AN OVERVIEW OF VERTEBRAL AXIAL DECOMPRESSION

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Introduction

Low back pain is a growing epidemic among industrialized societies. In the United States it is the most common work related disorder. The cost to industry is staggering, with estimates running 20 billion dollars or more annually.[4,42] Total payments for a single Workman's Compensation claim may be as high as $100,000.

Abenhain and Suissa studied the l-year incidence of work related low back pain in the province of Quebec for the year 1981.[1] Work absence due to back pain has an incidence of 1.4%. Seventy-four percent of work related injuries return to work within l month. 7.4% were out of work for more than 6 months. 75% of the direct total cost was borne by 10% of the absentees. Recurrence rates were 20% at l year and 36% after 3 years. Men had higher recurrence rates than women; drivers and nurses had higher recurrence rates than other occupations.

The recovery rate of the Quebec workers is similar to other countries. After l year 4.3% remained absent from work. Incidence rates of compensated back injuries by industrial sector showed that foresters and miners are at the top with 4.9% and 3.3% respectively.

Back Pain - A Diagnostic and Therapeutic Dilemma

Effective diagnosis and therapy requires thorough knowledge of spinal biomechanics. Our approach to back pain has been centered on a patho-anatomical model but unfortunately the model frequently fails to comply with the clinical picture. The Quebec Task Force Report stated: "There is so much variability in making a diagnosis that this initial step (i.e. clinical assessment) routinely introduces inaccuracies which are then further confounded with each succeeding step in care."[43] Adding to the confusion is the belief by too many physicians, patients and insurers that high tech imaging is the standard for establishing a diagnosis. However, the high rates of false positive and false negative findings point to the inadequacies of these studies in identifying the pain generating lesions[8,19,20,48,49]. Nachemson states: "A confirmatory imaging study is indicated only if surgery is contemplated. Clinical symptoms and findings remain the most important basis for diagnosis."[28]

The natural history of low back pain with and without radiculopathy has been described.[11,37,47] Spontaneous regression takes place in 80% to 90% of patients with low back pain by 6 weeks and a significant percentage of patients with sciatica report a satisfactory response to conservative medical management.

Studies on disc surgery emphasize inappropriate patient selection as the cause for surgical failure.[11,16,30,44] In Kramer's address to the International Spine Society he emphasized that the surgical failed back syndrome is the worst possible scenario a spine surgeon faces.([22] In North America the incidence for this iatrogenic disease is about 15%, compared to 5% with most European countries.(28) Comparisons between the United States and Europe indicate that the frequency of surgery in the U.S. is four times greater.[11] Statistics from the Back Pain Outcome Assessment Team compiled from 1979 to 1987 indicate a rapidly growing number of disc excision and fusion operations performed each year, further escalating the cost.[11,44]

Studies of the various surgical procedures largely lack validity and controlled prospective studies are rare .[7] A randomized study by Revel demonstrated percutaneous discectomy has little value[32] and the same is true for laser discectomy.

Chemonucleolysis is superior to saline injection but inferior to surgical discectomy. While chemonucleolysis had its followers for a period f time, it has fallen into disrepute because of the serious side effects including anaphylaxis and myelitis and should no longer be considered an option. There are not any studies demonstrating the superiority of one particular surgical intervention and there is no support for adding a fusion to a routine discectomy. [11,27,28]

The VAX-D Therapeutic Table

The VAX-D therapeutic table (Vertebral Axial Decompression) addresses the functional and mechanical aspects of discogenic pain and disease. The table was invented by Dr. Allan Dyer, former Deputy Minister of Health from Ontario and a pioneer in the development of the external cardiac defibrillator.

The table is designed to apply distraction tension to the patients lumbar spine without eliciting reflex paravertebral muscle contractions. The patient lies in a prone position, the upper body is over the stationary portion of the table, and the body is restrained by the patient holding on to adjustable handgrips which can be released at anytime for safety.

The table is a split table design, whereby distraction tensions are applied to the patient through a pelvic harness attached to a tensionometer and by separation of the movable part of the table. The distraction-relaxation cycles are automated or variably timed.

Distraction tensions and rates are continuously monitored and measured by the tensionometer and the output is shown on a digital gauge and captured on a pen-write printout. The table exerts its effects through decompression of the intervertebral discs.

Dr.'s G. Ramos and W. Martin of the Departments of Neurosurgery and Radiology at the HCA Rio Grande Regional Hospital, McAllen, Texas studied intradiscal pressure during VAX-D therapy.[30]

The patient population was comprised of individuals with unresolved low back pain who were referred for neurosurgical consultation. Previous management programs included conventional bedrest, medications, physical therapy, and or chiropractic treatments.

Depending on the diagnosis and findings of the examinations, patients were assigned to one of the following study groups: Intradiscal Pressure Study and Clinical Outcome Assessment Study. Patients with a subligamentous herniation at L4-5 who were candidates for percutaneous discectomy were included in a study of intradiscal pressure manometry. The pressure measurements were recorded by two different methods; an Ohmeda pressure transducer connected to a Hewlett Packer pressure monitor via a saline bridge and a Camino fiberoptic intracranial transducer adapted for intradiscal measurements. Both transducers were recalibrated after each procedure utilizing a Pneumatic Calibration Analyzer.

The transducers were Placed in the L4-5 disc under A-P and lateral fluoroscopy. With the catheter in place the patient was placed prone on the VAX-D table. Various decompression tensions from 50 to 100 pounds were applied. The distraction tensions and the resulting changes in intradiscal pressure were observed on digital readout and recorded on a graph tracing produced by the chart recorder.

Intradiscal pressures were significantly reduced to minus 150-160 mm Hg. It was observed that a threshold distraction tension was necessary to develop negative pressures in the disc. The extent of decompression measured in mm Hg follows an inverse relationship to the tensions applied.

The significance of this study cannot be overemphasized. The reduction of intradiscal pressure to negative levels has far reaching therapeutic implications. Prior to the introduction of VAX-D, a non surgical method for disc decompression was unavailable. In numerous studies conventional traction has never demonstrated a reduction of intradiscal pressure to negative ranges, on the contrary many traction devices actually increased intradiscal pressure most likely secondary to reflex muscle spasm .[5]

Indications and General use of the VAX-D Therapy Table VAX-D is indicated for patients with low back pain that has been unresponsive to conventional therapy for 6-8 weeks. Patients with radiculopathies are also candidates. The presence of a neurological deficit does not affect patient eligibility since studies have revealed the outcome in patients with neurological deficits was not affected by surgical or medical management [15] The presence of a rapidly progressive neurological deficit is an indication for surgery. Patients presenting with a fusion and the post surgical failed back syndrome may also be candidates.

Contraindications for VAX-D therapy include infection, neoplasm, osteoporosis bilateral pars detect or Grade 2 spondylolisthesis if unstable, fractures, the presence of surgical hardware in the spine, and the cauda equina syndrome. Patients with lateral stenosis and central stenosis may respond if severe secondary changes are not present in the vertebra. The patient should be evaluated by a therapist or physician prior to initiating therapy and routine spine films are necessary to rule out any contraindications. A CT scan or MRI is not necessarily a prerequisite before therapy.

The daily therapy sessions are administered by a trained VAX-D technician. All VAX-D technicians are encouraged to complete a certification exam. Treatments are administered on a daily basis for approximately twenty sessions and are routinely given Monday through Friday. An occasional patient may require a short maintenance period where 2 to 3 treatments a week are given for 2 to 4 weeks post therapy . The average patient has required 20-25 sessions. Each session is comprised of 15 cycles, each cycle being 1 minute in distraction and 1 minute in relaxation.

The table is designed to be operator friendly. With the patient standing, a specially made pelvic harness is fitted and tightened on the patient. The patient: lies prone on the table with the lower portion of the belt placed at the level of the table separation point. The adjustable handgrips are positioned such that the elbows remain straight. Repositioning and tightening of the pelvic harness is completed at this point. The harness is attached to a movable pretension housing that maintains a baseline tension of 20 lbs. throughout the rest phase. Once the pretension is set the treatment cycles may begin. The Ramos study indicated that 50 lbs. of tension was the threshold tension necessary to develop negative intradiscal pressures. The p.s.i. is slowly increased until tensions of 60-80 lbs. are developed, this may take 3 to 4 days of herapy. Some patients have required 90-100 lbs. of tension for a full therapeutic effect.

Pain distribution frequently changes during or immediately after therapy. A phenomenon called centralization first observed by McKenzie [24] has been noticed during a course of VAX-D therapy. Centralization is the process by which the pain pattern migrates from a peripheral distribution to a more central or proximal location and is an indication of a favorable clinical outcome. Centralization of pain patterns may be associated with increased central back pain, but this should be

interpreted as a positive sign and is likely secondary to stretching of the posterior longitudinal ligament as the lateral distortion of the disc retracts to a more concentric position. Centralization is a predictable prognostic indicator for symptomatic discs and annular competence[12] The observed occurrence of centralization during VAX-D therapy in a patient who initially could not centralize their pain pattern implies healing of the anulus as a result of VAX-D therapy.

As higher distraction tensions are reached few patients may report an increase in pain of a different quality. Overstretching of the soft tissues in the back likely represents the cause of this pain and the patient should be treated by decreased distraction tensions, so as not to traumatize the soft tissues.

The development of a sharp, burning, radiating pain during therapy could represent the stretching of an entrapped nerve. Since the breakdown of scar tissue is an objective, the patient should continue but distraction tensions should be reduced such that any pain elicited does not last more than 15-20 minutes post therapy. Distraction forces are then slowly increased over the ensuing days.

No serious side effects have been reported with VAX-D therapy. A limiting factor affecting the patient's tolerance to therapy is stress to the shoulder girdle and rotator cuff. This may be mitigated by placing a roll under the axilla of the affected side. Should a patient have discomfort from any cause, they may release the handgrips at any time. This adds an important safety factor to the treatment.

Mechanism of Action

An understanding of spinal biomechanics is necessary to appreciate VAX-D's mechanism of action, to effectively treat and diagnose spinal disorders, and to objectively review old and new therapies.

The literature is replete with biomechanical data. Vogel and Stahl have carried out in vitro experiments on intradiscal movements with symmetrical and asymmetrical loading.[21] With symmetrical loading the nucleus expands and is retained by the anulus. By contrast, if the disc is subjected to an asymmetrical load, the nucleus migrates to the area of least load or resistance.(38) With removal of the load the nucleus moves from an eccentric to a more concentric position within the disc. Relocation can be accelerated by compression in the opposite direction or by distraction. [21] The anulus of a normal disc can restrain the nuclear movement, but when the elastic properties of the anulus are compromised the structures become susceptible to injury. Fissures and ruptures develop which allow the nucleus to migrate.

Fissures are normally present by 30-35 years of age and increase with advancing age. Fragment sequestra appear as a result of age and trauma. These fragments can move independently and result in protrusions and disc prolapses. Migration of

nuclear material and sequestra is influenced by compressive forces, shearing, and increased intradiscal pressure.

Epidemiological data and scientific data have demonstrated that prolonged or repetitive flexion loads stress the posterior anulus resulting in discogenic pain and in some patients disc herniation.[3,25] Adams and Hutton carried out experiments with gradual loading of the disc and concluded that disc prolapses can occur with a sustained flexion load.[2] Hickey and Hukins performed experiments with bending and torsion and demonstrated that the anulus failed posteriorly.[17] Shirtzi-Adl demonstrated that disc fiber layers are most loaded in flexion and least in extension.(40) Nachemson's research on intradiscal pressures showed pressures were highest with flexion.(26) The outer third of the anulus is innervated by the sinuvertebral nerve. Any asymmetrical load associated with elevated intradiscal pressure can result in overstretching and fatigue of the anulus, thereby stimulating the mechanoreceptors in the outer third of the annular wall. Eventually fissure's will develop in the anulus which can lead to herniation Of the centra] mass of the nucleus. By reducing intradiscal pressure with VAX-D therapy, a therapeutic and prophylactic effect can be realized.

Numerous studies utilizing discography have helped us to understand the role of the disc as a pain generator. Provocational discography is the standard test for discogenic pain.[41] Its reliability has been questioned and opponents generally refer to the work of Holt, but his study has been refuted on methodological grounds.[9,18,41]

Recently a pathological marker of symptomatic disc disruption called the high intensity zone (HIZ) was demonstrated on MRI using spin echo gradient heavy T2 imaging. [6,38] An HIZ is evident in the posterolateral view on the sagittal section, which on provocation discography corresponded to a Grade 3 radial tear. The high signal intensity represents fluid within the fissure that may be causing pain either by chemical irritation or mechanical traction of the sinuvertebral nerve. By its cyclic action and ability to reduce intranegative pressure, VAX-D therapy could displace the fluid to the internal portion of the nucleus thereby ameliorating pain and enhancing healing of the anulus.

Donelson demonstrated it is possible to predict anular competence with the McKenzie mechanical assessment protocol.[I2] In his study patients were separated into centralizer's and non centralizer's. Discography was performed in both groups. Centralizer's tended to have an intact anulus or Grade 1-2 tears. Non centralizer's had a disrupted anulus, that is fissures to the outer third of the anular wall or Grade 3 tear. This is very exciting news for those who appreciate the centralization phenomenon because it allows us to clinically assess the competency of the anulus. Patients who centralize on initial evaluation may be treated with specific exercise. Patients who do not centralize on initial

examination are excellent candidates for VAX-D therapy. With such an approach, the patients disposition regarding effective therapy is known immediately, which arguably translates to reduced disability and reduced cost.

VAX-D therapy has been shown to convert non centralizer's to centralizer's during or after successful VAX-D therapy. This implies VAX-D is condusive to anular healing.

Asymmetrical loading of the disc and increased intradiscal pressure is partly responsible for internal derangement, disc degeneration and herniation. Changes in intradiscal pressure also play a prominent role in affecting nourishment of the disc since the disc is an avascular structure and receives its nourishment primarily by diffusion.

Intradiscal pressure that is greater than capillary pressure in the vertebral body impedes oxygen diffusion to the disc which in turn impedes healing.[I3] Reducing intradiscal pressure with VAX-D creates a dffusion gradient into the disc allowing nourishment to proceed. Solutes such as oxygen have a steep concentration gradient across the disc, with the peripheral concentration 20-30 times more than the concentration at the center of the nucleus. The availability of oxygen may be inadequate to meet the metabolic requirements required to heal a damaged anulus, and higher concentrations of lactate have been measured within the central portion of the disc. By reducing intradiscal pressure, VAX-D therapy creates a diffusion gradient thereby enhancing solute transfer. High levels of lactate could facilitate chondrocyte cell death as well as increase the activity of degradative enzymes further promoting the loss of the proteoglycan cell matrix. A vicious cycle is produced, accelerating disc degeneration. The mechanical effects of fluid loss during a compressive load are followed by a slow rate of disc deformation termed creep. The rate of creep is faster in a damaged or degenerated disc than a normal disc. Both vibration (overstress) and inactivity (understress) affect the rate of creep and disc degeneration. Pigs who were subjected to vibratory creep had lower levels of oxygen and sulfate transport, and higher levels of lactate within the disc.[13] Somewhere between the overstress of vibration and the understress resulting from inactivity is an optimum mechanical environment. Through its action, VAX-D may be capable of restoring that environment, enhancing healing of the disc and retarding degeneration.

The pathophysiology of nerve root compression has been described by Rydevik [33,34,35] The nerve root ganglion has an extensive venous plexus, which if obstructed, results in venous hypertension and endoneural edema, leading to hypoxia, ischemia and pain. External decompression with VAX-D therapy can be expected to relieve venous hypertension and reverse the pathognomonic process.

By significantly reducing intradiscal pressure, VAX-D promotes retraction of the herniation into the disc. VAX-D therapy could possibly shear a herniation from its connection to the central nucleus, creating a severed fragment within the spinal

canal. This sequestered disc is susceptible to small vessel invasion and digestion as a result of contact with the epidural space. Reinforcing this theme is the study by Modic et al who studied the natural history of disc herniation by MRI in patients with acute radiculopathy and discovered that large (6 mm) sequestered hernias were the first to undergo spontaneous resolution.[23]

I have studied sensory nerve dysfunction measured before and after VAX-D therapy, in order to determine the effect of VAX-D on nerve root compression. The results from this study are very significant and the data will be published in the future.

Inflammation very likely plays a role in disc pathology and herniation but the response to anti-inflammatories is rather disappointing. Saal found high levels of Phospholipase A2 in human disc samples removed at surgery in patients with radiculopathy.[36] As the enzyme responsible for liberation of arachidonic acid from cell membranes, Phospholipase A2 is the rate limiting step in the production of prostaglandin's and leukotrienes. Controlled studies have shown that anti-inflammatories are not useful for acute sciatica,[46] but since solute transfer to the disc may be enhanced by VAX-D therapy, the administration of anti-inflammatories during VAX-D therapy may result in higher concentrations of the drug within the disc, neutralizing inflammatory mediators responsible for nerve root inflammation and some forms of discogenic pain. As previously mentioned the fluid within the HIZ is thought to be an inflammatory fluid and VAX-D may be able to effectively pump this fluid into the central nucleus where it is not possible for it to exert an inflammatory effect. The fissure may be able to approximate its borders and heal once the fluid is pumped out.

VAX-D vs. Traction

The VAX-D table is an external decompression device and this separates it from conventional traction. Studies verifying decompression of the disc and nerve root are now available for VAX-D. I reviewed the literature and I could not find any available data that conventional traction reduced intradiscal pressure to the negative range nor are there any studies on conventional traction showing beneficial effects in nerve root compression and conditions associated with discogenic dysfunction.

Many patients who receive VAX-D therapy have had chronic back pain and failed numerous modalities including traction. Their positive response to VAX-D therapy, after having failed conventional therapy and traction confirms VAX- D's assertion that it is not conventional traction.

Clinical Studies The Acute Low Back Distress Study was conducted by the John P. Robarts Research Institute, London Ontario. The efficacy of VAX-D therapy was established with this study. The parameters measured were severity and duration of pain and disability, including analgesic requirements, and the presence and

degree of neurological involvement. One hundred and ten patients were entered into the study.

The treatment was considered a success if the baseline aggregate score for pain and disability was reduced by 50% after 10 treatments of VAX-D therapy. Sixty-six percent of the patients achieved success according to the study protocol. Prior to therapy the aggregate score for pain and disability was 5.1 and after 10 treatment sessions in the successful group it was 1.2.

The Clinical Outcome Assessment Study was conducted at McAllen HCA Hospital by Dr. G Ramos.[3] Fifty-two patients completed VAX-D therapy as the primary modality. Thirty-eight patients (73%) achieved a positive outcome with remission of their low back pain symptoms and a return to functional levels of activity. Ninety percent of the recovered group were suffering from disc herniations, the majority (89%) being subligamentous while 11% had extruded herniations. Neurological deficits did not compromise the response to therapy.

Review of the patients clinical findings for those who achieved remission showed that 33% exhibited neurological delicits and 73% had sciatic pain prior to therapy with VAX-D.

Dr. E. Gose, Dr. W Naguszewski, and Dr. R Naguszewski have completed an outcome study of VAX-D therapy from over twenty medical centers that included over 700 patients. Patients with back pain, with or without leg pain were included in the study as well as the failed surgical back patient. All patients had a diagnosis of a herniated disc, degenerative disc or facet syndrome. The authors are very enthusiastic about the outcome and have prepared a detailed report of their findings which been accepted for publication in another respected medical journal.

An outcome study at Columbia hospital, Tulsa OK is currently being conducted that shows a level of success consistent with the above study.

Summary

VAX-D therapy addresses the biomechanical aspects of discogenic disease and achieves its objective through decompression. It should be utilized in patients with low back pain, with or without radiculopathy who have failed conventional therapy (physiotherapy and chiropractic), and should be utilized prior to addressing surgery. By addressing the altered biomechanics responsible for disc disease, the VAX-D therapeutic table not only alleviates pain but has been shown to exert a beneficial effect on a major determinant in the equation responsible for discogenic disease, that is elevated intradiscal pressure.

Further analysis of future and unpublished research should be considered to further validate the therapeutic benefit of VAX-D therapy, however, these clinical

studies have shown it to be effective in back pain syndromes with or without radiculopathy including herniated discs and internal disc disruption.

The chronic back pain patients and surgical patients are very costly to society. Since many of these patients are responsive to VAX-D therapy, this unique non-obtrusive means of managing the common forms of debilitating low back pain associated with discogenic disease could represent a considerable savings.

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REFERENCES

1. Abenhain L, Suisse S. Rossignol M. Risk of recurrence of occupational back pain over three year follow-up. British J Ind Med 1988; 45:829-33.

2. Adams A, Hutton WC. Gradual disc prolapse. Spine 1985; 10:524-31.

3. Adams M, Green T, Dolan P. The strength in anterior bending of lumbar intervertebral discs. Spine 1994; 19:2197-2203

4. Akeson WH, Murphy RW. Low back pain. Clin Ortho 1977; 129:2-3.

5. Andersson GB, Schults AS, Nachemson AL. Intervertebral disc pressures during traction. Scand J Rehabil Med 1983; 9:88-91.

6. Aprill C, Bogduk N. High intensity zone: a diagnostic sign of painful lumbar discs on magnetic resonance imaging. The British Journal of Radiology 1992; 65:361-369.

7. Bloch R, 7. Bloch R, Davis DO, Dina TS. Methodology in clinical back pain trials. Spine 1987; 12(5): 430-08.

8. Boden SD, Patronas NJ, Weisel SW. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg {Am} 1990. 72:403-08.

9. Bogduk N, Modic MT. Controversy: lumbar discography. Spine 1996.21 :402-04

10. Bush K, Cown N, Katz DE, Gishen P. The natural history of sciatica associated with disc pathology. A prospective study with clinical and independent radiologic follow-up. Spine 1992; 17(10):1205-12.

11. Deyo RA, Cherkin DC, Loeser JD, Bigos SJ, Ciol MA. Morbidity and mortality in association with operations on the lumbar spine, The influence of age, diagnosis, and procedure. J Bone Joint Surg {AM} 1992; 74 (4):53643.

12. Donelson R. Aprille C, Medcalf R, Grant W. A prospective study of centralization of lumbar and referred pain. A predictor of symptomatic discs and annular competence. Spine 1997; 22(10):1115-1122.

13. Frymoyer JW. The adult spine. Principles and practice. Raven Press, New York 1991.

14. Gray ICM, Main CJ, Waddell G. Psychological assessment in general orthopaedic practice. Clin Orthop 1985; 194:258-63.

15. Hakelius A. Prognosis in sciatica. Acta Orthop Scand (suppl) 1970. 129: 1 76.

16. Herron LD, Turner T. Patient selection for lumbar laminectomy and discectomy with a revised objective rating. Clin Orthop 185; 199: 145-52.

17. Hickey D, Hukins D. Relation between the structure of the annulus fibrosis and the function and failure of the intervertebral disc. Spine 1985; 5: 106114.

18. Holt EP The question of lumbar discography. J Bone Joint Surg 1968; 50A:720-5.

19.Jensen MC, Brant-Zawadski, Obuchowski N, Modic MT, Malksalan D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl JMed 1994.331 :69-73.

20. Komberg M. Discography and magnetic resonance imaging in the diagnosis of lumbar disc disruption. Spine 1988; 14:908-18.

21. Kramer J. Intervertebral disk diseases. Causes, diagnosis, treatment and prophylaxis. George Theime Verlag Stuttgart, New York, 1990.

22. Kramer J. Presidential address: natural course and prognosis of intervertebral disc diseases. Spine 1995; 20(6):635-39.

23. Modic M, Ross J, Obuchowski N, Browing K, Cianflocco AJ, Mazanac D. Contrast enhanced magnetic resonance imaging in acute

lumbar radiculopathy: a pilot study of the natural history. Radiology 1995. 195(2):429-35.

24.McKenzie RA. The lumbar spine: Mechanical diagnosis and therapy. Waikane, New Zealand: Spinal Publications, 1981.

25. McNally DS, Adams MA, Goodship AE. Can intervertebral disc prolapse be predicted by disc mechanics. Spine 1993. 18: 1525- 1530.

26. Nachemson AL. The influence of spinal movements on the lumbar intradiscal pressure and on the tensile stresses in the annulus fibrosis.
Acta Ortho Scand. 1963; 33:183.

27. Nachemson AL. Fusion for low back pain and sciatica. Acta Orthop Scand 1985; 56(4):285-86.

28. Nachemson AL. Lumbar disc herniation-conclusions. Acta Orthop Scand (Suppl 251)1993;64.

29. North RB, Campbell JN, James CS, Conover-Walker, Wang H, Paintadosi S, Rybock JD, Long DM. Failed back surgery syndrome:
5-year follow up in 102 patients undergoing repeated operation. Neurosurgery 1991; 28(5):68591.

30. Ramos G, Martin W. Effects of vertebral axial decompression on intradiscal pressure, JNeurosurg 1994; 81:350-353.

31. Ramos G. Personal communication.

32. Revel M Payan C, Vallee C, Laredo JD, Lassae B. Roux C, Carter Salomon C... Delmas E, Roucoules J. Beauvais C, Savy JM
Chiecheportiche V, Bourgeois P, Smadja M, Hercot O, Wybier M, Cagan GM, Blum-Boisgard C, Fermanian J. Automated percutaneous
discectomy versus chemonucleolysis in the treatment of sciatica. A randomized multicenter trial. Spine 1993; 18(1):1-7. 33.

33. Rydevik B, Brown MD, Llundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984; 9:7-15.

34. Rydevik B, Myers R, Powell HC. Pressure increase in dorsal root ganglion following mechanical compression. Closed compartment
syndrome in nerve roots. Spine 1989; 14:574-76.

35. Rydevik B, Holms S, Brown NM. Nutrition of spinal nerve root. Trans Orthop Res Soc 1984; 9:276.

36. Saal JS, Franson R, Dobrow R, Saal JA, White A, Goldwaite N. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. Spine 1990; 15(7):674-78.

37. Saal JA, Saal JS, Herzog R. The natural history of lumbar intervertebral disc extrusions treated non operatively. Spine 1990; 15(7):683-86.

38. Schellhas K, Pollei SS, Gundry C, Heithoff K, Lumbar disc high-intensity zone. Correlation of magnetic resonance imaging and discography. Spine 1996; 21:79-86.

39. Schnebel B, Simmons J, Chowing J, Davidson R. A digitizing technique for the study of movements of intradiscal dye in response to flexion and extension of the lumbar spine. Spine 1988; 13:309-12.

40. Shiratzi-Adl A. Biomechanics of the lumbar spine in sagittal /lateral moments. Spine 1994; 19:2407-12.

41. Simmons JW, Aprille CN, Dwyer AP, Brodsky AE. A reassessment of Holt's data on the "question of discography." Clin Orthop 1988; 237:1204.

42. Spengler D, Bigos S, Martin N, Seh J. Fisher L, Nachemson A. Back injuries in industry: A retrospective study. Overview and cost analysis. Spine 1986; 11:241-45.

43. Spitzer WO, Leblanc FE, Dupuis M. Scientific approach to the assessment and management of activity-related spinal disorders. Spine 1987; 12:7S.

44. Turner JA, Ersek M, Herron L, Deyo R. Surgery for lumbar spinal stenosis, attempted meta-analysis of the literature. Spine 1992; 17(1): 1-8.

45. Vanharanta H, Sachs BJ, Spivey MA, Guyer RD, Hochshuler SH, Rashbaum RF, Johnson RG, Ohnmeiss D, Mooney V. The relationship of pain provocation to lumbar disc deterioration as seen by CT/discography. Spine 1987; 12(3):295-98.

46. Weber H, Holme I, Amlie E. The natural course of acute sciatica, With nerve root symptoms in a double blind placebo controlled trial evaluating the etfect of piroxicam (NSAID). Spine 1993; 18: 1433-8.

47. Weber H. Spine update. The natural history of disc herniation and the influence of intervention. Spine 1994. 19(19):2234-38.

48. Weisel SW, Tsourmas, Fefer HL, Citrin CM, Patronas N. A study of computer assisted tomography: The incidence of positive CAT
scans in an asymptomatic population. Spine 9:549-51.

49. Zuckerman J. Derby R. Hsu K et al. Normal magnetic imaging with abnormal discography. Spine 1988; 13:1355-9. J Neurosurg
81:350-353, 1994