NEUROVERTEBRAL INFLUENCE UPON THE AUTONOMIC NERVOUS SYSTEM: SOME OF THE SOMATO-AUTONOMIC EVIDENCE TO DATE -

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ABSTRACT: *Objective*: To present a broad overview of the literature in relation to the volume and variety of published material referring to spine-related neural reflexes upon organic symptoms, signs and conditions - the somato-autonomic influence. This presentation particularly emphasises somato-autonomic reflexes and to a degree, somato-autonomic-visceral reflexes mediated through the spinal influence of the neuraxis. It seeks to catalogue the evidence of the potential for further influence upon the *function* of internal anatomical structures - that is, other than those which may be regarded as purely musculoskeletal. The study further highlights the significant formal original neurophysiology research activities by chiropractors and medical researchers. These activities tend to explain the phenomena of this neurovertebral influence upon autonomic and internal function. *Data Sources*: Citations were extracted from a number of sources including: The Index to Chiropractic Literature, PubMed, Reference lists of previously published papers and textbooks, and two osteopathic electronic indexes. Over 500 papers were assessed and in a few cases only the abstracts were obtainable. *Data Synthesis*: There appears to be a developing interest in the influence of the autonomic nervous system (ANS) as depicted by the number of medical texts currently emerging. The most extensive work to date has been by Sato et al, where their studies correlate with spine-related concepts so pertinent to this presentation. The volume, variety and depth of material listed does not appear to have been presented previously. The inter-professional co-operative research projects are noted. *Conclusion*: It is noted that the volume of material presented tends to further define the neurological basis of the many clinical observations, and may provide additional explanation for the subjective patient reports of positive responses to manual manipulative intervention. Effectively at this stage, this both underpins and builds upon a long-established empirically based rationale.

INDEX TERMS: MeSH: AUTONOMIC NERVOUS SYSTEM; CHIROPRACTIC; MANIPULATION, SPINAL. (OTHER):SOMATO-AUTONOMIC REFLEX; SOMATO-VISCERAL REFLEX; ORGANIC CONDITIONS; VISCERAL DYSFUNCTION; VERTEBRAL ADJUSTMENT.

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INTRODUCTION

Gray's Anatomy states that the *"...function (of the nervous system) is to control and coordinate all the other organs and structures and to relate the individual to its environment."* [1]

Gray's statement emphasises the all-encompassing, integrative role of the nervous system – a continuing concept effectively expounded by Sherrington in 1906.[2]

This paper seeks to survey the literature relating to somatic impact upon the autonomic nervous system, and the effect that has on internal organic function or dysfunction. That aim would also assess evidence relating to aberrant somatic neural input, be that stimulatory or suppressive, and in turn assess, modify, or normalise such influences upon neural physiology and neuropathophysiology.

Various hypotheses exist on this matter of spine-related neural dysfunction. These range from involvement of massive irritation due to bombardment of noxious mechanoreceptors brought on by localised pathomechanics, to irritation and inflammatory response at the radicular level - or a combination of these various factors.[3-7] Carrick, in his significant original research has demonstrated,[8] and Terrett through an hypothesis,[9] have proposed concepts of neural influence at a more central level.

In reference to segmental neurospinal dysfunction, it is important to appreciate that at least for the purpose of this paper, a *vertebral subluxation* is not just a strict mechanical displacement of a vertebra. It is more accurately termed a *vertebral subluxation complex* (VSC) to encompass all the involved elements including functions and structures. A significant component in this complex is intersegmental articular mechanical dysfunction. This may comprise aberrant movement, fixation (hypomobility) or hypermobility between

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adjacent facets, as well as articular muscular and ligamentous changes triggering neural firing of mechanoreceptors, proprioceptors, effectively nociceptive noxious input. The VSC would then include disturbances of these structures and their function, especially their effect upon articular physiology (function) and the integral neurophysiology. Inflammatory and circulatory disturbances of the articular environment could also be associated. It is this total pathophysiological complex that would provide the opportunity through which manual intervention by way of a vertebral adjustment may be directed in order to influence internal body physiology.[10] It is submitted again that *segmental dysfunction* more than osseous displacement, may be the primary physical-mechanical feature involving any associated neural aberration in this situation but, that is only one part of the complex. Only a dry skeleton could have osseous disruption without more complex involvement.

HISTORICAL

In his 1910 text, Palmer founded what became the chiropractic profession on the basis of the importance of a neurospinal influence upon physiology. He cited the *"nervous system known as the automatic functions"*[11] - now known as the ANS. In 1954, Müller produced a text entitled *"Autonomics in Chiropractic"*, again highlighting the importance of the ANS to the profession. His text noted that *"The essential role of the autonomic nervous system as an integrator and controller of body functions is a fact all are agreed upon. That structure or function is disturbed, sometimes seriously, however this correlation is deranged from any cause is becoming more widely recognized by all schools of healing. It is the very bedrock upon which the premise of chiropractic is based."* [12] More recent research has become much more intensive, with sophisticated studies and advanced neurophysiology research into such topics concerning *the critical interaction between the musculoskeletal system and the autonomic nervous system*.[13-20]

Interestingly, as if aware of complex neural physiology through noxious stimulation, Palmer maintained that rather than nerves being "squeezed or pinched"**,** neural *energy* was "...accelerated, (and)...the volume and force is augmented."[21] It seems he was apparently aware of the bombardment of noxious impulses from disturbed proprioceptors or mechanoreceptors at that time, well before the complexities of such reflexes was appreciated as deeply as they are today.

For some 50 years early last century, the neurophysiologist Sherrington pioneered studies of neural reflexes. He stated that "To describe the action of nerve (sic) as integrative is, although true, hardly sufficient for a definition" - implying that neural influence was extensive.[2]

In the 1930's, Cannon addressed the issue of a relationship between the sympathetic division of the ANS with homeostasis,[22] and Huber and Crosby explored the "Somatic and visceral connections of the diencephalon."[23] A theme followed by Sollman in Germany in 1958.[24]

Other early authors such as Alverez, Breig, Pottenger, Kuntz and Sachs published significant texts for their time on the topic of neurovisceral disorders.[25-29] But their concepts now seem to receive less emphasis.

In Russia, Speransky conducted extensive research in neurophysiology, his text was translated and published in 1935 entitled, *A Basis for the theory of Medicine*... He cites Charcot as noting *"that not every injury to a nerve results in dystrophic lesions of the tissues, and that these lesions are connected not with the cessation of the functioning of the nerve, but with its irritation.*" He also cites Mitchell et al as stating that *"partial injury to nerves is more dangerous in this respect than complete severance."* [30]

More recently, the medical specialists Bannister (1988), Korczyn (1990), Appenzeller (1995), Goldstein (2001) Jänig (2006) have made significant contributions in the field of the autonomic nervous system.[31-35]

Currently, there are journals based on the ANS. One entitled *Autonomic Neuroscience: Basic and Clinical*, is produced by Elsevier and edited by Geoffrey Burnstock. This was previously published under the title of *The Journal of the Autonomic Nervous System* until the year 2000. *Clinical Autonomic Research* is the official journal of the Clinical Autonomic Research Society. There are also a number of other journals on the topic of neurophysiology and neurology. To this writer's knowledge, there is no specific journal based on the somatic-autonomic-visceral complex.

Burnstock has authored and co-authored many papers on neurophysiology as listed on Pubmed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi). His curriculum vitae would be one of the most extensive in this respect. His discovery of a "third nervous system" - the purinergic, in addition to the adrenergic and the cholinergic, was a most significant contribution to neurophysiology.[36,37]

Earlier, Johnston *et al* in particular explored clinical aspects of the ANS in some depth. Their exploration of the underlying role of the ANS has been highlighted in at least two pioneering scientific texts. From the mid-1960's to the mid-1980's, they were some of the earliest to broaden the importance of the clinical aspects of autonomic dysfunction.[38, 39]

SEMINAL WORK

This author is convinced however, that the most notable work in this field is that conducted by Akio Sato and colleagues in Japan. Their widely published works culminated in a text *The Impact Of Somatosensory Input On Autonomic Functions*, published over 328 pages in the *Reviews of Physiology, Biochemistry and Pharmacology* in 1997.[16] These extensive studies in neurophysiology conducted through the laboratories at the Department of the Autonomic Nervous System at the Metropolitan Institute of Gerontology, in Tokyo, Japan, provide a contemporary foundation for understanding clinically observed phenomena noted by manual practitioners. Sato has also been involved in the publications of numerous papers and at least two texts.

It should be noted that both familial and trauma induced *dysautonomia (also called autonomic dysfunction syndrome*,[40] usually refers to a more serious form of autonomic breakdown, as does the term *partial dysautomia,*[41] or *sympathetic dysfunction (Reflex Sympathetic dystrophy)*.[42] However, there can be milder versions of the trauma-induced autonomic symptoms. Gazit et al concluded that " *...autonomic nervous system-related symptoms of the patients have a pathophysiological basis, which suggests that dysautonomia is an extraarticular manifestation in the joint hypermobility syndrome.*" [43]

A number of authors have noted a connection between joint hypermobility and autonomic nervous system dysfunction (ANSD).[44] ANSD usually refers to a more acute and serious clinical situation, even though it is hypothesised to involve joint proprioception.[45] In the manual therapies however, interest in articular integrity is normally concerned with *hypo*mobility or joint fixations and its potential to affect the ANS.[3]

This historical section was included to show that a degree of recognition, and supportive evidence for the somato-autonomic concepts have existed for some years. But it is the more subtle clinical presentations which this paper seeks to focus upon.

METHOD

This overview seeks to assess somato-autonomic topics in neurophysiology published by chiropractic and osteopathic researchers, and medical authors in chiropractic and other journals.(Table 1) While not a meta-analysis, this paper summarises the dates and themes of these papers, their authors' professions, and the professions associated with journals of publication. This paper is offered in a manner which would encourage the reader to view the references as an integral part of a study of the hypotheses concerned.

It has also been the intention of this study to present examples of the wide variety of areas researched, and to portray the numerical volume of published items. Consequently, an atypical format (tabular) was adopted to cover such broad factors.

Many early journals were published before the advent of the MeSH terms and the 'Key Words' adjunct. Currently, some medical as well as some chiropractic journals still do not uniformly utilise these systems. A selection of early citations have been included along with the more recent references to note the fact that a degree of relevant physiological research had been in evidence for many years although it is only chiropractors, osteopaths as well as a limited number of medical doctors and physiotherapists who appear interested in this aspect of clinical care.

In both PubMed and Google, a search was made under the terms *somato autonomic*, *somatic autonomic*, *somato autonomic visceral* and *dysautonomia*. While there were quite a number of papers on these topics, interest seems to have been renewed more recently. During this search it was noted that there was a direct interest between acupuncture and somato-autonomic-visceral reflexes - a connection also noted by Sato *et al*.[107]

A search of library sources revealed considerable published material concerning neurospinal-related visceral physiology and pathophysiology. In recent years more formal papers have been appearing, together with original physiological research studies which tend to clarify the rationale of these spine-related-ANS somatovisceral concepts.

In particular, three electronic avenues which were employed were,

- *The Index to Chiropractic Literature (www.chiroindex.org)* and,
- *The Osteopathic Index (www.osteopathic-research.com).*
- *http://ostmed.hsc.unt.edu/ostmed/index.html*

The primary source of material for this paper was from the examination of the reference lists of previously published material. Citations have been gleaned from these sources as well as PubMed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi), Index Medicus and textbooks. The four volumes of the Chiropractic Research Archive Collection (CRAC) index series published by the Canadian Memorial Chiropractic College between circa 1980 and 1990, were also a valuable resource of data for this purpose.[108-111] Copies of original papers were obtained where possible, and abstracts where this was not feasible.

The electronic *Index to Chiropractic Literature* was examined, while another electronic index MANTIS, also has an extensive electronic reference base of the manual spinal sciences, but was not drawn upon at this time - *The Chiropractic Index – MANTIS (www.healthindex.com/MANTIS) - (ex-Chirolars)*.

The PubMed index on CAM was found not to be of assistance on this issue. (*The CAM Citation Index (Complementary and Alternative Medicine Citation Index) (www.nlm.nih.gov/nccam/camonpubmed.htm#),*

The author's own database collected over some years comprises more than 1200 citations on specified spine-related conditions. It also includes neurophysiology references on spine-related conditions. These are further divided into the different professions, and list some 127 different pathophysiological organic conditions. As an example of the volume of the material in the general literature - albeit at different standards of evidential levels, at least 21 chiropractic and 8 osteopathic papers concerning respiratory conditions have been located. In addition, there are at least a further 35 medical references also published in relation to spine-related respiratory conditions, these date from 1925 to 1995.

It also notes supportive medical neurophysiological research which has been cited in relevant papers and which report neurospinal influence upon organic function and dysfunction.(Table 2)

The nature of this presentation has been to highlight the variety, volume and depth of available evidence. It especially seeks to explicate the neurophysiological research conducted by chiropractors as well as that in chiropractic institutions and that sponsored by chiropractic research organisations. Where it is possible, it also notes inter-professional joint authorship and inter-professional co-operative research projects involving chiropractors as well as medical and osteopathic researchers in the neurosciences. However, this is not always clear in the publications.

REVIEW

"There is increasing evidence that manual therapies may trigger a cascade of cellular, biomechanical, neural and/or extra-cellular events as the body adapts to the external stress. Collectively (the research suggests) that spinal manipulation can alter the activity of nearby mechanical sensitive neurons...and in turn can lead to responses by the central and autonomic nervous systems...(which) may in turn

Table 1

CONTRIBUTIONS TO SPINAL NEUROPHYSIOLOGY, NEUROANATOMY and CLINICAL OBSERVATIONS:

PRIMARY AUTHOR	FUNCTION	ORGAN/KEYS	JOURNAL	YEAR
Bogduk N.	Cervical spine	Headaches	JMPT	1992[46]
Bolton PS, *et al.*	Adrenal glands	C-Vertebral movement	Auton Neurosci	2006[15]
Bolton PS, Budgell BS.	Spinal manipulation	Axial sensory beds	Med Hypotheses	2006[47]
Bolton PS.	Reflex effects/PNS	Vertebral subluxations	JMPT	2000[48]
Bolton PS.	Somatosensory	Neck /CNS	JMPT	1998[49]
Bolton PS, *et al.*	Neck afferents/ Respiration	Sympathetic ns, Respiratory ns	Brain Res Bull	1998[50]
Bolton PS, Holland CT.	Neck/CNS Mechanoreceptors	Normal afference Cervical motion	J Neurosci Methods	1998[51]
Bolton PS, Tracey DJ.	Neck/sensory	Medullary relay Thalamus	Exp Brain Res	1992[52]
Bolton PS, Tracey DJ.	Neck mechanoreceptors	Dorsal column nuclei/Thalamus	Brain Res	1992[53]
Bolton PS, Tracey DJ.	Somatosensory Propriospinal	Spinothalamic Upper cervical cord	Exp Brain Res	1992[54]
Briggs K, Boone WR.	Pupillary Changes	Somatovisceral/SMT	Exp Brain Res	1988[55]
Brophy GM. *et al.*	Vestibulospinal	Lumbar parasp musc	Neurosci Lett	1997[56]
Budgell BS, Bolton PS.	CSF pressure in rats	CSF	JMPT	2007[57]
Budgell BS, *et al.*	Neck mechanoreceptors	Heart variable rate	Auton Neurosci	2001[58]
Budgell BS.	Interspinous stimulation	Gastric Motility	J Auton Nerv Syst	2000[59]
Budgell BS, *et al.*	Reflex effects	VSC/ANS	JMPT	2000[13]
Budgell BS, *et al.*	Interspinous stimulation	Bladder motility	JMPT	1998[60]
Budgell BS, *et al.*	Interspinous stimulation	Adrenal function	Neurosci Res	1997[61]
Budgell BS, Sato A.	Cervical subluxation	Cerebral circulation	JMPT	1997[62]
Budgell BS, Sato A.	Modulations/Nociception	ANS	Prog Brain Res	1996[14]
Budgell BS, *et al.*	Interspinous stimulation	Cardiovascular	JNMS	1995[63]
Carrick FR.	Cervical Manipulation	Brain function	JMPT	1997[8]
Christian GF, *et al.*	SMT	Immunoreaction	Spine	1988[64]
Cramer GD, Darby SA.	Neuroanatomy	Neuraxis/ANS	Text	1995[65]
DeBoer KF, *et al.*	SMT/GI	Myoelectric activity	Man Med	1988[66]
Dishman JD, *et al.*	SMT	Reflex attenuation	Spine	2000[67]
Dishman JD, *et al.*	SMT/massage	Motoneuron excitability	Elect Clin Neurophys	2001[68]
Dishman JD, *et al.*	Motor evoked potentials	Transcranial stimulation	JMPT	2002[69]
Edwards IJ, *et al.*	Somatoautonomic reflex	Cardiorespiratory/BP	J Neurosci	2007[70]
Foreman SM, Croft A.	Cervical syndromes	ANS/Whiplash	Text	2002[71]
Fujimoto T, *et al.*	Cervical spine mechano	Heart rate/BP	J Auton Nerv Syst	1999[72]
Giles LGF.	Vertebrogenesis	ANS Syndromes	JMPT	1992[73]
Giles LGF.	Neuroanatomy	Tethered cord	Surg Radiol Anat	1991[74]
Giles LGF.	Neuroanatomy	Intervertebral foramen	JMPT	1994[75]
Giles LGF.	Neurovascular	Spinal canal & IVF	JMPT	2000[76]
Giles LGF.	Neuroanatomy	Intervertebral foramina	Neuro-orthop	1992[77]

Grimm DR, *et al.*	Musculoskeletal injury	ANS	JMPT	2005[20]
Grod JP, *et al.*	Proprioception	Neck pain	Arch Phys Med Reha	2002[78]
Hack GD, *et al.*	Neuroanatomy	Dura mater/rec cap min	Spine	1995[79]
Haavik-Taylor H, *et al.*	Transient modulation	Intracortical inhibition	Chiropr J Aust	2007[19]
Haavik-Taylor H, *et al.*	Neck/SMT	Somatosensory evoked potential	Clin Neurophysiol	2007 [18]
Igarashi Y, *et al.*	Arrhythmia	SMT	Chiropr J Aust	2000[80]
Kaushal B, *et al.*	Neurophysiology	Sensory convergence	Eur J Chiropr	2002[81]
Kurosawa M, *et al.*	Somatosensory	Spinal cord blood flow	Auton Neurosci	2006[82]
Murphy B.	Neural plasticity	SMT	ASRF	2004[83]
Murphy DR.	Somato-autonomic	Cervical Neurogenesis	Text	1999[84]
Niesluchowski W.	Scoliosis	Brain asymmetry	JMPT	1999[85]
Ruch WJ.	Radiological	ANS, SNS	Text	1997[86]
#†Sato A.	Somatosympathetic	Reflexes	NINDS/US Dept HEW	1975[87]
#Sato A, *et al.*	Mechanoreceptors	Sympathetic NS	JMPT	1984[88]
#Sato A.	Somatosensory/C/V G/I,G/U	Somatovisceral reflexes	JMPT	1992[89]
#Sato A.	Noxious/Innocuous	Somatovisceral Reflexes	JMPT	1995[90]
#*Sato A, *et al.*	Somatosensory modulation	ANS	Physiol Biochem Pharm	1997[16]
#Sato A, Budgell B.	Somatoautonomic Reflexes	Multiple	In: Haldeman S (ed)	2005[91]
Seaman DR, *et al.*	Neuropathophysiological	Dysafferentation	JMPT	1998[92]
Watanabe N, Polus B.	Mechanical input	ANS	Chiropr J Aust	2007[93]
Wantanabe N, *et al.*	Posture/autonomic regulation	Cardiovascular	Chiropr Osteop	2007[94]
Whelan TL, *et al.*	Salivary cortisol levels	Stress response/ SMT	JMPT	2002[95]
Wiles MR.	Electrogastrogram	Cervical SMT	JMPT	1989[96]
Yates BJ, *et al.*	Vestibulo-respiratory reflexes		In: Trouth CO *et al* (eds	1987[97]
Yochum TR, Rowe LJ.	Radiology	General	Text	1987[98]
OSTEOPATHIC				
Johnson WL, *et al.*	VSC	Somatic/visceral input	JAOA	2001[99]
^Korr IM.	Elect skin resistance patterns	Visceral disease	Fed Proc	1949[100]
^Korr IM, Wright HM.	VSC	Cutaneous SNS patterns	J Neural Transmission	1964[101]
^Korr IM.	Segmental NS	Disease processes	Text	1970[102]
^Korr IM.	Neurophysiology	General	Text	1978[103]
Patterson MM, *et al.*	Somatovisceral	Viscerosomatic reflexes	Text	1992[104]
^Wright HM, Korr IM.	Sympathetic NS	Segmental facilitation	JAOA	1955[105]
^Wright HM.	Local vasomotor disturbance		JAOA	1956[106]

(Includes: original research, chiropractic authors & or chiropractic publications)

Although not a chiropractic publication, this reference makes a significant contribution and includes a mention of the complex neurophysiology of spinal manipulation.

Sato works are directly related to somato-autonomic principles.

† The Research Status of Spinal Manipulative Therapy.

^ Neurophysiologists who worked extensively with the osteopathic profession.

JMPT = Journal Manipulative Physiological Therapeutics.

JAOA = Journal American Osteopathic Association.

(Where authors have been prolific, a limited number of their papers have generally been included).

Table 2

RELATED/SUPPORTIVE NEUROPHYSIOLOGY RESEARC			
AUTHOR(S)	TITLE KEYS	JOURNAL	YEAR
Adachi T, *et al.*	Cutaneous stimulation/Cerebral blood flow	Neuro Report	1990[112]
Araki T, *et al.*	Somato-adrenal medullary reflex	J Auton Nerv Syst	1981[113]
Arce A, *et al.*	Autonomic denervation/lymphocyte response	J Auton Nerv Syst	2001[114]
Barron W, *et al.*	Articular receptors/cardiovascular reflexes	J Physiol	1973[115]
Bolser DC, *et al.*	Viscerosomatic reflexes/spinothalamic tract.	J Neurophysiol	1991[116]
Brennan TJ, *et al.*	Somatovisceral reflexes/spinothalamic tract	J Neurophysiol	1989[117]
Cao WH, *et al.*	Somatosensory/c/f noxious –v- non-noxious	Jpn J Physiol	1992[118]
Chiu JH, *et al.*	Sphincter of Oddi/Somatovisceral reflex	Dig Dis Sci	1999[119]
Coote JH, *et al.*	Viscero-viscero sympathetic reflex	Neurosci Lett	1984[120]
Coote JH, *et al.*	Somato-sympathetic/cardiac symptoms	Brain Research	1978[121]
Coote JH.	Somatic afferents/muscle/joints/heart circulation	Brain Research	1975[122]
Coote JH, *et al.*	Thoracic white rami/somatic/visceral excitation	J Physiol	1969[123]
De Landsheere C, *et al.*	Spinal cord stimulation/angina	Am J Cardiol	1992[124]
Dmitrieva L, *et al.*	Somatic afferents/somatovisceral/muscle	[Zh Evol Biokhim Fiz]	2000[125]
Edney DP, *et al.*	Neck muscles afferents/neural gaze.	J Comp Neurol	1986[126]
Edwards IJ, *et al*	Somatoautonomic excitation & inhibition	J Neurosci	2007[77]
Elenkov IJ, *et al.*	ANS/brain/immune system.	Pharmacol Rev	2000[127]
Fujino M ,*et al.*	Somatic afferent stimulation/adrenal sympathetics	Neurosci Lett	1987[128]
Gilbey MP, *et al.*	Sympathetico-visceral	Baillieres Clin Endocr	1993[129]
Gouveia RG, *et al.*	ANS/Cluster headaches	J Headache Pain	2005[130]
Hikosaka O, *et al.*	Cervical spine/abducens motor neurones	Exp Brain Res	1973[131]
Hobbs SF, *et al.*	C1/2/Propriospinal/Viscerosomatic/ spinothalamic	J Neurophysiol	1992[132]
Hotta H, *et al.*	Lumbar spine/Vasa nervorum	Neurosci Lett	1991[133]
Hyngstrom AS, *et al.*	Neuromodulation/joint angulation	Nature Neurosci	2007[134]
Jänig W.	ANS/Homeostatis	Text	2006[36]
Jinkins JR.	Somato-autonomic/Neurogenic syndromes	Text	1997[135]
Jinkins JR, *et al.*	ANS/Lumbar spine/disc	Am J Roentg	1989[136]
Kerr FWL.	Cervical spine/trigeminal tract/solitary nucleus	Exp Neurol	1961[137]
Kimura A, *et al.*	Somato-autonomic reflexes	Jpn J Vet Res	1997[138]
Kimura A, *et al.*	Somatocardiovascular/Cervical cord	Neurosci Res	1995[139]
Kirchner F, *et al.*	Spinal sympathetic inhibition	Brain Research	1975[140]
Kiyomi K.	Somatoautonomic responses	In: Korr IM.	1978[141]
Kurosawa M, *et al*	Adrenal sympathetics/somatic stimulation	Neurosci Lett	1987[92]
Lindquist C, *et al.*	Mechanical sensitivity of nerve fibres	Brain Res	1973[142]
Maeda M.	Somatosensory/vestibular	Prog Brain Res	1979[143]
Menetrey D, *et al.*	Somatovisceral/Tractus solitarius	J Comp Neurol	1987[144]

Nagatomi R, *et al.*	ANS/brain/immune system.	Immunol Rev	2000[145]
Nash MS.	Depressed immune function	J Spinal Cord Med	2000[146]
Norman J, *et al.*	Cutaneovisceral reflex/Heart rate/Vagus	J Physiol	1973[147]
Norman J, *et al*	Somatosympathetic afferents/PR/BP	J Physiol	1973[148]
Ohtori S, *et al.*	Neuroanatomy/Somatosympathetic	Spine	2001[149]
Ohtori S, *et al.*	Lumbar noxious stimulation/Fos/Brain	Spine	2000[150]
Roca PD.	Ocular manifestations/Whiplash injuries.	Annals Ophthalmology	1972[151]
Sato A, *et al.*	Gastric motility/skin nociception	Brain Res	1975[152]
Sato A, *et al.*	Heart rate changes/somatosympathetic	J Auton NS	1981[153]
Sato A, *et al.*	Nociception/knee/BP/PR	Neurosci Lett	1984[154]
Sato A, *et al.*	Nociception/knee/Catecholomine/adrenal	J Physiol	1986[155]
Sato A.	Somatic afferent stimuli/Adrenals	Adv Biophys	1987[156]
Sato A.	Autonomic reflexes/somatic nociception	Masui	1987[157]
Sato A, *et al.*	Modulation of visceral function	Japanese J Phys	1987[158]
Sato A.	Somatosensory/ANS	Neurosci Behav Physiol	1997[159]
Sato A, *et al.*	Cardiovascular/somatosensory/ANS	Neurosci Behav Physiol	1997[160]
Sato A, *et al.*	Digestive secretions/motility/somatosensory/ ANS	Neurosci Behav Physiol	1997[161]
Sato A, *et al.*	Urinary/somatosensory/ANS	Neurosci Behav Physiol	1997[162]
Sato A, *et al*	Sudomotor/somatosensory/ANS	Neurosci Behav Physiol	1997[163]
Sato A, *et al*	Endocrine/somatosensory/ANS	Neurosci Behav Physiol	1997[164]
Sato A, *et al*	Immune/somatosensory/ANS	Neurosci Behav Physiol	1997[165]
Schmidt RF	Articular Nociception/Health & Disease	Brain Res	1996[166]
Sumiya E, *et al.*	Viscerosomatic inhibition	Jpn J Physiol	1997[167]
Sun MK, *et al.*	Nociceptor input/CNS/Medulla	J Physiol	1991[168]
Takahashi Y. *et al.*	Lumbar spinal reflex/lower abdomen	Spine	2000[169]
Vaida JS.	Posture/sudomotor/sympathetic efferents	Indian J Physiol Pharm	1994[170]
Wright HM, *et al.*	Segmental vasomotor variations	Fed Proc	1953[171]
Wright HM, *et al.*	Vasomotor Disturbance	Acta Neuroveg	1960[172]

lead to observed changes in circulating levels of various neuropeptides and regulatory proteins." [173]

Studies of somato-autonomic function and dysfunction are steadily emerging with quality research which continues to be carried out. Chiropractic neurophysiologists in conjunction with medical neurophysiologists are conducting much of this. (Tables 1 & 2) Some authors may have a number of papers on a particular topic, a limited selection only was adopted in this paper.

While recognising that articular adjustments – a localised and specific form of SMT, do have potential for certain physiological effects, further substantiation of spinal manipulative influence upon visceral conditions is steadily emerging.[174] That is, initial clinical and anecdotal observations are being explored by more extensive and formal studies. A wider acceptance of such concepts has probably developed to the same stage as that of the evidential literature support for manipulative approaches to mechanical lumbar spine conditions of thirty to forty years ago, and now also adopted by other professions.

This growing volume of elucidating scientific research on spine-related neuro-autonomic conditions has been summarised by Slosberg when he reports:

"...that repetitive stimulation of small myelinated and unmyelinated somatic afferents can dramatically increase reflex pre- and post-ganglionic sympathetic discharge." And notes further that *"...electrically stimulated articular nerves from knee joints of anesthetized cats* (led to) *two sympathetic responses of different latencies in the inferior cardiac nerves resulting in increases of heart rate and blood pressure."* He states further that *"...studies suggest that the alteration of afferent articular input due to joint dysfunction and nociception excitation, in conditions of noxious mechanical deformation or chemical irritation, may provoke significant changes in efferent and autonomic responses."* [175]

Much of this research is centred around major noxious stimulation of the autonomic nervous system from vertebrogenic (somato-autonomic) sources into the spinothalamic tract. Due to the integrative nature of the neuraxis, this is now known to fire into the tractus solitarius, interomedial lateral cell column (IML) of the spinal cord, other spinal cord tracts, through the brain stem nuclei and into the cortical areas.[72] (Tables 1 & 2)

Edwards and colleagues suggest that *"…the InM (Intermedius Nucleus) contains neurochemically diverse neurons and sends both excitatory and inhibitory projections to the NTS (Nucleus Tractus Solitarii). These data provide a novel pathway that may underlie possible reflex changes in autonomic variables after neck muscle spindle afferent activation."* While somato-autonomic mechanisms are recognised, there is currently only limited evidence exploring the long-term neurological effects of more minor traumatic effects emanating from spinal structures under such adverse conditions.[77]

Jinkins further clarifies the concepts by stating that *"The clinical state of neurogenic spinal radiculopathy accompanying nerve root spinal nerve, and dorsal root ganglion injury, may be associated with definite somatic and autonomic syndromes."* He notes further that *"The combined clinical complex includes "autonomic reflex dysfunction...and ..."generalized alterations in autonomic viscerosomatic tone."* [135]

Medical texts vary in their depth of commitment to the concept of somatovisceral disorders. Bourdillon states tentatively that, *"If autonomic reflex activity is accepted as a vehicle for many of the manifestations of joint and somatic dysfunction, then one can postulate that coronary vasospasm might be a result of somatic dysfunction and, if so, that it might respond to manual management."* [176]

An interesting study by De Landsheere *et al* incorporating PET monitoring found that stimulating the spinal cord (electrically), reduced exercise induced angina in patients, without conventional drugs or surgery. They found that the stimulation resulted in decreased ECG signs of myocardial ischemia, but not myocardial perfusion.[177]

It could be argued that if disturbances of the spine through such trauma as cervical whiplash (in its varying degrees), has recognisable neurological consequences, it is reasonable and possible to assume that normalising such a disturbed cervical spine should then also tend to ameliorate the neurological sequelae.

Whiplash injuries in particular can result in a number of neurological disturbances.(Table 3) In her text, Jackson mentions various autonomic symptoms which could result from disturbances to the cervical spine and cervical sympathetic supply.[194] They include:-

A sensation of exophthalmos	Blurring of vision	'Dazed and addled'
'Drop attacks'	Dysphagia	Nausea
Palpitations	Paresthesias	Tachycardia
Tinnitus	Vertigo	Vomiting
Weakness of extremities		

These citations provide evidence which tends to suggest that trauma of the cervical spine in particular, can have distinct neurological sequelae. It would seem logical that

TABLE 3

POSSIBLE CERVICOGENIC NEUROLOGICAL CONCOMMITANTS OF WHIPLASH

Brown S.	Visual accommodation	Clin Experiment Ophthalmol	2003[178]
Burke JP *et al*.	Visual system	Graefes Arch Clin Exp Ophth	1992[179]
Chrisman OD, *et al.*	Otological	Clin Orthop Rel Res	1962[180]
Croft AC.	Tissue injury/incl nerve	Text	2002[181]
Foreman SM.	Nerve system trauma	Text	2002[182]
Freeman MD, *et al.*	Whiplash associated disorders	Quebec Task FOrce	1998[183]
Grimm DR *et al.*	ANS	JMPT	2005[20]
Heikkilâ H, *et al.*	Cervicocephalic kinästhesia	Scand J Rehabil Med	1996[184]
Hinoki.	Vertigo	Acta Otolaryngol Suppl	1984[185]
Johansson BH.	Multiple	Pain Res Manage	2006[186]
Kivioja J, *et al.*	Immune response	Clin Immunol	2001[187]
Mallinson AI, *et al.*	Dizziness c/f non-whiplash	Am J Otol	1998[188]
May TS.	Headache	Am Acad Neurol	2007[189]
Pang LQ .	Otological	Laryngoscop	1971[190]
Radanov BP, *et al.*	Cognitive deficits	Spine	1992[191]
Roca PD.	Visual system	Annals Ophth	1972[192]
Storaci R, *et al.*	Oculomotor	Eur Spine J	2006[193]

TABLE 4

CHIROPRACTIC- RELATED RESEARCH PAPERS
ASPECTS OF BIOCHEMISTRY AND PHYSIOLOGY

Anaphylaxis [200]	Inflammatory cell changes[215]
ACTH [201]	Interleukin-2 [219]
ß-endorphins [201-204]	Lectins [220]
Biophotons [205]	Helix pomatia agglutinin (HPA)
Blood pressure [206-208]	Phaseolus vulgaris leucoagglutin (PHA-L)
Calcetonin [202]	Killer cells [221,222]
Catecholamine [209]	Lymphocytes[223]
CD 4 cells [210]	Macrophages [224]
Chemiluminescence [211]	Melanomas[212]
Conjunctival nevi[212]	Melatonin[225]
Cortisol [201,213,214]	Neuroimmunomodulation [226]
Endocrine [215]	Neutrophils [211,227-229]
Endotoxic shock. [200]	Oxygen radicals [224]
Heart rate [209]	Phagocytosis [227,230]
Immune complex deposition[216]	Prostaglandin [231,232]
Immune response [215,217]	Respiratory burst [228-230,233]
Immunoglobulin M (IgM)[218]	Serum aldosterone[234]
Immunohistochemistry [212]	Substance P [227,228]
Immunoreactive ACTH [201]	Tumor necrosis factor [228]

This table demonstrate s the wider aspects of chiropractic research. (Note: Many of these studies concern an immunological response. More than one term may appear in a single paper. Papers are not necessarily related to spinal manipulation but more the broad nature of chiropractic research.)

mechanical amelioration of mechanical trauma as in cervical facet hypomobility, hypermobility and noxious sensory input from related soft tissue scarring and damage, as well as joint irritation and inflammation, may well ameliorate or alleviate at least some of the associated neurological sequelae. It would seem reasonable to extend these same principles to other levels of injury in the spine. It is reasoned here, that there are varying degrees of whiplash other than the more extreme forms of trauma, less severe mechanical insult or indeed irritation, particularly of a chronic nature which would also have neurologically noxious input, and may explain a number of different insidious signs, symptoms and conditions.

In an extensive study, Hinoko noted that whiplash disturbed 'proprio-autonomic reflexes', not only of the cervical sympathetic nerves, but also that of cervical and lumbar proprioceptors.[195] Others have also noted proprioceptive disturbance in the form of vertigo and head re-positioning aberrations following whiplash.[184,196-197]

Grimm and colleagues highlighted 'interaction between cutaneous and vasomotor sympathetic neuron' response to acute musculoskeletal injury.[20] They monitored "*cardiovascular modulation, baroreceptor sensitivity, sudomotor response (skin conduction) and peripheral skin temperature*." Their findings were indicative of changes to somato-autonomic function.

The physiotherapy profession has more recently also become interested in the neurological aspects of manual therapy as a treatment of non-musculoskeletal conditions.[198] Grieve[199] acknowledges 'autonomic nerve involvement' in such signs and symptoms as:-

Dyspnoea	Flushing	Nausea
Pallor	Pulse alterations	Reduced respiratory excursion
Sweating	Vomiting	

In considering neurospinal influence, it is worth noting the rather wide range of research papers which assess the chiropractic profession's interest in biochemical markers as well as exploring connections between spinal manipulation and this measurable biochemistry. These have been monitored in a number of studies, many of which are related to the immune system. Nevertheless, they reflect rather dynamic interest in measurable biochemistry markers.(Table 4) Apart from the immunological interest in a number of these chiropractic papers,[216,226] a *neuroimmunological* relationship through the ANS has been recognised.[127,146,165,187,235]

Table 5

ANIMAL RESEARCH ON VISCERAL DYSFUNCTION, NEURAL DISTURBANCE AND THE VERTEBRAL SUBLUXATION

AUTHOR(S)	RESEARCH/TITLE	JOURNAL/YEAR
Brennan P, Kokjohn K, Triano J, *et al.*	Immunological correlates Spinal mobility Model: Dog	Proceedings Intl Conference Spinal Manip. 1991 [215]
Bolton PS, Holland CT.	Vertebral displacement Model: Cat	Soc Neurosci Abstr. 1996 [238]
Bolton PS Holland CT.	Vertebral motion Model Cats	J Neuroscience Meth 1998 [51]
Bolton PS Budgell BS, Kimpton A.	Cervical vertebral movement Model Rats	Auton Neurosci 2006 [47]
Budgell BS, Hotta H, Sato A.	Reflex bladder motility Stimulation of interspinous tissue Model: Rat.	J Manipulative Physiol Ther 1998 [60]
Budgell BS, Sato A Suzuki A, Uchida S.	Adrenal function Stimulation interspinous tissues Model: Rat	Neurosci Res 1997 [61]
Budgell BS Bolton PS.	Cerebrospinal fluid pressure Model: Rat	J Manipulative Physiol Ther 2007 [57]
Burns L*, Chandler LC, Rice RW.	Pathogenesis of visceral disease VSC (vertebral lesions) Model: Rabbits	Am Osteop Assoc, (Pub) Text Chicago 1948. [237]
Cleveland CS.	Researching the VSC Model: Rabbit.	Sci Review Chiropr 1965 [236]
Crawford JP, Hickson G, Ward M.	Immune complex deposition Acute synovitis/knee Model: Rabbit	J Manipulative Physiol Ther 1986 [216]
DeBoer KF.	Gastrointestinal myoelectric activity VSC/vertebral lesions: Model: Rabbit	Europ J Chiropr 1984 [239]
DeBoer KF, Schultz M, McKnight ME.	Gastrointestinal myoelectric Spinal manipulation Model: Rabbits.	Man Med 1988 [66]
Deboer KF, Hansen J, Dhami M.	Interaction of oxygen radicals and macrophages/ gossypol injection for inflammatory response. Model: Rats, Hamsters	J Manipulative Physiol Ther (Abstract) 1990 [224]
Kaushal B Hayek R, Ali S, *et al.*	T1-T4 sensory afferents Model: Rats	Eur J Chiropr (Poster Prestn) 2002 [81]
Kurosawa M, Watanabe O. Maruyama H, Budgell B.	Dorsal spinal cord blood flow innocuous cutaneous stimulation Model: Rats	Auton Neurosci 2006 [82]
Sato A Swenson RS.	Sympathetic nervous system Spinal column stress Model: Rats	J Manipulative Physiol Ther 1984 [88]
Waddell SC, Davidson JS, *et al.*	Immune response/endotoxic shock Cervical sympathetic trunk Model: Rats	J Manipulative Physiol Ther 1992 [200]

This table reflects chiropractic researchers or animal research papers in chiropractic journals.
(*Dr Burns has published at length on her extensive research, particularly in the J Am Osteop Assoc and the AT Still Research Institute Bull, the most recent is circa 1953.)

Some acknowledgement should also be placed on research into the effects of spine-related physiology, pathomechanics, and pathophysiology in an animal model. Two of the earliest studies on this topic were conducted by Cleveland (Circa 1965) and Burns et al in 1948.[236,237] More recent research by Brennan *et al* amongst others utilising mammalian subjects, has been particularly extensive.(Table 5)

Another paper of interest was that by Kimura and Sato entitled "Somatic regulation of autonomic functions in anaesthetised animals – neural mechanisms of physical therapy including acupuncture", depicts extensive somato-autonomic circuits.[138]

A recent landmark study by Bakkum *et al*., found that "preliminary data suggest that chronic vertebral hypomobility at L4-L6 in the rat affects synaptic density and morphology in the superficial dorsal horn of the L2 spinal cord level."[240]This finding would fit within the chiropractic model of segmental fixation within the VSC.

Physical postural distortions, anomalies, and dysfunction of the spine have also been attributed to adverse influence on the function of visceral structures innervated from disturbed vertebral levels.(Table 6)

Over almost fifty years between 1905 and 1952, Goldthwaite and medical colleagues in particular, espoused the postural implications and complications of visceroptosis and vascular stasis associated with a wide range of visceral conditions. In their extensively referenced text Goldthwaite *et al.*, stated that "*It is through this autonomic system that disturbances or improvements in visceral function are mediated by changes in the mechanics of the body*." [245] A number of mechanisms contribute to significant proprioceptive input, with direct structural influence through the centralised neural channels. This would suggest that a significant change in posture and postural mechanics may influence neuropathophysiology. Lennon *et al* summarise this aspect as follows:

"Observations of the striking influence of postural mechanics on function and symptomatology have led to our hypothesis that posture affects and moderates every physiologic function from breathing to hormonal production. Spinal pain, headache, mood, blood pressure, pulse, and lung capacity are among the functions most easily influenced by posture."

"The most significant influences of posture are upon respiration, oxygenation, and sympathetic function. Ultimately, it appears that homeostasis and autonomic regulation are intimately connected with posture. [249]

Table 6

AFFECT OF POSTURAL ABBERATIONS ON THE ANS.

Black FO *et al*.	1983	Vestibular [241]
Bouhuys A, *et a*l.	1962	Gas distribution in lungs [242]
Bouhuys A.	1963	Asthma [243]
Gagey BP, *et al*.	1996	Body axis [244]
Goldthwait JE, *et al.*	1952	Multi (Text) [245]
Gökpinar E, *et al.*	1998	Non-toxic goitre [246]
Gooch AS, *et al*	1967	Cardiac/Posture [247]
Grubb BP, *et al.*	2006	Tachycardia [41]
Kado DM, *et al*.	2004	Geriatric mortality [248]
Korr IM.	1949	Sudomotor [100]
Lennon J, *et al.*	1994	Multi [249]
Lewit K.	1980	Respiration [250]
Martin-Du Pan RC, *et al.*	2004	Multi [251]
Schey WL.	1976	Multi[252]
Storaci R, *et al.*	2006	Oculomotor dysfunction [193]
Ussher NT.	1933	Multi [253]
Ussher NT.	1940	Multi [254]
Vaida JS, *et al.*	1994	Sudomotor [170]
Watanabe N, *et al*.	2007	Autonomic regulation/cardiovascular [94]
Watson DH, *et al.*	1993	Cervicogenic headaches/muscles [255]
Wright HM, *et al.*	1966	Skin temperature [256]
Wyke BD.	1979	Senile disequilibrium [257]

Table7

VASOMOTOR RESPONSE TO SPINE-RELATED ABBERATIONS

AUTHOR	YEAR	JOURNAL
Adachi T, *et al.*	1990	Neuro Report[112]
Appenzeller O, *et al.*	1965	J Neurol Neurosurg Psychiatr[250]
Figar S, e*t al.*	1967	Acta Neuroveg[251]
Figar S, *et al.*	1967	Acta Univ Carolinae[252]
Figar S, *et al.*	1965	Acta Univ Carolinae[253]
Giles LGF.	2000	JMPT[76]
Gongal'skii VV, *et al.*	1992	Neirofiziologiia (Ukraine)[254]
Grimm DR *et al.*	2005	JMPT[20]
Hotta H, Nishijo K, *et al.*	1991	Neurosci Lett[133]
Knutson GA.	2001	JMPT[255]
Kurosawa M, *et al.*	2006	Auton Neurosci[82]
Mc Knight ME, DeBoer KF.	1988	JMPT[256]
Passatore M, *et al.*	1996	Acta Neurobiol Exp[257]
Potts JT.	2002	Clin Exp Pharmacol Physiol[258]
Potts JT, Paton JF, Mitchell JH, et al.	2003	Neurosci[259]
Potts JT, Spyer KM, Paton JFR.	2000	Brain Res Bull[260]
Sato A, Sato Y, Schmidt PE.	1997	Rev Physiol Biochem Pharm[261]
Shortt TL, Ray CA.	1997	Am J Physiol[262]
Sun MK, Spyer KM.	1991	J Physiol[168]
Wright HM.	1956	J Am Osteop Assoc[106]
Wright HM, *et al.*	1960	Acta Neuroveg[171]

In addition, cutaneous and vasomotor autonomic reflexes can provide further examples of spine-related somato-autonomic activity.(Table 7)

Wilson noted that "static contraction of skeletal muscle activates small-diameter afferent nerve fibres which evoke a reflex increase in sympathetic nerve activity and cardiovascular function." They cite others as making the same observation.[271]

As highlighted by D'Aubigne in the preface to Kapandji's text (and in the text's title), is the term central to this subject - the term 'physiology of a joint'. It comprises not only the cell physiology of all associated articular tissue, but also the biomechanical aspects of normal articular movements – joint physiology, structures and function.[272] Physiology can be defined as the *"a branch of biology that deals with the functions and activities of life or of living matter (as organs, tissues, or cells) and of the physical and chemical phenomena involved."* Pathophysiology of a joint then is some abnormality of the function of associated tissues, including especially neural tissues and articular mechanics – a *derangement of function* or "*the physiology of abnormal states; specifically : the functional changes that accompany a particular syndrome or disease*." [273]

Whatmore and Kohli define the term dysponesis and that *"most diseases consist of physiologic reactions that lead to organic dysfunction. These physiologic reactions constitute the response of the organism to some noxious agent, whether microbial, chemical, or mechanical.*" [274]

In another component of the subluxation complex (myopathology or myopathophysiology), Edwards *et al* proposed that autonomic variables can be influenced by afferent muscle spindle activation, particularly from the posterior muscles of the neck. Further, that cardiorespiratory variables rely on interaction between the somatic and autonomic systems, essentially somato-sympathetic reflexes.[70]

Hyngstrom *et al*, found further that "intrinsic electrical properties of spinal motoneurons vary with joint angle(s)..." and that "*dendrites of spinal motoneurons amplify inputs to a marked degree through persistent inward currents (PICs)…(where) dendritic amplification is subject to neuromodulatory control from the brainstem.*" [134]

It is submitted that if autonomic influence can be so readily disturbed locally, it would seem reasonable that associated visceral function could also be influenced, and that the material presented would tend to support that hypothesis. Inasmuch as the correction (vertebral adjustment) of a physically and physiologically disturbed spinal element (a VSC) may provide an acknowledged physiological mechanism to ameliorate or normalise the connected autonomic disturbance and linked organic dysfunction, that hypothesis could then be reasonably supported.

DISCUSSION

> *'The autonomic nervous system is intimately responsive to changes in the somatic activities of the body and while its connections with the somatic elements are not always clear in anatomical terms, the physiological evidence of visceral reflex activities stimulated by somatic events is abundant."* [275]

Central to this discussion is the fact that the autonomic nervous system "*is a visceral and largely involuntary sensory and motor system (where) virtually all visceral reflexes are mediated by local circuits in the brain stem or spinal cord*" [276] As stated, much of this activity is interceded through the spinal cord neurologically and the intervertebral foramina anatomically. If it is accepted that the ANS effectively influences all functions in the body, then there are six key elements to be addressed on this topic of external manual influence and spine-related pathophysiology. They are:-

1. Neural dysfunction. Whether within the vertebral subluxation complex (segmental dysfunction) there can be influence, irritation, interference or modulation (stimulation or inhibition) with afferent and/or efferent neural transmission - Dorlands Dictionary defines such a role as reflexogenic - *producing or increasing reflex action*.[277]
2. Organic dysfunction. Whether any resultant altered neural transmission can adversely alter or influence innervated structural or organic physiology;
3. The *degree* to which any neural-induced pathophysiology of viscera may take place;
4. Neural Pathophysiology. Whether subsequent manual spinal adjustment (SMT) can positively influence neuropathophysiology of neural dysfunction;
5. Organic pathophysiology. Whether manual adjustment may influence the innervated visceral dysfunction or other structures by that positive influence, and if so;
6. How that influence may be monitored and modified to best effect for a particular patient's response.

It is submitted that if a spinal, or indeed a peripheral articular adjustment does not affect, influence or have input upon the nervous system in any way, then effectively, manual manipulative therapy professions would not have a *raison d'être* in advancing such a neurospinal model. The reduction by articular adjustment of a bombardment of noxious neural stimuli from aberrant articular or associated soft tissue nociceptive[152,166,270,278] input, could be deemed an example of a positive influence. Clinical examples would be in the cases of resolution of the vascular component of cervicogenic headaches,[65] or the positive influence upon a dysfunctional lumbar spinal segment associated with PMT or dysmenorrhea. – conditions that are a dysfunction (pathophysiological) rather than pathological.[279,280]

If neural disturbance (Table 8) via hyperstimulation, suppression, or irritation is present within a subluxation complex, and deliberate neutralisation or normalisation of such aberrations through a vertebral segmental adjustment takes place, then the vertebral adjustment would be deemed to have had a positive neural influence. The differentiation and degree of a VSC affecting a somatic-somatic or a somatovisceral reflex (or both at once), must be the subject of interesting future research.

Another area of research interest could involve differentiating the variation in biological response to the VSC. For example, one response might be somato-somatic and another somato-visceral, even though by definition both would have a segmental neurological factor at the same vertebral level. A more specific example would be why a C1/C2 VSC in one patient produces headaches, in another migraines, and in yet another, no headache at all - just localised facet or muscle pain.

Extensive research currently being undertaken by the chiropractic neuroscientist Bolton et al, seems to corroborate the hypothesis of definitive compromise of neural physiology due to articular facet disturbance.[282,283] Their research involves complex neurological aspects of both the VSC and the vertebral adjustment.

In relation to other spinal levels, there are a number of symptoms which can reflect spine-related neurological involvement.[284] Common examples would include pain as in vertebrogenic sciatica and brachial plexus neuropathy.[285,286] Symptomatically, these conditions may be associated with various other neural symptoms including paresthesias, formication, muscle weakness, hyporeflexia, hyperalgesia and hypalgesia, through to muscle atrophy and trophic changes.[287] In osteopathic research, Karason and Drysdale demonstrate a somatovisceral reflex involving increased cutaneous circulation at the L5/S1 level, following administration of spinal adjustments (high-velocity low-amplitude – HVLA). These were conducted 'outside the region of the sympathetic outflow.' [288]

Further examples would be spinogenic dyspepsia.[289-292] Another could be the ciliospinal reflex, which incorporates both elements of a transient basic somatovisceral reflex. Chusid and McDonald classify the ciliospinal reflex as a visceral reflex. They also note that in the presence of Horner's Syndrome which involves the T1 and T2 sympathetic segment, the ciliospinal reflex is lost.[293]

It may be a rudimentary example, but if it is noted that just a sharp stimulating slap on the back can affect neural activity (nociceptive, mechanoceptive) and invoke sympathetic responses such as pupillary dilation, increased pulse rate and adrenal secretion, then a somato-autonomic neural mechanism would be demonstrated. If that slap on the back can produce a generalised somato-autonomic response, then a controlled, specific, neuromechanical stimulation via a specific vertebral adjustment to a mechanically disturbed spinal segment may have the potential towards a more localised and predictable physiological response. It is noted that other factors such as psychological aspects, may also be involved in this example.

Physical therapy techniques including exercises, and at times manual manipulative musculoskeletal techniques, have also been employed in the management of a variety of visceral conditions involving the sympathetic nervous system. These include respiratory and cardiovascular rehabilitation.[294]

Recognition of the existence of neural and visceral pathophysiology or physiological dysfunction are central to this model. While consideration is given to the possibility of vertebrogenic visceral dysfunction and vertebrogenic visceral symptoms, the advent of actual vertebrogenic visceral pathology needs additional research. The possibility of simulated symptoms of visceral conditions has been discussed elsewhere.[295]

Even with a diagnosis of a *simulated* organic condition, the patient's complaint is likely to be present in a symptomatic form. Unless the symptom is recognised as *simulation,* then previous diagnoses, treatments, or medication are likely to be based on those symptoms and not necessarily directed at the condition itself. That is, a treatment may well have been based in error on an actual organic condition, but one that is only simulated. One wonders if this could be a potential shortcoming in some Cochrane Collaboration studies, which appear to be based on the assumption of an accurate diagnosis in the first place - followed by treatment based on that diagnosis.

On the other hand there is a need for much more fundamental research to fully substantiate the spinovisceral role, even though reasonable evidence does exist. Conversely, there is a far greater paucity of evidence which would *contradict* claims by patients that they subjectively experience benefit from SMT. There seems to be a notable lack of double-blind controlled studies which *negate* positive claims regarding autonomic symptoms by patients under chiropractic care. Indeed, to justify the more recent adoption by *manipulative medicine* and *manipulative physiotherapy* of manual spinal therapy in relation to musculoskeletal conditions, there would appear to be a lack of evidential studies which provide the legitimacy for and justification of that involvement.

A connection has been discussed between mechanical spinal disturbance and resultant influence upon autonomic function.[13] This has been further depicted by way of highlighting sensory and mechanical disturbance from lumbar and cervical spinal facet changes (Table 1), and from whiplash (Table 3). It is also seen neurologically through the autonomic symptoms associated with migraine and other headaches, [296-298]and pain.(Table 9)[399-410] There does however seem to be only limited research into the ANS effects from the varying durations and severities of more minor chronic pain situations. Although Johnston[38,39] and Bannister[31] have discussed the finding that autonomic function, autonomic dysfunction and degrees of autonomic failure can be of varying degrees of severity. Carrick has shown that specific influence on cervical spinal structures has led to a reasonable hypothesis regarding spinal influence upon brain function, as monitored by blind-spot mapping.[8] Disturbance of specific aspects of the extensive autonomic network may also explain some visceral symptoms and pathophysiological conditions of organic dysfunction as listed in this paper.

SUMMARY

"Health practices such as acupuncture and spinal manipulation frequently employ stimulation of somatic tissues in the treatment of visceral symptomology. The efficacy of these practices may well be based in somato-autonomic reflexes. An understanding of how afferent input modulates autonomic function, therefore, has considerable meaning beyond its academic interest." [14]

Traditionally, medical interest in neural function in man has been centred on the more serious expressions of ANS dysfunction.[31,311] However it is in relation to the more subtle signs and symptoms which this paper sought to illustrate – and especially, to look at the *neurospinal* connections – even in infants.[312]

It is a stimulating chore to attempt to differentiate between the papers mentioned. The cited extensive writings in the referenced literature by Bolton, Brennan, Budgell, Burns, Carrick, Pickar, Korr, Patterson, Swenson and particularly Sato *et al*., do provide a deeper awareness and appreciation of neuro-vertebral concepts. Their contribution has been significant in providing greater insight into the wide influence of disturbed somato-autonomic-visceral reflexes.

Based on the indications presented, it would be fair to suggest that to date, the neurological implications of SMT may be more intricate, extensive and sophisticated than even DD Palmer may have believed.[313] However, more research is required for further understanding and to elucidate the complex of neurovisceral-pathophysiological phenomena. It is hoped that true scientists would thoroughly consider all material before drawing conclusions on the role of chiropractic in this somato-autonomic area.

As indicated, basic scientific evidence does exist in support of a spine related influence upon the ANS. It suggests distinctly possible vertebrogenic factors in affecting such autonomic activity in the disturbance of some forms of heart rate, blood pressure and neural activity in renal, adrenal and gastrointestinal function. The potential for a possible physical, as opposed to a chemical form of intervention, is exciting. Given the volume, variety and indeed depth of the material mentioned here, one could be regarded as ultra-conservative if one concluded meekly that there does appear to be at least some potential in support of an hypothesis of spine-related neuro-autonomic pathophysiological dysfunction. Sato *et al* stated that *"...the decreases in blood pressure and renal nerve activity during manipulation of the spine are thought to be supraspinal reflexes."* [314]

CONCLUSION

"In contrast to the impressive body of knowledge concerning the effects of visceral afferent activity on autonomic functions, there is, generally speaking, much less information available on the reflex regulation of visceral organs by somatic afferent activity from skin, the skeletal muscle and their tendons, and from joints and other deep tissues. The elucidation of the neural mechanisms of somatically induced autonomic reflex responses, usually called somato-autonomic reflexes, is, however, essential to

Table 8

SPINE-RELATED MODULATION OF THE ANS EXCITATORY &/or INHIBITORY		
Budgell B, Sato A.	Modulation	1996[14]
Dhami MSI, Coyle BA.	Stimulatory	1986[225]
Dishman JD, Ball KA, Burke J.	Excitatory	2002[69]
Edwards IJ, Dallas ML, Poole SL, *et al.*	Excitatory & Inhibitory	2007[70]
Fidelbus J.	Somatosympathetic reflexes	1989[226]
Fujimoto T, *et al.*	Stimulatory	1999[72]
Fujino M, *et al.*	Stimulatory	1987[128]
Haavik-Taylor H, Murphy B.	Inhibitory	2007[19]
Hinoki M.	Excitatory	1984[185]
Hyngstrom AS, Johnson, *et al.*	Excitatory/Amplification	2007[134]
Kirchner F, Kirchner D, Polosa C.	Inhibitory	1975[140]
Lennon J,*et al.*	Modulation	1994[249]
Ohtori S, *et al.*	Stimulatory	2000[150]
Pickar JG.	Excitatory & Inhibitory	2000[281]
Sato A.	Excitatory	1987[157]
Sato A, Schmidt RF.	Modulation	1987[158]
Sato A, Sato Y, Schmidt RF.	Excitatory & Inhibitory	1987[16]
Sato A.	Excitatory	1997[159]

developing a truly scientific understanding of the mechanisms underlying most forms of physical therapy, including spinal manipulation and traditional as well as modern forms of acupuncture and moxibustion." – Sato A et al [315]

In essence, this has been an attempt to highlight the published literature surrounding the hypotheses appropriate to the manual manipulative therapies in general, and the science of chiropractic in particular. It draws connections between the effect upon the autonomic nervous system due to pain, postural disturbance, and the mechanical disruption of trauma, with whiplash being the most easily demonstrated. There are indications in some of the references that trauma does not have to be extreme to produce such symptoms. It has attempted further, to look at the reflexogenic effects of factors affecting the ANS. It then follows the next association - that of the effect of ANS irritation upon the physiology of structures innervated by an ANS modified by such changes, especially those regarded as spine-related.

It would seem reasonable to hypothesise that if disturbance or mechanical insult to the spine can lead to ANS-related dysfunction or symptoms, then alleviation of that disturbance or insult should also tend to ameliorate those same symptoms, and thereby encourage disturbed physiology in involved structures to return towards normal function.

The volume and variety of evidence presented in this cursory overview, would tend to suggest there should be open mindedness when considering the possibilities of manual management of a number of somatovisceral conditions. An attitude of outright rejection may tend to limit chiropractic within a musculoskeletal field. This could deny science a whole area of opportunity, and deprive certain patients of options for a potential source of minimally invasive natural health management. Once all the available evidence is considered, and further research has had the opportunity to explain the clinically observed phenomenon of positive outcomes, proper assessment can then take place. Despite the material presented here, one is still reticent in making broad claims.

To consider the historical base for chiropractic, without the vital inherent correlations with the nervous system, is essentially to not fully appreciate chiropractic as it was originally intended - and where it is currently pioneering this natural model of health care.

Co-operation between the health professions, together with a serious, constructive and unbiased research effort into this topic, would raise the potential for a more effective, non-invasive means of influencing internal physiology and potentially, pathophysiology.

It would seem appropriate to be able to influence the nervous system and physiological dysfunction through the least invasive intervention possible and through readily accessible neural procedures. If that potential is through SMT, then it behoves the scientific community to explore that potential and develop it to its extreme. Rejecting the concept without thorough investigation is unscientific and close-minded.

While much research is currently underway, many areas remain to be explored in order to further develop the somato-

Table 9

PAIN AND THE ANS		
Benarroch EE.	Pain /ANS	2001[299]
Cortelli P Pierangeli G.	Chronic pain/ANS	2003[300]
Cramer GD, Darby SA.	Spinogenic pain	1995[301]
Grod JP, Diakow PR.	Neck pain/proprioception	2002[302]
J‰nig W.	Sympthetic NS/Pain	2006[303]
LeBoeuf-Yde C, *et al.*	Low back pain/Health	2003[304]
Michaelis M, Janig W.	Sympathetic NS/Pain	1998[305]
Nathan PW.	Sympathetic NS/Pain	1988[306]
Passatore M, *et al*.	Sympathetic NS/Pain	2004[307]
Rix GD, Bagust J.	Proprioception/headache/neck pain	2001[308]
Sato A, Sato Y, Schmidt RF.	Somatic nociception	1997[309]
Sterling M, Jull G, Wright A.	Sympathetic NS/Cervical pain	2001[310]

autonomic-visceral hypothesis. To this author, the weight of the evidence so far is such that a significant neurospinal connection cannot be ignored or discounted. Beyond the basic neurophysiological research, the positive clinical indications so far suggest quite inspiring promise.

"The human nervous system is the most complex physical system known to mankind: it consists of many billions of interactive units whose constantly changing patterns of activity are ***reflected in every aspect*** *of human behaviour and experience."* Gray's Anatomy [316]

An extensive list of references is available by contacting the author on
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