

Vertebral axial decompression therapy for pain associated with herniated or degenerated discs or facet syndrome: An outcome study

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The outcomes of vertebral axial decompression (DECOMPRESSION) therapy for patients with low back pain from various causes are reported. Data was collected from twenty-two medical centers for patients who received DECOMPRESSION therapy for low back pain, which was sometimes accompanied by referred leg pain. Only patients who received at least ten sessions and had a diagnosis of herniated disc, degenerative disc, or facet syndrome, which were confirmed by diagnostic imaging, were included in this study; a total of 778 cases. The average time between the initial onset of symptoms and the beginning of this therapy was 40 months, and it was four months or more in 83% of the cases. The data contained the patients' quantitative assessments of their own pain, mobility, and ability to carry out the usual 'activities of daily living'. The treatment was successful in 71% of the 778 cases, when success was defined as a reduction in pain to 0 or 1, on a 0 to 5 scale. Improvements in mobility and activities of daily living correlated strongly with pain reduction. The causes of back pain and their relationship to this therapy are also discussed. [Neurol Res 1998; 20: 186-190].

Keywords: Low back pain; herniated disc

INTRODUCTION

For most patients, the cause or causes of persistent low back pain remains poorly understood. Although imaging procedures, including CT and MRI, are able to accurately define structural pathology, the correlation of these anatomic findings with physiology, back pain, and other clinical complaints is imprecise¹. Although surgical decompression, epidural blocks, and spinal instrumentation can sometimes help patients suffering from back pain, these treatments do not completely take the biomechanical function of the disc into account, and may leave patients unrelieved of their suffering. In addressing the dysfunction of the disc with discectomy or surgical instrumentation, the biomechanical and physiological function of the disc is permanently disrupted. Mechanical low back pain is usually aggravated by activities that increase axial loading on the spine, such as sitting, standing, and lifting. Patients may describe

some relief with walking, but more particularly, by lying down, which unloads the spine and reduces intradiscal pressure (2,3). The causes of mechanical low back pain may include degenerative disc disease, degenerative spondylosis with limitation of range of motion, facet arthropathy, relative lateral recess stenosis from a combination of the above, microenvironment pressure changes affecting the thecal and epidural space from disc bulging, subligamentous and/or extruded herniation, and segmental instability.

Pain generation from degenerative disc disease is probably multifactorial. A number of potential mechanisms are specifically addressed by the lumbar vertebral body separation achieved during therapy. With aging, disc desiccation occurs, disc height is lost, and this process is accelerated with activities which produce high physical loading of the lumbar spine (4). Osteophytes develop along the anterolateral and posterior border of the vertebral bodies, and facet arthropathy increases as degenerative disc change advances (5). Normal vertebral body separation is lost as the disc degenerates. Redundancy of the posterior longitudinal ligament and ligamentum flavum combine with osteophyte encroachment upon the neuroforamen or central canal, resulting in stenosis at these sites, which is increased by axial loading of the spine.

The blood supply to the nerve roots of the cauda equina is sensitive to compression. Even at pressures of only 5-10 mmHg, the flow in over 20% of the venules was completely stopped (6). Flow in all the capillaries stopped at pressures between 20 and 50 mmHg. A pressure of 30 mmHg is slightly less than one pound per square inch, so solute transport is easily reduced. Even vertebral distractions (increased separation) of 1 or 2 mm per disc would reduce ligamental redundancy and help to restore canal/foraminal patency, reduce venous congestion and increase axoplasmic flow. Furthermore, the effects of lumbar spine lengthening may be sustained for a period of time after lumbar distraction has been stopped.



Figure 1: Patient undergoing treatment on the DECOMPRESSION Therapy Table

Twomey (7) placed lumbar vertebral columns removed from 23 male cadavers under 9 Kg of sustained traction for 30 min and measured an average increase in length of 9 mm. Thirty minutes after traction was removed, 13 of the 23 specimens had returned to baseline length, but the remaining 10 spines showed residual elongations ranging from 0.3 mm to 4 mm. Additionally, the data suggested that sustained traction had had a longer lasting effect on elderly spines. The mechanism of this residual deformation was not elaborated upon by the author, but disc rehydration may have been a factor since each column was soaked in normal saline and remained saturated by periodic additions of saline to a close fitting bag surrounding each column during the study.

That lumbar traction, if adequately applied, can effect physical change in patients suffering from back pain is well described by Gupta and Ramarao (8). They used water soluble contrast medium and epidurography to study 14 patients with prolapsed intervertebral disc syndrome before and after 10 to 15 days of continuous traction. Ten patients showed definite clinical improvement, with reduction in back pain and sciatica. Nine of these patients showed complete resolution of the defect on epidurogram and one of them showed partial reduction. The authors concluded that disc protrusion may be safely treated by traction. Mathews also demonstrated the effectiveness of lumbar traction in two patients by epidurography. Disc protrusions were decreased and an average vertebral distraction of 2 mm per disc space was shown in radiography (9). Judovich found that a traction force of approximately 26% of the body weight was needed just to overcome the resistance between the lower half of the patient and a (nonsplit) table (10).

Intuitively, lumbar traction should be successful in alleviating many of the conditions which cause low back pain and associated radiculopathy.

Unfortunately, studies of clinical efficacy have yielded equivocal results.

Previously, the successful application of lumbar traction has been limited by patient tolerance and the design of mechanical devices. Patients had difficulty tolerating the forces needed to relieve pain if delivered continuously.

Furthermore, the thoracic corsets worn by patients to prevent movement on the table were uncomfortable, restricted respiration, and can compromise venous return to the heart. Technological advances have now led to the development of equipment that has been found to achieve decompression of lumbar discs without stimulating the reactive reflexes of the lumbar musculature that can otherwise overcome efforts to effectively distract vertebral bodies.

The DECOMPRESSION therapy table is shown in Figure 1. The split table design eliminates frictional resistance between the patient and the table and allows controllable effective axial distraction tensions to be applied to the lumbar vertebral column. The equipment applies distractive forces in a gradual, progressive fashion, designed to achieve distraction of the vertebral bodies without eliciting reactive reflex muscular resistance. A portion of a typical chart recording of the tensile force applied to a patient's spine as a function of time is shown in Figure 2. Each decompression phase, during which the tension is increased, normally lasts for one minute. The force is increased more slowly in

the latter part of the decompression phase. The tension is then gradually decreased, over a period of 30 sec, to about 20 pounds, which is maintained during the rest phase. Another cycle then starts. The avoidance of paravertebral muscle contraction, stimulated by homeostatic proprioceptor and axon reflex mechanisms allows the distraction of the vertebral bodies necessary to achieve decompression of the intervertebral disc. The therapy is administered via an automated logic control mechanism which systematically applies distractive tensions and rest periods in a cyclic fashion. The typical therapy session consists of 15 cycles of tension and relaxation. This periodic process allows patients to withstand stronger forces than can be tolerated when static techniques are used and it promotes accommodation and relaxation during the therapy session. The upper body is fixed by means of the patient grasping adjustable hand grips, designed to eliminate the use of a thoracic corset. Consequently, there is no risk of circulatory or respiratory compromise. The pelvis is secured with a specially designed harness that adjusts snugly and applies forces primarily to the lateral pelvic alae, thus minimizing anterior-posterior pressures and reactive muscle spasm during the distractive period of each cycle.

DECOMPRESSION treatment has been shown (11) to decompress the nucleus pulposus to pressures below - 100 mmHg. This creates a tremendous potential diffusion gradient across the disc space, which is otherwise an avascular structure. Glucose and oxygen enter the disc at the end plate region while sulphate ions needed for the production of new glycosaminoglycans enter from the annulus fibrosis (12). Thus therapy may augment nutrient flow into the disc, facilitating structural restoration of the disc and promoting disc rehydration, since proteoglycans bind water (13). These effects may be cumulative with repetitive therapy sessions.

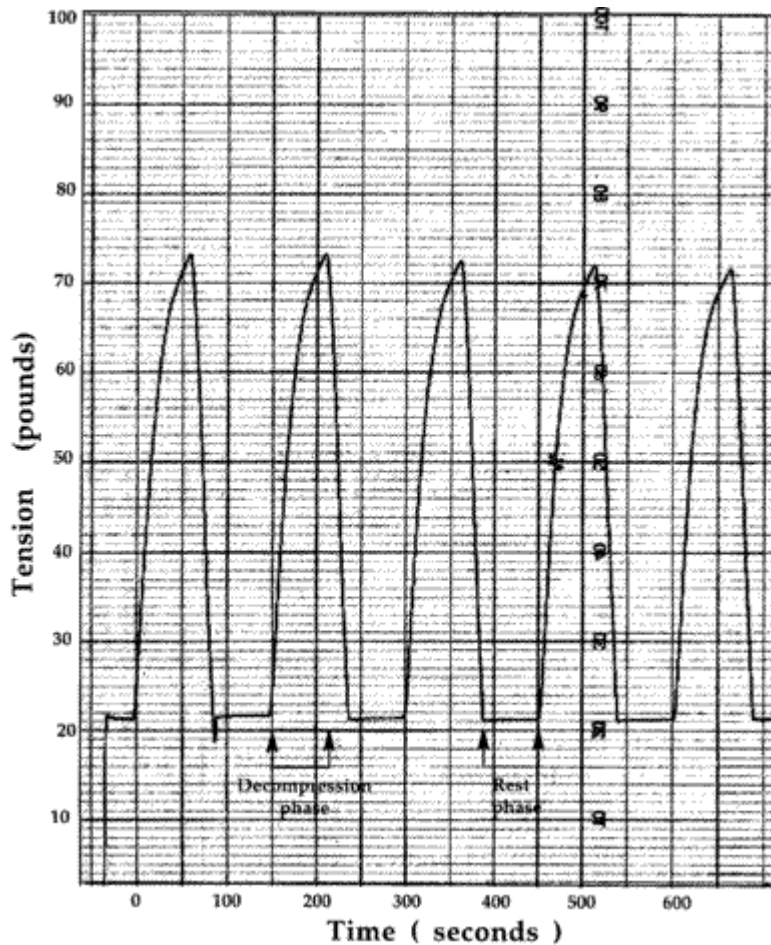


Figure 2: chart recording of tension versus time for five cycles of the typical 15-cycle DECOMPRESSION Therapy session

MATERIALS AND METHODS

Data was collected from twenty two medical centers in the USA for patients who received DECOMPRESSION therapy for low back pain. Only patients who received at least 10 treatments and had a diagnosis of herniated disc, degenerated disc, or facet syndrome, which was confirmed by imaging studies, were included in the study. The average number of treatments was 17 for facet syndrome, 19 for degenerative disc disease, and 20 for other diagnoses. The data contained the patients' assessment of their own pain, mobility, and ability to walk and sit. The pain scale ran from no pain (0) to severe pain (3). The mobility limitation scale was: No limitation (0), slightly limited (1), very limited (2), and completely immobile (3). The activity limitation scale was: walks frequently (0), walks occasionally (1), chairfast (2), and bedfast (3). The treatment schedule, including the use of other modalities, the duration and frequency of DECOMPRESSION therapy, and medication was also recorded, as well as the patient' s history. The symptoms were recorded at the beginning, mid-point, and

end of the treatment schedule. The patients' satisfaction with the treatment was quantified as: not satisfied (0), slightly satisfied (1), very satisfied (2), and completely satisfied (3).

The data were divided into five groups:

1. The first group which contained 34 cases, included all patients with extruded herniated discs, whether or not additional lesser problems were present.
2. The second group contained 195 cases of multiple herniated discs, without extrusion, with or without degenerative disc disease.
3. The third group consisted of 382 patients with a single herniated disc, regardless of degenerative disease.
4. The fourth group contained 147 cases of degenerative disc disease, without herniation.
5. The fifth group contained 19 cases with facet syndrome. Five cases of facet syndrome which had a pain reduction to 0 or 1 before 10 treatments, and one that had a reduction to 2, received less than 10 total treatments, so they were not included in the data base.

RESULTS

If treatment success is defined as a reduction in pain to 0 or 1 on a 0 to 5 scale, the treatment was successful in 71% of the 778 cases. The success rate varied from 53% for the patients with extruded herniated discs, to 73% for patients with a single herniated disc. It was 72% for people with multiple herniated discs and 68% for facet syndrome. On a pain scale of 0 to 5, the people with extruded herniated discs had an average pain of 4.16 at the beginning of treatment and an average of 1.82 after treatment, a reduction of 56%. The cases of multiple herniated discs went from 4.13 to 1.18, a reduction of 71%. The patients with a single herniation had a reduction from 4.16 to 1.09, or 71%. The degenerative disc cases reduced from 3.93 to 1.17, a 70% reduction. The patients with facet syndrome had a reduction of 4.00 to 1.13, a 72% reduction in pain. Overall, 71% of the patients experienced a reduction in pain to 0 or 1. The reduction in the average pain score was also 71%. One percent of the patients reported increased pain, 7% had no change, 92% improved by 1 unit or more, 87% improved by 2 units or more, and 70% improved by 3 units or more. A summary of these findings is shown in Table 1.

Table 2 shows how the average pain, mobility, and activity scores for the entire group of 778 patients improved during treatment. Although 51% of the pain reduction occurred during the first half of the course of treatment, 56% of the mobility improvement and 55% of the activity improvement occurred during the last half.

On a rating scale of 0 to 3, increases in spine mobility of one grade or more was seen in 77% of the patients with mobility limitations. Functional increases of 1 or more grades in the activity score was recorded in 78% of the patients who, before treatment were either unable to walk or capable of only limited walking.

The coefficient of linear correlation¹⁴ between mobility and pain scores was 0.72. Between pain and activity the correlation was 0.60, and between activity and mobility it was 0.59. On a scale of 0 to 3, the average satisfaction with treatment was 2.4, which lies between 'very satisfied' and 'completely satisfied'. In this study, 31 patients had previous lumbar disc surgery. MRI scans showed scar tissue that could potentially entrap nerve roots. Despite this, 84% of this group's pain scores and 71% of their mobility scores and 61% of their activity scores improved by one unit or more with therapy, and 65% of their pain scores were reduced to 0 or 1. Vertebral axial decompression was well tolerated.

Vertebral axial decompression therapy outcomes: Earl E. Gose et al.

Table 1: Pain outcomes for venous diagnoses

Diagnosis	No. of Cases	Pain Before treatment	Pain after treatment	% of pain reduction	% of successes
Extruded herniation	34	4.16	1.82	56	53
Multiple herniation	195	4.13	1.18	71	72
Single herniation	382	4.16	1.09	71	73
Degenerative disc disease	147	3.93	1.17	70	72
Facet syndrome	19	4.00	1.13	72	68
Average over 778 cases:	>		4.10	1.21	71

Table 2: Variation of average pain, mobility, and activity scores during treatment, and final outcome measures for the entire group

	Pain (0-5 scale)	Mobility limitation (0-3 scale)	Activity limitation (0-3 scale)
Before therapy	4.10	1.81	1.24
At midpoint	2.62	1.30	0.80
After therapy	1.21	0.64	0.27
Overall improvement	71%	65%	78%
Improved by 1 unit or more	92%	77%	63%

DISCUSSION

We consider DECOMPRESSION therapy to be a primary treatment modality for low back pain associated with lumbar disc herniation at single or multiple levels, degenerative disc disease, facet arthropathy, and decreased spine mobility.

Physiology (pain and mobility) and pathology correlate imprecisely. We believe that post-surgical patients with persistent pain or "Failed Back Syndrome" should not be considered candidates for further surgery until a reasonable trial of vertebral axial decompression has been tried.

Low back mobility increased subsequent to therapy and correlated well with pain reduction. Both of these factors are important in areas such as Workers Compensation and personal injury. Estimates of permanent partial impairment rely heavily on mobility aspects, as seen in the AMA Guides to the Evaluation of Permanent Impairment, 4th edition. Although allowance for pain is made in the percentage of impairment, the determination of impairment is made by determination of spine mobility using the range of motion model. By definition no patient can be assigned any impairment rating until maximum medical improvement (MMI) is reached. We submit that patients can usually be brought to a higher level of MMI by this therapy because of the anticipated improvements in mobility.

In summary, the pain, activity, and mobility scores were all greatly improved after therapy. DECOMPRESSION by its unique design may more precisely address the physiology of persistent low back pain than other conventional therapies. We consider it to be a front line treatment for degenerative spondylosis, facet syndrome, disc disease and nonsurgical lumbar radiculopathy.

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AN OVERVIEW OF VERTEBRAL AXIAL DECOMPRESSION

By Dr. Frank Tilaro

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 1-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 1 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 1 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 1 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.(10,37,47) 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 of 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 DECOMPRESSION Therapeutic Table

The DECOMPRESSION 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 DECOMPRESSION 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 DECOMPRESSION 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 mmHg 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 DECOMPRESSION, 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 DECOMPRESSION Therapy Table

DECOMPRESSION 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 DECOMPRESSION therapy include infection, neoplasm, osteoporosis, bilateral pars defect 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 DECOMPRESSION technician. All DECOMPRESSION 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 therapy. 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 DECOMPRESSION 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 anular competence (12) The observed occurrence of centralization during DECOMPRESSION therapy in a patient who initially could not centralize their pain pattern implies healing of the anulus as a result of DECOMPRESSION 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 DECOMPRESSION 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 DECOMPRESSION'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. (24) 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 annulus 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 annulus is innervated by the sinuvertebral nerve. Any asymmetrical load associated with elevated intradiscal pressure can result in overstretching and fatigue of the annulus, thereby stimulating the mechanoreceptors in the outer third of the annular wall. Eventually fissure' s will develop in the annulus which can lead to herniation of the central mass of the nucleus. By reducing intradiscal pressure with DECOMPRESSION 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, DECOMPRESSION therapy could displace the fluid to the internal portion of the nucleus thereby ameliorating pain and enhancing healing of the annulus.

Donelson demonstrated it is possible to predict anular competence with the McKenzie mechanical assessment protocol.(12) 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 annulus or Grade 1-2 tears. Non centralizer' s had a disrupted annulus , that is fissures to the outer third of the annular 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 annulus. Patients who centralize on initial evaluation may be treated with specific exercise. Patients who do not centralize on initial examination are excellent candidates for DECOMPRESSION therapy. With such an approach, the patients disposition regarding effective therapy is known immediately, which arguably translates to reduced disability and reduced cost. DECOMPRESSION therapy has been shown to convert non centralizer' s to centralizer' s during or after successful DECOMPRESSION therapy. This implies DECOMPRESSION is conducive to anular healing.

Asymmetrical loading of the disc and increased intradiscal pressure is partly responsible for internal derangement, discdegeneration 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.(13)

Reducing intradiscal pressure with DECOMPRESSION creates a diffusion 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, DECOMPRESSION 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, DECOMPRESSION 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 DECOMPRESSION therapy can be expected to relieve venous hypertension and reverse the pathognomonic process.

By significantly reducing intradiscal pressure, DECOMPRESSION promotes retraction of the herniation into the disc. DECOMPRESSION 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 undergo spontaneous resolution.(23)

I have studied sensory nerve dysfunction measured before and after DECOMPRESSION therapy, in order to determine the effect of DECOMPRESSION 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 leukotriene. 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 DECOMPRESSION therapy, the administration of anti-inflammatories during DECOMPRESSION 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 DECOMPRESSION 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.

DECOMPRESSION vs. Traction

The DECOMPRESSION 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 DECOMPRESSION. 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 DECOMPRESSION therapy have had chronic back pain and failed numerous modalities including traction. Their positive response to DECOMPRESSION therapy, after having failed conventional therapy and traction confirms DECOMPRESSION' 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 DECOMPRESSION 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 DECOMPRESSION 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.(31) Fifty-two patients completed DECOMPRESSION

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 deficits and 73% had sciatic pain prior to therapy with DECOMPRESSION.

Dr. E. Gose, Dr. W Naguszewski, and Dr. R Naguszewski have completed an outcome study of DECOMPRESSION 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

DECOMPRESSION 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 DECOMPRESSION 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 DECOMPRESSION 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 DECOMPRESSION 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.

About the author

Dr. Frank Tilaro is a board certified internist who has been practicing orthopaedic medicine for the last 12 years. Dr. Tilaro has restricted his practice to orthopaedic medicine. He is a member of the American Back Society, the McKenzie Institute, the American Association of Orthopaedic Medicine and the

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