The Treatment of Discogenic Low Back Pain: An Integrated Approach

Frank Tilaro MD

Paper Presented to the McKenzie North American Conference June 2-4, 2000

The literature recognizes the disc as the probable pain generator in the majority of patients with low back pain. Kuslich S, et al. performed progressive local anesthesia on patients with low back pain and noted their response to tissue stimulation during operations on the lumbar spine [9]. He found the annulus and nerve root to be the pain generator in the majority of cases. The facet joint capsule was an infrequent pain generator. Smyth and Wright placed nylon threads into various lumbar tissues while performing certain lumbar spinal operations and exerted tension on the threads[18]. This study indicated that the annulus was the most common site of low back pain and the compressed nerve root was responsible for sciatica. Hirsch C. carefully placed needles into the lumbar spine of awake patients and stimulation of the posterior portion of the annulus resulted in pain in the majority [6]. Mooney V. concludes that the source of chronic low back pain in the majority of cases is the disc [12]. Nachemson A. implicates the disc as the most likely cause of pain and provides indirect proof[13].

We accept the fact the disc acts hydrostatically or not [11]. Donelson, Aprill, Metcalf, and Grant utilized CT discography after McKenzie assessment in an attempt to correlate symptomatic discs with anular competence [3]. Seventy-four percent of the centralizers had positive discograms, of which ninety-one percent had an intact annulus. Of the patients who peripheralized, only sixty-nine percent had positive discograms while only fifty-four percent had an intact annulus. In patients who did not respond at all, only twelve percent had a positive discogram. This data is useful since it distinguishes discogenic from non-discogenic pain, as well as differentiating the competent from the non-competent annulus. Since the proper diagnosis is obtainable on a consistent basis the final challenge is to find specific therapy for the disc 'in patients who are not candidates for mechanical (manual) therapy. The purpose of this presentation is to introduce you to new technology that has demonstrated effectiveness on a consistent basis.

The VAX-D Therapeutic Table (Vertebral Axial Decompression) reduces intradiscal pressure to a minus 150 mm Hg., effectively decompressing the disc [16]. With conventional traction intradiscal pressures either increase, remain the same or slightly decrease[14]. Conventional traction devices elicit reflex muscle contraction thus interfering with decompression[1]. The time energy distraction curve for these devices is a linear response. The VAX-D table has a time energy distraction curve that is logarithmic, and we believe this is the reason decompression occurs with VAX-D[19].

The first clinical trial with VAX-D was the *Acute Low Back Pain Distress Study* performed in London, Ontario.[19] This was a randomized controlled trial comparing VAX-D to sham treatment. Unfortunately patients receiving the sham treatment could not be convinced they were receiving the real thing and the dropout rate was excessive. The patients in the sham treatment arm were subsequently treated with routine physical therapy modalities. The patients

treated with VAX-D showed a significant difference in outcome i.e. earlier resolution in symptoms and earlier return to work. A multi-center retrospective study in 778 patients with low back pain (average duration of pain was 43 months) demonstrated significant relief of symptoms immediately following VAX-D therapy.[5] VAX-D has been shown to reduce sensory nerve dysfunction in patients with compressive radiculopathy using a CPT Neurometer.[20]

Currently four studies are being prepared for submission for publication:

- a study using Dermatomal Somato Sensory Evoked Potentials (DSSEP's) to assess neve root decompression after VAX-D therapy agrees with the results from the CPT study;

- a prospective study using two dosing schedules has demonstrated dose dependency for VAX-D indicating a biological response;

- a four year post VAX-D study has shown its lasting benefits;

- and a randomized trial has further demonstrated its efficacy in patients with chronic symptoms.

VAX-D treatment is a stand alone therapy. The protocol calls for daily treatment sessions five days per week for four weeks and then once a week for four weeks. Patients may continue to take medications during treatment. Physical activity, including remaining at work, is encouraged if possible, but patients are instructed to avoid postures that increase intradiscal pressure. Most patients are treated between fifty-five and seventy-five pounds of tension. A treatment session is fifteen cycles and each cycle is one minute in distraction and one minute in relaxation. A chart recorder prints each distraction - relaxation cycle, demonstrating the logarithmic function. The recordings are continuously monitored since any deviance from the standard logarithmic curve indicates decompression may not be occurring.

The indications for VAX-D are patients with discogenic pain who have not responded to standard medical therapy. The contraindications include gross instability (spondylolisthesis Grade 2, bilateral pars defects, trauma), patients with any hardware in the spine, tumor or infection of the spine, and cauda equina syndrome.[19] Patients with neurologic deficits have been successfully treated with VAX-D.

VAX-D exerts its therapeutic effect through reduction of intradiscal pressure. This could affect both biomechanical and biochemical events. The disc is thixotrophic.[2] Reducing intradiscal pressure allows the disc to take advantage of this property, facilitating nuclear migration toward the center of the nucleus. Negative intradiscal pressures create a large diffusion gradient for oxygen and nutrients. Diffusion is the primary source of nutrition for the avascular disc. A steep oxygen gradient occurs across the disc, concentrations in the center of the nucleus 20 to 30 times less than the periphery.[7] This may explain why degeneration begins in the central portion of the disc. Proteolytic enzymes called matrix metalloproteinases reside in the disc and have been implicated in disc degeneration[4,10,12,17,18] . The matrix metalloproteinases are regulated by specific inhibitors (TMVS), cytokines (Interleukin-1) and growth factors.

Elevated intradiscal pressure can interfere with diffusion by reducing the gradient. As disc metabolism becomes more anaerobic, there is an accumulation of lactic acid, loss of chondrocyte and fibroblast function, and activation of the metalloproteinases. It is presumable that VAX-D, by reducing intradiscal pressure, may have some effect on this biochemical chain of events.

Clinically, patients who respond to VAX-D respond in a slow progressive manner and may continue to respond after finishing their course of VAX-D therapy. It has been shown experimentally that elevated lactate levels in the disc prohibit disc proteoglycan synthesis.[8,15] This can be partially reversed by aerobic conditioning of the disc. Destruction of the proteoglycan matrix leads to disc degeneration. The disc cannot herniate with compressive forces alone in the absence of disc degeneration[2].

Once the proteoglycan matrix is compromised, compressive forces are transferred to the annulus and facet joints, resulting in anular failure and facet arthropathy. Changes in T2 imaging suggesting rehydration of the disc have been noticed after VAX-D, suggesting an effect on proteoglycan synthesis but formal studies are absent.

Those of us familiar with both the McKenzie assessment and treatment protocol and VAX-D have demonstrated that some patients unsuitable for mechanical therapy respond to VAX-D therapy. Patients who partially centralize, have a short lived reduction of pain, or whose pain rapidly returns after therapeutic exercises have also benefited from the addition of VAX-D therapy.

During the early stages of VAX-D therapy, especially if the patient continues to express symptoms, we avoid loading the disc. Our experience has been that besides exacerbating symptoms, some patients may become more refractory to treatment. With stabilization of symptoms we may attempt to gently load the disc. In the majority of cases repetitive flexion tends to reproduce their symptoms and extension provides relief. At this point patients may begin therapeutic exercises. This usually occurs in the latter third or fourth week of VAX-D therapy.

To summarize, I now believe we have the tools to complete the cycle that defines conservative care for the patient with low back pain. We have the capabilities to arrive at an accurate diagnosis on a consistent basis. We have the know how to treat patients who are candidates for mechanical therapy. The capability to treat any patient with low back pain by mechanical therapy (i.e. McKenzie method) is our ultimate goal since that patient can then treat themself. Lastly, I believe we have found the missing link (VAX-D) that allows us to treat patients who are not candidates for mechanical therapy and convert them to a patient who may be treated mechanically.

References

I . Andersson G, et al. Intervertebral Disc Pressures During Traction. Scand J. Rehab Suppl 1983;9:88-91
2. Bogduk, N. Clinical Anatomy of the Spine. Churchill Livingston
3. Donelson R, Aprill C, Mcdcalf R, Grant W. A Prospective Study of Centralization of Lumbar and Referred Pain. Spine 1997;22-.1115-1122
4. Fujita K, Nakagawa T, Mrabayashi K, Nagai Y. Neutral Proteinases in Human Intervertebral Disc. Role in Degeneration and Probable Origin. Spine 1993-118:1766-1773
5. Gose E, Naguszewski P, Naguszewski W. Vertebral Axial Decompression Therapy for Pain Associated with Hemaited or Degenerated Discs or Facet Syndrome: An Outcome Study. Neurological Research 1998;20:186-190
6. Hirsch C. An Attempt to Diagnose the Level of Disc Lesion Clinically by Disc Puncture. Acta Orthop Scan 1948; 1 8:132-140
7. Holm S, Maroudas J, Uraban PG, Selstram G. Nachemson A. Nutrition of **the** Intervertebral Disc: Solute Transport and Metabolism. Connective Tissue Research 198 1;8:101-119
8. Holm S, Nachemson A. Variations in the Nutrition of the Canine Intervertebral Disc Induced by Motion. Spine 1983;8:866-874
9. Kuslich S, et al. The Tissue Origin of Low Back Pain and Sciatica: A Report of Pain Response to Tissue Stimulation During Operations on the Lumbar Spine Using Local Anesthesia. Ortho Clinics North America 1991;22:181
10. Matsui Y, Maeda Nt Nakagami W, lwata H. The Involvement of Matrix MetaHoproteinases and Inflanunation in Lumbar Disc Herniation. Spine 1998;23:863-69
11. McKenzie R. The Lumbar Spine. Mechanical Diagnosis and Therapy. Spinal Publications 1991
12. Mooney V. Presidential Address International Society for the Study of the Lumbar Spine, Dallas, 1986. Where is the Pain Coming From? Spine 1987; 12:754-759
13. Nachemson A. The Lumbar Spine. An Orthopaedic Challenge.Spine 1976; 1: 59
14. Nachemson A, Elfstrom G. Intravital Dynamic Pressure Measurements in Lumbar Discs. Scand J. Rehabil Med (Supp) 1970; 1: 440
15. Ohshinia H, Urban JPG. The Effect of Lactate and pH on Proteoglycan and Protein Synthesis Rates in the Intervertebral Disc. Spine 1992; 17:1079-1082
16. Ramos G, Martin W. Effects of Vertebral Axial Decompression on Intradiscal Pressure. J Neurosurg 1994;81:350-353
17. Sedowofia K, Tomlinson 1, Weiss J, Hilton R, Jayson M. Collagenolytic Enzyme Systems in Human Intervertebral Disc. Their Control, Mechanism, and Their Possible Role in the mediation of Biomechanical Failure. Spine 1982;7:213-222
18. Smyth MJ, Wright V. Sciatica and the Intervertebral Disc. An Experimental Study. J Bone Joint Surg (Am) 1958;40:1401-1418
19. Tilaro F. An Overview of Vertebral Axial Decompression. Can J Clin Med 1998;5:2-8
20. Tilaro F, Miskovich D. The Effects of Vertebral Axial Decompression on Sensory Nerve Dysftuiction. Can J of Clinical Med 1999;6:2-7