Asia-Pacific Chiropractic Journal

A CLINICAL SERIES

Neurodynamics of vertebrogenic somatosensory activation and Autonomic Reflexes - a review:

The introduction to a clinical series

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Abstract: Neurological factors associated with the vertebral subluxation are presented together with recognition of this clinical entity by the chiropractic, medical and osteopathic professions.

Noxious activation of somato-autonomic reflexes is an established physiological response to sensory phenomenon.

While neuro-phenomenon is recognised physiologically, it appears due recognition of the vertebrogenic origin in somatosensory physiology seems to be limited in clinical medicine.

Indexing terms: Vertebral subluxation; Neurophysiology; Somatosensory; Autonomic nervous system

Introduction

The explanation for many biological phenomena may depend on a contextual understanding.There can however be significant evidence in support of hypotheses. This series if offered in order to discuss some of the neurophysiological evidence associated with the complex vertebral subluxation concepts.

A brief overview is presented of some of the available material involving vertebrogenic pathophysiology. This series seeks to explore existing published evidence of the effects of disturbed somatic structures influencing neurological function and impacting on visceral function. This phenomenon has been designated here as a *Somato-Autonomic Visceral Complex* (SAVC). The data recognises intricacies involving the vertebral subluxation complex as a primary influence in this phenomenon, as well as being a particularly cogent portal that seeks to neutralise the noxious neural input.

> *... Aberrant somatosensory reflexes are seen as one of the elements involved in vertebral subluxations'*

This review is aimed at presenting and discussing in particular the medical evidence that supports the chiropractic hypothesis of the vertebral subluxation. Under current available evidence, that hypothesis is offered as the most likely explanation for the phenomenon reported by patients and recorded in the literature by chiropractors, osteopaths and medical practitioners. (1, 2, 3)

For the purpose of this report a subluxation is defined as:-

> *A subluxation is an articular dysfunction, typically but not limited to the spine and pelvis, characterised by anatomical and neurophysiological signs and symptoms.*

The manual or instrumental correction of a vertebral subluxation or other articulation is identified as an *adjustment* which emphasises its specificity.

An adjustment may be defined as:-

> The physical application of a highly developed finely tuned advanced form of manual or instrument intervention directed to restore joint and neural physiology in order to ameliorate associated signs and symptoms.

The Vertebral Subluxation Complex: Beyond the mechanical

There has been a tendency to regard the *Vertebral Subluxation Complex* (VSC) primarily as elements of an articular biomechanical dysfunction with or without osseous displacement. Evolving research and clinical case reports recognise a significant integrated neural element associated with these physical and pathophysiological articular disturbances. These are essentially neuropathophysiological with identifiable signs and symptoms. The somatosensory afferent influence upon the autonomic nervous system (ANS), and consequently upon visceral physiology is now well recognised as somatovisceral reflex pathophysiology. (4)

Despite some notable exceptions, conventional allopathic clinical models have shown comparatively limited interest in any means to influence human physiology other than through medication. Meanwhile the chiropractic and osteopathic professions have focused on clinically recognised disturbances of this autonomic influence due to disrupted somatic structures, particularly from a neurophysiological and clinical perspective. (5)

The input from noxious vertebral activation may be regarded neurologically as distinctly sensitive and influential. Such input may originate from sensory elements in a range of somatic structures through to a range of spinal somatic structures, particularly the sensory-rich facets. Through somatosensory, somato-autonomic, somatovascular, and somatovisceral activation of reflexes, the influence may play a number of pathophysiological roles. For these reasons it is necessary to note that the VSC is appreciably more comprehensive than the very limited version of just a partially displaced bone as in the previous more traditional definition of a subluxation. (6, 7, 8)

In view of these considerations, it would then seem prudent, to neutralise adverse neural influence by eliminating the noxious sensory input.

A classic example of this *somato-autonomic complex* would be cervicogenic headaches which exemplify the neural involvement. Other examples would include lumbogenic sciatica and thoracogenic intercostal neuralgia along with other recognised functional vertebrogenic conditions such as dysphagia, dyspepsia and simulated conditions shadowing neural pathways. (9, 10)

This rationale for the science sustaining the subluxation concept draws primarily on the standard biological and medical sciences. This series draws heavily from these with a deliberate use of citations in order to demonstrate the strength of the evidence in the face of claims that it does not exist,

This treatise is proposed in order to submit evidence-based material upon which a rationale for a contextual model of a vertebral subluxation complex may be based. Practitioners in the manipulative sciences have noted for over a century that while managing back and neck pain that on occasion, a range of other symptoms and conditions may also appear to ameliorate. These involve activated somato-autonomic, somatosensory, somatovisceral and somatosomatic reflex signs and symptoms. Positive outcomes have led to published case reports, research and word-of-mouth referrals by patients.

Davis summarises the somatosensory disturbance in his example following whiplash when he states '*Patients with chronic whiplash syndrome may have a generalized central hyperexcitability from a loss of tonic inhibitory input (disinhibition) and/or ongoing excitatory input contributing to dorsal horn hyperexcitability. Dysfunction of the motor system may also occur, with or without pain.*' (11)

A range of everyday clinical signs and symptoms can at times indicate noxious somatosensory reflex input from aberrant somatic structures. These somatic structures such as functionally disrupted joints may be identified as subluxation complexes, or if a spinal segment, a *vertebral subluxation complex* (VSC). These spinal complexes may be static or dynamic with an integrated afferent and efferent neural element. It should not be regarded as a purely osseous disturbance.

Manual correction or adjustment which seeks amelioration of this VSC through specific manipulation of the particular articulations has been clinically found to positively influence associated disturbance of the *autonomic nervous system*, as identified in the many case reports in chiropractic and osteopathic texts and journals. (12, 13, 14, 15, 16)

However, cases of clinical evidence explaining and supporting these observations are available in the published literature. This has been primarily garnered by the chiropractic an osteopathic professions and to a limited degree in the medical literature. (17-19)

Aberrant somatosensory reflexes are seen as one of the elements involved in vertebral subluxations. Somato-autonomic afferent reflexes have traditionally only received limited attention in clinical applications. In 1997 Sato stated that in relation to '*autonomic reflex regulation, insufficient attention has been paid to the role of somatic afferents.*' (1) Relatively limited appreciation of this aspect of neurophysiology seems to have merged into orthodox allopathic practices, except for the manipulative sciences. As such, it could be said that this statement from some 20 years ago is still current, particularly from a therapeutic point of view. (20)

Early use of the term *autonomic nervous system* (ANS) was first proposed by Langley in 1898. (2) Three years earlier Palmer rationalised the importance of this aspect of the nervous system which he subsequently called the body's automatic functions. (3) Bannister stated that the ANS influence was delivered to '*every visceral organ in the body*.' (4) Jänig et al also state that '*virtually all organs and tissues except skeletal muscle fibres receive an autonomic innervation*' and that '*innervation of most blood vessels is only by sympathetic (vasoconstrictor) axons*.' (21, 22, 23, 24)

Autonomic visceral reflexes primarily regulate the body's non-somatic structures and functions. In this neurophysiological role, these functions are regarded as homeostatic reflexes and can be activated by visceral, special senses, humoral, or somatic stimulation depending on the site, strength, or spinal level of somatic activation. (6, 7)

Sato and Swenson applied lateral pressure on rats' vertebrae and detected definitive somatovisceral reflexes affecting blood pressure, heart rate, and renal nerve activity. This simulated mechanical input tends to replicate stimulation of the spine creating noxious activation from the fixation or displacement of spinal segments. Fixations are seen by chiropractors as a frequent factor in many VSCs with the potential to affect the function of visceral structures via the

ANS. Lantz states that '*every connective tissue component of an articulation is affected by immobilisation. This immobilisation is termed by chiropractors as a fixation and comprises a central element of the vertebral subluxation complex along with segmental displacement.*' (25, 26, 27, 28, 29)

Somatic structures, particularly articular surfaces and especially vertebral articular facets are rich in mechanoreceptors. Patterson states that the spinal column is so extensively and richly innervated with mechanoreceptors '*it seems to operate as one vast proprioceptive organ.*' These sensitive receptors continually feed sensory information through the ANS to the brain in order to maintain homeostasis as well as monitor and coordinate body movement and status including both protopathic (nociception) and proprioception (position) senses. This evidence suggests that both chronic and acute noxious insults have the potential to develop somato-autonomic reflex activity and subsequently somatovisceral pathophysiology associated with the vertebral subluxation complex. (30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42)

Articulations disturbed by trauma, inflammation, hypermobility, hypomobility (vertebral impedance), stasis, aberrant movement, or micro-displacement (43) of articulations in particular lead to somatosensory activation. This can result in a barrage of neural firing from a range of different activated mechanoreceptors. These may lead to associated somato-autonomic neural reflex arcs involving innervation of organs and muscles. Under the facilitation hypothesis this hyper-stimulated irritation may then stimulate the intrinsic segmental muscles (e.g. rotators) resulting in their hypertonicity and vertebral segmental restrictions (VSC) which could further contribute to excitation of somatovisceral reflexes, and potentially organ dysfunction. (12, 13, 44)

Somatovisceral reflexes

In somatovisceral reflexes, somatic sensory input from peripheral stimuli enters the sympathetic portion of the ANS either through dorsal root ganglia, cranial nerve ganglia, or nuclei. The reflex then emerges from the lateral horn of the spinal cord, or from a cranial nerve nucleus, to a ganglion as a presynaptic efferent branch passing through as a postganglionic fibre to the target structure. The return reflex arc may then be completed via the afferent branch. Anatomically, the afferent branches of the somatovisceral reflex circuits follow similar pathways as many somatic and special senses activate autonomic responses. It may also be noted that viscerosomatic reflexes can result in irritated muscular splinting (as in muscular guarding associated with acute appendicitis) which may in turn also stimulate further contraction of the intrinsic segmental spinal muscles. (44, 45, 46, 47) This neural activation may lead to further noxious somatic irritation and stimulation of somato-autonomic reflexes as elements of the vertebral subluxation complex.

In noting that the stimulation of spinal nerves can affect visceral organs, Sato et al further define this spinal segmental reflex being '*... elicited when spinal nerves originating at specific segmental levels are stimulated. The segmental afferent nerves modulate visceral organs via autonomic efferent nerves or modulate them indirectly by affecting visceral afferent input.*' (30) As with much conventional research, Sato et al used animal subjects with appreciable correlation with medical physiology in this extensive work which cites some 750 basic scientific papers. (48, 49)

In merging the concepts of the pathophysiological and pathomechanical phenomena involving activated vertebrogenic somato-autonomic and somatovisceral reflexes, the more comprehensive term of *Somato Autonomic Visceral Complex* (SAVC) is offered as a contextual description. This serves to imply the broader significance of this mechanical and neurophysiological phenomenon, as they relate to the manipulative sciences. To normalise this physical-mechanical (somatic) disturbance, the term *Somato-autonomic Complex* (SAC) implies a physical corrective influence to certain visceral functions. (20)

Where there is a dominant spinal enablement, it is further suggested that this SAVC complex may be more accurately identified with the definitive term of Vertebral Autonomic Visceral Complex (VAVC). This would differentiate the spine from the somatic influence originating from other somatic structures initiating autonomic reflexes. This would identify VSCs as a source of influence both physiologically and therapeutically in the manual sciences.

The pathoneurophysiological impact of noxious somatic activity upon somato-autonomic reflexes should be more widely recognised clinically. In addition to innocuous physiological tone, (31) a barrage of chronic neural impulses from noxious somatic input originating from highly activated mechanoreceptors, proprioceptors, and nociceptors, may be further facilitated within a vertebral subluxation impact on autonomic functions. This may be further influenced by such initiating factors including severity, duration, type, and recurrence or persistence patterns. Stimulated receptors contained in cartilaginous facet surfaces, ligamentous, capsular, spinal musculature, and tendon tissue would activate such reflex registration. This reflex arc may also encompass and be reflected in the tone of individual muscles. (50, 51, 52, 53, 54, 55)

Examples of somato-autonomic disturbances can be clinically noted in instances of cervicogenic headaches, heart rate variability, and a range of nociceptive syndromes. Given these precedents, it is then reasonable to assert that similar reflexes involving other spinal segmental levels may impact on other internal organs as somatovisceral pathophysiology and syndromes. (56, 57, 58, 59)

Variable factors of activation could include the articular segmental level, duration, type and severity of somatic disturbance, as well as health status and age of the patient. Passatore et al noted that not only may the more severe grades of whiplash associated disorders (WADs) affect the spino-medullary regions, but lower grades may also disrupt vertebrobasilar circulation. They opined that both '*major and minor neurological symptoms*' of regions supplied by these arteries may be affected as a result of even minor trauma.(60)

Definitive evidence of a somatic impact on the autonomic nervous system was noted by Hakim and Grahame in 2004 and Durand and Daniels in 2020. (61, 62) These authors recommended wider recognition of this articular dysfunction as it has been '*overlooked by physicians*'. They found joint hypermobility in 174 hospitalised patients presenting many different autonomic-related symptoms, including:

- Presyncope - fainting, feeling faint, dizziness, light-headedness;
- Cardiorespiratory - chest pain, shortness of breath, palpitations;
- Gastrointestinal – nausea, stomach ache, diarrhoea, constipation; and
- Fatigue, joint pains, anxiety, depression, migraine, allergy, rash, nocturia, dysuria, flushing, night sweats, fever, lymph gland pain, poor sleep.

Although nerve root compression (NRC) may be an extreme example, the associated symptoms and pathology could be assessed as severe. However, in the medical literature, there seems to be relatively little consideration of degrees of NRC such as minor irritation. One can imagine a sudden leap from no compression to severe compression. Consequently it is suggested that there may also be varying degrees of nerve root compromise, from chronic or minor noxious sensory irritation to moderate input through to severe sensory bombardment, resulting from tissue damage due to structural change. (62)

These sensory and mechanical articular disturbances would be common clinical presentations for chiropractors. Such findings may also be a part of serious injury at other levels and overshadowed by the severity of the primary site(s).

In his authoritative volume on the spine, Hadley also noted a range of possible vertebrogenic symptoms related to a chronic cervical syndrome. These include sudden sharp facial or throat

pain, sensory disturbances such as vertigo, tinnitus, and diminished hearing as well as superficial vasomotor disturbances. (63)

These symptoms have the potential to impact a patient's quality of life, personal tensions, and possibly relationship stress. As such, spinogenic conditions may expose psychosocial issues which may also need to be considered clinically. (64, 65, 66)

Other clinical signs

Other clinical signs of these fixated articulations can be readily detectable and accessible to manipulative practitioners. Under this model, normalising their function would be expected in order to ameliorate associated neural reflex activity and the associated signs and symptoms. (41, 42, 43, 44, 45) In addressing these segmental fixations (subluxations), Mieritz et al found that aberrant segmental motion was smoother following spinal manipulation. (67, 68, 69, 70, 71, 72)

Clinically, localised neck and lower back pain, articular tenderness, sciatica, and headaches (as in cervicogenic headache) characteristically represent this neurological association. There can however be a number of other neurological signs and symptoms with examples such as paraesthesia, muscle weakness, altered reflexes, and dyskinesia. (73)

The published evidence from the manipulative sciences, including clinical observations, recognise the manual manipulative removal or modification of the noxious somatic input which are deemed to positively influence the variety of signs, symptoms, and pathophysiology. (17, 38, 74)

It has taken decades for some traditional allopathic theorists to formally accept that disturbed spinal segments could even exist as a vertebral subluxation, let alone be adjusted. Gradual acceptance has now been acknowledged by some as mechanical lower back and neck pain. More recently, the concept of cervicogenic headaches has become recognised as a pathophysiological condition which can be manually addressed. Currently, a similar discordant resistance persists regarding somatovisceral pathophysiology, but now recognised by some as being amenable to manual care. Such resistance is not new in conservative medicine. (75, 76, 77, 78)

The term *cervicogenic headache* has now been afforded formal recognition as a vertebrogenic condition. It is a recognised category under the 2017/18 edition of the World Health Organisation's (WHO) ICD 10 as diagnostic code R51. Further, a *cervicocranial syndrome* is allocated as code M53.0. In addition, the 2013 edition of the International Classification of Headache Disorders (ICHD-3) classifies a *Cervicogenic Headache* as code 11.2.1. This designation can now be regarded officially as a '*medical*' term. Moreover, the *Subluxation complex* (vertebral) is also finally recognised and coded as item M99.1 in the 2017/18 WHO's ICD-10. (79, 80, 81)

The influence of somatovisceral and somato-autonomic reflexes are intrinsic to neurophysiology. It would seem logical for noxious spinal initiated reflexes to be the central elements which may offer a means through which it is possible to reduce the noxious input and its associated pathophysiological influence on visceral functions. For decades these principles have been applied directly in chiropractic and osteopathic spinal models of manipulative care, with the aim to improve or restore physiology at the involved level when it is indicated. This has been the rationale for removing the somatic irritant to restore or normalise the somato-autonomic reflex cycle.

Both mechanically and functionally disrupted vertebral segments have been shown to be neurologically and clinically influential and largely correctable in neutralising associated spine-related consequences, depending on the severity of involvement. The degree to which this may be a factor at the pathological level has yet to be clearly demonstrated, although both recent and early research evidence of this does exist in both human and animal subjects. (38, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91)

The axial proximity of prolific mechanoreceptors in segmental articulations, their potential for subtle, chronic, and hyper stimulation, together with their mechanical import to the spine and central processing reflex centres through the spinal cord, would tend to indicate the potential for significant neurological influence. As such, this would have potential to provide a somatic gateway to influence the ANS through manual input. (4, 92)

An irritated Autonomic Nervous System (ANS) can be associated in a wide range of disturbed physiological (pathophysiology) functions. These can be factors in such examples as inflammation, pain, immunity, endocrine production, cardiac and gastrointestinal activity. Despite awareness of this over many decades, as recently as 2014, Amiya and colleagues recognised the mechanism but acknowledged that such '*detailed mechanisms of this interrelationship have not been clearly explained.*' (93, 94, 95)

A decade before Sato's 1997 statement, (29) Wallin stated that '*If spinal reflex responses are true mass responses, i.e. if they extend also to visceral sympathetic nerves, this factor would attain greater relative importance.*' (94) Sato and colleagues raised this profile, and demonstrated how aberrant somatovisceral reflex responses can indeed impart such physiological influence. (96)

Conclusion

The prospect of manipulative care providing benefit for some spine-related conditions to some patients in a conservative minimally-interventionist manner, the potential to affect particular cases of pathophysiological visceral dysfunction, is plausible. It may also be possible as a predisposing factor or association to some more complex conditions, and should be subjected to extensive ongoing meaningful research. This should expand the chiropractic and osteopathic sources and concepts that are already available in order to more fully appreciate the neurophysiological subtleties involved in the subluxation complex.

> 'This chapter focuses on the description of the location, structure and function of those somatosensory receptors whose activation has been shown experimentally and clinically to have predictable effects on one or another autonomic effector organs… and make use of their potential autonomic effects for therapeutic reasons.' Sato Sato Schmidt (48) p 8

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Cite: Rome P. Waterhouse JD. Neurodynamics of vertebrogenic somatosensory activation and Autonomic Reflexes - a review: The introduction to a clinical series. Asia-Pacific Chiropr J. 2021;1.4. URL apcj.net/papers-issue-2-4/#RomeWaterhouseIntroduction

References

1. Rome P, Waterhouse J, Maginness G, Ebrall P. Medical management of infantile colic and other conditions with spinal manipulation: a narrative review of the European medical literature. J Contemp Chiro. 2019;2:60-75.
2. Rome PL, Waterhouse JD. Evidence informed vertebral subluxations – a diagnostic and clinical imperative. J Phil Princip Prac Chiropr. 2019; Dec/12-34
3. Rome PL. Commentary: medical evidence recognising the vertebral subluxation complex. Chiropr J Aust. 2016;44(4):304-7
4. Pickar JG. Neurophysiological effects of spinal manipulation. Spine J. 2002;2(5):357-71.
5. Welch A, Boone R. Sympathetic and parasympathetic responses to specific diversified adjustments to chiropractic vertebral subluxations of the cervical and thoracic spine. J Chiropr Med. 2008;7(3):86-93
6. Leith JL, Koutsikou S, Lumb BM, Apps R. Spinal processing of noxious and innocuous cold information: differential modulation by the periaqueductal gray. J Neurosci. 2010;30(14):4933-42.
7. Navid MS, Lelic D, Niazi IK, et al. The effects of chiropractic spinal manipulation on central processing of tonic pain – a pilot study using standardised low-resolution brain electromagnetic tomography (sLORETA. Sci Rep. 2019;9;6925. doi: 10.1038/ s41598-019-42984-3
8. Rome P, Waterhouse JD. Noxious Somato-Autonomic reflex influence upon smooth muscle: Its integration with vascular tone and perfusion. A review. Asia-Pac Chiropr J. 2020;1.2:online only. URL https://apcj.rocketsparkau.com/ noxious-somato-autonomic-reflex-influence-upon-smo/
9. Grieve GP. Thoracic joint problems and simulated visceral disease. In: Grieve GP (Ed) Modern Manual Therapy of the Vertebral Column. Churchill/Livingstone, New York, 1986:390.
10. Murtagh J. Spinal dysfunction. Chapter 24. General practice. 5th Edn. North Ryde. McGraw-Hill 2012. http:// murtagh.mhmedical.com/content.aspx?sectionid=116026062&bookid=1522&Resultclick=2
11. Davis C. Chronic pain/dysfunction in whiplash – associated disorders. J Manipulative Physiol Ther 2001;24(1):44-51.
12. Gatterman MI. Foundations of chiropractic subluxation. 2nd ed. St. Louis, Miss. Elsevier Mosby; 2005.
13. Leach RA. The chiropractic theories, principles and clinical applications. 3rd edn. Baltimore: Williams & Wilkins. 1994.
14. Redwood D, Cleveland CS. Fundamentals of Chiropractic. St Louis, Mosby.2003
15. Chila A. Foundations of osteopathic medicine. 3rd edn. Philadelphia, Lippincott Williams and Wilkins. 2010
16. King HH, Jänig W, Patterson MM. (Eds) The science and clinical application of manual therapy. Edinburgh. Churchill Livingstone Elsevier 2011
17. Index to Chiropractic Literature. https://www.chiroindex.org/#results
18. National Library of Medicine. https://pubmed.ncbi.nlm.nih.gov/advanced/
19. Osteopathic Medical Digital Repository. https://ostemed-dr.contentdm.oclc.org/digital/search/advanced
20. Sato A. Neural mechanisms of autonomic responses elicited by somatic sensory stimulation. Neurosci Behav Physiol 1997;27(5):610-621
21. Langley JN. On the union of cranial autonomic (visceral) fibres with the nerve cells of the superior cervical ganglion. J Physiol 1898;23(3):240-270
22. Palmer DD. The Science, art and philosophy of chiropractic. Portland. Portland Printing House. 1910:865
23. Bannister R. In: Autonomic failure: A textbook of clinical disorders of the autonomic nervous system. 2nd edn.Oxford. Oxford University Press. 1988;1
24. Jänig W, Keast JR, McLachlan EM, Neuhuber WL, Southard-Smith M. Renaming all spinal autonomic outflows as sympathetic is a mistake. Auton Neurosci 2017;206:60-62
25. Functions of the autonomic nervous system.. In: Boundless anatomy and physiology. https://courses.lumenlearning.com/ boundless-ap/chapter/functions-of-the-autonomic-nervous-system/
26. Pomeranz B, Macaulay RJ, Caudill MA. Assessment of autonomic function in human by HR spectral analysis. Functions of the autonomic nervous system.. In: Boundless anatomy and physiology. Am J Physiol 1985;248(1 Pt 2):H151-H153
27. Janjowska EA, Ponikowski P, Piepoli MF, et al. Autonomic imbalance and immune activation in chronic heart failure – pathophysiological links. Cardiovasc Res 2006;70(3):434-445
28. Sato A, Swenson RS. Sympathetic nervous system response to mechanical stress of the spinal column in rats. J Manipulative Physiol Ther. 1984;7(3):141-147
29. Lantz CA. Immobilisation degeneration and fixation hypothesis of chiropractic subluxation. Chiropr Res J 1988;1(1):21-46
30. Patterson MM. Somatic dysfunction in osteopathic medicine. http://www.chiro.org/LINKS/ABSTRACTS/ The_Role_of_Subluxation_in_Chiropractic.shtml

31. Blecher R, Krief S, Galili T, et al. The proprioceptive system masterminds spinal alignment: insight into the mechanism of scoliosis. Dev Cell 2017;42(4):388-399. e3. doi: 10.1016/j.devcel.2017.07.022

32. Briggs L, Boone WR. Effects of chiropractic adjustments on changes in pupillary diameter: a model for evaluating somatovisceral response. J Manipulative Physiol Ther 1988;11(3):181-189

33. Hölzl R, Möltner A, Neidig CW. Somatovisceral interactions in visceral perception: abdominal masking of colonic stimuli. Integr Physiol Behav Sci 1999;34(4):269-284

34. LeBoeuf-Yde C, Axén I, Ahlefeld G, et al. The types and frequencies of improved nonmusculoskeletal symptoms reporte after chiropractic spinal manipulative therapy. J Manipulative Physiol Ther 1999;22(9):559-564

35. Lephart SM, Borse PA. Proprioception: the sensations of joint motion and position. http://www.hqh.com/webfiles/HighestQualityHealthNZ/files/Acceleration_Training_-_Information_on_Proprioception.pdf. Extracted Dec 13, 2017

36. Nansel D, Szlazak M. Somatic dysfunction and the phenomenon of visceral disease simulation: a probable explanation for the apparent effectiveness of somatic therapy in patients presumed to be suffering from true visceral disease. J Manipulative Physiol Ther 1995;18(6):379-397

37. Budgel B, Hotta H, Sato A. Spinovisceral reflexes evoked by noxious and innocuous stimulation of the lumbar spine. J Neuromuscul Syst 1995;3:122-131.

38. Budgell B, Sato A. Modulations of autonomic functions by somatic nociceptive inputs. In: Progress in Brain Research. Amsterdam: Elseview. 1996;13:525-539

39. Sato A. Somatovisceral reflexes. J Manipulative Physiol Ther 1995;18(9):597-602

40. Tsao H, Galea MP, Hodges PW. Reorganisation of the motor cortex is associated with postural control deficits in recent low back pain. Brain 2008;131:2161-2171

41. Wyke B. Articular neurology – a review. Physiotherapy 1972;58(3):94-99

42. Zimny ML. Mechanoreceptors in articular tissues. Am J Anat 1988;182(1):16-32

43. Hodges PW, Cholewicki J, Jacek D, van Jaap H. Is spine 'stability' the best term – or should it be stiffness. In: Spinal control: the rehabilitation of back pain E-book. State of the art and science. London. Elsevier. 2013

44. Jänig W, Häbler HJ. Specificity in the organisation of the autonomic nervous system: a basis for precise neural regulation of homeostatic and protective body functions. Prog Brain Res 2000;122:351-367

45. The autonomic nervous system 15.2 Autonomic reflexes and homeostasis. Anatomy & Physiology. Rice University. Open Stax CNX. Feb 26, 2016 http://cnx.org/contents/14fb4ad7-39a1-4eee-ab6e-3ef2482e3e22@8.24

46. Nicholson S. Your back and abdominal muscles and the dangers of muscle spasm and guarding. Low Back Pain Program. https://lowbackpainprogram.com/back-and-abdominal-muscles/

47. Humes DJ, Simpson J, Acute appendicitis, BMJ 2006;333(7567) 530-531

48. Sato A, Sato Y, Schmidt RF. The impact of somatosensory input on autonomic functions. In: Blaustein MP, Grunicke H, Pette D, Schultz G, Schweiger M. (eds). Rev Physiol Biochem Pharmacol. Berlin: Springer;1997;130:4

49. Budgell B. Autonomic responses to spinal pain. Rigakaryoha Kagasku 2000;15(3):81-87.

50. McDowall D, Emmanuel E, Chaseling M. Tone as a health concept: An analysis. Complementary Ther Clin Prac. 2017;29:27-34.

51. Swinkels A, Dolan P. Spinal position sense is independent of the magnitude of movement. Spine. 2000;25(1):98-104.

52. de Vries J, Ischebeck BK, Voogt LP, et al. Joint position sense error in people with neck pain: a systematic review. Man Ther. 2015;20(6):736-744

53. Holt KR, Haavik H, Lee ACL, Murphy B, Elley CR. Effectiveness of chiropractic care to improve sensorimotor function associated with falls risk in older people: a randomised controlled trial. J Manipulative Physiol Ther. 2016; DI:https://doi.org/10.1016/j.jmpt.2016.02.003

54. Smith DL, Haug MJ, Walsh MS. The effect of posture on neck proprioception and head/neck stabilisation in asymptomatic participants. J Canad Chiropr Assoc. 2019;63(2):100-10

55. Haavik H, Murphy B. The role of spinal manipulation in addressing disordered sensorimotor integration and altered motor control. J Electromyogr Kinesiol. 2012;22(5):768-776.

56. Uchida S, Kagitani F. Autonomic nervous regulation of ovarian function by noxious somatic afferent stimulation. J Physiol Sci. 2015;65:1-9

57. Korim WS, Egwuenu E, Fong AY, et al. Noxious somatic stimuli diminish respiratory-sympathetic coupling by selective resetting of the respiratory rhythm in anaesthetised rats. Exper Physiol 2012, 97;DOI: 10.1113/expphysiol.2012.066365

58. Clement C, Keay K, Podzebenko K, Gordon B, Bandler R. Spinal sources of noxious visceral and noxious deep somatic afferent drive onto the ventrolateral periaqueductal gray of the rat. J Comp Neurol. 2000;425ֵ3):323-344

59. Verne GN, Robertson ME, Price DD. Representations of pain in the brain. Curr Rheumatol Rep. 2004;6(4):261-265

60. Passatore M, Roatta S. Influence of somatic nervous system on sensorimotor functions: whiplash associated disorders (WAD) as a model. Eur J Appl Physiol 2006;98:423-449

61. Hakim AJ, Grahame R. Non-musculoskeletal symptoms in joint hypermobility syndrome: indirect evidence for autonomic dysfunction. Rheumatology 2004;43(9):1194-1195. https://doi.org/10.1093/rheumatology/keh279

62. Durand WM, Daniels AH. C4 root compression leads to phrenic nerve palsy with shortness of breath, chest pain, and elevated hemidiaphragm. Lancet . 2020 Oct 10;396(10257):1101. doi: 10.1016/S0140-6736(20)32027-4.

63. Hadley LA. Anatomico-roentgenographic studies of the spine. Springfield,. Charles C Thomas.1976;438.

64. Jamison JR. A psychological profile of fibromyalgia patients: a chiropractic case study. J Manipulative Physiol Ther 1999;22(7):454-457

65. Langworthy JM, Breen AC. Psychosocial factors and their predictive value in chiropractic patients with low back pain: a prospective inception cohort study. Chiro Osteop 2007;15:5

66. Rhudy JL, McCabe KM, Williams AE. Affective modulation of autonomic reactions to noxious stimulation. Int J Psychophysiol 2007;63(1):105-109

67. Boal RW, Gillette RG. Central neuronal plasticity, low back pain and spinal manipulative therapy. J Manipulative Physiol Ther 2004;27(5):314-326

68. Budgell BS. The reflex effects of subluxation: the autonomic nervous system. J Manipulative Physiol Ther. 2000;23(2):104-106.

69. Leach RA. (12) pps 89-119, 201-290, 373-394)

70. Sato A, Sato Y, Schmidt RF. (47) (pps 38-54)

71. Seaman DR, Faye LJ. The vertebral subluxation complex. In: Gatterman MI. (11) p 195-244.

72. Mieritz RM, Hartvigsen J, Boyle E, Jakobsen MD, Aagaard P, Bronfort G. Lumar motion changes in chronic low back pain patients: a secondary analysis of data from a randomized trial. Spine J 2014;14(11):2618-2627

73. Goodley PH. What happens when joints dysfunction. Chapter 6. In: Release from pain. Essentials of orthopaedic medicine. 2nd edn. Telz Stone, Israel. Self-Published. 2005. [e –book). [http://drgoodley.com/goodley/live/]

74. Sato A, Schmidt RF. The modulation of visceral functions by somatic afferent activity. Jpn J Physiol. 1987;37(1):1-17.

75. Winters RW. Accidental medical discoveries. New York: Skyhorse Publishing;2016.

76. Vincent MB. Cervicogenic headache: a review comparison with migraine, tension-type headache, and whiplash. Curr Pain Headache Rep. 2010;14(3):238-43

77. Becker WJ. Cervicogenic headache: evidence that the neck is a pain generator. Headache. 2010;50(4):699-705

78. Yi X, Cook AJ, Hamill-Ruth RJ, Rowlingson JC. Cervicogenic headache in patients with presumed migraine: missed diagnosis or misdiagosis. J Pain. 2005;6(10):700-3

79. World Health Organisation's publication, World Classification of Diseases (ICD-10) 2017/18. http://www.icd10data.com/search?s=cervicogenic

80. The International classification of headache disorders, 3rd edn. 2013. Cephalalgia. 2013;33(9):629-808. http://www.ihs-headache.org/binary_data/1437_ichd-iii-beta-cephalalgia-issue-9-2013.pdf

81. World Health Organisation's publication, World Classification of Diseases (ICD-10) 2017/18. http://www.icd10data.com/search?s=M99.1

82. Agocs S. Cleveland's rabbits: the use of animals to study vertebral subluxation. Chiropr Hist. 2016;36(1))63-69.

83. Burns L, Chandler LC, Rice RW. Pathogenesis of visceral disease following vertebral lesions. .Am Osteop Assoc, Chicago 1948. (Note Dr Burns has also published at length on her extensive research, particularly in the J Am Osteop Assoc and the AT Still Research Institute Bulletin.

84. Cleveland CS. Researching the subluxation of the domestic rabbit: a pilot research program conducted at the Cleveland Chiropractic College. Pub Cleveland Chiropractic College, Kansas City, Missouri, 24pps. (See also Sci Review Chiropr. Aug 1965; (4):5-28 .

85. Cramer G, Henderson C, Khalsa P, Pickar J. Animal models. In: Basic science research related to chiropractic spinal adjusting: the state of the art and recommendations. J Mnipulative Physiol Ther 2006;29(9):726-761.

86. Henderson CNR. Animal models in the study of subluxation and manipulation: 1964-2004. In:Gatterman MI. (11) p 47-103.

87. Jinkins JR, et al. Autonomic dysfunction associated with disc extrusions. Am J Neuroradiol 1989;10:219-231.

88. Jinkins JR. The pathoanatomic basis of somatic, autonomic and neurogenic syndromes originating in the lumbosacral spine. In: Giles LGF, Singer KP. Clinical anatomy and management of low back pain. Jordan Hill, Oxford-Butterworth-Heinemann. 1997;255-272

89. McGregor M, Wiles MR, Grice AS. The present use of guinea pigs in chiropractic research. J Canad Chiropr Assoc. 1980;24(3):101-107.

90. Schey WI. Vertebral malformations and associated somatovisceral abnormalities. Clin Radiol 1976;27:341-353.

91. Rosner A. Getting down to brass tacks: the neurophysiology of spinal manipulation. Dynamic Chiropr 2015;33(17). https://www.dynamicchiropractic.com/mpacms/dc/article.php?id=57469

92. Vernon H. Historical overview and update on subluxation theories. J Chiropr Humanit. 2010;17(1):22-32.

93. Amiya E, Watanabe M, Komuru I. The relationship between vascular function and the autonomic nervous system. Ann Vasc Dis. 2014;7(2):109-119.

94. Touj S, Houle S, Ramia D, et al. Sympathetic regulation and anterior cingulate cortex volume are altered in a rat model of chronic back pain. Neuroscience 2017;352:9-18

95. Borovikova LV, Ivanova S, Zhang M, et al. Vagus nerve stimulation attenuates the systemic inflammatory response to endotoxin. Nature 2000;405:458-462.

96. Wallin BG. Intraneural recordings of normal and abnormal sympathetic activity in man. In Bannister R. In: Autonomic failure: A textbook of clinical disorders of the autonomic nervous system. 2nd edn. Oxford. Oxford University Press. 1988;192