

THE ACADEMY OF CHIROPRACTIC ORTHOPEDISTS



Editorial Board

Bruce Gundersen, D.C., F.A.C.O.
Editor-In-Chief

Original Articles Editor

Steve Yeomans, D.C., F.A.C.O.
Reprints Editor

Rick Corbett, D.C., F.A.C.O.
Case History Editor

Ronald C. Evans, DC, FACO
Clinical Pearls Editor

Douglas G. Harden, DC, FACO
Literature Review Editor

Current Events Editor

Editorial Review Board

James R. Brandt, DC, FACO
Jeffrey R. Cates, DC, FACO
Susan L. Chung, DC, FACO
Dan Dock, DC, FACO
Ronald C. Evans, DC, FACO
B. Timothy Harcourt, DC, FACO
John F. Hayes III, DC, FACO
Martin Von Iderstine, DC, FACO
Joseph G. Irwin, DC, FACO
Charmaine Korporaal, DC, FACO
Matthew H. Kowalski, DC, FACO
Joyce Miller, DC, FACO
Lee D. Nordstrom, DC, FACO
Douglas Ortman, DC, FACO
Gregory C. Priest, DC, FACO
Jeffrey M. Wilder, DC, FACO

e-Journal

Quarterly Journal of ACO – June 2004 –

Original Articles

A Clinical Trial on Non-Surgical Spinal Decompression Using Vertebral Axial Distraction Delivered by a Computerized Traction Device

By Bruce Gundersen, DC, FACO; Michael Henrie, MS II, Josh Christensen, DC.

INTRODUCTION

Hypothesis: Axial traction of the spine produces remission of symptoms in specific conditions that have not responded to traditional manipulative protocols when computerized decompression traction, electrical stimulation and biofeedback exercise stabilization are applied under a controlled regimen.

The study is a pilot project and was not considered by an IRB for the initial phase. Continued investigation is suggested. The equipment for the study was provided by Calhoon Health Products. No fees for treatment were charged to any patients and no subjects were paid to participate in the study.

REVIEW OF THE LITERATURE

There are many studies on traction in the current literature. We have sited 20 indicating a broad interest in this concept and a continued search for alternatives to surgical decompression of the spine. The articles with a brief synopsis are listed at the end with the reference. The primary clinical point of the literature review is that compression of the neuronal elements of the spine seems to be a leading cause or generator of the pain in chronic

situations. Decompression has proven effective and various forms of decompression are elaborated. In conclusion from analyzing these articles, vertebral axial distraction can be accomplished several ways and reports of reduction of intradiscal pressure, reduction of disc herniations, and associated symptoms are cited.

CURRENT RESEARCH

A trial was designed to measure the improvement on low back and leg pain and neck and arm pain patients. Patients who had reported symptoms in those areas were notified of the project and invited to participate. Other providers of physical medicine were notified as well and encouraged to have patients with similar unresponsive conditions inquire. All patients admitted to the study had a lengthy history of pain with multiple episodes of chiropractic manipulation and physical therapy with limited success.

METHODS

A combination of questionnaires were used to compute an intake score for each patient. The score was computed using the formula, the sum of the total score from each questionnaire. Categories of severity were created as follows: 0-150; 151-175; 176-200; and > 200.

Protocols were determined based on total intake score and ranged from 3 to 6 treatment sessions per week. Traction protocols were determined based on patient history and symptoms, chronicity and extent of radicular signs. Treatment frequency was determined by total points: under 150 - 3 days per week, 151 to 175 - 4 days per week, 176 to 200 - 5 days per week and over 200 - 6 days per week.

The Axial Disc Compression Traction Therapy unit, manufactured by Chattanooga, was utilized in this study. Directions contained in the D.T.S. Information manual, copyright 2002 by Jay Kennedy were followed.

In this study, there were nine men and 5 woman ranging in age between 26-64. The range in chronicity for LB/Leg pain was 6 months to 29 years and neck to arm pain 1 year to 7 years. Exclusion criteria included, those with spinal fusions from hardware implant, those with non-disc related central spinal stenosis, those over age 70 or under age 18.

Intake measurements include modified Oswestry Low Back Pain Disability Questionnaire (Fairbanks, 1980) and the Neck Disability Index (Vernon and Mior, 1988) Activities Discomfort Scale (Turner, 1983) and a quadruple visual analogue pain scale (Yeomans, 2000). Each item was scored and the total recorded and compared to the exit scores. For this project, no objective tests were obtained on intake or exit, only standardized outcomes assessment tools.

THE PROCEDURE



Patients who qualified to enter into the study were measured and fitted to the traction unit. Both prone and supine protocols were considered for lumbar decompression. The prone position is usually recommended but can be modified per patient ability to tolerate the position. Cervical decompression is done in the supine position. Precise positioning for each patient is critical for outcomes to be optimized. A 100% compliance was expected from each subject accepted into the study in order to optimize the statistical analysis.



The specific treatment protocol was determined by the doctor after assessing the intake examination and evaluation. The computer controls the variations in the traction allowing for spinal decompression and

attempting to reduce the muscle reaction and subsequent compression that can occur with some types of traditional or conventional traction devices. The preprogrammed patterns for ramping up and down the amount of axial distraction allows for optimal levels of spinal decompression and disc hydration when possible.



Proper patient positioning and specific technique insure expected results.



RESULTS

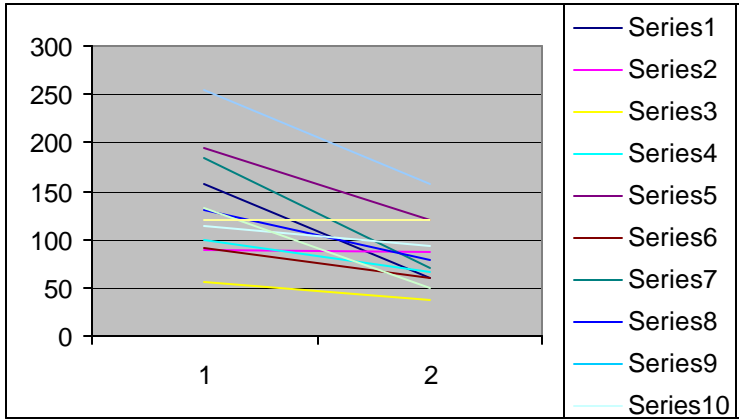
Of the 14 patients that were admitted into the study on May 17, 2004, the group was divided into the neck and arm pain group with 4 patients and the low back and leg pain group with 10 patients.

The three outcomes assessment tools were scored and totaled for each patient on intake and after three weeks of the study.

Spinal Decompression Study Results

	Average	135.33	83.17	0.36	63.75
Patient	Complaint	Intake Score	Exit Score	% Measured	% Reported
1	Low back and leg	158	60	0.62	75
2	Low back and leg	90	86	0.04	0
3	Low back and leg	56	37	0.34	85
4	Neck and Arm	99	66	0.33	95
5	Low back and leg	194	120	0.38	40
6	Neck and Arm	91	60	0.34	50
7	Low back and leg	185	70	0.62	85
8	Neck and Arm	131	78	0.40	70

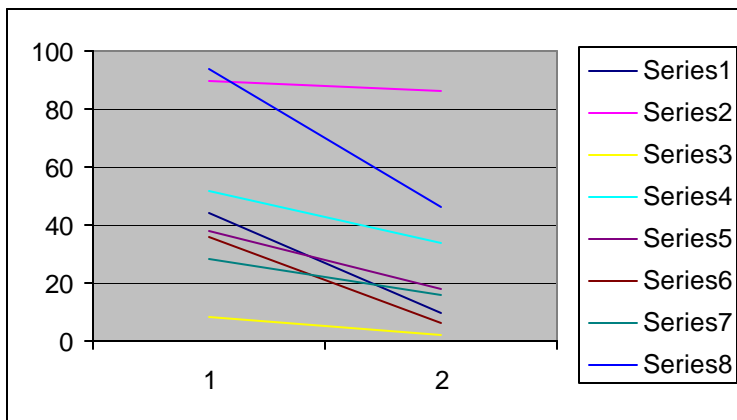
9	Neck and Arm	114	94	0.18	70
10	Low back and leg	133	49	0.63	100
11	Low back and leg	119	120	-0.01	10
12	Low back and leg	254	158	0.38	85



Using a single tool, the Revised Oswestry form for low back, it is noted that improvement parallels, in all but one case, the combination of the three tools.

Oswestry Low Back

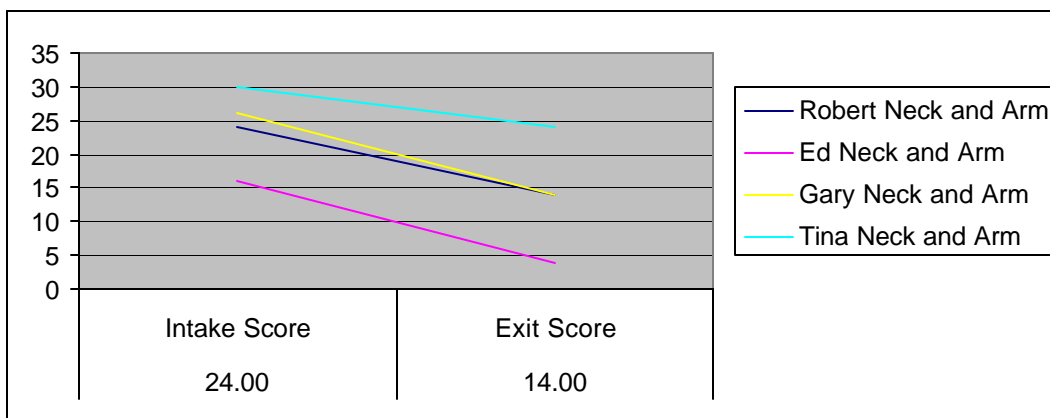
	Average	42.29	24.57	0.53
<u>Patient</u>	<u>Complaint</u>	<u>Intake Score</u>	<u>Exit Score</u>	<u>%Improvement</u>
1	Low back and leg	44	10	0.77
2	Low back and leg	90	86	0.04
3	Low back and leg	8	2	0.75
4	Low back and leg	52	34	0.35
5	Low back and leg	38	18	0.53
6	Low back and leg	36	6	0.83
7	Low back and leg	28	16	0.43
8	Low back and leg	94	46	0.51



The neck patients all responded well but not with as high an average as the low back patients.

Neck Oswestry

	Average	24.00	14.00	0.46
<u>Patient</u>	<u>Complaint</u>	<u>Intake Score</u>	<u>Exit Score</u>	<u>%Improvement</u>
1	Neck and Arm	24	14	0.42
2	Neck and Arm	16	4	0.75
3	Neck and Arm	26	14	0.46
4	Neck and Arm	30	24	0.20



Following the three-week initial phase of the study, the patient sample in this study continued to receive decompression at variable rates based on improvement. The outcome measurements are repeated at one month intervals to determine if the disability levels and perceived improvement parallel each other.

DISCUSSION

It is interesting to note that the measured results parallel the perceived or reported improvement in all but one case. That case would not be included in a long term study due to non-compliance but was included here because that is a regular obstacle in daily clinical practice.

Decompression of the spine is possible using axial distraction as a modality. Study limitations include remission of symptoms may also be linked to electrochemical effects and biomechanical stabilization. All but two of the patients in the study improved at least 30% or more in the first three weeks. Two did not. One drove 2 hours to and 2 hours from treatment sessions and was not expected to achieve much improvement notwithstanding. He did report considerable relief immediately after each session and understood that the driving more than negated any improvements. The other patient who did not measure any improvement did not comply with the protocol as outlined and would have been dismissed from the study due to poor treatment compliance.

Continued follow-up with this patient sample is recommended in Part II of this study at 1, 3, 6 and 12 month results with and without additional treatment. Studies on surgical decompression procedures of the spine are often designed to include a 2-3 year follow-up as well as reporting any associated morbidity during the study time for up to 5 years. Additional patients should be likewise admitted and studied and the 5 year plan should be instituted. Patients will also be instructed in regular use and frequency of the stabilization exercises.

This study utilized an outcomes based research design. Given the significant improvements reported in this study, it is hopeful that a randomized, controlled trial where sham traction (placebo) can be compared to decompression therapy. Also, separate subject groups can also be randomized to electrical stimulation, pelvic stabilization groups, and a combined therapies group.

CONCLUSIONS

Utilizing the outcome measures, this form of decompression reduces symptoms and improves activities of daily living. Long-term benefits were not studied but will be reported in another study. The future study will include regular follow-up measurements to determine if the remission continues with or without recurrence. Also, the future study will investigate whether or not periodic supportive treatment sessions are needed to maintain symptom satisfaction.

A BRIEF SYNOPSIS OF RESEARCH ON LUMBAR/CERVICAL TRACTION

1) Bogduk, N.: **The Anatomical Basis for Spinal Pain Syndromes.** JMPT 6:Nov.Dec1995. There is no scientific basis for the belief that muscles are a source of chronic pain generation. However controlled studies show how common disc and facet pain is accounting for more than 70% of chronic back pain.

2) Komari H, et al.: **The Natural History of Herniated Nucleus with Radiculopathy.** Spine 21: 225-229, 1996. A study group of 77 patients verified on pre- and post- traction MRI with signs and symptoms of herniation, received non-surgical intervention including pelvic traction (anything else? If not, delete "including." If so, include the other treatment approaches). Changes in herniation with good to excellent symptomatic improvements were noted in over 82% utilizing an outcomes based research design. The authors drew the conclusion that improving the disc's circulation or blood supply, accounts for healing of herniation.

- 3) Onel,D et. al.: CT Investigation of the effects of Traction on Lumbar Herniation.** Spine 14: 82-90,1989. A study group of 30 patients with lumbar disc herniations received traction in a CT scanner utilizing a >50% body weight for ~20 min. Hernia retraction was observed in 70% and good clinical improvements were seen in over 93%. The authors concluded improved blood flow was the source of healing. Additionally, they speculated that previous studies that reported traction doesn't create negative intradiscal pressures perhaps used too light a force.
- 4) Parsons, WB Cumming, JDA: Traction in Lumbar Disc Syndrome.** Can Med Jour 77:7-10,1957. A study sample of 100 patients with disc syndrome unresponsive to manipulation were treated with high force traction (+80lb). 86% of patients had good to excellent outcomes and 12 had poor outcomes. Most had pain for an extended time durations.
- 5) Saal, JA Saal, JS: Nonoperative Treatment of Herniated Lumbar Disc w/ Radiculopathy.** Spine 14 (4): 431-437, 1989. A study sample of 58 subjects had a conservative program that included traction. Overall 86% had good-excellent results with reduced leg symptoms.
- 6) Mathews, JA: Dynamic Discography: A Study of Lumbar Traction.** Annls of Phys Med, IX (7), 265-279, 1968. A study sample of 3 patients with a ruptured lumbar disc had contrast medium and radiographic images taken during and after a lumbar traction procedure. The protrusions were shown to lessen considerably utilizing 30-minute prone traction sessions and a dimpling of the outer annulus suggested a negative intradiscal force was created.
- 7) Lidstom, A Zachrisson M: PT of the low back pain and sciatica.** Scan J of Rehab Med, 2: 37-42, 1970. Intermittent supine traction with +50% body-weight, for ten, 20 minute sessions with added exercises showed considerable improvement in over 90% of the 62 patients.
- 8) Hood, LB Chrissman, D: Intermittent Traction in the Treatment of Rupture Disc Plays Ther** 48: 21, 1968. A study sample of 40 patients with neurological signs were treated with traction on a friction-free table utilizing 55-70 lbs for 20 minutes. Good to excellent results were seen in 55%.
- 9) Mathews JA et. al.: Manipulation and traction for Lumbago and Sciatica.** Physio Pract 4: 201, 1988. A controlled trial of traction with manipulative techniques. Traction force applied at approximately 100 lbs for 20 minutes resulted in substantial relief in over 85%.
- 10) Colachis S, Strohm BR: Effects of Intermittant Traction on Vertebral Separation.** Arch of Phys Med& Rehab, 50: 251-258, 1969. Subjects were subjected to a supine 'angled' traction force of up to 100 lbs. with x-ray examination. A rope angle of 18 degrees revealed separation greatest at L4-5 (Note: it is speculated that a more acute angle of approximately 10 degrees results in greater separation at L5-S1). The separation was obvious up to T12-L1 with total elongation of the spine approaching +5mm. The vertebra separation is greater on the posterior vs. anterior aspect of the vertebra.
- 11) Constatoyannis C, et. al.: Intermittent Cervical Traction for Radiculopathy Due to Large-Volume Herniations.** JMPT, 25 (3) 2002. A 3-week trial of intermittent cervical traction to patients with large volume herniations resulted in complete resolution of symptoms in 4 of 6 patients.
- 12) Shealy N, Leroy P: New Concepts in Back Pain Management.** AJPM (1) 20:239241 1998. The application of supine lumbar traction with adherence to several specific characteristics including progression to a peak force and altering the angle of 'pull' from 10 degrees (L5-S1) to 30 degrees (L3) enhanced distraction at specific levels.
- 13) Gose E, Naguszewski W&R: Vertebral axial Decompression for Pain associated With Herniated and Degenerated Discs or Facet syndrome: an Outcome Study.** Neuro Research, (20) 3, 186-190, 1997. A

retrospective analysis of over 770 cases, many assumed to be unresponsive to previous therapies showed a 71% good to excellent success rate with ~20 treatments on the prone VAX-D traction device. All patients were treated prone with 65-95 lbs. of force 3-5 times per week.

14) Weatherall VF: Comparison of electrical activity in the sacrospinalis musculature during traction in two different positions. J Ortho Sports Phys Ther(8):382-390, 1995. The use of EMG electrical activity was shown to be similar in the prone laying position vs. the supine position in a group of patients.

15) Letchuman R, Deusinger RH: Comparison of sacrospinalis myoelectric activity and pain levels in patients undergoing static and intermittent lumbar traction. Spine 18(10): 1361-1365, 1993. This study's objective was used to determine the degree of muscular guarding/contraction of lumbar paraspinals comparing intermittent vs. static traction. Improved comfort was noted greatest in the intermittent traction group.

16) Chin YG, Li FB, Huang CD: Biomechanics of traction for lumbar disc prolapse. Chinese Ortho; Jan(1): 40-2, 1994. Intervertebral pressure was recorded before and during traction. A reported 62% of prolapsed discs showed negative pressure prior to traction. Similarly, 64% reduced IDP with traction and was related to distraction distance. In 19% of prolapsed discs, the pressure actually increased, demonstrating the disruption to the hydrostatic mechanism occurring with complete annular damage and prolapse.

17) Nanno M: Effects of intermittent cervical traction on muscle pain. EMG and flowmetric studies on cervical paraspinals. Nippon Med J; Apr;61(2):137-47, 1994. Cervical intermittent traction was shown to be effective in relieving pain, increasing frequency of myoelectric signals and improving blood flow in the affected muscles.

18) Chung TS, Lee YJ, et al. Reducibility of cervical herniation: evaluation at MRI during cervical traction with a nonmagnetic device. Radiology Dec; 225(3):895900,2002. 29 patients and seven healthy volunteers had intermittent traction while in MR. Substantial increase in vertebral length was seen. Full herniation reduction in 3 and partial in 18 was reported.

19) Dietrich M, et al: Non-linear finite element analysis of formation and treatment of disc herniation. Proc Inst Mech Eng; 206(4):225-31, 1992. The author's analysis shows loads not greater than those occurring in everyday life can cause loss of stability of the disc and allow lateral nucleus displacement. The model indicates conservative therapy by traction may result in retraction of hernia by about 40%.

20) Ramos G, Martin Wm: Effects of axial decompression on intradiscal pressure. J Neuro 81: 350-353, 1994. Significant negative pressure (-100mm Hg) was recorded at L4/5 disc in three volunteers as axial traction was administered. Negative pressure was recorded at -50 pounds tension perhaps representing a minimal threshold force. Patients were prone and harnessed.

REFERENCES

1. Mackenzie R: Mechanical diagnosis and therapy for disorders of the low back. In Taylor JR, Twomey LT (Eds.): Physical therapy of the low back. Churchill Livingstone, New York, 2000 p.26 & 143.
2. Kushlich SD, Ulstrom RN, Michael CJ: Tissue origin of low back pain and sciatica. Orthop Clin North Am (22): 181,1991.
3. Nachenson AL, Elfstrom G: Intravital dynamic pressure measurements of Lumbar discs. Scand J Rehabil Med 2 Supp 1:1-40, 1970.
4. Yong, Hing K., Kirkaldy-Willis WH: ' Pathophysiology of degenerated discs of the lumbar spine. Phila. The Ortho Clinics of N. Am. Vol.(14) #3 July 83, p.p. 493504.
5. Bogduk N., Twomey L.: Clinical anatomy of the Lumbar Spine. ChurchillLivingstone New York. 1992. p.p. 68-69 & 151-173
6. Twomey LT.: Sustained lumbar traction: An experimental study of long spine segments. Spine 1985; (10): 146-149.

7. Judkovich BD.: Lumbar traction therapy-elimination of physical factors that prevent stretch. JAMA 1955; 159.
8. Gose E, Naguszewski L.: Vertebral axial decompression therapy: an outcome study. Neuro Resarch. (20)#3, April 1998.
9. Mathews JA.: Dynamic Discography: A study of lumbar traction. Annals of Phy Med, IX (7) 1968, p.p. 265-279.
10. Colachis SC.et al: Effects of intermittent traction on vertebral separation. Arch of Phy Med & Rehab 1972 (50), p.p.251-258.
11. Shealy CN. Borgmeyer V.: Decompression, reduction, stabilization of the Lumbar spine: A cost effective treatment for lumbosacral pain. AJPM 1997, 7(2), 663 -665.
12. Winkle D, et al.: Diagnosis & treatment of the lumbar spine. Aspen: Maryland: 1996 p.p. 303-313.
13. Degenerative disorders of the spine. In: Hochschuler SH, Cotler HB, (Eds.) Rehab of the Spine. Mosby MO. 1993 p.p.464-465 & p.260,
14. Cyriax JH: Illustrated Manual of orthopaedic med, London, Butterworths, 1983 p.p. 30-40.
15. Schiotz E, Cyriax JH: Manipulation, past and present. London, Heinemann, 1975
16. Biomechanics. In: Hochschuler SH, Cotler HB, (Eds.) Rehab of the Spine. Mosby MO.1993 p.p. 146.
17. Kushlich S: Tissue origin of mechanical low back pain and sciatica. In: Hochschuler SH, Cotler HB, (Eds.) Rehab of the Spine. Mosby, MO. 1993 p.p.595-599.
18. Natural history of the lumbar spine. In Taylor JR, Twomey LT,(Eds):Physical Therapy of the Low Back, Churchill Livingstone, New York, 2000, p.p 25-26 & 43-51.
19. Onel D et al. Computed tomographic investigation of the effects of traction on lumbar disc herniations. Spine 1989; 14(1):82-9
20. Hides J, Stokes, M, et al: Evidence of lumbar multifidus wasting ipsilateral to symptoms in patients with low back pain. Spine 1995, 19(2): p. 165
21. Anderson DBJ, Nachemson, AL. Intervertebral disc pressures during traction. Scand J of Rehab Supple 1983; (9):88-91.
22. Colachis S, Strom J: Cervical traction. Arch Phys Med 1965 (64):815.
23. Harris P: Cervical traction: Review of literature and treatment guidelines. Phys Ther (57):910, 1977.
24. Braaf MM, Rosner S. Recent concepts in treatment of headache. Headache, (5):3844 1965.
25. Cyriax J: The treatment of lumbar disc lesions. British Medl Jour Dec.23 14341438.
26. McElhannon JE: Physio-therapeutic treatment of myofascial disorders. Anaheim Hills, CA.: James McElhannon.
27. Deyo RA, Loeser JD, Bigos SJ. Herniated lumbar intervertebral disc. Ann Intern Med 1990:(112): 598-603.
28. Breig A, Troup J: Biomechanical considerations in the straight leg raise test. Spine 1979; (4):242.
29. Mazion JM, Haynes LM: Mazion's illustrated reference of orto/neuro/physio diagnostic techniques. Casa Grande, Mazion publisher, 4th ed, 1980
30. Greenstein GM: Clinical assessment of neurological disorders. St. louis, Mosbyyear book, Inc. 1995.
31. Weber H: The natural history of disc herniation and the influence of intervention. Spine 1994 (19): p.p. 2234-2238.
32. Saal JA, Saal JS: The non-operative treatment of HNP with radiculopathy: an outcome study. Spine 1989 (14): p.p. 431-437.
33. Komori H, Shinomiya K, et al., The natural history of HNP with radiculopathy. Spine (21): 225-229, 1996.
34. Quain MB, Tecklin JS.: Lumbar traction: it's effect on respiration. Phys Ther 1985; 65 (9): 1343-6.
35. Krause M, et al: Lumbar traction: evaluation of effects and recommended application for treatment. Man Ther 2000, May;5 (2): 72-81.
36. Gillstrom P, Erickson K,: CT exam of influence of autotraction on herniation of lumbar disc. Arch Orthop Trauma Surg 1985; 104(5):289-93.
37. Beurskens AJ et al: Efficacy of traction for non-specific back pain. Spine 1997 Dec 1 ;(23): 2756-62.
38. Laban MM et al: Intermittant cervical traction: a progenitor of lumbar radicular pain. Arch Phys Med Rehab 1992 Mar;73 (3):295-6.
39. Pellecchia GL: Lumbar traction: a review of the literature. J Orthop Sports Phys Ther 1994 Nov;20 (5): 262-7.

40. Austin R: Lumbar traction a valid option. *Aust J Physio* 1998; 44 (4):280.
41. Constantoyannis C, et al: Intermittent cervical traction for radiculopathy due to large-volume herniated discs. *JMPT* 2002 Mar; 25 (3).
42. Adams M, Bogduk Net al: *Biomechanics of back pain*. Churchill Livingstone NY, 2002: p,p 163-167
43. Grieve G: *Mobilization of the spine*. Churchill Livingstone NY, 1991: p.p 273283.
44. Martin M, Ramos G: Effects of vertebral axial decompression on intradiscal pressure. *J Neur* 81: 350-353, 1994.
45. Richardson C, Jull Get al: *Therapeutic exercises for spinal segmental stabilization in low back pain*. Churchill Livingstone NY, 1999.
46. Dullerud R, Nakstad P: CT changes after conservative treatment for lumbar disc herniation. *Acta Radiol Sept*;35(3):415-9, 1994.
47. Quain MB, Tecklin JS: Lumbar traction: its effect on respiration. *Phys Ther Sept*;65 (9):1343-6, 1985.
48. McGill S: *Low back disorders (evidence-based prevention and rehabilitation)*. Human Kinetics, 2002.
49. Hseuh TC, Ju MS: Evaluation of the pulling angle and force on intermittent cervical traction. *JFMed Assoc* 1991 Dec;90(12): 1234-9
50. Saal JS, Saal JA: Nonoperative management of herniated cervical IVD with radiculopathy. *Spine* 1996 Aug 15;21(16): 1877-83.
51. Weatherall VF: Comparison of electrical activity in the sacrospinalis musculature during traction in two different positions. *J Ortop Sports Phys Ther* 1995;(8): 382390.
52. Letchuman R, Deusinger RH: Comparison of sacrospinalis myoelectric activity and pain levels in patients undergoing static and intermittent lumbar traction. *Spine* 1993;18(10): 1361-1365.

ATTRIBUTION

Special thanks to those who contributed to this study and t his article: Chattanooga Group, Lynn Calhoon, Ronald C Evans, Steve Yeomans, and Curtis Gundersen.

Delay in Crawling: Is this a sign of long-term developmental delay?

Joyce Miller, BSc, DC, DABCO (US) FCC(UK)

Abstract:: The child who is late to crawl often presents to the chiropractor with a non-specific history and examination and offers a diagnostic challenge. The child's developmental patterns, neurological findings and heredity are important determinants for the correct prognosis. Developmental delay in the absence of aberrant neurological findings is termed Dissociated Motor Development (DMD) and has an excellent prognosis for achieving normal function.

Introduction: Thirteen-month-old Sam presents to the chiropractic clinic with inability to crawl, stand or take steps. He has been labeled a "lazy" child. His parents are very concerned that there is something seriously wrong as a friend's child has Cerebral Palsy and this was first discovered when her little boy was late to crawl and walk.

Findings: Past medical history: Sam was born at 38 weeks with vaginal delivery and forceps extraction. He was slow to sit and roll over, but reached these milestones without medical concern. Hip scans were taken a 10 months and read as normal. He eats well, sleeps well and is not unduly fussy although his mother feels that he is frustrated at his lack of mobility.

Examination revealed a child of normal intellectual and social development for his age who could not be induced to crawl or to take weight on his legs. When held in vertical suspension, he had flexed hips and knees

and would appear to “sit in the air.” Muscle tone and strength were normal. Neurological examination was normal. Pelvic distortion and hip hypomobility were found as biomechanical dysfunctions.

Discussion: Sitting in air with hips held in flexion and abduction with either knees extended (figure 1) or knees flexed (figure 3) is considered a sign of dissociated motor development (Lundberg). DMD is defined as gross motor delay in the absence of neurological signs. No pathogenetic implications are suggested.



Figure 1 Air Sit A: Child in suspension holds hips in flexion and abduction



Figure 2 Natural Position: Suspended child relaxes and flexes legs alternately



Figure 3 Air Sit B: Suspended child holds hips and knees in flexion

Sitting in air posture is a sign that the child will ultimately achieve normal maturational development (DiMario). It is wise for the chiropractor to depend on a full assessment, however, to determine whether there are any pathological implications in the child’s developmental delay. Figures 4 and 5 list indications to help guide the clinician to the appropriate prognosis.

Figure 4. Indications of Dissociated Motor Delay

- Normal neurological development
- Heredity of bottom shuffling or scooting instead of crawling
- Non-relevant perinatal period
- Developmental pattern of learning to sit and roll late
- Adaptive crawling (shuffling) techniques adopted

Figure 5. Indications of Pathogenetic Developmental Delay

- Aberrant neurological findings
- Developmental delay in fine motor, mental or self-help areas as well as gross motor
- History of Perinatal injury
- Muscular hypotonia
- Retained primitive reflexes
- Absent spinal or extremity functional lesion that may explain the cause

Etiology: What causes this delay? Perhaps it is the physical and emotional trauma of a difficult birth, perhaps an environment less conducive to the need for locomotion (older siblings who serve the infant) or perhaps neuronal connections slow to develop. Whatever the cause, chiropractors are in a position to assess and correct any biomechanical dysfunction. Coordination and proprioceptive exercises (such as cross crawl) may aid in coordination (and perhaps neuronal connections) to improve the prognosis.

Conclusion: Chiropractors are often consulted when children are slow to crawl or walk. It is important to be able to determine whether there are serious long-term implications (such as cerebral palsy) or if the child is likely to “catch-up” with their peers in time. The chiropractor should also treat the biomechanical dysfunctional lesions to aid in attaining developmental milestones. (Sam went on to crawl normally, to take weight on his legs and to walk at 15 and ½ months of age).

References:

Adolph KE, Vereijken B, Denny MA, (1998). Learning to crawl. *Child Development* 69(5), 1299-1312.

DiMario FJ Jr (2003) Dissociation of motor maturation. *J Child Neurology* 18(6): 391-3.

Lundberg A (1979) Dissociated motor development—developmental patterns, clinical characteristics, causal factors and outcome, with special reference to late walking children. *Neuropaediatric* 10(2): 161-82.

MID CERVICAL INTERVERTEBRAL ANGULATION (DURING VENTROFLEXION) AS COMPENSATION FOR ABERRANT UPPER CERVICAL MOVEMENT – A CLINICAL OBSERVATION

Adrian H Bosman; Heiner Kittel

Introduction: Hypomobility of one vertebral segment upon another has been described and accepted by a broad spectrum of clinicians. Gillet(1) using early movement palpation techniques described it and Jackson(2) suspected that when it occurred in the cervical spine it would result in a compensatory mechanism attempting to make up the lost movement. She noted that in ventroflexion of the cervical spine an angle was created at the C5/C6 intervertebral space, which spread with time, progressively higher as degenerative changes occurred at each compensating intervertebral level. Breig(3) et al concluded that this angulation between vertebrae was due to pathological biomechanics of the spine – causing a lever action during ventroflexion, resulting in distortion of the root sleeves and accompanying disc degeneration. Even slight angulation set up tension in the dura with resultant root sleeve distortion. Jackson is of the same opinion. The symptoms experienced as a result of these changes are all too familiar.

Methodology: Observing and analysing the recordings made of cine X-rays; lateral views of flat film X-rays.

Results. Using cine X-ray in clinical practice Bosman(4) too noticed that the degeneration always seemed to accompany the angulation. In addition however, after observing and analysing the recordings made of the cine X-rays of cervical movement(5) we began to develop criteria for what was felt could be normal and aberrant movement – in this case hypomobility – between C0, C1 and C2.

Discussion: The criteria indicating normal movement are that the distances between the base of the Occiput and the posterior arch of C1 and the posterior arch of C1 and the spinous process of C2 must remain (to all intents and purposes) equal whether the neck is in the neutral, ventroflexed or extended position. However should C1, on a lateral X-ray film, be shown to approximate the Occiput or conversely the spinous process of C2 in the neutral, extension or flexion views, this we think indicates aberrant movement and might be the cause of the mid cervical compensatory changes that Jackson alluded to and the angulation which Breig concluded was pathological biomechanics.

Conclusion: If these criteria for normal high cervical spinal movement should prove to be correct then in cases where the movement is shown to be disturbed, manipulation might be effective in its correction. Although only anecdotal, some x-ray and clinical evidence supports this. Further study under controlled conditions is suggested.

References. 1. GILLET, H *Belgian Chiropractic Research Notes*. 7th Edition 1968.

Page 46; Personal Communication.

2. JACKSON, RUTH *The Cervical Syndrome*. 2nd Edition pages 21-26, 28

3. BREIG, ALF. *Biomechanics of the Central Nervous System*. 1960: pages 23, 43 & 47.

4. BOSMAN, A H. *The Spine from Youth to Age*. Symposium; Arthritis and Rheumatism Council. London. Jan 13, 1972. *Normal and Abnormal Movement The High Cervical Spine*.

5. Video tape of Cine X-ray film; Flat plate lateral view X-rays.

Dr Adrian H Bosman. P O Box 1845, Plettenberg Bay 6600. Tel/Fax 044 533 4113

Abstracts

Abstract/Commentary

by Michael D. Smithers, D.C.

Male Osteoporosis: Risk Factors and Pathophysiology

Douglas C. Bauer, M.D., Medscape 2002, Orthopaedics Specialties

At the International Osteoporosis Foundation (IOF) World Congress in Lisbon, Portugal, osteoporosis (OP) in men was presented by Dr. John Bilezikian, who is a Professor of Medicine at Columbia University in New York. Dr. Bilezikian reviewed recent demographic data released by the National Osteoporosis Foundation (NOF), and stated that the number of fractures related to osteoporosis in men continues to rise with 10-12 million men in the United States having osteoporosis or osteopenia.

Pathophysiology

Although men suffer a substantial number of osteoporosis related fractures, the risk of fracture is less than in women of similar age. There are a number of significant protective factors that account for these differences and provide important insights into osteoporosis in men. First, compared with women, peak bone mass is on average 7% to 10% higher in men, presumably an effect of androgens (testosterone related hormones). Secondly, men have larger bones than women do. Larger bones have a greater cross-sectional area and other biomechanical attributes independent of bone density that contribute to a reduced susceptibility to fracture. Male bone quality, such as fewer trabecular perforations, also contribute to reduced fracture risk independent of bone mass. Thirdly, men do not have a clearly defined precipitous decline in sex hormones ("andropause") and the consequent rapid bone loss that women experience during menopause. However, in middle aged and older men, serum testosterone and estrogen levels do gradually decline. The relative importance of estrogens (compared with testosterone) in older men is increasingly recognized, but the exact role of each hormone is not completely understood. Lastly, older men are less likely to fall than older women. The reason for the reduced risk of falling is unknown, but it may relate to greater preservation of muscle mass and/or neuromuscular function in older men compared with women.

Risk Factors for Osteoporosis in Men

Dr. Bilezikian noted that 40% to 60% of men with osteoporosis have an identifiable cause or risk factor. The major risk factors in men are corticosteroid use, alcohol abuse, and hypogonadism (as assessed by free testosterone levels). Other risk factors for osteoporosis in men include a variety of medical conditions such as renal or liver disease, cancer (particularly myeloma), and gastrointestinal problems that result in calcium and vitamin D deficiency. He cautioned about using standard laboratory values for vitamin D, typically 9-52 ng/dL in most US laboratories, and suggested using a lower cutoff of 20-25 ng/dL for diagnosis of osteopenia/pososis.

Diagnosis

Osteoporosis in men is typically diagnosed in 1 of 2 ways: after a low-trauma fracture, or less often, by the presence of an abnormally low bone mineral density (BMD) as seen through a bone density study. Low and moderate trauma fractures indicate impaired skeletal strength and, as with women, confer a high risk of further fractures. Low BMD in men, particularly measured at axial sites such as the hip or spine, strongly predicts future fractures as it does in women.

Treatment

Testosterone replacement should be used when there is clear hypogonadism. On the basis of large studies that have demonstrated both efficacy and safety in men, bisphosphonates, such as Fosamax (alendronate) and Actonel (risedronate) are an appropriate treatment for many men with osteoporosis. Subcutaneous parathyroid hormone augmentation, already in use in Europe, is expected to be approved for clinical use in the United States in the near future.

Hormonal Effects in Men

Research studies have recently discussed men lacking the alpha estrogen receptor or lacking the aromatase enzyme required to convert testosterone to estrogen. These men have low bone mass, high rates of bone turnover, and unfused epiphyses. The discovery of such men has led to a number of important discoveries about the relative importance of estrogen and testosterone in the aging male skeleton. Numerous epidemiologic studies of older men have found that estrogen levels, particularly bioavailable estrogen, correlate better with bone mass than do testosterone levels. More recent studies support the role of estrogen in both the acquisition of peak bone mass in men and the maintenance of bone mass in older men. In a French study, P. Szulc and colleagues, reported that estradiol levels were important determinants of bone mass and bone turnover. In this research project, sex hormones, bone mineral density, and bone dimensions were measured in 934 men aged 19-85. The investigators found that with increasing age, external bone diameter increased 10% to 12%, endosteal diameter increased 13% to 16%, and estimated cortical width decreased 16%. After adjusting for the effects of age and body size, total testosterone levels correlated with external bone diameter whereas estradiol levels correlated with endocortical diameter and cortical width. Their findings suggest that estradiol acts mainly as a weak inhibitor of bone resorption, while testosterone acts mainly as a weak stimulator of bone formation. An Italian study reported on the relationship between sex hormone levels, bone turnover, and bone loss over 2 years of follow-up in a cohort of 200 men aged 55-82. Men with estradiol levels below the median had higher levels of bone turnover and showed higher rates of bone loss at the hip. Conversely, androgen levels did not correlate with either bone loss or bone turnover. These results confirm those demonstrated in previous reports and suggest that low estrogen levels in men result in higher bone turnover and greater bone loss over time.

Risk Factors for Osteoporosis in Men

In addition to low bone mass, other key risk factors for osteoporosis in women include greater age, reduced body weight, smoking, a positive family history, and previous fractures, particularly of the spine. The role of risk factors in men is much less studied, but several presentations in Lisbon addressed the issue.

A Belgian project summarized the existing data on the relationship between distal radius or spine fracture and subsequent hip fractures in both men and women. Among postmenopausal women with a history of distal radius fracture, the relative risk (RR) for hip was 1.53 (95% confidence interval), whereas the corresponding relative risk in men was significantly higher (RR = 3.26). Among women with a spine fracture, the relative risk for subsequent hip fracture was 2.22, compared with a relative risk of 3.54 in men. The authors acknowledged that, because distal radius fracture is 10 times less common in men than in women, its utility as a predictor of hip fracture in men is limited. However, these results conflict with a prior presentation from a Rotterdam, Netherlands, study that compared risk factors for vertebral fracture in a cohort of 3000 men and women over age 55. The researchers found that a prevalent vertebral fracture increased the risk of subsequent vertebral fractures to a lesser extent in men compared with women (male RR = 2.8, vs. female RR = 4.1).

Summary

- 1) In men, estradiol acts mainly as a weak inhibitor of bone resorption, while testosterone acts mainly as a weak stimulator of bone formation.
- 2) Low estrogen levels in men result in higher bone turnover and greater bone loss over time.
- 3) Previous forearm and spine fractures in men should be noted when performing risk assessment for osteoporosis and fracture.
- 4) The risk of hip fracture seems to be similar for men and women at the same absolute level of bone mineral density.

Case History

Lumbar Disc Replacement

Jeffrey R. Cates, DC, MS, FACO, DABCC

Several weeks ago, a patient presented himself to my office looking for relief for his ongoing low back pain. I was pleasantly surprised to note that he was encouraged to attempt a clinical trial of chiropractic care by both his primary medical physician and his orthopedic surgeon. After a reasonable trial of chiropractic treatment it was clear that the patient had notable relief but residual back pain persisted and exacerbations continued to haunt him. He elected to obtain another opinion at a well respected University based medical facility. Upon returning from that consultation the patient related that he was given the opportunity to participate in an artificial disc replacement study and request I council him on the risks and benefits. These types of requests are likely to become common across the United States in the near future and the chiropractic physician needs to be prepared to address the issue of lumbar disc replacement with their patients. To that end, I thought I would share the results of my review with you.

Artificial discs have been used in Europe for well over a decade. The Food and Drug Administration is currently evaluating these devices for use in the United States and it is likely that these devices will soon be readily available here in the United States. ¹ Chiropractors will need to understand available research so that we might council our patients about the pros and cons of using this new technology.

There are various types of lumbar disc replacements. ² Some of the more common brands are Charité, Prodisc, Acroflex, and Maverick. It is thought that this technology may have the potential to replace arthrodesis as the treatment of choice for degenerative disc disease. ³ Several clinical studies from Europe, Asia, and the United States show there to be a relatively good outcomes in up to 90% of the cases with high patient satisfaction reported. ⁴⁻⁸ Intervertebral disc space can be increase as much as 19.7%. ⁷ Complication rates are seen in up to 9% of case and include problems such as vertebral fracture, implant malposition, retrograde ejaculation, and radicular pain. ⁶ The most common complication reported was device migration. ^{7, 9, 10}

The knowledge base is growing rapidly as the number and quality of available studies increases. Most of the clinical studies note that the success rates look promising but caution us that more high quality research studies are needed. McAfee et al produced the first prospective randomized design study which was published in April of 2003. This study revealed that success rates using disc arthroplasty was comparable to lumbar fusion-interbody fusion cage and BMP or interbody autograft and pedicle screw instrumentation. ¹¹ A systematic review by de Kleuver et al, published in April 2003, notes that total disc replacements should be considered experimental and should only be used in strict clinical trials. ¹²

In the future, disc replacement surgery may become as common as knee or hip surgery. The chiropractic physician should continue to monitor the literature and developments relating to these devices and be prepared to counsel their patients accordingly.

References

1. Blumenthal SL, Ohnmeiss DD, Guyer R, et al. Artificial intervertebral discs and beyond: a North American Spine Society Annual Meeting symposium.;2(6):460-463.
2. Traynelis V, Haid J, Regis W. Spinal Disc Replacement: The Development of Artificial Discs. 04/06/2004. Available at: <http://www.spineuniverse.com/displayarticle.php/article1245.html>, 2004.
3. Huang RC, Girardi FP, Cammisa FP, Wright TM. The implications of constraint in lumbar total disc replacement. Aug 2003;16(4):412-417.

4. Zigler JE, Burd TA, Vialle EN, Sachs BL, Rashbaum RF, Ohnmeiss DD. Lumbar spine arthroplasty: early results using the ProDisc II: a prospective randomized trial of arthroplasty versus fusion. Aug 2003;16(4):352-361.
5. Zigler JE. Clinical results with ProDisc: European experience and U.S. investigation device exemption study. *Spine*. Oct 2003;28(20):S163-166.
6. Tropiano P, Huang RC, Girardi FP, Marnay T. Lumbar disc replacement: preliminary results with ProDisc II after a minimum follow-up period of 1 year. Aug 2003;16(4):362-368.
7. Jin D, Qu D, Zhao L, Chen J, Jiang J. Prosthetic disc nucleus (PDN) replacement for lumbar disc herniation: preliminary report with six months' follow-up. Aug 2003;16(4):331-337.
8. Delamarter RB, Fribourg DM, Kanim LE, Bae H. ProDisc artificial total lumbar disc replacement: introduction and early results from the United States clinical trial. *Spine*. Oct 2003;28(20):S167-175.
9. Griffith SL, Shelokov AP, Buttner_Janz K, LeMaire JP, Zeegers WS. A multicenter retrospective study of the clinical results of the LINK SB CharitÃ© intervertebral prosthesis. The initial European experience. *Spine*. Aug 1994;19(16):1842-1849.
10. Cinotti G, David T, Postacchini F. Results of disc prosthesis after a minimum follow-up period of 2 years. *Spine*. Apr 1996;21(8):995-1000.
11. McAfee PC, Fedder IL, Saiedy S, Shucosky EM, Cunningham BW. SB CharitÃ© disc replacement: report of 60 prospective randomized cases in a US center. Aug 2003;16(4):424-433.
12. de_Kleuver M, Oner FC, Jacobs WC. Total disc replacement for chronic low back pain: background and a systematic review of the literature. *European Spine Journal : Official Publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. Apr 2003;12(2):108-116.

ANKLE SPRAIN WITH A DIFFERENT TWIST

submitted by James R. Newcomb, DC, FACO

HISTORY

A 30 year-old married overweight female owner of a retail business presented with right ankle pain and swelling. She recalled twisting her ankle one week earlier while walking across a gym mat. The ankle pain decreased after two days but pain increased in the posterior calf. She described the pain as constant (76% - 100%), throbbing, burning, aching and constricting. She rated the pain at 5 on a scale of 10.

EXAMINATION

Posture and gait were antalgic. Edema extended from the antero-lateral ankle to the Achilles tendon. There was acute but non-point tenderness over the cuboid bone, calcaneofibular ligament and Achilles tendon. Weight bearing and ambulation were painful. Ankle ranges of motion were painful and limited in eversion and plantar flexion. When questioned further about the tripping incident, she denied falling completely down but recovered her posture after the right leg and foot extended sharply in the postero-lateral direction.

DIAGNOSES

The diagnoses were Grade II inversion ankle sprain with dorsiflexion of the foot and resultant lateral subluxation of the cuboid bone and posterior talocalcaneal displacement.

PROGNOSIS

The prognosis for the recovery of pre-injury function and comfort was very good.

TREATMENT

Pulsed interferential therapy was applied across the ankle joint to disperse edema. This was followed by medial adjustment of the cuboid and anterior adjustment of the talus. Figure eight compression ankle support was utilized to help maintain the correction. Patient instructions included limiting both weight bearing and ambulation. The return visit in three days found her much improved. Re-check at 11 days found her near normal. Release exam at 7 weeks was asymptomatic (see comments).

COMMENTS

Inversion ankle sprains usually involve some plantar flexion with anterior talus movement. The combination of inversion and dorsi-flexion changes the usual force directions. Using the time-honored principle of reversing the trauma forces brought quick pain relief and return of function. The unusual motion used to avoid falling accounts for this unusual combination of forces.

Release exam at 7 weeks found the ankle non-tender and pain free with normal motion, function and comfort. The patient returned to trampoline exercise at 5 weeks (without order) and was developing plantar heel pain consistent with early plantar fasciitis. She was counseled on foot care and released from ankle treatment.

JRN

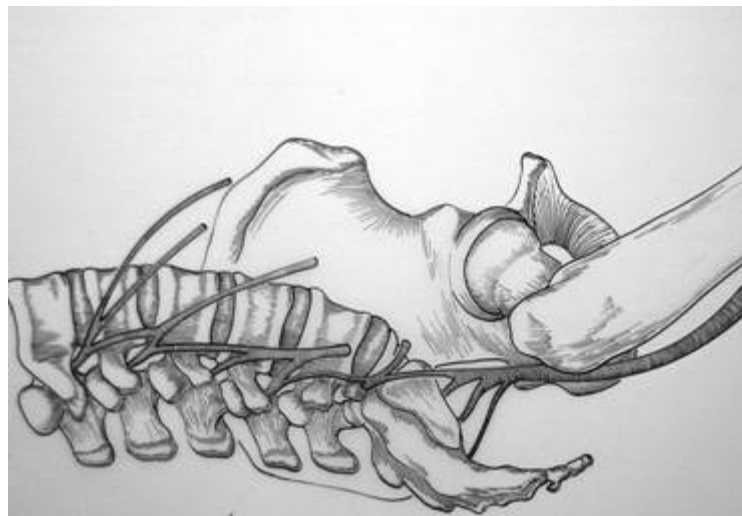
Clinical Pearl

A Clinical Pearl...the Cox Sign

Ronald C. Evans, DC, FACO, FICC

Several maneuvers tighten the sciatic nerve and compress an inflamed nerve root against a herniated lumbar disc. With various straight-leg raising tests the L5 and S1 nerve roots move several millimeters at the level of the foramen. The L4 nerve root moves a smaller distance, and the cephalic roots show little motion.

During the unilateral raising test, tension develops sequentially. It first develops in the sciatic foramen, followed by tension of the sacrum. Next, as the nerve the pedicle, tension develops in this Finally, tension occurs in the intervertebral foramen. The



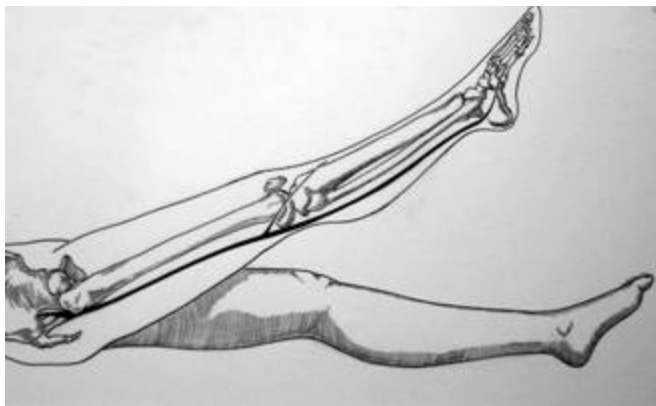
straight-leg
greater
over the ala
crosses over
area.
straight-leg

raising test will cause traction on the sciatic nerve, lumbosacral nerve roots, and dura mater. Adhesions within

these areas may be due to herniation of the intervertebral disc or to extradural or meningeal irritation. Pain that is felt by the patient comes from the dura mater, nerve root, adventitial sheath of the epidural veins, or the synovial facet joints. The test is positive if pain extends from the back, down the leg along the sciatic nerve distribution.

A central protrusion of an intervertebral disc will lead to pain primarily in the back. A protrusion in the intermediate area will cause pain in the posterior aspect of the lower limb and lower back. A lateral protrusion will cause primarily posterior leg pain.

Unilateral straight-leg raising is full at 60 to 70 degrees. At this level the nerves are completely stretched, primarily the L5, S1, and S2 nerve roots, having an excursion of several millimeters. Pain after 60 to 70 degrees is probably joint pain from the lumbar area or sacroiliac joints. The examiner compares both legs for any differences.



In the dynamics of unilateral straight-leg raising, the slack in sciatic arborization is taken up from zero to 35 degrees. There is no dural movement. When approaching 35 degrees, tension is applied to the sciatic nerve roots. In the range of 35 to 70 degrees, the sciatic nerve roots tense over the intervertebral disc. The rate of nerve root deformation

diminishes as the angle increases. Above 60 to 70 degrees, there is practically no further deformation of the root that occurs during further straight-leg raising and the pain probably originates in the joint.

Young patients (less than 30 years old) with herniated discs have marked propensities for positive straight-leg raising tests. Although the test itself is not pathognomonic, a negative test at least rules out the possibility of a herniated disc. After age 30, a negative straight-leg raising test no longer precludes this diagnosis.

Cox sign occurs during straight-leg raising when the pelvis rises from the examination table instead of the hip flexing. Cox sign is present when patients have a prolapse of the nucleus into the intervertebral foramen. The critical arc of movement is from 0 degrees to 35 degrees. The larger the space occupying mass

(prolapsed nuclear material) the smaller the angle that can be reached in lifting the leg from the examination table.

The Cox sign is usually correlated with the existence of an antalgia sign, bowstring sign, heel/toe walk test, Kemp's test, Lewin punch test, Lewin snuff test, Milgram's test, and Neri's sign.

It is important to note that Cox sign is a consistent findings associated with disc prolapse. The sign is easily overlooked in the patient's pain presentation. A false negative may occur if the examiner does not observe the movements of the buttocks on the affected side. The sign is present the moment hip flexion motion is locked and the buttock rises from the examination table.

Bibliography

- Breig A, Troup JDG: Biomechanical considerations in straight-leg-raising test: Cadaveric and clinical studies of the effects of medial hip rotation, **Spine**, 4:242, 1979.
- Brody IA, Williams RH: The signs of Kernig and Brudzinski, **Arch Neurol**, 21:215, 1969.
- Brown DE, Neumann RD: *Orthopedic secrets*, Philadelphia, 1995, Hanley & Belfus, Inc.
- Cox JM: **Low back pain mechanism, diagnosis and treatment**, ed 5, Baltimore, 1990, Williams and Wilkins.
- Dambro MR, Griffith JA: *Griffith's 5 minute clinical consult*, Baltimore, 1997, Williams & Wilkins.
- Dyck P: The femoral nerve traction test with lumbar disc protrusion, **Surg**
- Edgar MS, Park WM: Induced pain patterns on passive straight-leg-raising in lower lumbar disc protrusion, **J Bone Joint Surg**, 56B:658, 1974.
- Evans RC: *Illustrated essentials in orthopedic physical assessment*, St. Louis, 1994, Mosby-Year Book, Inc.
- Hawkins RJ: *An organized approach to musculoskeletal examination and history taking*, St. Louis, 1995, Mosby-Year Book, Inc.
- Kapandji IA: **The physiology of the joints, vol 3, the trunk, and the vertebral column**, Edinburgh, 1974, Churchill Livingstone.
- Magee DJ: *Orthopedic physical assessment*, ED 3, Philadelphia, 1997, WB Saunders Company
- Scham SM, Taylor TKF: Tension signs in lumbar disc prolapse, **Clin Orthop**, 75:195, 1971.
- Urban LM: The straight-leg-raising test: A review, **J Orthop Sports Phys Ther**, 2:117, 1981.
- Waddell G, McCulloch JA, Kummel E, Venner RM: Nonorganic physical signs in low back pain, **Spine**, 5(2):177, 1980.
- Wilkins RH, Brody IA: Laseque's sign, **Arch Neurol**, 21:219, 1969.

#2

"Never forget that the best tools that we as orthopedic specialists have are our eyes, ears and hands. Too often we, and other medical specialists, depend on technology instead of clinical skills to assess our patients, and hands-on evaluations are becoming less and less frequently utilized. A machine or medical gadget may be helpful in many cases, but remember: use our physical senses to evaluate our patients. Technology may be impressive, but it is no substitute for a thorough physical examination; technology is only an adjunct to our care."

Greg Priest, DC,FACO

Review of the Literature

Current Events

Academy Certification Examination will be held on October 9, 2004 at Northwestern Health Sciences University in Bloomington, Minnesota. This is in conjunction with The American College of Chiropractic Orthopedists (ACCO) and the Council of Chiropractic Orthopedists (CCO of ACA). If you know someone who has completed their postgraduate orthopedics, and has not obtained certification have them contact Cheryl at 515-981-9427. You can direct them to the Academy web site at www.dcorthoacademy.com for information. The website is update frequently with new information.

eLetter From The President

If you are not receiving this, please contact Ed Payne and request to be added to the list.

epayne@fcer.org

Attribution

Ed Payne, FCER