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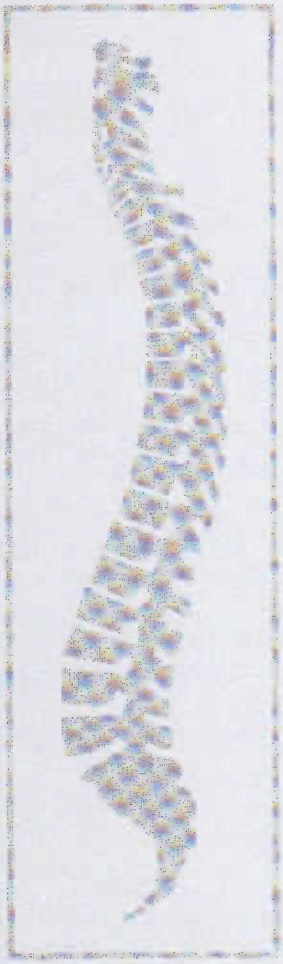

Professional Notes

How Common is Cervicogenic Headache

Nilsson N (1995) *The Prevalence of Cervicogenic Headache in a Random Population Sample of 20-59 Year Olds*, Spine 20(17)1884-1888.

A new study just published in *Spine* confirms two previous studies in reporting that cervicogenic headache is as common as migraine.

Cervicogenic headache, where the true cause of the pain is in the structures of the cervical spine, was only first formally recognized and classified by medicine in 1988. (*Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain* International Headache Society Classification Committee Cephalalgia 1988; 8(7):1-93). The IHS diagnostic criteria are:

A. Pain localized to neck and occipital region. May project to forehead, orbital region, temples, vertex, or ears.

B. Pain is precipitated or aggravated by special neck movements or sustained neck posture.

C. At least one of the following occurs:

1.Resistance to or limitation of passive neck movements.

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Chiropractic Management of Visceral Disorders

Introduction

1. 100 years ago, on September 18, 1895, Daniel David Palmer relieved deafness for a janitor in Davenport, Iowa by the unusual method of treating a sore and malpositioned thoracic vertebra. He used a spinal adjustment or manipulation to slide the joints into normal place and motion, and called his new method of treatment *chiropractic*. Sceptics should note that John Bourdillon, a medical manipulator, is one of many others who have recorded similar experiences.[1]

Throughout the subsequent history of chiropractic many patients having signs and symptoms of many other disorders apparently remote from the spine - and diagnosed medically as angina, asthma, dysmenorrhoea, hypertension, peptic ulcer, etc. - have had remarkable recoveries following chiropractic management.

Early chiropractors sought to explain their results by claiming that their treatments relieved pinched spinal nerves and restored the vital flow of energy from the central nervous system to segmentally related internal organs. A later explanation, following the work of Korr,[2] has been that spinal subluxation causes sustained abnormal somatovisceral reflexes, which may make target organs less resistant to disease. What should be said today, 100 years on?

2. Those with a dogmatic mind, of whom there are far too many in health care, may already be reacting to this article on account of the title and subject matter alone. Those with a scientific mind will be curious to think about, and plan studies to test, the following type of experience.

Mr. A.T., a 56 year old dairy farmer complaining of chest pain that feels like "a tire around my chest", is referred by his family physician to a cardiologist. Examinations including imaging, ECGs, and exercise stress tests, reveal many classic signs and symptoms of myocardial ischemia - deep chest and arm pain, paleness, sweating, cardiac dysrhythmia, and coronary arteriosclerosis.

Mr. A.T. is told he has a heart problem and is treated accordingly. He is advised to stop strenuous physical work and change his occupation. However the medications prescribed, nitrates, then beta-blockers, are ineffective. Faced with the sale of his farm and anxious to find something that might help, he seeks the advice of a chiropractor.

Chiropractic examination reveals joint subluxation, with restricted joint range of motion and muscle tension in the lower cervical and upper thoracic spine. Palpation of the two top segments in the thoracic spine reproduces the cardiac pain. Xrays show marked narrowing of the intervertebral foramina at C6/C7.

Following a short course of spinal manipulation, designed to restore normal function to the spinal segments and paraspinal muscles and to relieve irritation to the spinal nerve roots, joint function is normal and the pain fully relieved. Mr. A.T. returns to his normal farm work and lifestyle, and has no further symptoms.

3. This case is adapted from one cited by Kunert, a German cardiologist, in a paper titled '*Functional Disorders of Internal Organs due to Vertebral Lesions*'.[3] It is similar to one presented to the New Zealand Commission of Inquiry into Chiropractic. Kunert says:

".... we have records of numerous cases similar to the one described here, in which a definite connection appears to exist between a functional disorder in an internal organ and a spinal lesion..... lesions of the spinal column are perfectly capable of simulating, accentuating, or making a major contribution to (organic) disorders. There can, in fact, be no doubt that the state of the spinal column does

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1996 International Meetings: European Chiropractors' Union Annual Convention, May 16 -18, 1996, Geneva, Switzerland. *Contact:* Anne Kemp, ECU Secretary, 9 Cross Deep Gardens, Twickenham, Middlesex, TW1 4QZ, Tel: (44)(0)181 891 2546, Fax: (44)(0)181 744 2902. FCER's International Conference on Spinal Manipulation, October 17 - 19, 1996, Bournemouth, England. *Contact:* Emma Davis, FCER, 1701 Clarendon Boulevard, Arlington, VA 22209 U.S.A.

have a bearing on the functional status of the internal organs." Lewit, the prominent neurologist and manual medicine specialist, provides detailed support for this view in his leading text.[4]

4. As a matter of logic these are the possible explanations for Mr. A.T.'s case:

a) The spinal or somatic problem (subluxation) is simulating or mimicking heart disease. Some pathology is present, but this is not the cause of the problem. (This is analogous to the well-documented situation in which a patient has clear evidence of a herniated lumbar disc but this is not in fact the cause of his back and leg pain. The pain is relieved by manipulation aimed at sacroiliac or lumbar facet dysfunctions, but the herniation remains.[5])

b) Heart disease and pain have caused a reflex reaction in the spine and paraspinal muscles. The resulting spinal dysfunction (subluxation) then exaggerates or mimics cardiac pain.

c) Heart disease has caused subluxation as in (b), the underlying disease has now subsided, and the spinal lesion gives symptoms simulating continuing heart disease.

d) Spinal subluxation is causing heart disease, maybe through altered somatovisceral reflexes which, either alone or together with other stressors, cause ischemia and disease.

On any of the above four explanations it is important to Mr. A.T. and other patients that cardiologists be aware of the possible role of a spinal lesion, and that there can be a specialist examination for the potential role of such a lesion in presumed cardiac pain.

Following the huge advances in neuroscience in the past 20 years, but alive to the fact that so much remains unknown, what explanation should chiropractors give for Mr. A.T.'s case today?

Fittingly, on the eve of the centennial month, a detailed literature review and opinion on this very issue has just been published in the Journal of Manipulative and Physiological Therapeutics (JMPT), the profession's leading scientific journal. The authors are Dale Nansel PhD and Mark Szlazak DC,[6] faculty members of Palmer College of Chiropractic West in San Jose, California, one of the two affiliated colleges established by DD Palmer and his family. This Report summarizes then responds to this landmark article.

B. Visceral Disease Simulation

6. Advancing a series of arguments based on logic and evidence, Nansel and Szlazak say there is now a compelling case for somatic dysfunction (musculoskeletal dysfunction and its neurophysiological effects) simulating or mimicking visceral disease, but no persuasive evidence to support somatic dysfunction as a cause of visceral disease in internal organs. They support explanations a) to c) in paragraph 4, but not d). They suggest:

i) The hard shell of the musculoskeletal system is meant to protect the vulnerable visceral organs and functions. If this is so, and the human organism possesses such "elegant and powerful mechanisms of self-healing", is it likely that the "relatively modest insult" of spinal mis-alignment or fixation would undermine regulation of visceral function?

It might cause pain, and that might sometimes *mimic* a visceral disorder. The pain might lower overall resistance to and therefore *aggravate* visceral disorders - but would spinal dysfunction be *designed to cause* visceral dysfunction?

ii) If somatovisceral reflexes exist for successful adaptation to emergency situations (e.g. constricting blood flow to the skin and gut, and increasing flow to the muscles for 'fight or flight' response), would they be coupled with effects that "put certain tissues at risk of sympathetically mediated ischemic necrosis"?

Further, the same autonomic responses exist in simple exercise. Is the body designed so that exercise risks the healthy innervation of viscera?

Current concepts regarding the physiology of the autonomic nervous system and a large body of experimental evidence have not been able to demonstrate that the sympathetic nervous system is capable of creating "ischemic responses of any real consequence" even under "sustained maximal sympathetic activity." (In other words, Korr[1] and Sato[7] and other investigators may have been able to show various somatovisceral responses to mechanical stimulation of the spinal nerve roots but these do not reach the level of clinical significance).

This, they say, is what you would expect logically, given the body's amazing powers of self-regulation and self-healing, and also because "an impressive array of sophisticated local tissue regulatory mechanisms designed to ensure adequate delivery of oxygen and nutrients ... preceded the evolutionary development" of nervous system regulatory mechanisms designed for whole body 'fight or flight' responses and exercise.

(Nansel and Szlazak then provide a detailed technical analysis of why cardiovascular physiologists can now say that the net effect of the activity of the sympathetic nervous system under stress is an increase in the delivery of oxygenated blood to *all* tissues of the body, not just the skin, muscle, heart and brain.)

iii) If somatic dysfunction can cause visceral disease, would not patients with spinal problems display regionally-related visceral disorders? Shouldn't there be some evidence showing correlations? There is none. For example "there is not the slightest suggestion that patients suffering from severe primary mechanical low-back pain for instance, are more prone to develop higher incidences of prostate or testicular carcinoma, colitis, ovarian cysts ... or any other category of regionally or segmentally related organ disease."

iv) If a single chiropractic adjustment, or course of adjustments, can relieve a patient with a true case of asthma or hypertension, presumably it can also

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cause these problems. Why has there never been evidence of an asthma attack
shortly following an unsuccessful chiropractic treatment and caused by it?

v) There is a "scientifically sound alternative interpretation" supported by "a truly impressive body of experimental and clinical scientific evidence". (The authors support this statement with 285 references, part of the new database of 351 references they have compiled.)

The alternative explanation is that the subluxation causes pain that mimics or simulates primary visceral disease. The collective results of the studies "demonstrate that somatic (musculoskeletal) pain, together with the autonomic reflex response it induces, is notorious in its ability to create complex patterns of signs and symptoms that can often be virtually identical to and, therefore, easily mistaken for those induced by primary visceral disease." The problem is misdiagnosis.

Nansel and Szlazak then discuss the evidence. They start with the experiments of Kellgren and Lewis in 1938. Injection of hypertonic saline solution into deep paraspinal tissues produced:

a) Diffuse regionally-related pain referral patterns; and

b) Somatic and autonomic reflex responses (e.g. increases in heart rate, blood pressure, reflex muscle spasm, etc.)

that were indistinguishable from the signs and symptoms of various primary visceral disorders. The extensive work of subsequent investigators is referenced.

They then look at the basic mechanisms causing this "organ disease mimicry". Tremendous advances of knowledge concerning the anatomy and physiology of pain perception (nociception) make it now "well-established that primitive *visceral* afferent nerves that transmit nociceptive information from internal organs, and equally primitive *somatic* afferents involved in the transmission of nociceptive information from deep connective tissue structures (e.g. muscle, fascia, tendons, ligaments, joint capsules, bone, etc) *converge on common pools of interneurons* within the spinal cord and brainstem". This converged information is then transmitted along other common central nervous system pathways.

The result can be signs and symptoms that, from a diagnostic point of view, may equally be visceral or somatic in origin. A medical specialist, because of his/her training, will suspect a visceral problem (e.g. angina) if there is confirming pathology (e.g. arteriosclerosis and dysrhythmia). A chiropractor will suspect a somatic problem (e.g. subluxation) if there is confirming functional pathology (e.g. reduced ranges of joint motion, muscle trigger points).

7. Nansel and Szlazak conclude:

a) It is not logical, and there is no evidence, that somatic structures cause segmentally related internal organ disease.

b) Simulated and aggravated visceral disease is logical, and is supported by extensive evidence. In Scandinavia Bechgaard has estimated that 10% of the patients at his coronary unit have somatic mimicry rather than true angina.[8]

c) Somatic visceral disease mimicry syndromes mean there should be increased cooperation between medical physicians and those, including chiropractors, who specialize in the evaluation and treatment of somatic dysfunction.

d) There is growing appreciation of this need across health care specialties, but access to the relevant literature has been extremely difficult because of the wide diversity of disciplines and the large variety of key indexing terms.

C. Discussion

8. Nansel and Szlazak, in essence, do two things:

a) Provide an explanation for successful chiropractic management of many presumed visceral disorders that is well-supported on current knowledge. This has the benefit of being appealing across the various health disciplines.

b) Cast severe doubt upon, without ruling out, the possibility of somatic dysfunction as a cause of true internal organ disease.

Let us now look at point b).

9. Firstly, despite their many references, Nansel and Szlazak overlook and make no comment on various studies essential to their subject matter, studies where:

a) Patients with confirmed visceral pathology and spinal subluxation/dysfunction have been treated with chiropractic manipulation; and

b) Benefits of treatment have included not only pain relief but also physiological changes that influence the visceral pathology.

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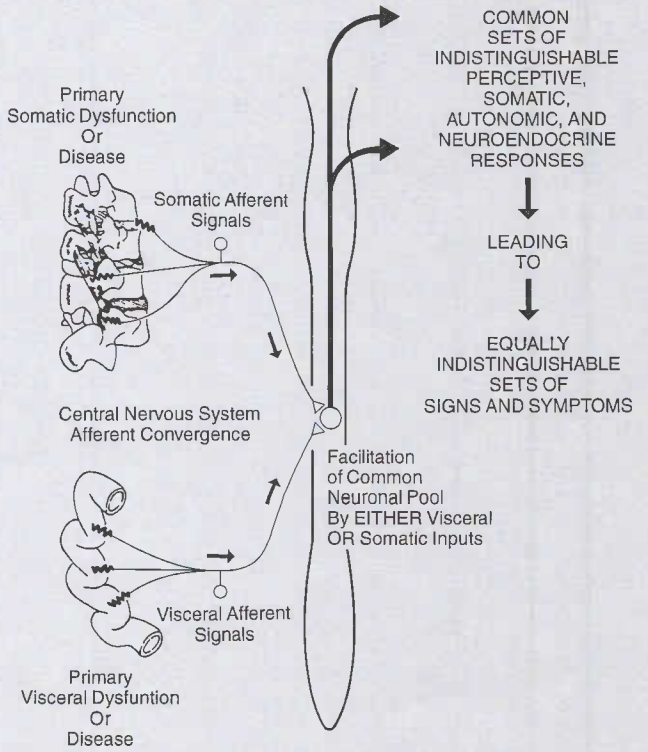
Figure 1
SIMULATED SOMATO-VISCERAL DISEASE MODEL



From Nansel & Szlazak, JMPT (1995)

See for example studies by:

i) Yates RG, Lamping DL et al on hypertension.[9]

ii) Kokjohn J, Schmid DM et al on decrease in prostaglandin levels in women with primary dysmenorrhoea.[10]

iii) Brennan PC, Triano JJ et al reporting enhanced neutrophil respiratory burst.[11]

iv) Miller WD[12] and Hviid C[13] demonstrating changed vital capacity and peak expiratory flow rates in patients with asthma.

v) Pikalov A and Vyatcheslav VK demonstrating improved remission of pathology in patients with duodenal ulcer.[14]

10. Consider the last study above, a pilot study but one which produced statistically significant results suggesting that somatic dysfunction predisposes the duodenum to disease and is a cause of true visceral disease. It was conducted by Andrei Pikalov MD PhD, formerly of the Medical Research Institute, Ministry of Internal Affairs, Moscow, Russian Federation, and now of Cleveland Chiropractic College, Kansas City, and colleagues because of promising results in earlier studies in this field by Lewit[15] and Rychlikova:[16]

a) 35 adults attending the Gastroenterological Department, Moscow Central Hospital with acute, uncomplicated duodenal ulcer confirmed by endoscopic examination were examined for vertebral subluxation. 23 patients had subluxation - signs including displacement and painfulness of vertebral spinous processes, restricted motion, and contracture and painful sensation of paravertebral muscles. Most frequently affected spinal segments were T9-T12.

b) The patients were assigned to two treatment groups:

i) Standard medical management - those without subluxation, and 12 or approximately 50% of those with subluxation, received standard drug therapy and dietary regime over 4-7 weeks.

ii) Spinal manipulation - 50% of those with subluxation received spinal manipulation and the standard dietary regime. The course of manipulation was up to 14 treatments over a 3 week period, but with frequency and technique at the discretion of the doctor depending upon the patient's condition.

c) Principal result or outcome was full clinical remission of the duodenal ulcer in terms of epithelialization (smooth healing of the lining of the duodenum) or cicatrization (healing by scar formation) as confirmed by endoscopic examination.

d) Results were that remission or healing took an average of 16.4 days in the manipulation group, approximately 10 days or 40% faster than the average of 25.7 days in the medical group. This was a statistically significant difference. Initial diameters of ulcerous defects were equivalent.

Pain was resolved in 3.8 days on average in the manipulation group. No comparison is given for the medical group.

e) The authors admit that the mechanisms of healing are unknown at present but, after discussion of the research, suggest the following:

i) "Normalization of segmental trophic innervation of the intestinal mucosal layer ".

ii) Normalization of "the action of the autonomic nervous system which influences both cellular metabolism and the vasomotor dynamics of the stomach and the duodenum".

iii) "Stimulation of the endogenous opiate system."

11. In this study we have patients with duodenal ulcer, not simulated or pseudo ulcer, and somatic dysfunction. Manipulation to relieve that somatic or spinal dysfunction not only relieves pain but has a healing effect significantly better than standard drug therapy. This is apparently mediated by the autonomic nervous system. If removal of the spinal problem healed the visceral disease (ulcer) it is logical to propose that the disturbance of the autonomic nervous system caused by the spinal problem may have been a cause of the ulcer - lowering the resistance of the epithelial wall of the duodenum to noxious stimuli.

The mechanisms may be speculative and unknown but, as scientists know, so are most things about human physiology. We are just beginning to understand how aspirin works. Until the last five years it was thought that inflammation was quite independent from the nervous system, but it is now known that there is a significant neurogenic component.[17,18]

Much remains undiscovered about the neurophysiology of the spine which, in the words of a leading researcher in this field, Rand Swensen DC MD PhD "has been slow in developing .. (and) has lagged behind understanding of neurophysiology of the more accessible tissues of the skin and extremities just as understanding of the orthopedics and biomechanics has lagged behind that of the limbs.[19]

12. The studies referred to in para 9 involve spinal cord stimulation and somatovisceral reflexes caused by spinal lesions and chiropractic treatment of them. Next, Nansel and Szlazak do not reference or discuss experimental studies which show that other forms of spinal cord stimulation cause somatovisceral reflexes which have a significant effect on physiological processes that result in true visceral disease.

In the field of heart disease take, for example, the study from Sweden by Mannheimer C Eliasson T et al[20] which reports that spinal cord stimulation decreases myocardial ischemia in patients with angina pectoris. In this study, published in 1993 in the British Medical Journal:

a) 20 patients with severe angina pectoris (15 had previously had coronary bypass surgery) were subjected to atrial pacing (in which heartbeat frequency is artificially increased repeatedly after rest intervals) to assess myocardial ischemia (insufficient blood and oxygen supply to the myocardium or central muscle layer of the heart).

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b) The aim of the study was to investigate whether spinal cord stimulation, with an implanted spinal cord stimulator, decreased the pain and myocardial ischemia caused by pacing and, if so, how.

c) A pacing catheter was used to increase the frequency of heartbeats by 10 beats per minute until the patient experienced moderate anginal pain (Level 1). Measurements and blood sampling were then taken. After 30 minute rest and 20 minutes spinal cord stimulation there was again pacing to Level 1, followed by measurements/sampling. Where the patient now experienced no pain at Level 1, pacing increased the heart rate until there was similar pain (Level 2). Results were again measured.

d) The results were that:

i) The spinal cord stimulation decreased myocardial oxygen consumption and, as a result

ii) Reduced ischemia and anginal pain.

This paper references several other studies on cardiology reporting that treatments with pain inhibiting effects such as transcutaneous electrical nerve stimulation (TENS), have an anti-ischemic effect in terms of decreased myocardial oxygen consumption and improved myocardial lactate metabolism.

13. With respect to Nansel and Szlazak's conclusions, we are not here discussing pain that is mimicking cardiac pain and heart disease. We are discussing somatic stimulation of the autonomic nervous system that produces a significant effect on true cardiac pain and its underlying cause, myocardial ischemia. This is not "pseudo-cardiac" disease but the source of true heart disease.

14. In the ways demonstrated above, and in the various arguments they advance, Nansel and Szlazak exhibit a spirit of certainty that does not account for all the evidence and leaves them vulnerable to future research findings in a field that everyone acknowledges is still poorly understood.

D. Conclusion

15. The functional state of the soma or musculoskeletal framework, which represents over 70% of the body, clearly has a major influence on internal function mediated by the nervous system. Mr. A.T. discovered this in dramatic fashion. The four logical mechanisms for this are given in paragraph 4.

The first three, which involve simulation or aggravation of visceral disorders, are now established and uncontroversial. These mechanisms, and the diagnostic challenges they represent, provide a strong case for a chiropractic role and interdisciplinary management in many cases where, on traditional medical management, the contribution of spinal problems has not been considered.

16. The fourth possibility, that somatic lesions may be a cause of visceral disease, remains controversial - but also remains quite open. There is some evidence. It is not strong, and mechanisms of action are unclear. Because the field of spinal neurophysiology in its infancy this is not surprising. Nansel and Szlazak are premature and wrong to cast such severe doubt.

However they have been thoughtful and intentionally provocative, which is always good. If they promote greater acceptance of the basic concept of vertebrovisceral relations by medical specialists, and critical analysis of the current scientific evidence by chiropractors, their article will have been more than justified.

17. There has been insufficient space to discuss important related areas such as the significance of non-symptomatic spinal lesions - many of Pikalov's ulcer patients had subluxation but no back pain. A holistic review of the causes of visceral disease would look not only at the role of the nervous system but also other internal environmental factors such as nutrition, toxins, energy and psychological state. It would address stress and Selye's general adaptation syndrome.

When we speak of the body and causes of disease in this early stage of our understanding we should be hesitant to be too assertive. Everyone in health care should be humbled by past mistakes and present inadequacies, and should leave space for what we will discover about human physiology in the years before us. We should try to cultivate the wonder and openness of true scientists, always remembering:

"The role of science is not to provide everlasting truth; but rather to provide a modest obstacle to everlasting error."[21]

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21. Anonymous, from Nansel and Szlazak - see Ref 6 supra.

Professional Notes: *continued from page 1*

2. Changes in neck muscle contour, texture, tone, or response to active and passive stretching and contraction.

3. Abnormal tenderness of neck muscles.

D. Radiologic examination reveals at least one of the following:

1. Movement abnormalities in flexion/extension.

2. Abnormal posture.

3. Fracture, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis).

The new study on prevalence in *Spine* is by Niels Nilsson DC MD and is based on a random survey of 826 adults in Odense, Denmark. Those who had suffered headaches for 5 days or more during the past month were examined, and 17.8% were found to have cervicogenic headache. This represented a prevalence of 2.5% in the general population.

There was no significant correlation between age and headache but there was according to sex - "women were about twice as likely as men to suffer frequent headaches".

Consider how many migraine societies and research foundations there are around the world. Logically there should now be at least similar emphasis on cervicogenic headache.

Much headache diagnosed as migraine will in fact be cervicogenic headache, and both forms of headache may be part of a continuum rather than separate categories. There is now a good body of scientific evidence of effectiveness of chiropractic management for both types of headache. (For a full discussion see the May 1995 issue of this Report titled *Primary Headaches and Cervical Spine Dysfunction*).

Disc Herniation and Leg Pain

Stern PJ, Cóté and Cassidy JD (1995) *A Series of Consecutive Cases of Low-Back Pain with Radiating Leg Pain Treated by Chiropractors*, J Manip Physiol Ther 18(6):335-341.

The authors of this new case series from a postgraduate chiropractic teaching clinic at the Royal University Hospital, Saskatoon, Canada, conclude that a course of non-operative treatment including manipulation can be effective and safe for the treatment of back and radiating leg pain.

- 71 consecutive patients diagnosed with leg pain from lumbar disc herniation, the majority of whom were referred by medical doctors for chiropractic manipulation, were examined. 59 received a course of chiropractic treatment comprising manipulation, interferential therapy and flexion exercises.
- 90% reported improvement, and 75% had success, defined as:

i) At least 50% recovery and/or return to work reported by the patient;

ii) A chart record of 3 things - clinically important degree of improvement; an increased range of spinal motion; and improved straight leg raise or femoral nerve stretch test.

- No patient had increased back and leg pain or other complications.

This research team is now proceeding with a full randomized controlled trial.

Trzajna, Chicotada, and Whiplash

Although often criticized, the term *whiplash* has become entrenched as a description for automobile cervical spine injuries. What terms are used in other languages?

Language	Country	Term	English Translation
Croatian	Croatia	Trzajna proveda vrata	Jerking neck injury
Finnish	Finland	Niskojen retkahdusvamma	Cervical whiplash
French	France	Coup du lapin	Rabbit's blow
German	Germany	Schleudertrauma	Slinging trauma
Icelandic	Iceland	Hálstognun	Neck strain
Italian	Italy	Colpo di frusta	Whiplash
Norwegian	Norway	Nakkesleng	Rapid neck hyperextension
Portuguese	Portugal	Chicotada	Whiplash
Spanish	Bolivia	Sindrome post	Post traumatic cervical
	Chile	traumatico cervicale	syndrome
	Mexico		
	Peru		
	Spain	Tiron muscular	Muscle pull
Swedish	Sweden	Pisknärt skada	Whiplash injury

Source: Evans RW *Headache* 1995:35:262-263.