J. Phys. Ther. Sci.
22: 345–354, 2010

The Efficacy of Spinal Decompression via DRX3000 Combined with a Spinal Mobilization and a Lumbar Stabilization Exercise Program for Patients with Discogenic Low Back Pain

SANG-YEOL MA, PhD, PT[1)], HYEONG-DONG KIM, PhD, PT[2)]

[1)]*Department of Physical Therapy, Sewoori Hospital*
[2)]*Department of Physical Therapy, College of Health Science, Korea University: Jeongneung 3-dong, Sungbuk-gu, Seoul, 136-703 Republic of Korea.*
TEL: +82 2-940-2830, FAX: +82 2-940-2830, E-mail: hdkimx0286@yahoo.com

Abstract. [Purpose] The purpose of this study was to determine the effects of motorized spinal decompression using the DRX3000 system (Axiom Worldwide, Tampa, FL, USA) combined with spinal mobilization as well as lumbar stabilization exercises on patients with discogenic low back pain (LBP). [Subjects] A total of 30 adults with discogenic LBP (mean age, 34.06 ± 6.41 years; age range, 28–48 years; 14 males, 16 females) volunteered to participate in this study. [Methods] A 4-week course of spinal decompression treatment combined with motorized flexion-distraction mobilization and lumbar stabilization exercises were administered to the participants for 6 days per week for the first two weeks, and four times per week for two additional weeks. The entire treatment consisted of 20 visits over a 4-week period. Comparisons of changes in the Oswestry Disability Index (ODI) and straight leg raise (SLR) test at pre-intervention, after 10 treatment sessions, and at discharge (after 20 treatment sessions) were analyzed. [Results] There were significant improvements in the outcome measures of ODI score and SLR test after 10 and 20 sessions of spinal decompression treatment combined with spinal mobilization and lumbar stabilization exercises as compared with the pre-intervention. [Conclusion] Spinal decompression treatment combined with spinal mobilization and lumbar stabilization exercises significantly improved the clinical outcome measures of ODI score and SLR test in patients with LBP secondary to intervertebral disc herniation.
Key words: Low back pain, Spinal decompression therapy, Spinal mobilization

(This article was submitted Mar. 26, 2010, and was accepted Apr. 10, 2010)

INTRODUCTION

Chronic low-back pain (LBP) is common in the general population and is considered to be a significant problem in industrialized countries[1)]. Lifetime prevalence of LBP varies ranging from 61–83% in younger age groups and 53–75% in older groups[2)]. Symptoms often recur within 1 year in 60–80% of the population, even if the original presentation resolves in most cases; some patients suffering from LBP may experience chronic, unremitting symptoms[3)] including sciatic conditions, which may exist in one quarter of those experiencing LBP[4)]. LBP is also an important economic issue and a challenge to the health care systems of developed countries, as they result in major medical costs. For example, in the United States, the cost attributed to LBP is estimated to be more than $100 billion annually[5,6].

There are many mechanical issues in the lower back which may lead to LBP, including injury to muscles and ligaments in the lumbosacral region, facet joint or sacroiliac joint arthropathy or discogenic disease due to disc degeneration[7]. Discogenic disease typically presents as pain in the lower back, buttocks, and hips and complaints of weakness, numbness or tingling, radicular pain, and claudication[8,9]. Biomechanical compromise of the lumbar motion segment caused by a degenerative change in an intervertebral disc may lead to a decrease in nucleus pulposus pressure in persons with chronic LBP[10,11]. The resulting compressive stress would be transferred from the nucleus to the posterior annulus leading to a 34% fall in nucleus pulposus pressure[12]. It has been also shown that the tensile modulus, the Poisson's ratio, failure stress, and strain energy density of the annulus fibrosus are also negatively affected by disc degeneration[13]. Thus, structural defects in the annulus fibrosus could result in a failure at lower loads and further structural disruption, and this phenomenon has been shown to lead to LBP[13]. Although the degenerative intervertebral disc problem is difficult to treat without recourse to surgery, except for the most moderate cases[14–17], due to the structural nature of the disease[18], many noninvasive treatment options such as physical therapy, exercises, medications, back school, manipulation, or acupuncture have been developed to reduce neurological symptoms as well as leg or back pain related to disc disease or to enhance disc physiology in the hopes of retarding or reversing disc degeneration[19–24].

One of the treatment options is axial traction, an attempt to relieve LBP by acting on the disc and nerve roots, which can be applied in a variety of ways. These applications include traction via a motorized pulley (motorized traction), manual traction by a therapist (manual traction), traction delivered by the patient exerting traction force via pulling or pushing on bars at the head of a table with the patient in Fowler's position on a specially designed table (autotraction), and through a suspension apparatus (gravitational traction)[25]. In all cases, with the exception of the motorized lumbar traction, it is difficult to maintain the treatment for a certain period of time due to fatigue of the patient or therapist as well as the patient's intolerance to the position and/or the force applied[26]. Therefore, the use of motorized lumbar traction by encompassing a split table to eliminate the friction force between the bed and the body has now become more common in clinical practice because of its greater success at standardization of repeatability in trials[25]. When traction is applied, pull force of traction may elicit a protective proprioceptive response resulting in contraction of the paravertebral muscles, which could reduce the traction force[26].

Recently, several axial decompression devices newly developed for non-surgical treatment of chronic (and/or acute) LBP have been introduced to the market and are being widely used in a variety of clinical settings. These lumbar traction systems include the DRX3000/DRX9000 (Axiom Worldwide, Tampa, FL, USA), the vertebral axial decompression system (VAX-D) (Vat-Tech, Inc., Palm Harbor, FL, USA), SpineMED (CERT Health Sciences, LLC, Baltimore, MD, USA), and the Accu-Spina System (North American Medical Corporation, Aventura, FL, USA). Although some randomized trials of spinal compression therapy have suggested that symptoms of chronic LBP with or without sciatic pain might be relieved by intermittent axial traction methods using systems such as the DRX9000 and VAX-D[27–30], and these treatments continue to be used in practice, there is a paucity of evidence in published data to support the efficacy of vertebral axial decompression treatment in individuals with discogenic LBP[31].

Spinal mobilization and manipulation have recently received growing acceptance as effective treatments in the field of spinal care. Spinal mobilization is defined as the application of a series of manual force to the spinal segments which are within the patient's control to stop[32]. It often involves traction through the use of specially designed treatment tables, but spinal mobilization does not use high velocity and low-amplitude manual thrusts to the spinal joints[33]. Flexion-distraction mobilization, an instrument-assisted procedure, is one of the most common forms of treatment for LBP and is used among physical therapists, chiropractors, osteopathic physicians, and medical physicians[34]. The main goal of this technique is to open the posterior aspect of the functional spinal segment to allow greater sagittal diameter within the central and intervertebral canals[35,36]. Thus, spinal mobilization or manipulation can increase the range of motion of the spinal joint, and affect modulation of sensory input to the nervous system[37,38], the motor control system, and the pain processing system[39]. Although a few studies have reported that spinal manipulation is effective in the treatment of discogenic LBP and sciatica[40–43], there are few randomized controlled studies which have evaluated the effectiveness of spinal mobilization in treatment of discogenic LBP.

Recently, there has been growing clinical use of therapeutic exercises that target the improvement of spinal and trunk stability. This type of exercise approach is known as lumbar stabilization exercise. Lumbar stabilization exercises are often used to protect affected lumbar spine structures from further injury, recurrent pain episodes, and degenerative change, as well as reduce the intensity of the pain and disability in LBP[44–48]. There is strong evidence that lumbar stabilization exercises are likely to be beneficial for pain and function in patients with recurrent and chronic LBP[49,50].

Although LBP with or without sciatica is the most frequently reported type of pain by adults in industrialized countries, its management varies widely and the efficacy of many interventions for LBP secondary to herniated nucleus pulposus (HNP) remains elusive[51]. No studies to date in the clinical literature have specifically examined the effects of the combination of spinal decompression therapy via motorized axial decompression device and spinal mobilization as well as lumbar stabilization exercises on the treatment of discogenic LBP. Therefore, the purpose of this study was to determine the effects of spinal decompression therapy using DRX3000 combined with spinal mobilization and lumbar stabilization exercises on the treatment of patients with discogenic LBP.

SUBJECTS AND METHODS

A total of 30 adults with discogenic LBP (mean age, 34.06 ± 6.41 years; age range, 23–48 years; 14 males, 16 females) volunteered to participate in this study. To be included in this study the subjects were required to meet the following criteria: 1) participants were more than 18 years of age with discogenic LBP; 2) participants had to have one of the following diagnoses: herniated disc, bulging or protruding intervertebral discs confirmed by magnetic resonance imaging (MRI) or computed tomography (CT), conventional radiograph of the lumbar spine, and clinical examination; 3) participants must have had imaging evidence of herniated disc or bulging or protruding intervertebral discs at a motion segment unit consistent with their current symptoms, since oftentimes structural imaging of herniated disc of MRI and/or CT and symptoms are poorly matched[52–54]; and 4) all participants must have reported limited activities of daily living due to LBP that had an average score greater than 30/100 on the Korean version of the Oswestry Disability Index (ODI) which is considered to be a moderate disability[55,56].

Subjects using prescription of anticoagulants, corticosteroids, or opiate-based pain medication were excluded from the study, as were those with any of the following conditions: a history of lumbar spine surgery, pregnancy, severe osteoporosis, unstable spondylolisthesis, recent lumbar vertebral compression fracture below L1, local spinal osteomyelitis, meningitis, aortic aneurysm, primary malignant or metastatic spinal neoplasm, pelvic/abdominal malignancy, local bilateral pars defects, severe peripheral neuropathy, hemiplegia, paraplegia, cognitive dysfunction, cauda equina syndrome and disc pathology with sequestration. Subjects were also excluded if they were currently receiving workers' compensation.

Participants were recruited at a regional spine care center where the current study was performed. Participants were screened to check for inclusion/exclusion criteria. Any questions regarding this study were addressed. All participants signed an informed consent form approved by the local University Institutional Review Board prior to participation. Participant characteristics, primary diagnosis and MRI findings of participants are summarized in Tables 1 and 2.

The DRX 3000 spinal decompression system consists of a split table that is used to reduce friction on the lumbar muscles and a computer programmed to provide cycling distractive forces along the axis of the lumbar spine. The DRX3000 device has built-in-air bladders and disc angle pull adjustments so that the intensity of intermittent distraction force is increased gradually to avoid muscle contractions in reaction to being stretched in the later part of spinal decompression. With proper pull angle settings to target the affected lumbar segment,

Table 1. Participants' characteristics

Characteristics	Values
Age (years)	34.06 ± 6.41
Sex (male/female)	14/16
Height (cm)	165.90 ± 7.89
Weight (kg)	62.90 ± 12.16
Side involved: left/right (%)	36/64
Location of pain	%
Pain in back/buttocks only	27
Pain below buttocks, above knee	33
Pain below knee	40
Duration of symptoms (months)	%
Less than 2	100
Previous history of LBP (% yes)	0

Note: Values are means ± SD (standard deviations); N = 30; LBP: Low Back Pain.

Table 2. Primary diagnosis and MRI findings of participants

Category	Values (%)
Primary diagnosis	
Herniated disc	43
Herniated disc and degenerative disc	57
Disc involved confirmed with MRI	
L4-L5	77
L5-S1	23
Changes in disc confirmed with MRI	
Protrusion and disc space narrowing	100

Note: MRI: magnetic resonance imaging.

traction can be applied to an isolated spinal disc slowly and cycle between brief moments of tension and relaxation (oscillation) delivered by a motor pulley.

The clinician attached a chest and shoulder harness to the upper body and then positioned the participant lying supine on the DRX3000 table with the hips and knees flexed and the lower legs supported on a stool. The pull angle setting was 15 degrees for the L4-L5 level and 10 degrees for the L5-S1 level. The initial weight setting was 25% of participant's body weight. The pulling weight was increased by 1.02 kg per session as tolerated and the final pulling weight was 50% body weight plus 4.54 kg to 9.07 kg. The distraction and relaxation times were set to 60 seconds and 30 seconds, respectively, and half of the pulling force during the distraction period was maintained during the relaxation period. Each participant was scheduled for treatment 6 days per week for the first two weeks, and four times per week for two additional weeks. The total number of visits were 20 times over a period of 4 weeks and treatment was delivered for 30 minutes in each session. Fifteen minutes of superficial heating (hot pack) were provided followed by 5 minutes of ultrasound treatment (SM-250, Samson Med, Seoul, Korea) using a frequency of 1 MHz with a 5-cm^2 sound head at an intensity of 1.5 W/cm^2 in continuous mode, and 15 minutes of interferential current treatment (SM-850P, Samson Med, Seoul, Korea) at an intensity of approximately 25 mA prior to DRX3000.

All participants also received flexion-distraction mobilization which was performed by the use of a specially designed treatment table (MF 90, Wellness System, Seoul, Korea) with a moveable headpiece, a stationary thoraco-lumbar piece, and a moveable lower extremity segment which allows a clinician to manually move the patient through passive spinal flexion and extension. The clinician placed the participant prone on the treatment table, and then attached the ankles to the treatment table using the cuffs that provide distractive force. The clinician standing to the participant's side placed one hand over the lumbar region at the level of interest for stabilization, applying counter-traction forces, while the other hand controlled the caudal section of the table through the desired range of motion. Treatment was delivered for 5 minutes, 6 days per week for the first two weeks, and four times per week for two additional weeks so that the entire treatment consisted of 20 visits over a 4 week period.

Lumbar stabilization exercises using a sling device (Redcord, Staubo, Norway) in order to improve the dynamic stability of the lumbar spine through retraining the co-contraction pattern of the deep trunk muscles and abdominal muscles were also carried out. During treatment, each movement pattern was precisely guided by the clinician and once successful coordination of the action of the deep trunk muscles and abdominal muscles was achieved, more demanding tasks which require the large prime movers of the trunk were gradually integrated into the program. Each participant was treated 6 days per week for the first two weeks, and four times per week for two more weeks. The entire lumbar stabilization exercise program consisted of 20 visits over a 4-week course.

Table 3. Overall mean (± SD), mean differences (± SD) from pre-intervention

Measure	PI	10 sessions after TI	Discharge
ODI score*	35.40 ± 9.60†‡	23.18 ± 12.18†¶	18.07 ± 12.02‡¶
ODI score difference from PI		–12.12 ± 1.03	–17.33 ± 1.00
SLR test (degree)*	35.23 ± 9.01†‡	57.53 ± 20.97†	64.30 ± 19.40‡
SLR test difference from PI (degree)		22.3 ± 2.55	29.07 ± 2.29

*Significant main effect for time (p<0.01). †Significant difference between pre-intervention and 10 sessions after TI (p< 0.01). ‡Significant difference between pre-intervention and discharge (p<0.01). ¶Significant difference between 10 sessions after TI and discharge (p<0.01).
Note. Values are means ± SD (standard deviations). ODI score range: 0 (none disability) to 50 (severe disability). ODI: Oswestry Disability Index; SLR: straight leg raise; PI: pre-intervention; TI: treatment initiation.

Outcome measures included straight leg raise (SLR) test of the affected side and ODI. For the SLR test, with the participant lying supine, the leg was passively raised by the clinician to the end range while the knee was maintained in extension. Measurement was conducted on the tibial crest, distal to the tibial tuberosity using an inclinometer. It has been shown that this test has good interrater reliability (intraclass correlation coefficient, 0.87–0.96)[57]. Reduced ability to manage in everyday life due to LBP was estimated by using the 60-point ODI (0 being no overall pain and 50 being most severe back pain total score)[55–57]. The ODI score was determined by the participant who checked 10 items with scores ranging from 0 (no back pain during activity) to 5 (severe pain during activity). ODI has a test-retest reliability coefficient of 0.92[58] and a validity of 0.62[58].

A one-way repeated analysis of variance (ANOVA) was used to determine whether there were any significant differences in outcome measures. Single degree of freedom mean contrasts were used to determine the source of any significant effects ($p<0.05$)[59]. The independent variable was time (pre-intervention, 10 sessions after initiation of treatment, and discharge, respectively). The dependent variables included SLR degree and ODI scores. The software package SPSS 14.0 KO (SPSS, Chicago, IL, USA) was used for statistical analyses.

RESULTS

All recruited subjects completed 20 treatment sessions of traction combined with motorized flexion-distraction mobilization and lumbar stabilization exercises. All subjects who participated in the study were included in the data analysis. Subjects reported their current symptoms of LBP for less than 2 months prior to the intervention and no adverse events were noted during the course of the study.

Comparisons of changes in the ODI scores and SLR test at pre-intervention, 10 sessions after initiation of treatment, and at discharge (within 3 days of the last visit) were analyzed for participants with discogenic LBP. There was a significant main effect of time for the ODI score ($F(2, 28) = 175.44$, $p<0.01$). Significant improvements were noted for the ODI scores at 10 sessions after initiation of treatment and at discharge as compared with the pre-intervention score ($p<0.01$). The mean ODI score at 10 sessions after initiation of treatment and at discharge were 35 % and 49 % lower than the mean pre-intervention score ($p<0.01$ respectively; Table 3). A significant improvement was also noted for the mean ODI score at discharge as compared with the mean score at 10 sessions after initiation of treatment ($p<0.05$). The mean ODI score at discharge was 22 % lower than that at 10 sessions after initiation of treatment ($p<0.01$; Table 3). There was also a significant main effect of time in the SLR test ($F(2, 28) = 136.27$, $p<0.01$). The mean SLR value at 10 sessions after initiation of treatment and at discharge were 163 % and 183 % of the mean pre-intervention value ($p<0.01$, Table 3). The mean measure at discharge was also 112 % of the mean at 10 sessions after initiation of treatment ($p<0.01$). Table 3 presents details of the outcome measures of ODI and SLR.

DISCUSSION

This study provides useful information as to how patients with discogenic LBP responded to 4 weeks of spinal decompression treatment combined with motorized flexion-distraction mobilization and lumbar stabilization exercises. Generally, participants in this study demonstrated significant improvements in ODI and SLR after 10 and 20 sessions of spinal decompression treatment via DRX3000 apparatus combined with spinal mobilization and lumbar stabilization exercises. These results are consistent with findings of previous studies[29,60,61], which reported visual analog scale and/or a disability scale improvements for patients with discogenic LBP after treatment with spinal decompression therapy combined with other treatment protocols such as heat, cold, and/or muscle stimulation.

The most common source of chronic LBP and nerve root entrapment syndromes most likely arises from damage to intervertebral discs[62], and discogenic pain may be due to progressive annular fibrosus breakdown and tearing leading to posterior herniation of the nuclear pulposus causing pain, or damage to the internal disc structure[63]. Previous studies[64,65] have demonstrated that spinal decompression decreases intradiscal pressure which, in turn, may allow herniated discs to be reoxygenated, rehydrated and renutrified, facilitating disc metabolism and restoration.

In our current study patients with discogenic LBP received a prescription of a lumbar stabilization exercise program for 4 weeks that addressed impairments of abdominal and lumbar trunk musculature. Improvements in ODI and SLR were observed after application of this treatment along with other treatment programs. This may be due to the resultant enhanced activities of abdominal and lumbar trunk musculature. Previous studies[66–72] have demonstrated that in patients with LBP there is a disruption of the structure and function of the deep trunk muscles such as the transverse abdominis (TrA) and lumbar multifidus (LM). TrA and LM contractions normally occur in an anticipatory manner prior to limb movement in healthy adults, whereas contraction of TrA and LM is delayed or attenuated in patients with LBP[67,73–77]. In addition, fatty infiltration and atrophy of Type I and II muscle fibers occur in the LM of persons with LBP[70–72,78–80]. It has been reported that lumbar stabilization exercise programs can normalize the functional and morphological changes in patients with LBP through improving the strength, endurance, and/or motor control of the abdominal and lumbar trunk musculature[81–86].

In this study, patients with discogenic LBP received flexion-distraction mobilization over a 4-week period that addressed the symptoms of LBP with associated lower extremity pain arising from lumbar disc herniation. Improvements in ODI and SLR were also noted with application of the treatment using flexion-distraction mobilization combined with other treatment protocols such as motorized traction and lumbar stabilization exercises. There are several possible explanations for the mechanical, physiologic, and anatomical effects of flexion-distraction mobilization procedures on the herniated intervertebral disc. It has been shown that flexion increases the central canal diameter and the intervertebral canal in the lumbar spine[87], as well as the size of the intervertebral foramen, up to 31% in the cervical spine[88]. Flexion-distraction manipulation has also been demonstrated to increase the central canal diameter in the cervical spine[89]. Additionally, distraction combined with flexion temporarily increased the lumbar disc spaces and facet joints[90]. Previous studies[91,92] have reported that distraction treatment manipulation can reduce disc protrusion in the lumbar and/or cervical spine. There is also evidence that flexion distraction can increase intradiscal pressure[90,91], and reduce hypertonicity of multifidi muscles that are assumed to play an important role in the maintenance of dysfunction and pain associated with the disc[93].

There were several limitations to our study. We examined a relatively small sample of patients with discogenic LBP, therefore future studies need to examine a larger subject group for better representation of the general population. Subjects were not classified based on a variety of symptoms or radiological imaging such as MRI or CT. This study sample consisted of adults with ages ranging from their 20's to their 40's; thus, the study population may not be representative of older adults who may have different characteristics of discogenic LBP. In addition, no MRI or CT imaging was taken to evaluate the changes in the herniated disc after the completion of 4 weeks of treatment.

In conclusion, spinal decompression treatment combined with motorized flexion-distraction mobilization and lumbar stabilization exercises for 4 weeks significantly improved the clinical outcome measure of ODI and SLR in patients with discogenic LBP. It is difficult to make a casual relationship between the intervention and outcomes of this study because there was no control group in this study, and improvements may have been due to placebo or spontaneous recovery. Therefore, it is necessary to conduct further investigations using randomized controlled trials using a large group of patient and MRI and/or CT imaging before and after the intervention to evaluate the changes in the herniated disc.

REFERENCES

1) Maetzel A, Li L: The economic burden of low back pain: a review of studies published between 1996 and 2001. Best Pract Res Clin Rheumatol, 2002, 16:23–30.
2) Svensson H, Andersson GBJ: The relationship of low–back pain, work history and work environment, and stress: a retrospective cross-sectional study of 38- to 64-year old women. Spine, 1989, 14: 517–522.
3) Hides JA, Richardson CA, Jull GA: Multifidus muscle recovery is not automatic after resolution of acute, first episodes low back pain. Spine, 1996, 21: 2763–2769.
4) Andersson GBJ: Epidemiologic aspects of low back pain in industry. Spine, 1981, 6: 53–60.
5) Frymoyer JW, Durett CL: The economics of spinal disorders. In: Frymoyer JW, editor. 2nd ed, The adult spine: principles and practice, Vol 1, Philadelphia: Lippincott-Raven, 1997, pp144–149.
6) Katz JN: Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am, 2006, 88 (Suppl 2): 21–24.
7) Macario A, Pergolizzi JV: Systematic literature review of spinal decompression via motorized traction for chronic discogenic low back pain. Pain Pract, 2006, 6: 171–178.
8) Goh KJ, Khalifa W, Anslow P, et al.: The clinical syndrome associated with lumbar spinal stenosis. Eur Neurol, 2004, 52: 242–249.
9) Spengler DM: Degenerative stenosis of the lumbar spine (current concepts review). J Bone Joint Surg, 1987, 69A: 305–308.
10) Sato K, Kikuchi S, Yonezawa T: In vivo intradiscal pressure measurement in healthy individuals and in patients with ongoing back problems. Spine, 1999, 24: 2468–2474.
11) Acaroglu ER, Iatridis JC, Setton LA, et al.: Degeneration and aging affect the tensile behavior of human lumbar anulus fibrosus. Spine, 1995, 20: 2690–2701.
12) Nachemson A: In vivo discometry in lumbar discs with irregular nucleograms. Some differences in stress distribution between normal and moderately degenerated discs. Acta Orthop Scand, 1965, 36: 418–434.
13) Adams MA, McNally DS, Dolan P: 'Stress' distributions inside intervertebral discs. The effects of age and degeneration. J Bone Joint Surg Br, 1996, 78: 965–972.
14) Amundsen T, Weber H, Nordal HJ, et al.: Lumbar spinal stenosis: conservative or surgical management?: A prospective 10 year study. Spine, 2000, 25: 1424–1435.
15) Benoist M: The natural history of lumbar degenerative spinal stenosis. Joint Bone Spine, 2002, 69: 450–457.
16) Herno A, Airaksinen O, Saari T, et al.: Lumbar spinal stenosis: a matched-pair study of operated and non-operated patients. Br J Neurosurg, 1996, 10:461–465.
17) Johnsson KE, Rosén I, Udén A: The natural course of lumbar spinal stenosis. Clin Orthop Relat Res, 1992, 279: 82–86.
18) Truumees E: Spinal stenosis: pathophysiology, clinical and radiologic classification. Instr Course Lect, 2005, 54: 287–302.
19) Hale ME, Dvergsten C, Gimbel J: Efficacy and safety of oxymorphone extended release in chronic low back pain. Results of a randomized, double-blind, placebo- and activecontrolled phase III study. J Pain, 2005, 6: 21–28.
20) Shen FH, Samartzis D, Andersson GB: Nonsurgical management of acute and chronic low back pain. J Am Acad Orthop Surg, 2006, 14: 477–487.
21) Long A, Donelson R, Fung T: Does it matter which exercise? A randomized control trial of exercise for low back pain. Spine, 2004, 29: 2593–2602.
22) Leibing E, Leonhardt U, Koster G, et al.: Acupuncture treatment of chronic low-back pain–a randomized, blinded, placebo-controlled trial with 9-month follow-up. Pain, 2002, 96 :189–196.
23) Gay RE, Bronfort G, Evans RL: Distraction manipulation of the lumbar spine: a review of the literature. J Manipulative Physiol Ther, 2005, 28: 266–273.
24) van der Roer N, van Tulder MW, Barendse JM, et al.: Cost-effectiveness of an intensive group training protocol compared to physiotherapy guideline care for sub-acute and chronic low back pain: design of a randomised controlled trial with an economic evaluation. BMC Musculoskelet Disord, 2004, 5: 45.
25) Harte AA, Baxter GD, Gracey JH: The efficacy of traction for back pain: a systematic review of randomized controlled trials. Arch Phys Med Rehabil, 2003, 84: 1542–1553.
26) Tekeoglu I, Adak B, Bozkurt M, et al.: Distraction of lumbar vertebrae in gravitational traction. Spine (Phila

Pa 1976), 1998, 23: 1061–1063; discussion 1064.
27) Gionis TA, Groteke E: Spinal Decompression. Orthopedic Technology Rev, 2003, 5: 36–39.
28) Gose EE, Naguszewski WK, Naguszewski RK: Vertebral axial decompression therapy for pain associated with herniated or degenerated discs or facet syndrome: an outcome study. Neurol Res, 1998, 20: 186–190.
29) Shealy CN, Koladia N, Wesemann M: Long-term effect analysis of IDD therapy in low back pian: a retrospective clinical pilot study. Am J Pain Manage, 2005, 15: 93–97.
30) Naguszewski WK, Naguszewski RK, Gose EE: Dermatomal somatosensory evoked potential demonstration of nerve root decompression after VAX-D therapy. Neurol Res, 2001, 23: 706–714.
31) Daniel DM: Non-surgical spinal decompression therapy: does the scientific literature support efficacy claims made in the advertising media? Chiropr Osteopat, 2007, 15:7.
32) Magee DJ, Zachazewski JE, Quillen WS: Pathology and intervention in musculoskeletal rehabilitation. St. Louis, Saunders, 2009, pp371.
33) Bronfort G, Haas M, Evans R, et al.: Evidence-informed management of chronic low back pain with spinal manipulation and mobilization. Spine J, 2008, 8: 213–225.
34) Christensen M, Delle Morgan D: Job analysis of chiropractic: a project report, survey analysis and summary of the practice of chiropractic within the United States. Greeley (Colo), National Board of Chiropractic Examiners, 1993, pp78.
35) Kruse RA, Imbarlina F, De Bono VF: Treatment of cervical radiculopathy with flexion distraction. J Manipulative Physiol Ther, 2001, 24: 206–209.
36) Cox J: Low back pain: mechanism, diagnois and treatment (6th ed). Baltimore, Williams & Wilkins, 1999, pp261–272.
37) Cramer GD, Tuck NR, Knudsen JT, et al.: Effects of side-posture positioning and side-posture adjusting on the lumbar zygapophysial joints as evaluated by magnetic resonance imaging: a before and after study with randomization. J Manipulative Physiol Ther, 2000, 23: 380–394.
38) Whittingham W, Nilsson N: Active range of motion in the cervical spine increases after spinal manipulation (toggle recoil). J Manipulative Physiol Ther, 2001, 24: 552–555.
39) Pickar JG. Neurophysiological effects of spinal manipulation. Spine J, 2002, 2: 357–371.
40) Vroomen PC, de Krom MC, Slofstra PD, et al.: Conservative treatment of sciatica: a systematic review. J Spinal Disord, 2000, 13: 463–469.
41) Shekelle PG, Adams AH, Chassin MR, et al.: Spinal manipulation for low-back pain. Ann Intern Med, 1992, 117: 590–598.
42) van Tulder MW, Koes BW, Bouter LM: Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. Spine, 1997, 22: 2128–2156.
43) Koes BW, Scholten RJ, Mens JM, et al.: Efficacy of epidural steroid injections for low-back pain and sciatica: a systematic review of randomized clinical trials. Pain, 1995, 63: 279–288.
44) Richardson C, Jull G, Hides J, et al.: Therapeutic exercise for spinal segmental stabilization in low back pain. Scientific basis and clinical approach. Churchil Livingstone, Harcourt Brace and Company Limited, London, 1999.
45) Koumantakis GA, Watson PJ, Oldham JA: Trunk muscle stabilization training plus general exercise versus general exercise only: randomized controlled trial of patients with recurrent low back pain. Phys Ther, 2005, 85: 209–225.
46) Niemisto L, Lahtinen-Suopanki T, Rissanen P, et al.: A randomized trial of combined manipulation, stabilizing exercises, and physician consultation compared to physician consultation alone for chronic low back pain. Spine, 2003, 28: 2185–2191.
47) Niemistö L, Rissanen P, Sarna S, et al.: Cost-effectiveness of combined manipulation, stabilizing exercises, and physician consultation compared to physician consultation alone for chronic low back pain: a prospective randomized trial with 2-year follow-up. Spine (Phila Pa 1976), 2005, 30: 1109–1115.
48) Sung PS: Multifidi muscles median frequency before and after spinal stabilization exercises. Arch Phys Med Rehabil, 2003, 84: 1313–1318.
49) Cairns MC, Foster NE, Wright C: Randomized controlled trial of specific spinal stabilization exercises and conventional physiotherapy for recurrent low back pain, Spine 2006, 31: E670–E681.
50) Standaert CJ, Weinstein SM, Rumpeltes J: Evidence-informed management of chronic low back pain with lumbar stabilization exercises. Spine J, 2008, 8: 114–120.
51) van Tulder MW, Assendelft WJ, Koes BW, et al.: Method guidelines for systematic reviews in the Cochrane Collaboration Back Review Group for Spinal Disorders. Spine, 1997, 22: 2323–2330.
52) Weishaupt D, Zanetti M, Hodler et al.: MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. Radiology, 1998, 209: 661–666.
53) Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al.: Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med, 1994, 331: 69–73.
54) Boden SD, Davis DO, Dina TS, et al.: Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg Am, 1990, 72: 403–408.

55) Fairbank JC, Couper J, Davies JB, et al.: The Oswestry low back pain disability questionnaire. Physiotherapy, 1980, 66: 271–273.
56) Fairbank J, Pynsent P: The Oswestry Disability Index. Spine, 2000, 25: 2940–2953.
57) Waddell G, Somerville D, Henderson I, et al.: Objective clinical evaluation of physical impairment in chronic low back pain. Spine, 1992, 17: 617–628.
58) Kim DY, Lee SH, Lee HY, et al.: Validation of the Korean version of the oswestry disability index. Spine (Phila Pa 1976). 2005, 30: E123–E127.
59) Keppel G: Design and Analysis: A researcher's handbook. Englewood Cliffs: Prentice-Hall, 1982, pp 169–183.
60) Beattie PF, Nelson RM, Michener LA, et al.: Outcomes after a prone lumbar traction protocol for patients with activity-limiting low back pain: a prospective case series study. Arch Phys Med Rehabil. 2008; 89: 269–274.
61) Macario A, Richmond C, Auster M, et al.: Treatment of 94 outpatients with chronic discogenic low back pain with the DRX9000: a retrospective chart review. Pain Pract, 2008, 8: 11–17.
62) Kuslich SD, Ulstrom CL, Michael CJ: 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. Orthop Clin North Am, 1991, 22: 181–187.
63) Anderson MW: Lumbar discography: an update. Semin Roentgenol, 2004; 39: 52–67.
64) Matsui Y, Maeda M, Nakagami W, et al.: The involvement of matrix metalloproteinases and inflammation in lumbar disc herniation. Spine, 1998, 23: 863–868.
65) Fujita K, Nakagawa T, Hirabayashi K, et al.: Neutral proteinases in human intervertebral disc. Role in degeneration and probable origin. Spine, 1993, 18: 1766–1773.
66) Ferreira PH, Ferreira ML, Hodges PW: Changes in recruitment of the abdominal muscles in people with low back pain: ultrasound measurement of muscle activity. Spine. 2004, 29: 2560–2566.
67) Hodges PW, Richardson CA: Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine, 1996, 21: 2640–2650.
68) Hodges PW, Richardson CA: Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord, 1998, 11: 46–56.
69) Hodges PW, Richardson CA: Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Arch Phys Med Rehabil, 1999, 80: 1005–1012.
70) Yoshihara K, Shirai Y, Nakayama Y, et al.: Histochemical changes in the multifidus muscle in patients with lumbar intervertebral disc herniation. Spine, 2001, 26: 622–626.
71) Yoshihara K, Nakayama Y, Fujii N, et al.: Atrophy of the multifidus muscle in patients with lumbar disk herniation: histochemical and electromyographic study. Orthopedics, 2003, 26: 493–495.
72) Zhao WP, Kawaguchi Y, Matsui H, et al.: Histochemistry and morphology of the multifidus muscle in lumbar disc herniation: comparative study between diseased and normal sides. Spine, 2000, 25: 2191–2199.
73) Hodges PW, Richardson CA: Contraction of the abdominal muscles associated with movement of the lower limb. Phys Ther, 1997, 77: 132–142; discussion 142–144.
74) Hungerford B, Gilleard W, Hodges P: Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine, 2003, 28: 1593–1600.
75) Macdonald D, Moseley GL, Hodges PW: Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain, 2009, 142: 183–188.
76) Hodges PW: Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. Exp Brain Res, 2001, 141: 261–266.
77) Hodges PW, Moseley GL, Gabrielsson A, et al.: Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res, 2003, 151: 262–271.
78) Kang CH, Shin MJ, Kim SM, et al.: MRI of paraspinal muscles in lumbar degenerative kyphosis patients and control patients with chronic low back pain. Clin Radiol, 2007, 62: 479–486.
79) Mengiardi B, Schmid MR, Boos N, et al.: Fat content of lumbar paraspinal muscles in patients with chronic low back pain and in asymptomatic volunteers: quantification with MR spectroscopy. Radiology, 2006, 240: 786–792.
80) Kjaer P, Bendix T, Sorensen JS, et al.: Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? BMC Med, 2007, 5: 2.
81) Hides JA, Richardson CA, Jull GA: Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine, 1996, 21: 2763–2769.
82) Rissanen A, Kalimo H, Alaranta H: Effect of intensive training on the isokinetic strength and structure of lumbar muscles in patients with chronic low back pain. Spine, 1995, 20: 333–340.
83) Danneels LA, Vanderstraeten GG, Cambier DC, et al: Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. Br J Sports Med, 2001, 35: 186–191.
84) Tsao H, Hodges PW: Persistence of improvements in postural strategies following motor control training in people with recurrent low back pain. J Electromyogr Kinesiol, 2008, 18: 559–567.
85) Tsao H, Hodges PW: Immediate changes in

feedforward postural adjustments following voluntary motor training. Exp Brain Res, 2007, 181: 537–546.
86) Hebert J, Koppenhaver S, Fritz J, et al.: Clinical prediction for success of interventions for managing low back pain. Clin Sports Med, 2008, 27: 463–479.
87) Inufusa A, An HS, Lim TH, et al.: Anatomic changes of the spinal canal and intervertebral foramen associated with flexion-extension movement. Spine, 1996, 21: 2412–2420.
88) Muhle C, Resnick D, Ahn JM, et al.: In vivo changes in the neuroforaminal size at flexion-extension and axial rotation of the cervical spine in healthy persons examined using kinematic magnetic resonance imaging. Spine, 2001, 26: E287–E293.
89) Chen IH, Vasavada A, Panjabi MM: Kinematics of the cervical spine canal: changes with sagittal plane loads. J Spinal Disord, 1994, 7: 93–101.
90) Gudavalli MR: Biomechanics research on flexion-distraction procedure. In: Cox JM, editor. Low back pain: mechanisms, diagnosis and treatment (6th ed). Philadelphia (PA), Lippincot Williams & Wilkins, 1999, pp263–268.
91) BenEliyahu DJ: Magnetic resonance imaging and clinical follow-up: study of 27 patients receiving chiropractic care for cervical and lumbar disc herniations. J Manipulative Physiol Ther, 1996, 19, 597–606.
92) Neault CC: Conservative management of an L4-L5 left nuclear disk prolapse with a sequestrated segment. J Manipulative Physiol Ther, 1992, 15: 318–322.
93) Schneider MJ: The traction methods of Cox and Leander: the neglected role of the multifidus muscle in low back pain. Chiropr Tech, 1991, 3: 109–115.