

Prospective Randomised Controlled Study of VAX-D and TENS for the Treatment of Chronic Low Back Pain

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Abstract

Low back pain is one of the most significant medical and socioeconomic problems in modern society. International guidelines call for evidence-based management for the pain and disability associated with musculoskeletal disorders. The purpose of this randomised controlled trial is to address the question of efficacy and appropriateness of VAX-D (Vertebral Axial Decompression) Therapy, a new technology that has been shown in clinical research to create negative intradiscal pressures, and has been shown to be effective in treating patients presenting with chronic low back pain (>3 months duration) with associated leg pain. Successful outcome was defined as a 50% reduction in pain utilising a 10cm Visual Analogue Pain Scale and an improvement in the level of functioning as measured by patient-nominated disability ratings. Patients were randomly assigned to VAX-D or to TENS which was used as a control treatment or placebo. The TENS treatment demonstrated a success rate of 0% while VAX-D demonstrated a success rate of 68.4% ($P < 0.001$). A statistically significant reduction in pain and improvement in functional outcome was obtained in patients with chronic low back pain treated with VAX-D. (Neurol Res 2001; 23:780-784)

Introduction

Low back pain is a major cause of disability in today's society. According to the National Health and Medical Research Council (NHMRC), each year approximately 600,000 Australians present with low back pain as a recent illness. Although a high percentage of patients with acute low back pain recover within 4-6 weeks, a significant number of patients suffer from recurrences. Von Korff has studied the natural history and found that approximately 60% will have recurrences. (1) In a study of back pain in primary care, Von Korff and Saunders found that 60% to 75% improve in the first month, 33% report intermittent or persistent pain at year one, and 20% of patients describe substantial limitations at this time. (2) Klenerman et al demonstrated that 7.3% of individuals with acute low back pain who had not recovered by two months still reported high levels of pain and disability at twelve months after onset. (3) Chronic low back pain is increasing faster than any other disability, and 5-7% of the population will report their back problems as being a chronic illness. Fifty percent of work loss caused by back pain is accounted for by duration of disability for longer than 4 weeks. In Australia chronic low back pain affects more than 1,900,000 individuals and costs Australia more than 10 billion dollars each year.

International guidelines call for evidence-based management for the pain and disability associated with musculoskeletal disorders. Today's primary care practitioners have a comprehensive responsibility in the management of their patient's low back conditions, and they must be aware that recurrences after the presenting episode are likely. The

literature suggests that for those who have not recovered by two months, management efforts should begin. (4)

Acute disc injury and discogenic pain is one of the primary processes leading to low back pain and lumbar radiculopathy, although the pathophysiologic mechanisms are still not well understood. It is believed that increases in disc pressures resulting from heavy lifting, vibrational and postural forces etc. are important factors in the pathogenesis of low back pain. The effects of disc hydraulics in herniations or protrusions may cause a mechanical deformation of the nerve roots and a compression-induced impairment of the vasculature. In addition, it has been found that the biochemical properties of the nucleus pulposus may induce a toxic or inflammatory reaction in the nerve root.

There have been many studies indicating that the disc and its associated pathology are identified as a primary cause of low back pain and lumbar radiculopathy. Hirsch stimulated various lumbar tissues in awake patients with the use of carefully placed needles. (5) Stimulation of the posterior portion of the annulus produced low back pain in many individuals. Furthermore, he was able to eliminate the pain by the injection of a minute volume of local anaesthetic into the annulus. Smythe and Wright placed nylon threads into various lumbar tissues while performing lumbar spinal operations. (6) During the postoperative period, they pulled on the threads and asked the patients to describe the location of any pain produced. The annulus fibrosus was the most common site of low back pain, and the compressed nerve root was responsible for sciatic pain. Tension placed on a normal nerve root resulted in no pain.

Falconer and associates published their observations made during exploration of the lumbar spine under local anaesthesia. (7) Murphy reported similar results in his small series of surgical cases. (8) Both authors concluded that the annulus and nerve root were the pain generating tissues. Wiberg in 1950, operating on 200 patients using local anaesthesia of the skin and muscles only, reported that pain emanated from the disc. (9) Kublisch operated on 193 patients using local anaesthesia and drew certain conclusions about the likely origin of back and leg pain. (10) Sciatica could only be produced by stimulation of a swollen, stretched, or compressed nerve root. Back pain was produced in the majority of cases by stimulating the outer layer of annulus fibrosus and the posterior longitudinal ligament.

If the disc is a major source of low back pain then applying specific target therapy for the treatment of disc pathology should improve patient outcomes. VAX-D is a primary, non-surgical treatment for the management of patients with disabling low-back pain and neurological symptoms associated with herniated and degenerative disc disease. Research has shown that the VAX-D table is a decompression device that is capable of reducing intradiscal pressures to negative levels. (11)

Successful reduction of intradiscal pressures with VAX-D represents a technological advance that should provide a means of addressing compressive disc pathology. Creating negative intradiscal pressure is likely to affect both the biomechanical and biochemical causes of discogenic pain. Patients suffering from discogenic pain and/or associated sciatic pain are seeking conservative treatment without the risks associated with injections and surgical procedures.

VAX-D incorporates advanced technology that permits the application of distractive tensions without eliciting reflex muscle guarding. Conventional traction devices have not demonstrated this ability or the ability to reduce intradiscal pressures to negative levels. Studies published in the medical literature report that intradiscal pressure either remains unchanged or increases during traction. (12) It has also been demonstrated that paraspinal muscles are not able to fully relax during conventional traction.

The beneficial effects of VAX-D decompression in the relief of peripheral nerve dysfunction has been previously reported in the literature, (13) and a multi-center outcome study reported that VAX-D treatment was successful in 71% of the 778 cases studied. (14).

This study was designed to evaluate the effect of VAX-D on chronic low back pain.

Material and Methods

In association with Quintiles, the world's largest health care consultancy organisation for data analysis in clinical trials, a protocol was developed and then approved by the Human Research Ethics Committee at the University of Wollongong, New South Wales, Australia.

It was predetermined that the treatment would be considered a success if the patient attained a fifty percent (50%) decrease in pain, numerically on the Visual Analogue Scale (VAS). Absolute changes in pain score determined by VAS over time were analysed with repeated measures analysis of variance and *t*-test. In addition, improvements in disability were recorded on a patient nominated disability rating. Any level of improvement in disability was acceptable. The instruments for determination of these outcomes were supplied by the National Musculoskeletal Initiative of Australia. The study itself was to be conducted in the medical clinics of the VAX-D Spinal Institute and so to prevent bias in the data collection Quintiles were engaged to collect and analyse the data. TENS was selected as an appropriate placebo treatment as a means of establishing a plausible but (probably) ineffective control for an unblinded treatment.

Through advertisement in local papers forty-four patients with chronic low back pain greater than 3 months in duration, with associated leg pain, and a confirmed disc protrusion or herniation on CT Scan or MRI were selected and randomised into the two treatment methods, either VAX-D or TENS. The patients were randomised in sequential order and treatments were determined by a predefined central randomisation list.

The average duration of pain in the patient population was 7.3 years. The conditions for receiving either treatment including travelling to and from the clinic and duration of therapy were designed to be the same for both populations. Inclusion criteria for the study were: age 18-65 years; a minimum VAS score of 2; candidates must live within 45 minutes of the clinic location; capable of thoroughly understanding the information given and following protocol. All candidates signed an informed consent form.

Exclusion criteria were: osseous stenosis; unstable spine (bilateral pars defect or Spondylolisthesis of Grade II or greater); spinal surgical implants; shoulder problems which prevent compliance with VAX-D therapy; spinal pain due to tumor, infection, or

inflammatory disease; pregnancy; and previous VAX-D therapy.

Patients randomised to VAX-D were treated according to the manufacturer's protocol. Patients lie on the split table device in a prone position. VAX-D utilises handgrips that the patient grasps with arms extended above the head to stabilise (restrain) the shoulder girdle and upper body. This is thought to be the most effective means of assuring that tensions applied to the pelvis are transmitted accurately along the linear axis of the spinal column during the procedure. The fact that the patient may release at any time during the treatment provides an important safety factor. A special harness designed to apply forces primarily to the lateral pelvic alae is fitted and tightened around the patient. The pelvic harness is connected to a tensionometer at the caudal end of the table. The function of the tensionometer is to provide constant feedback to the programmed logic control and operating system. During the VAX-D session a continuous chart recording is generated plotting the controlled time/energy progress of the entire procedure.

Table 1: Demographic data

Characteristic	Statistic	All	VAX-D	TENS
No of Patients	n	44	22	22
Age (years)	Mean	42	41	43
	Range	22-57	27-57	27-55
Sex				
Female	n	21	11	10
Male	n	23	11	12
Race				
White	n	40	20	20
Asian	n	4	2	2
Chronicity				
Yrs of Pain	Mean	7.3	8.4	6.2
	Range	0.25-30	0.25-30	0.5-28

Tensions are applied to the lumbar spine in a cyclic fashion from the baseline tension up to the therapeutic range of fifty to ninety-five pounds. Each treatment session is thirty minutes in length and is comprised of fifteen cycles of decompression alternating with relaxation. Each decompression and relaxation phase may be individually varied as suitable for the particular treatment parameters.

A chart recorder prints the time energy curve for each decompression-relaxation cycle. This affords the technician a means of monitoring and adjusting the decompression process. Patients received VAX-D therapy five times per week for four weeks and then once per week for four weeks in accordance with protocol. All VAX-D treatments were administered by certified VAX-D technicians at four clinics in the Sydney area.

Patients randomised to TENS therapy received treatment at one of the four clinics. Electrodes were placed according to the manufacturer's protocol. Patients lay prone on a treatment table and received TENS for thirty minutes daily for twenty days then once a week for four weeks. All patients receiving TENS were monitored by a technician.

Neither group received any physical therapy modalities, epidural steroid injections or other treatments during the trial. Both patient groups were allowed to take non-narcotic pain relievers and anti-inflammatory medication if necessary.

A 10-cm Visual Analogue Scale (VAS) for pain and a four-point disability rating scale were used to assess patient response. The level of pain on the VAS was recorded on a 10cm line marked at one end 'No Pain' and marked at the other end 'The Worst Pain Imaginable'. The written instruction to the patient was to 'please place a mark on the line below to indicate your current level of pain'. The self-nominated disability rating scale required patients to list the four activities that were most affected by their low back pain. These were scored according to the following criteria: 1 = cannot do at all; 2 = can do but severely limited; 3 = can do but slightly limited, 4 = can do without limitation.

Data was collected at the initiation of the study prior to randomization and at the end of the eight week treatment period in a separate interview. Success was defined as (equal to or greater than) a 50% improvement in the patient's pain and any improvement in their disability rating.

Patients were free to withdraw from the study on their own volition at anytime. The study treatment could be terminated prematurely if any of the following events occurred: patient wished to terminate his/her participation for whatever cause (two cases); the investigator judged it was in the best interest of the patient to withdraw (zero cases); the patient was unable to comply with protocol (zero cases).

The efficacy-evaluable population used for statistical analysis of efficacy is comprised of all patients who were randomised to study treatment, received at least 10 study treatments, had efficacy data recorded after Baseline, and satisfied the inclusion/exclusion criteria.

The primary efficacy measure in this study was the proportion of successfully treated patients in each of the treatment groups. The difference in proportions of successfully treated patients in each treatment group was tabulated and compared using Fisher's Exact Test and 95% confidence limits.

Successfully treated patients were to be followed up at six months to determine whether the successful outcome was sustained.

Results

Forty-four patients were enrolled into the study. Twenty-two were randomised to each of the treatment groups. A summary of demographic characteristics for the 44 enrolled patients is presented in Table 1.

Two patients (4.5% of 44), Patient 029 and Patient 003, were regarded as having withdrawn/not completed the study according to the protocol. Patient 029, randomised to TENS, withdrew due to not wishing to continue and Patient 003, randomised to VAX-D, withdrew due to treatment no longer being required. No patients were withdrawn by the investigator. Patients 018 and 034 both randomised to VAX-D, did not comply with the study criteria and are therefore excluded from the efficacy-evaluable population. They both had a baseline VAS score less than 2 but this error of inclusion was not picked up until the completion of the trial. The efficacy-evaluable population therefore comprised of 40 patients: 19 patients randomised to VAX-D, 21 randomised to TENS.

A summary of the data collected at baseline and post-treatment in the efficacy-evaluable population is presented in Table 2.

Table 2: Efficacy-evaluable population

Characteristic	Statistic	VAX-D	TENS	
Number of Patients	n	19	21	
Number of treatments	Mean	24.1	18.0	
	Range	18-36	10-24	
Baseline pain (VAS)	Mean	5.99	5.44	
	Range	2.1-8.7	2.7-8.5	
Post treatment pain (VAS)	Mean	1.85	5.97	
	Range	0-5.6	1.8-8.5	
Decrease in pain (%)	Mean	69.1	-17.1	
	Range	11.1-100	-123-33.3	
Disability Rating				
	Pretreatment	Mean	2.2	2.2
		Range	1.5-3	1.75-3
	Posttreatment	Mean	2.9	2.2
Range		2.0-4.0	1.5-3	
Improvement in disability rating (%)	Mean	33.8	-2.23	
	Range	0-100	-36.4-50.0	
Successful cases	n	13	0	
	Percent	68.4	0	

In the efficacy-evaluable population the proportion of successfully treated patients was 13 out of 19 patients (68.4%) for the VAX-D treatment group compared to zero out of 21 (0%) for the TENS treatment group. There was a high statistically significant treatment group comparison p-value of <0.001. The 95% confidence interval for the difference in proportions of successfully treated patients, comparing VAX-D with TENS was 47.5% to 89.3%.

In the VAX-D group all patients recorded some improvement in their pain levels whereas in the TENS group 13/ 21 recorded an increase in pain.

At six-month follow-up, of the 13 successful cases, 2 have been lost to follow-up, 1 case suffered a significant other injury and of the remaining 10, seven have shown sustained success (ie. they still meet the criteria for successful outcome).

The results reported for the TENS group were less than that expected for a placebo control. The negative outcomes may have been due to the fact that the TENS patients (and the VAX-D patients) had to travel to and attend a medical clinic five days per week for four weeks, and one day per week for four weeks. This fact that both treatment groups had to travel to, and attend the clinic, was necessary to ensure that the only variable between the two groups was in the type of treatment that they received. The benefits of treatment in the VAX-D group clearly outweighed the negative effects of travelling, which became evident in the placebo group.

Discussion

Disc stresses coupled with ongoing increased intradiscal pressures from mechanical loading may lead to failures in the normal biomechanics of the disc and progress to degeneration, posterior displacement of the nuclear material, annular disruptions and herniations. Other causative factors in the course of disc degeneration are negative diffusion gradients, reduction of the fluid content of the nucleus pulposus, and abnormal disc metabolism. With positive disc pressures throughout the day that are above diastolic pressure, the metabolism of the disc becomes anaerobic thus impeding the normal reparative healing abilities.

Proteolytic enzymes (matrix metalloproteinases) reside in the disc and have been implicated in disc degeneration. (15) The matrix metalloproteinases are regulated by specific inhibitors (TIMPS), cytokines (Interleukin-1) and growth factors. (16) Spinal loading may interfere with diffusion into the disc by reducing the gradient across the vertebral endplate. As disc metabolism becomes anaerobic, there is an accumulation of lactic acid, fall in pH, loss of chondrocyte and fibroblast function, and activation of the metalloproteinases.

Although the mechanism of action may not be fully understood the thixotropic (17) properties of the nucleus material may facilitate nuclear migration toward the centre of the disc under negative pressures created by VAX-D.

It has been shown experimentally that elevated lactate levels and low pH in the disc prohibit disc proteoglycan synthesis and accelerates matrix degeneration (18).

Destruction of the proteoglycan matrix and fluid retention properties can lead to a degenerative cascade with loss of cellular reparative functions and vitality. The reduction of intradiscal pressures may enhance the diffusion gradient across the endplate into the avascular disc. It has been postulated that mechanisms that facilitate oxygen and nutrient uptake in the disc may exert a beneficial effect on the metabolism and restorative functions.

Successful reduction of intradiscal pressures with VAX-D therapy represents a technological advance in lumbar spinal treatment and is likely to affect both the biomechanical and biochemical causes of discogenic pain. The results from this study demonstrate that VAX-D is an effective treatment for the management of patients with chronic low back pain and is significantly superior when compared to TENS therapy. Analysis of the data demonstrated an attributable success rate of 68.4% for VAX-D. These findings are consistent with earlier studies by Gose E, Naguszewski W, Naguszewski R. (14)

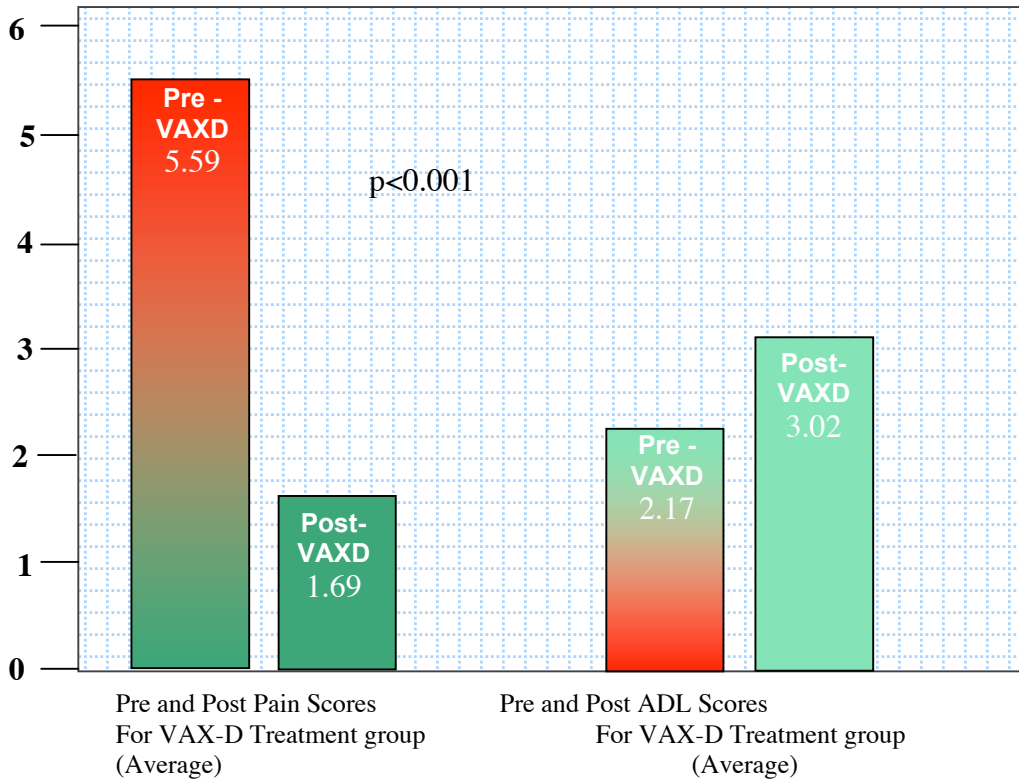
The results of this prospective study demonstrated that VAX-D can achieve a statistically significant improvement in pain and functional outcome in managing patients suffering from disc related chronic low back pain.

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Graph 1 - Results of VAX-D Treatment Group



References:

1. Von Korff M. Studying the natural history of back pain. Spine 1994; 19(18 Suppl): 2041S-2046S.
2. Von Korff M., Saunders J. The course of back pain in primary care. Spine 1996; 21: 2833-2837.
3. Klenerman L, Slade, PD Stanley IM. et al. The prediction of chronicity in patients with an acute attack of low back pain in general practice setting. Spine 1995; 20:478-484.
4. Bogduk N, Evidence Based Clinical Guidelines for the Management of Acute Low Back Pain; The National Musculoskeletal Medicine Initiative NHMRC; Nov 1999
5. Hirsch C. An attempt to diagnose the level of disc lesion clinically by disc puncture. Acta Orthop Scand 1948;18:132-140
6. Smythe MJ. and Wright V. Sciatica and the intervertebral disc. An experimental study. J Bone Joint Surg (Am) 1958;40:1401-1418
7. Falconer MA; McGeorge M, Begg AC; Observations on the cause and mechanism of symptom production in sciatica and low back pain. J Neuro Neurosurg Psychiatry 1948;11:13-26
8. Murphy F. Experience with lumbar disc. Clin Neurosurg 1973;20:1-8

9. Wiberg G. Back pain in relation to the nerve supply of the intervertebral disc. *Acta Orthop Scand* 1950;19:211-221
10. Kublich S, Ulstrom C, Michael C. The Tissue of Low Back Pain and Sciatica: A Report of Pain Response to Tissue Stimulation During Operations on the Lumbar Spine Using Local Anesthesia. *Orth Clinics of North Am* 1991; 22:181-187
11. Ramos G, and Martin W. Effects of vertebral axial decompression on intradiscal pressure. *J. Neurosurg* 1994; 81: 350-353.
12. Anderson G, Schultz A, Nachemson A. L. Intervertebral Disc Pressures During Traction. *Scand J Rehabil Supp* 1983; 9:88-91
13. Tilaro F, Miskovich D. The Effects of Vertebral Axial Decompression On Nerve Sensory Dysfunction; *Can J of Clinical Medicine* 1999 Vol 6 No 1: 2-7
14. Gose E, Naguszewski W, Naguszewski R. Vertebral axial decompression therapy for pain associated with herniated or degenerated discs or facet syndrome: An Outcome Study. *J Neurological Research* 1998 Vol 20:186-190.
15. Bogduk N. *Clinical Anatomy of the Lumbar Spine and Sacrum*. Third Edition. Churchill Livingstone 1997.
16. Fujita K, Nakagawa T, Hirabayashi K, Nagai Y. Neutral Proteinases in Human Intervertebral Disc. Role in Degeneration and Probable Origin. *Spine* 1993; 18:1766-1773
17. Nachemson A. Elfstrom G. Intravital Dynamic Pressure Measurements in Lumbar Discs. *Scand J. Rehabil Med (Supp)* 1970; 1:4-40
18. Matsui Y, Macda M, Nakagami W, Iwata H. The Involvement of Matrix Metalloproteinases and Inflammation in Lumbar Disc Herniation. *Spine* 1998; 23:863-69