# Clinical Research
**Studies and Publications**

The Effects of Vertebral Axial Decompression On Sensory Nerve Dysfunction in Patients with Low back Pain and Radiculopathy
Frank Tilaro, M.D. Dennis Miskovich, M.D.
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## ABSTRACT

Effective non-surgical decompression of the nerve root has not been available to this date. The vertebral axial decompression (VAX-D) therapeutic table has demonstrated an ability to significantly reduce intradiscal pressure to a negative 150mmHg., allowing for disc decompression. The purpose of this study was to determine if VAX-D therapy could externally decompress the nerve root. Patients with radiculopathy and abnormal sensory function determined by the Current Perception Threshold (CPT) Neurometer who had received VAX-D therapy were retrospectively studied. CPT readings on 22 peripheral nerves were taken before and after VAX-D therapy.

Only patients with initial abnormal CPT readings, symptoms of sciatica, positive SLR, and positive imaging studies were reported on. The results after therapy were as follows: 14/22 nerves (64%) returned to normal function, 6/22 (27%) improved, 1/22 (4.5%) had no improvement and 1/22 (4.5%) showed deterioration. The average neurometer grade before therapy was 6.36 and after therapy 2.09 (a score of zero indicates normal function). Overall improvement was 67% ($p<0.05$). Theoretical considerations regarding the mechanism of action are expounded upon in this paper.

## INTRODUCTION

Patients with nerve root compression secondary to a herniated disc are frequently treated surgically although there is evidence they may be managed conservatively. Spangfort's computer assisted analysis of surgically treated disc herniations concluded that disc herniations were best treated surgically (1). This data has been refuted by Weber who conducted a randomised controlled trial between surgically and conservatively treated patients (2). Hakelius reported that patients with neurological deficits did not have any difference in outcome whether treated surgically or conservatively (3). Saal and Saal studied the natural history of radiculopathy and conducted an outcome study in 64 patients with radiculopathy treated non-surgically and concluded that patients with disc herniations could be managed non-surgically (4,5). Bush has reported his results on the successful non-surgical treatment of radiculopathy (6).

Although the literature demonstrates success for treating herniated discs conservatively,

many patients still undergo a surgical procedure for patients with nerve root compression. To this date, a non-surgical method to decompress the nerve root has not been available. Non surgical decompression could have significant advantages over the surgical methods currently in use. These may be reduced cost, early back to work, lower morbidity, a reduction in post operative complications and elimination of the failed back syndrome. Medical decompression could constitute a reconstructive process since spinal biomechanics and metabolism should be favorably altered in order to achieve decompression. Surgery does not favorably alter the biochemistry and physiology of the disc.

The vertebral axial decompression (VAX-D) therapeutic table has demonstrated its effectiveness in treating low back pain with and without radiculopathy (7). The table asserts its effects through decompression of the intervertebral disc and has reduced intradiscal pressures to a negative 150mmHg (8). It's assumed that reduction of intradiscal pressures to such significant levels should produce nerve root decompression but this has not been specifically investigated. The purpose of this study was to determine if VAX-D therapy effectively decompresses nerve roots. There was no attempt to correlate the results of this study with the patients outcome.

## MATERIALS AND METHODS

A retrospective review of patient charts from an outpatient clinic was conducted. All patients had Current Perception Threshold (CPF) neurometer testing before instituting VAX-D therapy and immediately after completion of a course of therapy. Only patients with abnormal CPT grades who had sciatica, positive SLR, and imaging studies that correlated with the observed clinical syndrome were reported on.

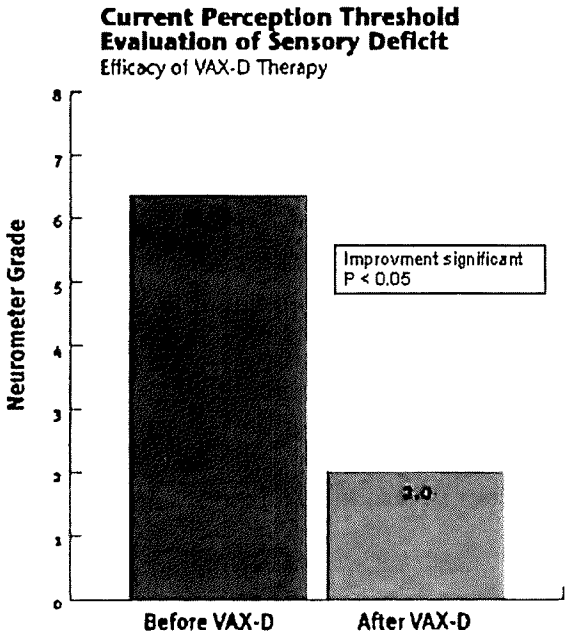
A total of 17 patients qualified, 22 nerves were studied since some patients had multilevel involvement. The nerves measured were the peroneal and sural nerves from the LA-5 and L5-S I nerve roots rsespectively. Sensory nerve dysfunction was measured by the CPT neurometer.

The CPT neurometer relies on transcutancous electric nerve stimulation at multiple sinusoidal frequencies to determine current perception thresholds (9). Its effectiveness has been well established in numerous studies (10,11,12). The CPT measure represents the minimal amount of a painless, neuroselective, transcutaneous electrical stimulus required to reproducibly evoke a sensation at least half the time it's presented. Three independent CPT measurements are obtained from each site tested using three different frequencies of electrical stimuli: 2000 Hz, 250 HZ and 5 Hz representing respectively the large myelinated, small myelinated and small unmyelinated fibers(13). Abnormally low CPT measures indicate a hypersensitive nerve function (seen in early stages of dysfunction) while elevated CPT measures indicate a loss of nerve function reflecting a hypoesthetic condition seen in advanced stages of dysfunction.

CPT measures are stated in units equivalent to 0.01 milliamperes (mA) of output intensity. Below 0.10 mA, CPT measures are resolved in increments of 0. I CPT. A CPT

of 100 indicates a stimulus output intensity of 1.0 mA, a CPT of 9.5 indicates an output intensity of 0.095 mA. The output range of the CPT device is 0.001 mA (CPT = 0. 1) to 9.99 mA (CPT = 999).

Figure 1




The Neuval CPT Evaluation and Database software evaluates and stores a patient's CPT value and generates a report detailing the condition of the nerves tested. These evaluations are based upon comparisons with standardized ranges of healthy CPT values and ratios included in the software. Two types of analyses are performed. Range analysis quantifies neuropathies from the hyperesthetic through the hypoesthetic stages by comparing values at 2 KHz, 250 Hz, and 5 Hz from a test site to normal values. Ratio analysis compares the different CPT measures for each of the 3 frequency readings within a nerve fiber. Two types of ratio analyses are performed on the patient's CPT values. The within site ratio analysis compares ratios of the different frequencies obtained from the same test site, in this study the test sites over the peroneal or sural nerves. The between site analysis compares ratios of the different frequencies obtained from contralateral sites i.e. values from the left sural or peroneal nerve compared to the right sural or peroneal nerve. The ratio analyses comparisons are sensitive in the earliest stages of neuropathy, whereas the range analyses represents greater functional impairment.

Range analysis quantifies neuropathies on a scale from -1 to +4, representing mild hyperesthesia to anesthesia. Within site ratios and between site ratios are assigned values between 1 to 2 which

represent the slightest sensory dysfunction to mild dysfunction. The grading system takes into account that in order of severity an anesthetic condition is more severe than a hypoesthetic condition, which is more severe than a hyperesthetic condition, which is more severe than a within site ratio abnormality, which is more severe than a between sites ratio abnormality.

The Neuval software analyses the CPT test values for range and ratio analyses scoring and assigns the test stimulus with the highest CPT score its corresponding Grade value while any additional scores are assigned their respective Adder grade values. Table 1 illustrates the CPT parameters for achieving a grade. A grade of 0 means no abnormality, grades I to 4.82 the very slightest to mild sensory dysfunction and grades 5 to 12 represents mild hyperesthesia to anesthesia.

Table 1: CPT Parameters

| Analysis | Score | Grade | Adder | Maximum grade | Commentary |
|---|---|---|---|---|---|
| Range | 4 | 10 | 1 | 12 | Anesthetic |
| Range | 3 | 9 | 0.45 | 9.9 | Severe hypoesthesia |
| Range | 2 | 8 | 0.41 | 8.82 | Moderate hypoesthesia |
| Range | 1 | 7 | 0.37 | 7.74 | Mild hypoesthesia |
| Range | -2 | 6 | 0.31 | 6.62 | Moderate hyperesthesia |
| Range | -1 | 5 | 0.27 | 5.54 | Mild hyperesthesia |
| W/S Ratio | 2 | 4 | 0.41 | 4.82 | Mild sensory disfunction |
| W/S Ratio | 1 | 3 | 0.35 | 3.70 | Very mild sensory disfunction |
| B/S Ratio | 2 | 2 | 0.39 | 2.78 | Slight sensory disfunction |
| B/S Ratio | 1 | 1 | 0.33 | 1.66 | Slightest sensory disfunction |
| All | 0 | 0 | 0 | 0 | No abnormal |

The clinical population of this study were patients who suffered from chronic dysfunction averaging 17.2 months in duration. These patients were refractory to various forms of conservative care including bed rest, traction, physical therapy, medications, chiropractic, and injections. This group of patients then received VAX-D therapy and CPT evaluation was performed before and after VAX-D therapy.

The VAX-D table provides a monitored distraction force to the lumbar spine with a controlled energy time function unique to this medical device, lowering intradiscal pressure to the negative range (pressures are reduced to a low of negative 150mmHg). The table was specifically engineered with this purpose in mind. Andersson performed a review of all traction devices and concluded that none of the devices reduced intradiscal pressure to the negative ranges, in fact some devices actually increased intradiscal pressure (21). The indications for VAX-D are low back pain with or without radicular symptoms, persisting for 8 weeks or more. Patients with low back pain associated with tumor, infection, osteoporosis, bilateral pars defects, spondylolisthesis Grade 2, surgical hardware, and the cauda equina syndrome are not considered candidates for VAX-D therapy. Patients with severe lateral stenosis or central stenosis are not ideal candidates for VAX-D therapy since these conditions represent a different disease process. Patients with the post surgical failed back syndrome or fusion may be treated with VAX-D.

Table 2: CPT Scores and Grades

| | Pre VAX-D Score | Pre VAX-D Score | Pre VAX-D Score | Pre VAX-D Grade | Post VAX-D Score | Post VAX-D Score | Post VAX-D Score | Post VAX-D Grade |
|---|---|---|---|---|---|---|---|---|
| Patient | 2 KHz | 250 Hz | 5 Hz | | 2 KHz | 250 Hz | 5 Hz | |
| 1 | -2 | -2 | -1 | 6.58 | -2 | -1 | 0 | 6.27 |
| 2 | 4 | 0 | 0 | 10 | 0 | 0 | 0 | 0 |
| 3 | -1 | 0 | 0 | 5 | 0 | 0 | 0 | 0 |
| 4 | 0 | -2 | 0 | 6 | 0 | 0 | 0 | 0 |
| 5 | -1 | -1 | 0 | 5.27 | -1 | 0 | 0 | 5 |
| 6 | 0 | 0 | -1 | 5 | 0 | 0 | 0 | 0 |
| 7 | 4 | 4 | 0 | 11 | 0 | 0 | 0 | 0 |
| 8 | -1 | 0 | 0 | 5 | 0 | 0 | 0 | 0 |
| 9 | 4 | 0 | 0 | 10 | 0 | 0 | 0 | 0 |
| 10 | -2 | -2 | -1 | 8.58 | 0 | 0 | 0 | 0 |
| 11 | -1 | 0 | 0 | 5 | 0 | 0 | 0 | 0 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 11 | -1 | -1 | 0 | 5.27 | -1 | 0 | 0 | 5 |
| 11 | 0 | -1 | 0 | 5 | 0 | 0 | 0 | 0 |
| 12 | -2 | -2 | -1 | 6.58 | -2 | -2 | -1 | 6.58 |
| 13 | -2 | -2 | 0 | 6.31 | 0 | 0 | 0 | 0 |
| 14 | 0 | 0 | -1 | 5 | 0 | 0 | 0 | 0 |
| 15 | -2 | -2 | -1 | 6.58 | -2 | -1 | 0 | 6.27 |
| 15 | -2 | -2 | 0 | 6.31 | 1 | -1 | 0 | 5.27 |
| 16 | 0 | -2 | -1 | 6.27 | 0 | 0 | 0 | 0 |
| 16 | -2 | -2 | 0 | 6.31 | -2 | -2 | -1 | 6.58 |
| 16 | -2 | 0 | 0 | 6 | 0 | -1 | 0 | 5 |
| 17 | 0 | 0 | -1 | 5 | 0 | 0 | 0 | 0 |

All exercise programs and physical therapy sessions are discontinued once the patient begins VAX-D therapy. Patients taking medications (NSAID's, non-narcotics, and narcotic medications) were allowed to continue with their medications. Patients in this study received treatments 3 to 5 times per week and averaged 23 total treatments.

## RESULTS

There were 13 males and 4 females in the study. The average age was 40.8 yrs and the average duration of symptoms was 17.2 months. Three patients had multilevel involvement. CPT scores ranged from 5 (mild hyperesthesia) to 11 (anesthesia) prior to VAX-D therapy.

The results after VAX-D therapy were as follows: 14/22 nerves (64%) returned to normal function, 6/22 (27%) improved, 1/22 (4.5%) had no improvement and 1/22 (4.5%) was worse (Table 2). Ninety-one percent (91%) demonstrated improved neurological function measured by the CPT Neurometer after VAX-D therapy. The average neurometer grade before therapy was 6.36 and after therapy 2.09 (Figure 1). Overall improvement was 67%, statistically significant at $p<0.05$. Sixty-four percent (64%) of the patients achieved complete recovery of neurologic function.

## DISCUSSION

The data from this study demonstrate that the VAX-D therapeutic table is capable of affecting spinal sensory nerve dysfunction in abnormal nerves secondary to a compressive radiculopathy. 16/22 nerves showed abnormalities in the large myelinated fibers, 13/22 demonstrated abnormalities in the small myelinated fibers and 8/22 demonstrated abnormalities in the small unmyelinated fibers. These fibers are the major

subpopulation of sensory nerve fibers that provide innervation to the test sites that were measured in this study.

The mechanism responsible for the signs and symptoms of sciatica are complex and not fully elucidated. Although compression of the nerve or nerve root is a consistent finding in sciatica, compression by itself does not explain all the observed symptomatology (11,24).

Many of the studies that have yielded data regarding the effects of compression come from studies on peripheral nerves. There are anatomical differences between peripheral nerves and the spinal nerve roots that render the spinal nerve roots more vulnerable to compression. The spinal nerve has both an intrathecal and extrathecal portion. The intrathecal portion is bathed by the CSF which offers some protection from compression. The extrathecal portion has very little collagen supporting tissue, which renders the fibers susceptible to the effects of compression. By comparison, the peripheral nerve fibers have a generous amount of supporting collagen tissue that afford it some protection from compression (11).

Compression of a peripheral nerve does not cause pain unless the nerve has been previously irritated (17,38). Compression of the dorsal root ganglion (DRG) can result in pain. Rydevik compared the dorsal root ganglion to a closed compartment syndrome when mechanically deformed. The DRG has a rich vascular supply and a tight capsule. Compression leads to edema and hemorrhage in the endoneurial space with subsequent elevation of interstitial tissue fluid pressure and reduced blood supply to the sensory nerve cells in the DRG (36). The DRG has the capability to act as a neuromodulator of pain through release of substance P, calcitonin gene related peptide and possibly other chemical mediators (44).

The compressive force and rapidity of onset are both important factors leading to nerve dysfunction (26-30). Olmarker reported that a rapid onset of compression induced a more pronounced effect than a slower onset both in terms of intraneural edema formation and impairment of the nutritional supply to the nerve roots. The compressive forces exerted by a herniated disc can be as high as 400mmHg.

Vascular compromise has been an attractive theory to explain the effects of compression. Rydevik has shown that with slow nerve compression there is compromise first of the venules, then capillaries, and finally the arterioles. In the animal model this occurred at 30mmHg. Compression studies performed on the pig cauda equina have shown that electrophysiologic changes occur at 30 mmHg., the same pressure that causes vascular compromise (35).

If the compressive force is great enough and or occurs rapidly, compression can cause significant nutritional compromise in the nerve root and peripheral nerves (8,29,32,34,42). The primary nutritional support is through the arterial tree and the secondary nutritional support is through the cerebrospinal fluid. Compromise of the arterial tree can be partially negated by continued nutrition via the cerebrospinal fluid. If compression affects the spinal nerve root proximal to the dorsal root ganglion both

support systems are compromised and diffusion of metabolites and nutritional support mechanisms are markedly reduced.

The mechanism through which the VAX-D table accomplishes decompression is related to reduction of intradiscal pressure. Both a direct mechanical effect and biochemical effect may be responsible. Reduction of intradiscal pressure could allow for retraction of the herniation. A possible shearing affect that interrupts the connection of the herniation to the central nucleus, given the arrangement of the annular fibers is a possibility with extruded hernias. This study indicates that the distraction of vertebral structures by VAX-D also reduces compression of spinal nerve roots.

Inflammation and the inflammatory response very likely play an important role in producing some symptoms associated with sciatica. The nucleus pulposus has demonstrated inflammatogenic properties and is capable of inducing an immune reaction (21,24,25,40). Saal has documented elevated levels of phospholipase A2 in herniated disc tissue (37). Phospholipase A2 is a precursor for arachidonic acid responsible for production of the inflammatory Prostaglandins and Leukotrines. Alterations in disc metabolism may be partly responsible for inflammation. The central portion of the disc is oxygen starved, there is a steep oxygen concentration gradient across the disc, concentrations being 20 to 30 times greater in the periphery (1O). High levels of lactate have been measured within the central nucleus (1O). The high levels of lactate may be responsible for activating destructive proteolytic enzymes and initiating the inflammatory cascade.

Since elevated intradiscal pressure adversely affects the diffusion gradient, nutrient and oxygen diffusion to the disc is impaired and anaerobic metabolism prevails. Anaerobic metabolism negatively affects the capabilities for healing and repair of all tissues. By significantly reducing intradiscal pressure the VAX-D therapeutic table creates a diffusion gradient enhancing the delivery of nutrients and oxygen to an oxygen starved disc, creating an aerobic environment, enhancing repair, and possibly interrupting the inflammatory cycle.

## CONCLUSIONS

Fourteen of twenty-two peripheral nerves (64%) showing abnormal dysfunction secondary to a compressive radiculopathy returned to normal function after a therapeutic course of VAX-D therapy. The data from this study implies that VAX-D therapy is capable of influencing sensory nerve dysfunction associated with a compressive radiculopathy. Motor dysfunction returns before sensory dysfunction in compressive radiculopathies so it is rather striking that we observed total remission in 64% of the cases with sensory dysfunction (2). It is possible that reduction of intradiscal pressure by VAX-D significantly alters the biomechanics and biochemistry of the disc and nerve root.

The author realizes there are shortcomings with nonrandomized retrospective analysis such as this study. In attempts to minimize selection bias, the patients reported on in this

and imaging results. Also, the final outcome was an objective measure which could not be influenced by subjective or other external factors.

The objective of this study was to make any observations on sensory nerve dysfunction attributed to VAX-D therapy. Patient outcomes were not measured although the majority of patients reported subjective relief of pain. Further studies investigating the effects of VAX-D therapy on sensory and motor dysfunction are encouraged.

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