the nerve is anterior and below piriformis muscle. The patient complains of pain, numbness and/or weakness in L4, L5 or S1 distributions. These may be associated with localized tenderness in piriformis muscle

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Neural-Scan is a direct sensory function test, which provides a reproducible (<0.2mA) functional assessment of the peripheral sensory nervous system by measuring the voltage intensity which initiates membrane potential changes, to propagate a nerve impulse. One problem with the diagnosis of the piriformis syndrome has been the lack of consistent objective diagnostic findings. We have found lumbar Neural-Scan tests reliable in detecting sciatica and, when correlated with signs and symptoms can confirm the diagnosis of piriformis syndrome.

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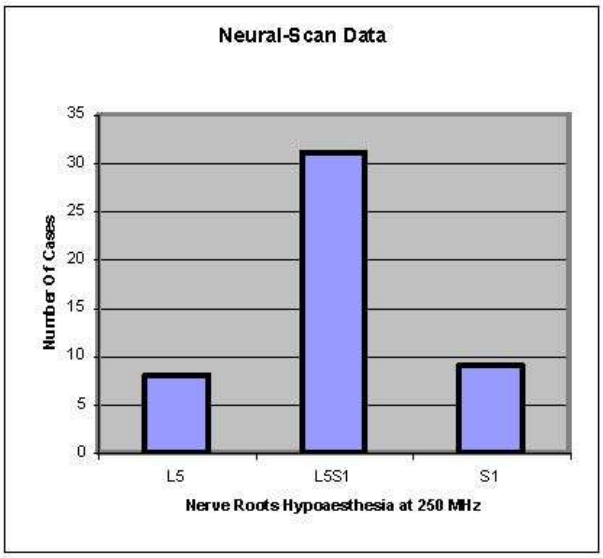
TABLE I: DEMOGRAPHIC CHARACTERISTICS OF 50 PATIENTS

FIGURE 1: PAST MEDICAL HISTORY (N=50)



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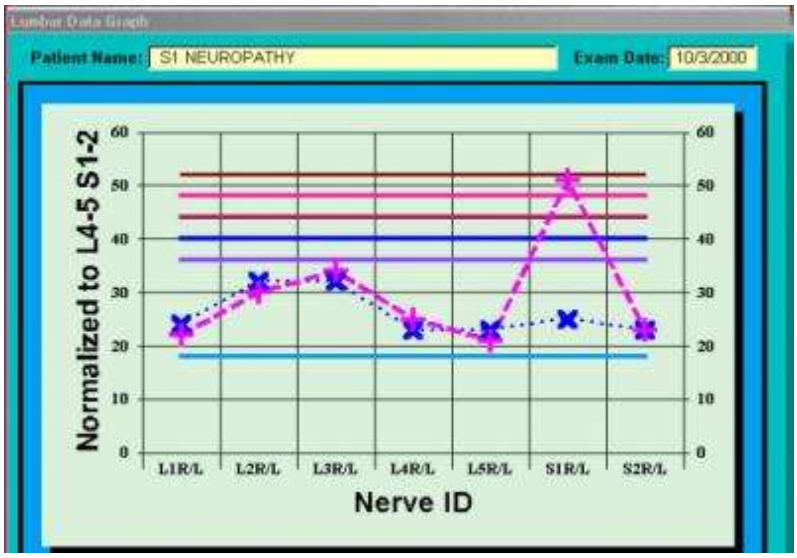

Before Treatment Mean SEM

TABLE II: MCGILL, OSWESTRY, ROLAND-MORRIS---BEFORE AND AFTER (N=27)

	BEFORE TX MEAN + SEM	AFTER TX + SEM	P VALUE
MCGILL	40.629 + 3.048	21.555 + 2.510	P <0.01
ROLAND MORRIS	16.074 + 0.935	11.740 + 1.021	P < 0.01
OSWESTRY	25.963 + 1.260	20.666 + 1.224	P < 0.01

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Pain Fiber NCS Objectively Supports and Quantifies Subjective Pain

Presented: American Academy of Pain Medicine San Diego February 2006 Abstract Form Category: Research - Clinical
Title:
Sensory A-delta fiber nerve conduction testing discriminates mild from severe chronic spine pain
Abstract Text:
Objective validation of the complaint of pain is a major problem in treating chronic spinal area pain, and chronic pain in general. While patient symptom description and imaging studies help to refine pain assessment, there is need for an objective test of pain perception, and measurement of pain magnitude.

In our study, we correlated sensory A-delta fiber nerve conduction testing with clinically determined mild and severe pain levels reported in patients with paraspinal pain. Two groups of 40 non-compensation patients were categorized as having mild or severe pain based upon a scoring system. Subjective symptoms, range of motion measurements, pain inventories, imaging studies, employment status, drug seeking behavior, and use of medications were assessed in determining an overall picture of pain severity. Sensory nerve conduction tests were done. The abnormalities were added to arrive at a score. Patients with mild pain had a mean score of 9 and those with severe pain had a score of 30. A chi square analysis of the results, indicated that the A delta combined scores correlated with pain severity at a p level < 0.01.

We found that sensory nerve conduction testing is a useful objective parameter in the evaluation of chronic spinal pain. This test is very inexpensive for patients, non-invasive, and a potential avenue for routine evaluation of chronic paraspinal pain.

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PAIN FIBER NCS REVEALS LUMBOSACRAL NERVE ROOT LESIONS ASSOCIATED WITH PROSATITIS AND PELVIC PAIN SYNDROMES

AMERICAN COLLEGE OF SURGEONS
OCTOBER 10, 2008

TITLE: Chronis Prostatitis Causing Pelvic Pain Due To Sacral Entrapment

INSTITUTION: Chicago Medical School, Center for Genitourinary Disorder, Rehab. Associate, Chicago, Dundee, Rockford, IL, USA

AUTHORS: Mohammed Badruddoja, MD., MS., FRCS., FRACS, Irving M. Bush, MD., FACS, Irshad Mohammad MB. VS, Aaron P. Bush B. Ed, Fred Aguilar CRN

INTRODUCTION: Sacral Entrapment Syndrome (SES) may be related to pain seen in Chronic Prostatitis. This can be diagnosed by the use of a pain fiber NCS device, which detects the hyper/hypo-function of pain-carrying delta fibers. Pain fiber NCS is used as a diagnostic modality to establish and locate the site of pain in Radiculopathy, Neuropathy, Spinal Stenosis, Pyriformis Syndrome, and Vulvadynia. The prostate gland is supplied by L4, L5, and S2, and entrapment of any of these nerves can be found by using pain fiber NCS.

METHODS: 53 patients with Chronic Prostatitis had Neural Scan studies. The diagnosis was established by history, physical examination, laboratory studies and prostatic ultrasound, after excluding all other causes of pelvic pain. None of the patients reported radicular symptoms.

RESULTS: 30/53 patients had evidence of S1 and S2 entrapment. In addition 14 patients had evidence of S1, L4, and L5 nerve root compression. 9 Patients had normal Neural Scans. 15 Patients were treated with appropriate nerve root block, by trained pain specialist, which was effective in 9/15 patients. 29 patients were treated with uroplasty (neuromodulation), once a week for 12 weeks. 21/29 patients were asymptomatic after treatment. 3 patients relapsed when they were evaluated 10 weeks after therapy.

CONCLUSION: Pelvic Pain due to Chronic Prostatitis is very likely due to entrapment of S1 and S2 nerve. Patients with positive pain fiber NCS studies are good candidates for treatment with nerve block or uroplasty (neuromodulation).

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Prototype device - without a potentiometer, was termed V-sNCT

Title: Predicting Nerve-Root Pathology with V-sNCT, Voltage-Actuated Sensory Nerve Conduction Threshold

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Abstract
Voltage-actuated sensory nerve conduction threshold (V-sNCT) is a quantitative test of sensory function. This study compares the sensitivity, specificity, and predictive value of V-sNCT compared to physical examination for the presence of nerve-root adhesions visible on epidurogram. Predicted abnormal nerve roots by V-sNCT and/or physical examination were compared with abnormal nerve roots documented by abnormal epidurogram in forty-nine patients with L5/S1 radicular back pain. Sensitivity, specificity, and predictive value for V-sNCT predicting nerve root pathology were 94.6%, 70.2%, and 91.0%, respectively, compared to 61.7%, 72.3%, and 87.6%, respectively, for prediction by physical examination alone. In addition, area under ROC Curve and relative risk for V-sNCT were significantly more predictive of nerve-root pathology than physical examination ($p<0.05$). Prediction of abnormal nerve-root pathology with V-sNCT is superior to prediction from neurological examination.

Introduction
Sensory neurological examination is an important part of any physical examination. This part of the physical examination takes on even more importance in the workup of the patient with chronic pain. During World War II, Dr. George Riddoch, a neurologist in the British Army, developed a logical approach to the sensory examination with the concept of identifying "signature" surface areas highly correlated with specific anatomic dermatomes, which, in turn, are associated with specific nerve roots.[1] Later, the concept of current perception threshold (CPT) was developed to quantitate level of sensory deficit.[2] Problems developed with this diagnostic technique, however, with significant variability associated with changing skin resistance.[3] Recently, the concept of voltage-actuated sensory nerve conduction (V-sNCT) has resulted in the development of a new instrument to quantitate sensory function (Medi-Dx 7000™, Neuro-Diagnostic Associates, Inc., Laguna Beach, CA). This instrument provides testing which is voltage mediated, and results are independent of changes in skin resistance. The purpose of this study was to assess how well V-sNCT worked in predicting nerve-root pathology as defined by filling defects on epidurogram prior to epidural lysis of adhesions.

Methods
After IRB approval and informed consent, patients with L5 or S1 radicular back pain scheduled for lysis of epidural adhesions[4] were studied. All patients underwent pre-procedure V-sNCT testing. All patients received catheter-directed lysis of adhesions, during which an epidurogram was done with 10 ml of Omnipaque-180 contrast. Predicted abnormal nerve roots identified by V-sNCT prior to the procedure were compared with abnormal nerve roots documented by abnormal epidurography of the nerve root. In addition, neurological examinations were conducted relating to the nerve roots tested. Neurological examination for L5 and S1 nerve roots were conducted as described by Hoppenfeld.[5] Neurological exam associated with L5 and S1 consisted of a motor, reflex (except for L5), and sensory test. Motor test for L5 was to test dorsiflexion of the big toe with the extensor hallucis longus. The examiner supported the patient's foot with one hand around the calcaneus and then placed his/her thumb in such a position that the patient must dorsiflex his/her great toe to reach it. The examiner opposed the dorsiflexion by placing his/her thumb on the nail bed of the great toe and fingers on the ball of the foot. Motor test for S1 was to test plantar flexion and eversion of the foot by opposing this motion with pressure on the head of the fifth metatarsal. There is no reflex to test for L5 function, but S1 was tested with the Achilles tendon reflex. Sensation for L5 was tested on the dorsum of the foot; sensation for S1 was tested at the lateral malleolus. In addition to the L5 and S1 nerve roots, major peripheral nerves tested included the superior and inferior gluteal nerves, as well as the sciatic, tibial and common peroneal nerves.

The statistical analysis tested the predictive power of V-sNCT compared to the predictive power of physical examination, using the abnormal epidurogram as the pathological standard. Analysis was with chi-square and ROC analysis,[6] with significance defined at $p<0.05$. Sensitivity, specificity, and predictive value for V-sNCT and physical findings as predictors of root pathology were calculated using the following formulas:

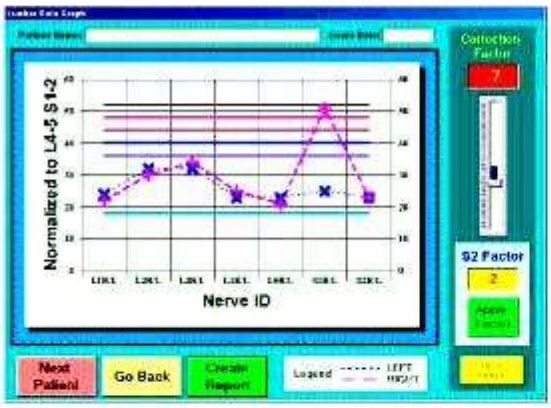
Sensitivity=Test True Positives / Real Positives
=100 x Predicted Abnormal / (Predicted Abnormal + False Normal)
Specificity =Test True Negatives / Real negatives
=100 x Predicted Normal / (Predicted Normal + False Abnormal)
Predictive Value=Test True Positives/All Positives
=100 x Predicted Abnormal/(Predicted Abnormal + False Abnormal)

Confidence intervals of the areas under the ROC curves were used to test for significant difference between prediction with V-sNCT and neurological examination at p<0.05. Also, relative risk and 95% confidence intervals were calculated for abnormal epidurogram, given an abnormal V-sNCT or an abnormal neurological examination at L5 and S1. Visual analog scores (VAS) were compared using Student's paired t-test. Significance was p<0.05.

Results

Forty-nine patients were studied. 28 males and 21 females. Age (mean±SEM) was 49±2, weight 86±3 kg, height 172±1 cm. Twenty-five patients had undergone previous back surgery; all had a diagnosis of lumbar radiculitis. VAS pain scores prior to procedure were 8.6±0.2 and one month after the procedure VAS pain scores were 4.4±0.4 (p<0.05). V-sNCT test results and epidurograms for a patient with an S1 lesion are shown in Figure 1 and Figure 2. Sensitivity, specificity, and predictive value for V-sNCT predicting nerve root pathology were 94.6%, 70.2%, and 91.0%, respectively, and an ROC curve was calculated (see Figure 3). Area under the ROC curve is 0.82±0.04 (p<0.001; 95% CI 0.76-0.90) for V-sNCT, compared to 0.61±0.04; 95% CI 0.60-0.74) for neurological examination (p<0.05).

Figure 1. V-sNCT and epidurogram prior to lysis of adhesions at S1. Note hypoesthesia of right S1 on the V-sNCT graph is correlated with lack of filling of the right S1 root on epidurogram.



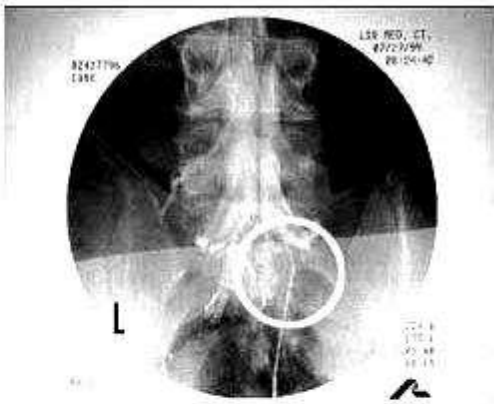
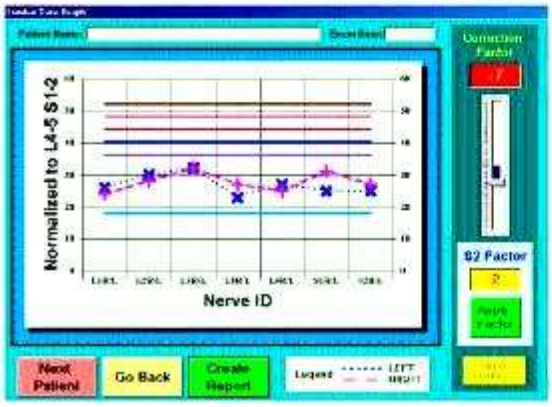


Figure 2. V-sNCT and epidurogram after lysis of adhesions at S1. Note that V-sNCT has returned to normal and right S1 nerve root fills with contrast.



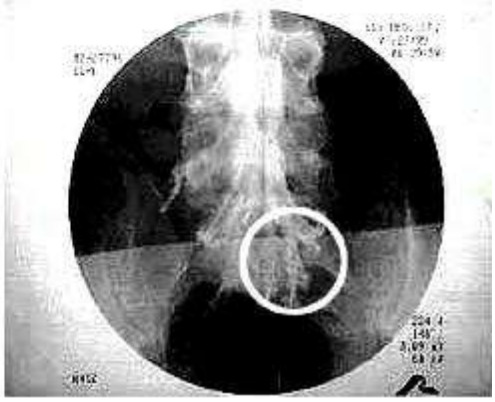
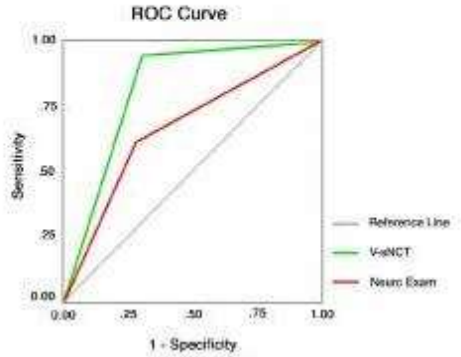
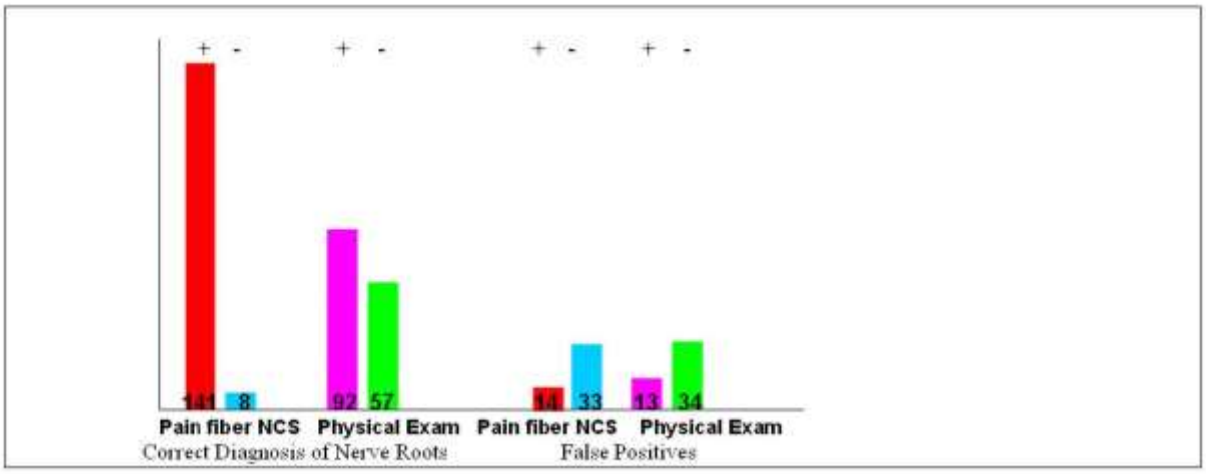


Figure 3. Receiver Operating Characteristic (ROC) curves comparing the effectiveness of V-sNCT and neurological examination in predicting nerve-root pathology. Better predictors are indicated with movement of the reference line to the upper left.





	V-sNCT	Neurological Examination
Sensitivity	94.6%	61.7%
Specificity	70.2%	72.3%
Predictive Value	91.0%	87.6%
Area under Curve (ROC)	0.82±0.04 (0.76-0.90)*	0.67±0.04 (0.60-0.74)
Relative Risk	4.67 (2.50-8.69)*	1.40 (1.17-1.66)

*p<0.05, compared to Neurological Examination

Discussion

Voltage-actuated sensory nerve conduction threshold (V-sNCT) is a direct quantitative sensory test which provides a reproducible functional assessment of the peripheral sensory nervous system by measuring that voltage intensity which initiates membrane potential changes to propagate nerve impulses. This study used the Medi-Dx 7000 to test the V-sNCT. A predecessor, the Neurometer assesses sensory function by measuring current output, which varies with changes in skin resistance. Although sensitivity with V-sNCT was superior to neurological examination, specificity was no different than the neurological examination. However, both the V-sNCT and neurological examination give the clinician the ability to test various branches of the peripheral nerves, which may result in further definition of the location of entrapment/pathology to specific nerve segments.

Conclusion

The purpose of this study was to compare the sensitivity and specificity of V-sNCT for the presence of nerve-root adhesions visible on epidurogram. Voltage-actuated sensory nerve conduction threshold provides the pain-management specialist with a sensitive and specific tool for prediction of nerve-root pathology. Advantages of V-sNCT include simplicity, decreased test time, small intra-patient variability, and decreased sensitivity to changes in skin resistance. It is a better predictive test than the neurological examination because it is a more sensitive test. In addition, because the patient is blinded to the V-sNCT results, it may be a very good test for malingering. This study demonstrates that prediction of nerve-root pathology with V-sNCT is superior to prediction of nerve-root pathology from neurological exam alone.

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