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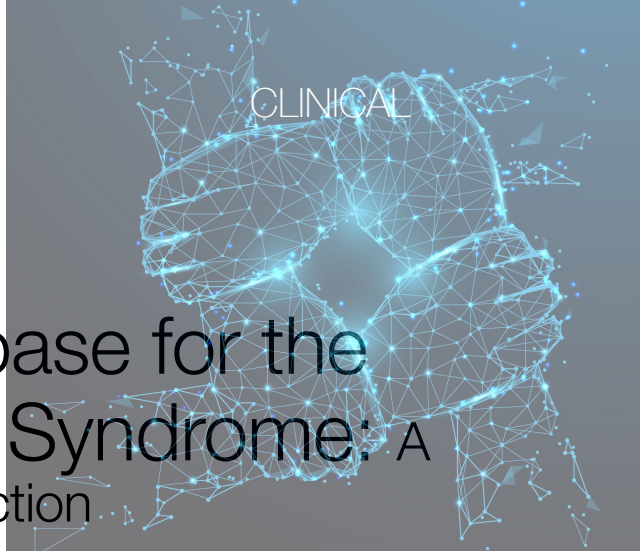

A neurological evidence base for the Vertebrogenic Dyspepsia Syndrome: A somatosensory link to visceral dysfunction

Peter Rome and John Waterhouse

Abstract: Literature is reviewed which portrays the neurophysiological basis and clinical recognition for the spinal manipulative rationale of a subluxation-related vertebrogenic form of dyspepsia. It discusses the relationship of the cervical spine and the vagus parasympathetic innervation, and the dorsal spine with its sympathetic and spinal nerve influence. Practitioners of chiropractic, medicine, osteopathy, acupuncture, and physiotherapy have all published on this topic. Two key spinal regions appear to be involved - the cervical spine's influence on the parasympathetic vagus, and the sympathetic innervation through the splanchnic innervation of the mid-dorsal spinal nerves.
No evidence could be located which contradicted the neurophysiology of this biomechanical model of care for the condition. With physiological evidence of positive outcomes, a vertebrogenic biomechanical focussed model of care is justified as one of the options for functional dyspepsia (FD).

Indexing terms: Vertebrogenic; functional dyspepsia; dyspepsia; subluxation; GERD; indigestion gastrointestinal.

Introduction

In animal subjects ' *... both noxious and innocuous stimulation of somatic afferents have been shown to evoke reflex changes in sympathetic efferent activity and, thereby, effector organ function. These phenomena have been demonstrated in such sites as the gastrointestinal tract, urinary bladder, adrenal medulla, lymphatic tissues, heart and vessels of the brain and peripheral nerves.*' (1)

The term functional dyspepsia (FD) has been a part of medical lexicon for about 250 years. It applies to an idiopathic form of indigestion. The early papers located which recognised the condition range from 1773. (2, 3) In 1908 six papers were published in the Royal Society of Medicine in its first volume, (4) with a second paper published in the BMJ of the same year. (5) Subsequently, according to the PubMed Timeline, relatively few papers were published on the topic until 1986 when numbers started to rapidly increase.

The term (FD) only seemed to gain favour about 1990. The fact that it has been deemed to be of no known cause has led to pharmaceutical treatment(s) being based on empirical observations of what seems to work for some. Some off-label medications are prescribed too. (6, 7, 8)

Dyspepsia has been identified by Oustamanolakis and Tack as being in two forms, FD and organic. The latter is subject to identifiable causes such as

> *... the idea that 'heartburn' (dyspepsia) has no mechanical aetiology is fatally flawed. Regulatory bodies which control how chiropractors communicate to patients must appreciate the evidence presented by Rome and Waterhouse and better inform their regulatory decision-making ...'*

ulceration, infections, cancer and food allergies. They ascribe a pathophysiological mechanism to FD which includes an autonomic nervous system-central nervous system dysregulation. (9)

The evidence of a vertebrogenic neurological factor in a functional conditional of dyspepsia is available in the literature from chiropractic, medicine and osteopathy and physiotherapy. Further, there is mounting recognition of a vertebrogenic factor in the management of a range of so-called non-musculoskeletal conditions in medical literature as well. (10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26),

This vertebrogenic model would consider some forms of '*functional dyspepsia*' appropriate for manual intervention. Those forms could include dysfunction of sphincter hyper- or hypo-tonicity and gastric motility (e.g. gastroparesis, achalasia) as examples. When indicated by symptoms and spinal clinical signs, manipulative management has been applied for these conditions on the basis of modifying the somato-autonomic effects. It is noted that the upper gastrointestinal sphincters (and certain others) can be activated under involuntary or voluntary control. (27, 28, 29, 30)

Medical papers state clearly that functional dyspepsia is an enigma in cause, management and outcomes. There is a range of medical treatments available for patients with this condition. It is often more a matter of trying various medications to see which one might relieve the symptoms. (31, 32, 33, 34)

Functional dyspepsia varies from 5%-25% of the general population with approximately 30% non-ulcer dyspeptic adults having impaired gastric emptying. (35) This percentage indicates a reasonably common idiopathic condition which traditionally, has no definitive aetiology and no generally accepted consistent pharmaceutical answer.

A number of visceral conditions have been designated as functional. That is, they do not necessarily exhibit pathological tissue changes or positive laboratory tests, but are exhibiting symptomatic dysfunction – perhaps somewhat of a catch-all term.

Clarification of terms

Central excitation

This term is defined in the Mosby medical dictionary as '*A model based on neurophysiologic findings that explains the symptoms of subluxogenic pain and discomfort that arise from non-spinal sites*.' That is, a symptom (or sign) attributed to a vertebral origin where the segment has been disturbed to the extent that activated noxious sensory firing may be carried by a somatovisceral or somato-somatic reflex. (36)

Central integration

Central integration indicates a focus on the changes in cortical intrinsic inhibitory interactions from somatosensory evoked potential stimulation. (37, 38)

Dysfunction

It is suggested that one form of visceral dysfunction can be due to subluxogenic neural pathophysiology which may occur through noxious somato-autonomic reflexes. The dysfunction may be considered on three levels forming a somato-autonomic-visceral triad which is essentially a subluxation complex. (39)

Dysfunction - vertebral

This is the primary somatic element of a vertebral subluxation complex. It comprises a disturbance of the function and/or the position of a spinal segment(s). Facet fixation is also a common feature. All have the potential to activate noxious neurogenic autonomic and somatovisceral reflexes. (40, 41)

Dysfunction - Neurophysiological

As a result of the vertebral dysfunction, the segmental disturbance can have a direct influence upon neural efferent elements through activating noxious sensory reflexes. This somatosensory stimulation has been shown to activate somato-autonomic nerve reflexes. These effectively influence the function of the innervated or target structure through somatovisceral or somato-somato reflex pathways. (42, 43, 44, 45)

Dysfunction - Innervated organ

Under this model, the hyperstimulation of neural innervation may lead to an organ's altered function – as in dysfunctional dyspepsia. Depending on the spinal level involved, it may be clinically observed as signs and symptoms of other autonomic somato-autonomic reflexes. For example, these effects may be reflected viscerally as symptoms of:-

- Dysphagia; (46)
- Dysphonia; (47)
- Dyspnea; (48)
- Dyspepsia; (49)
- Sphincter contraction; (50)
- Gastrointestinal motility; (51)
- Smooth muscle function. (52)

Functional

Such a vague term as *'functional'* alludes to symptomatic presentations associated with an organ or structure without apparent or demonstrable explanation(s). In a paradox, if something is functional it is considered to be operating normally.

In some conditions, the term '*functional*' could suggest that a cryptic neural element may exist – although often identified in an idiopathic association. The vertebrogenic factor is closely associated with chiropractic and osteopathy; which may provide that missing link in a general understanding of the complexity of a vertebral subluxation. Apart from the idiopathic appellation, the qualifying term for a condition may also be known as primary dyspepsia (49), essential dyspepsia, (53) and non-ulcer dyspepsia. (54)

In regard to the acknowledged idiopathic designation of functional dyspepsia, it has remained an enigma despite the 1,851 (115 so far to Sept 30 in 2021) papers listing the condition under that title on papers in PubMed and 1,873 in a Medline search. For the condition to remain such an enigma for so long, should warrant research of the vertebrogenic-subluxation model. (55, 56)

Where an aetiology is obscure, it seems that a functional nomenclature may be added as more descriptive of the effect rather than a cause. This appears to be abstruse and somewhat meaningless categorisation as the range of conditions where it apples is so broad. They range in part from such examples as:

- Functional asplenism; (57)
- Functional constipation; (59)
- Functional diarrhea; (60)
- Functional dysphagia; (46)
- Functional dysphonia; (47)
- Functional dyspepsia; (49)
- Functional dystonia; (61)
- Functional emesis; (62)

- Functional rhinoplasty; (58)
- Functional seizures; (63)
- Functional tremor; (64)
- Psychogenic (functional) Parkinsonism. (65)

Functional dyspepsia

On one of its current websites, FD is described by the Mayo Clinic as a term ' … f*or recurring signs and symptoms of indigestion that have no obvious cause.*' (32) In 2018, Chavoustle and Silver stated that it ' *… is described as "functional" because there is nothing structurally wrong with the upper digestive area, but bothersome symptoms persist.*' (66)

Other gastrointestinal dysfunction conditions have also been noted. (35, 67, 68)

In 2017 Talley et al stated that for this condition of functional dyspepsia, '*The pathophysiology of functional dyspepsia is not completely understood.*' (69)

In 2018, Madisch and colleagues identify a need for definitive diagnosis, '*symptom-oriented*' treatment for functional dyspepsia. However they stated inter alia that '*There is as yet no causally directed treatment for functional dyspepsia.*' (33)

In 2019, Masuy et al state clearly that '*None of the available therapies is effective in the majority of patients without being associated with major side effects. Developing new treatment options is challenging due to the heterogeneity of functional dyspepsia, the lack of readily identified target mechanisms and the poor association between pathophysiological disturbances and symptoms.*' They state further that '*To date only limited treatment options are available and conflicting results in terms of efficacy have been reported. Consequently, nonpharmacological treatment options are increasingly being explored for functional dyspepsia*.' (70)

Rangan at Harvard Health was more definite when in 2020 he posted that FD '*is a common condition… without a clear cause*.' He also states that ' *… many patients do not experience significant symptom improvement with … the various medical treatment available to try*,' and that ' *… even the most effective FD medications only resolve symptoms in one out of six patients*.' (71)

More recently in 2020, Ford et al stated that FD is not completely understood and is difficult to treat in most patients. They stated that '*Approximately 80% of individuals with dyspepsia have no structural explanation for their symptoms and have functional dyspepsia. Functional dyspepsia affects up to 16% of otherwise healthy individuals in the general population.*' However, in what could be regarded as closer to a chiropractic neurological model, they state '*it is probably related to disordered communication between the gut and the brain, leading to motility disturbances, visceral hypersensitivity, and alterations in gastrointestinal microbiota, mucosal and immune function, and CNS processing*.' (72)

It appears that it has yet to be determined whether the associated symptoms (including hyperacidity - for which a proton pump inhibitor may be prescribed to suppress the gastric acidity - essentially hydrochloric acid) are signs, symptoms, contributors or causes of FD. Due to the intransigence of some cases, and as a last resort, tricyclic antidepressants and psychological therapy may be recommended medically. Under a subluxation model, many FD signs and symptoms could ultimately be considered vertebrogenic effects, and therefore potentially amenable to manipulative care. (73, 74)

Signs and symptoms of FD include: (75, 76)

- Epigastric pain
- Heartburn
- Bloating

- Excessive belching
- Nausea after meals
- Early satiation
- Vomiting
- Postprandial fullness

The *Mayo Clinic*'s website states that functional dyspepsia is common and can have long-lasting symptoms which 'have no obvious cause.' (77)

The *Canadian Society of Intestinal Research* acknowledged that functional dyspepsia had '*no observable or measurable structural abnormalities found to explain persistent symptoms*,' and further that '*The cause of functional dyspepsia is unknown.*' This would be consistent with the conventional medical policy of hesitancy in recognising the wider neural ramifications of subluxated or dysfunctional vertebrae. (78)

In reviewing current medical treatments, there is an implication of somewhat of a double standard in that medically, patients with FD may be treated even though the cause is unknown. Yet because chiropractic and osteopathy base their management of their vertebrogenic model outside the traditional medical model, some critics maintain that they should not accept patients for management of visceral symptoms. This appears contrary when the medical model may only be addressing a symptom, and yet admits not knowing the cause, especially, and when the clinical evidence of a vertebrogenic factor does exist in the medical literature, albeit latent. (33, 46, 51, 79)

In noting that FD presents a diagnostic, management and clinical dilemma for physicians, a comprehensive 2013 review Yarandi and Christie noted that '*hypersensitivity has been suggested to be perceived at a central sensory level, with glutamate as the potential neurotransmitter involved. This theory suggests that increased presynaptic release of glutamate in the central sensory areas facilitates transmission of visceral sensory signals, leading to an amplified response to non-painful stimuli and perception of pain. In addition, central hypersensitivity can potentially lead to activation of previously silent visceral nociceptors through recruiting more spinal neurons to the pain pathway.*' (34)

Given the notion that spinal manipulation for some conditions is an enigma to cynics, it is surprising that some in medicine (80) would suggest denying patients the chance of potential relief by suggesting there is no evidence to justify a trial of care, when published evidence and patient demand indicates otherwise.

Due to the lack of consistency regarding aetiology, medical treatment and efficacy, and persistent acceptance of functional dyspeptic patients it would be justifiable for other professions to continue under the same criteria. To avoid the appearance of double standards, patients should be free to seek equal opportunity and access to a potential option of care for this condition. The pathophysiological evidence and patient satisfaction strongly support the rationale for considering manipulative care.

Somatic dyspepsia (Somatisation)

Medically, this term somatic dyspnea tends to imply a somatisation projection of physical symptoms through psychological distress. The assumed terminology is related to mental classifications of functional overlay, psychogenetic conditions, and psychosomatic concepts. (81, 82)

Idiopathic symptoms seem to have attracted the descriptive '*somatic*'. This term implies a psychological, psychosocial, or psychiatric element projected as a physical manifestation. The authors of this paper consider that many of these dyspeptic patients may be experiencing a

vertebrogenic form of dyspepsia. The apparent and often protracted periods of somatisation seem to be more the result of being a primary physical manifestation first, followed by psychological distress. (83)

Conditions that defy physical or laboratory diagnosis may at times attract this diagnosis. In 1978, Sarno alluded to psychogenic backache as the '*court of last resort*'. (84)

Apart from the idiopathic appellation, another condition has also been known as primary dyspnea, (85) essential dyspnea and non-ulcer dyspepsia. (86)

As the vertebral subluxation complex (VSC) model has limited acceptance in medicine, it is not surprising that a psychological basis for some unexplained conditions has been adopted when the VSC is not part of the broader conventional medical spectrum as noted by Maigne, Lewit, Schmorl and Junghanns. (87, 88, 89, 90, 91)

Somatosensory

Sato et al extensively explore the effects of noxious input from somatosensory activation leading to modulation of visceral functions including gastrointestinal motility [p. 168]and somatosensory modulation of digestive secretion, [p. 180]as well as somatosensory modulation of gastrointestinal blood flow, [p. 183]and the somato-vagal reflex. [p. 185] (92)

Sato et al acknowledge the role of spinal manipulation, physiotherapy and acupuncture in the influence upon somatosensory activated somato-autonomic reflexes. (93) In a further study, Sato and Schmidt stated that '*Somato-gastrointestinal reflexes are well known clinically, and the influence of somatic afferent stimulation on gastro-intestinal motility has been reported for dogs, cats, monkeys, and humans*.' (94) This principle is supported conversely through a decrease of the somatosensory stimulation by Battakova and Shraimanov in their paper titled '*Neurophysiological changes in the afferent somatosensory system indices in the case of vertebrogenic spine pathology in miners*'. (95)

Somatovisceral

Somatovisceral is a term which recognises ' … *the influence of the body framework (soma) or neuromusculoskeletal system on the function of the internal body systems and organs*.' It primarily relates to this influence through segmental neural reflexes which are activated from noxious somatic sensory input to involve neural central processing and visceral innervation at that associated level. (96)

Sato et al have extensively demonstrated a distinct neural reflex association by stimulating the somatic cutaneous sensory afferents in animals. (1)

In a reflection of Russian medical practices, the physicians Pikalov and Kharin noted in 1994 patients' relief from gastrointestinal symptoms by spinal manipulation. While the physiological basis for this eluded them, they postulated that the normalisation in segmental trophic innervation as a possible mechanism. They reported gastric pain relief in patients after an average of 3.8 days and '*clinical remission*' 10 days faster than traditional care. (97)

Subluxation

Subluxation complex is a more specific term than vertebrogenic and has been adopted in relation to both structural and functional disturbances which initiate conditions originating from a segmental level.

For joints to be either fixated or functionally disturbed there must be more than osseous elements affected. In order to assess all the factors involved such as sensory modifications, soft tissue alterations, neural activation, and the cumulative effect of these components the following definition is offered:

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

Peripheral joints and paraspinal articulations as well as vertebral facets may be subluxated. In the case of vertebral subluxations in particular, further specificity as to the source of spinal involvement needs to be identified for optimal outcomes. Neurologically, specific vertebral facet(s) would be the expected focus in FD associating the neurological innervation with the symptomatic gastrointestinal structure involved.

In addition to the sympathetic nerve influence through the thoracic splanchnic nerves, parasympathetic innervation via the vagus also has an impact on gastrointestinal function, and may be influenced through the cervical spine.

A series of medical papers between 2001 and 2012 by Vaňásková and others concluded that cervical vertebral dysfunction '*may cause visceral dysfunction*.' They focused on the upper gastrointestinal tract motility and dysphagia. Their treatment of choice was cervical manipulation. They stated, '*Musculoskeletal (manual) medicine techniques can reduce pain and normalize the dysphagia. Using scintigraphy, this can be objectively measured*.' (46, 98, 99)

Biedermann noted that in infantile colic '*The definition of a functional disorder (is one) caused primarily vertebrogenically enables paediatricians, physiotherapists, speech therapists, and others who address infants and schoolchildren to widen their scope of available therapeutic options and to include the "functional approach" in their therapeutic considerations.*' (100, 101)

There is a degree of overlap of this topic on dyspepsia and abdominal symptoms with infantile colic. A study by Klougart et al. found that in the digestive complaint - colic, 41% of infants required vertebral adjustment of the mid-thoracic spine - primarily T4/5 to T8/9. (102, 103)

Subluxogenic

This term may be defined as a sign or symptom originating from a physiologically disturbed joint, often a vertebral articulation. Further, Mosby's medical dictionary indicates that subluxogenic is associated with central excitation. (36)

This term was also used in the report of the extensive review for the US Department of HEW as *The Research Status of Spinal Manipulative Therapy*. (104)

In another form of subluxation-related disturbance, Vernon and Gatterman raise the matter of subluxogenic signs in relation to cervicogenic headaches, as did Bryner in relation to sub-occipital trauma. (105, 106) They recorded. '*22% of those with indigestion reported some relief after chiropractic care. Compared with those reporting no relief, mid-back pain was more common among those reporting indigestion.*'

In identifying the associated symptoms or condition, the subluxogenic expression essentially differentiates the effects from its origin. Identification of the segment involved enables correction to be focussed. There would be no point in trying to manipulate a segment not neurologically related to a condition.

Subluxogenic dyspepsia

Based on the creation of noxious somatosensory autonomic reflexes originating from an articular dysfunction and/or displacement - a subluxation complex may involve a neurogenic-related visceral dysfunction as a pathophysiological phenomenon. Again, the segmental level of involvement may indicate the possible associated symptom(s) and vice versa. (107, 108, 109, 110)

In FD, the evidence suggests that biomechanical disturbance of the mid-dorsal vertebrae through influence of sympathetic innervation, and/or cervical vertebrae via the parasympathetic

vagus innervation, to be sites of potential vertebrogenic subluxation-related influence on gastrointestinal physiology.

Vertebrogenic

Vertebrogenic pain and the vertebrogenic syndrome are both listed as item M54.89 in the 2021 ICD1-10-CM. This pain may be identified in two spinal forms, one demonstrating the morphological changes such as intrusive osteophyte formation with distinct diagnostic signs and symptoms. The syndrome initiated from anatomical and physiological (functional) disturbance with the creation of noxious somatosensory activation. Vertebrogenic dyspepsia would represent this second form, sometimes without evidence of morphological or laboratory changes, but based more on symptoms of 3-6 months duration, postprandial discomfort, early satiation and epigastric pain. (111, 112)

On a commercial website for Novus Biologicals, vertebrogenic pain syndrome may be described as '*an acute or chronic pain in the back, and can include thoracic, lumbar, sacral or adjacent regions. Pain can be of many qualities and causes, and treatments may include physical therapy, spinal manipulations, injections and in some cases, surgical correction*.' (There was no explanation forthcoming as to why the cervical spine was omitted.) (113) We note that pain of this nature may be regarded as noxious somatosensory input.

This 'vertebrogenic' term is appearing more frequently in recent times in a somatovisceral sense. It seems to have first arisen in a medical paper by Rychliková and Lewit in 1976. The functional version of vertebrogenic may be referred to as a subluxation complex due to physically altered segmental dynamics with disturbed neurological and other soft tissue elements. This would be differentiated from pressure or neural irritation initiated by osteophytic formation or other exostosis compromising the spinal cord or radicular neural elements. (114, 115, 116)

It appears inconsistent that some authors will recognise the physical manifestations of vertebrogenic subluxation factors in certain musculoskeletal disorders, but perhaps not the associated pathoneurophysiological ones. Exceptions to this would include recent reports by Garo-Falides and Wainwright as well as Vaňásková. (79, 98, 117, 118)

Vertebrogenic dyspepsia

This term has broad connotations as it also incorporates the noxious neural somatosensory elements from a range of somatic factors. Due to the relationship with the richly innervated spinal column, cord, radicular proximity and some cranial nerves, influence on the autonomic nerves can also be associated with the non-subluxation vertebrogenic disorders. These may include vertebral congenital anomalies, pathologies, scar tissue, fractures, and bulging or herniated discs.

It appears that the nomenclature for idiopathic dyspepsia has been replaced by *Functional Dyspepsia* as the terms appear to be used interchangeably. Conventional medicine's tardy acceptance of a subluxation model would explain its failure to identify the vertebrogenic model of FD. The research, clinical evidence, and prevalence of the condition noted in the chiropractic literature and European medical papers appear to have been largely ignored. (7, 86, 119)

As an indication as to chiropractic patient management of gastrointestinal conditions, an All Fields search of Index to Chiropractic Literature conducted on September 25 2021, revealed 122 papers listed covering such topics as dyspepsia [n=11], dysphagia [n=11], indigestion [n=4], gastric [n=10], gastroparesis [n=1], gastrointestinal [n=64] and GERD [n=22]. (120)

Review

Reluctance to adopt a subluxation model of this condition may explain the failure to identify the vertebrogenic model of FD has been acknowledged. (79, 98, 118)

The authoritative and independent *Rome Foundation* based in Rome, Italy, now favours the term *Disorders of Gut-Brain Interaction* (DGBI) in preference to functional gastrointestinal disorders. The Foundation's aim '*is to improve the lives of people with disorders of gut-brain interaction*.' However, while this term recognises a central neural element, it does not appear to include a neurogenic subluxation possibility. (121)

This body was formed in 1990 and has now established that there are four basic signs of functional dyspepsia. They are: early satiation, postprandial fullness, epigastric pain, epigastric burning, plus between meal-related and meal-unrelated. (Symptoms of nausea, vomiting, belching and heartburn although within the spectrum, were not included) (122)

Abdominal pain of spinal origin is a condition with etiological similarities to subluxation based intercostal neuralgia. A number of medical studies discuss treating these pain conditions with nerve blocks. A 1977 study by Ashby managed five patients experiencing a variety of abdominal pains. All five experienced relief following an intercostal nerve block. A case of mid-thoracic spinal dysfunction associated with pseudo-appendicitis was resolved the same way, demonstrating similar physiological principles of somatic origin. (79, 123, 124, 125, 126, 127, 128)

Red Flags

In addition, in practice, and with most conditions, red flags regarding dyspepsia are a consideration to be noted. (8, 129, 130) Further, one is mindful that a combination of mid-dorsal back pain may also be referred from gastrointestinal pathology. (131)

Research, neurophysiological

A more scientific basis for a somatogenic model of certain conditions is recognised in neurophysiology. Studies conducted by the *Department of the Autonomic Nervous System* at the *Metropolitan Institute of Gerontology* in Tokyo, and the *Laboratory of Physiology,* Tsukuna Japan, and the *Physiologisches Institut der Universität Würzburg* in Germany, are leading institutions in this research. Their studies substantiate the pathophysiological principles, in that as vertebrae are somatic structures, the neural element of a biomechanically disturbed somatic structure can lead to activation of noxious somatosensory and somato-autonomic reflexes which have physiological somatovisceral effects. (40)

Apart from numerous literature publications, their neurophysiological studies culminated in a textbook format in 1997. One particular section addressed pathophysiology of the gastrointestinal system. It stands as confirmation accounting for the empirical observations, clinical experiences, and clinical outcomes noted by the manual sciences for over 100 years before that text was published. It also substantiates and explains the rationale of continued patient demand for a manipulative model of health care provided by the manipulative professions. That is, to address subluxation-based physiologic dysfunction in forms of dyspepsia. In addition, the overlooked presence of such a base of vertebrogenic visceral conditions previously published, in non-English medical literature, tends to support recognition of the chiropractic and osteopathic models. (132, 133)

Sato et al noted that somatosensory modulation of gastrointestinal motility occurred in animals that experienced noxious mechanical somatic stimulation (51, 134, 135) They stated that in anaesthetised animals, gastric motility was also affected by '*natural somatic stimulation, especially by noxious mechanical stimulation (and had) been shown to depend on the segmental areas stimulated*'. (136)

In a further somatovisceral study, Sato and Terui found that '*Noxious stimulation…to the skin, chest and abdomen of rats, only the somatic stimulation of the abdomen changed the duodenal pressure and motility*.' (137)

As well as a somatovisceral association, Camilleri noted a viscerosomatic association. He explained a firm neural central connection in that with sensory input from the upper gastrointestinal tract '*the pathophysiology of the motor and sensory components of the functional gastrointestinal disorders augur well for major advances and novel therapies.*' (35)

A somatovisceral association was discussed by Song et al who found that in rats, gastrointestinal function may be affected by cervical spondylosis. (137)

A 2019 study on the effect of chronic stress on gastric vagal afferents in mice, supported '*growing evidence suggesting vagus nerve-related signal disruption from organs to the brain*', however they did not identify the spine as an element in that disruption. (139)

Research, medical

In 1979, Kametani et al applied somatic stimulation to the skin of rats to assess gastric motility inhibition and concluded that the gastric reflex was a spinal reflex. In 2014, Piché et al studied similar nociceptive effects at various levels from T2 to L6, and their different effects upon gastric motility and gastrointestinal blood flow. (140, 141)

Bortolotti et al. researched indications of motor pattern dysfunction ('*dysmotility-like*') in patients with idiopathic dyspepsia in 1995. This suggests neural influence upon the functioning of the smooth muscle in the gastric wall. Coffin et al also concluded that a neural influence where certain types of dyspepsia experienced a ' *... sensory dysfunction...associated with impaired reflex reactivity in the stomach.*' (142, 143)

Modified motility is reported as a commonly associated symptom attributed to disturbed or subluxated vertebral segment. (28, 99, 137, 141, 144, 145)

Clinical studies, medical

In 2017, Talley et al stated that pharmaceutically, there are various treatment options, however, they also state that these are available '*with varying levels or evidence and efficacy*'. As a treatment model, this suggests that it is a matter of trying one particular drug at a time to see if it provides relief. On that trial basis alone, and in view of the efficacy reports offered here, chiropractic or other manual care would seem justified as one of the options for patients. (8)

A number of non-English medical clinical reports on the topic of vertebrogenic gastrointestinal functional conditions raise the point of why so few similar papers appear in English language medical journals. European medical literature in particular, has carried a number of papers regarding the spinal manipulative management of digestive disorders. (97, 115, 116, 118, 146, 147, 148, 149, 150, 151, 152, 153)

In discussing the range of aetiologies of dyspepsia, Krag acknowledged that there can be mechanical, postural, pathological, or pathophysiological contributing factors. He stated that abdominal pain may be ' *... referred pain due to excessive refluxes provoking high threshold receptors in joints and muscles... referred pain from strictures of the vertebrate axis apophyseal joints, and that these can be released by a manipulation producing the characteristic "click".*' Krag noted studies of 149 patients involving somatogenic dyspnoea involving facet syndromes (30 cases), spinal nerve irritation (73 cases), and 46 with a slipped rib syndrome, although the site was not specified, the costovertebral articulation is a common site of involvement. (151)

Lewit devotes a chapter of his textbook where he discusses a range of visceral conditions under the topic of '*Vertebrovisceral correlations*'. He also noted that vertebral '*blockages*' (the fixation element of subluxations) at T4-T7 and particularly T5/T6 were characteristic of certain gastro-duodenal conditions. (89)

In his textbook on Orthopaedic Medicine, Maigne notes his manipulative cure or improvement of pseudo-ulcers on several occasions finding them to be painful syndromes of spinal origin. (90, 154)

The 1995 paper on vertebrogenic influence by Filippkin et al discussed imaging studies on 89 Russian children with vertebrogenic disease including neck injuries from birth. An earlier study of cervical spine birth injuries of the spine and medulla in 174 children, associated 'functional obstructions of the intestinal tract' in a range of conditions including:- (118,155)

- Pylorospasms
- Spastic hypotonic dyskinesia of the ileum
- Gastroesophageal reflux
- Reflux oesophagitis
- Oesophageal stenosi
- Dysrhythmic iliac peristalsis

Other European gastrointestinal dysfunction studies included such potential vertebrogenic conditions as:-

- Peptic ulcers, (97, 146, 147)
- Gastroenteritis, (146)
- Irritable bowel disease, (152)
- Gastric ulcer in adolescent, (115)
- Gall bladder pain ,(153)
- Oesophageal dysmotility, (50)
- Achalasia/Loss of oesophageal peristalsis, (50)
- GERD, (50)
- Sphincter hypertonicity, (50)
- Gastroparesis, (50)
- Biliary dyskinesia, (50)
- Intestinal motility. (50)

The stated aetiology of the conditions noted by Manabat and Cagir stated that '*Causes of intestinal motility disorders appear to be multifactorial, and only a few have been detected.*' In the absence of demonstrable tissue pathology, these conditions would appear to be ideal for a comparative study with spinal manipulation, particularly in those cases involving sphincter tone and gastric motility dysfunctions. (156)

Other vague terms which may involve vertebrogenic factors in gastric conditions are noted, these include:

- Nonspecific diagnosis, (31)
- Oesophageal dysfunction, (157)
- Disturbed oesophageal motility, (99)
- Duodenal motility, (137)
- Hypertensive oesophageal sphincter, (158)
- Motor disorder of the oesophagus, (159).
- Neurogenic dysphagia, (159)

- Sphincter dysfunction,(50)
- Swallowing disorders. (98)

Hammad states that 50% of hospital admissions with angina pectoris have non-cardiac disorders. He noted that in 38 patients with pseudo-angina where all cardiac testing was negative, 31.58% had an oesophageal spasm, 23.68% myofascial pain and 21.05% were vertebrogenic in origin. (160)

Research, chiropractic

There are indications of a degree of interprofessional collaboration taking place. It can be noted that apart from chiropractic authors being published in chiropractic journals, there are now chiropractic authors published in medical journals, medical authors in chiropractic journals and joint authorship in both professions' journals. (1, 97, 145, 161)

Budgell and Suzuki demonstrated gastric motility inhibition by noxious interspinous thoracic stimulation of capsaicin injection in anaesthetised rat subjects. They noted a segmentally organised somatovisceral reflex response involving sympathetic afferents, the coeliac ganglion and alpha adrenergic receptors. Budgell has been associated with publishing at least nine papers involving the gastrointestinal dysfunction and the ANS. (144, 162, 163, 164, 165)

A review paper by Pickar noted in experiments that '*inhibition of gastric motility was greatest when the mechanical stimulation was applied to the sixth thoracic vertebra*.' (161)

In 1980, Wiles conducted Electrogastrogram (EGG) studies on a small cohort. He noted that following manipulation of C1, an EGG wave pattern normalised following manipulation of C1 and there was an increase in basic gastric tone. (166, 167)

Clinical studies, chiropractic

Medical collaboration with the chiropractic healthcare service has been provided at the Texas Medical Institute. Over 13 years this clinic has a philosophy of offering 'successful conservative care that offers fast results' (from)'*a team of doctors who specialize in family practice, emergency medicine, and chiropractic care.*' Its website states that:

> *'Chiropractic care includes spinal manipulation and spinal decompression techniques that can treat disorders of the joints, neck, back, digestive system (including GERD and stomach ulcers), as well as muscle spasms and chronic headaches.' (168, 169)*

In a cohort of 1,567 patients experiencing indigestion, Bryner and Staerker found that 71% had experienced mid back pain in the previous six months. Of these, 46% experienced indigestion and mid-back pain simultaneously. (170)

A 1985 controlled research on rabbits, DeBoer et al monitored myoelectric activity (EMG) and noted suppression of gastrointestinal smooth muscle activity in response to manually subluxated upper thoracic vertebrae in 51.4% (19) of 37 rabbit subjects. (171)

Other studies have reported on such conditions as:

- GERD, (10,161,178)
- Heartburn ,(170,172)
- Gastric reflux, (172)
- Dyspepsia, (172)
- Functional dyspepsia, (173)
- Reflux oesophagitis, (174)
- Indigestion, (170)

- Constipation, (175)Indigestion, (179)
- Gastroesophageal reflux disease. (176, 177)

In 2011, Young et al, and Bull et al in 2003, noted that chiropractic care included lifestyle and dietary advice in the management of gastrointestinal conditions. Other professions include these elements as well. These papers accurately indicate that chiropractic is not a technique, and not bound by manipulation solely, but encompasses a wider range of considerations in managing the health care of patients. (180, 181)

Osteopathy

A 2012 study on functional dyspepsia by Mirocha and Parker recognised that some patients do not respond to conventional care. They then state that *'Somatic findings at midthoracic levels correspond to localized visceral dysfunction such as decreased peristalsis and vasoconstriction.'* They and others, suggest that spinal manipulation may assist this by normalising autonomic tone and other functions. (182, 183, 184, 185, 186, 187)

Other clinical gastrointestinal studies

Physiotherapy

Grieve reports that epigastric pain over the stomach and pancreas was produced by a T6-T7 nerve root irritation, and gall bladder '*disease*' by irritation of the T8-T9 nerve roots. (188)

In Grant's physiotherapy textbook, Bogduk and Valencia noted that vertebral structures of the dorsal spine can be associated with referred pain from visceral sources. They state that '*clinically, such referred pain may mimic the referred pain of visceral disease.*' In the same text, McNair and Maitland noted gastric related somatic disorders amongst other visceral symptoms. (189, 190)

A 2020 case-controlled study by Azizi et al on 62 patients concluded that manipulation of the thoracic spine relieved gastrointestinal pain for at least three months. (191)

Veterinary science

In veterinary animal studies, Maler stated that ' *... any animal with a spine may present with disorders secondary to vertebral subluxations...that can cause neurologic dysfunction and have widespread detrimental effects on our patients' health*.' She discussed the potential for a cervical subluxation/restriction being responsible for a number of gastrointestinal presentations and a variety of other conditions. Her patients involve in a range of pets including rabbits, guinea pigs, ferrets, reptiles, and birds. In regards to hoofstock, Maler noted that '*Gastrointestinal issues, especially colic-type symptoms of newborns, may respond favourably to chiropractic treatment added to standard medical therapy*.' (192)

Acupuncture

Acupuncture is also noted for applying somatovisceral principles. In a 2020 RCT reported in the *Annals of Internal Medicine*, Yang et al addressed three of the primary symptoms of functional dyspepsia – postprandial fullness, upper abdominal bloating, and early satiation. After 4 weeks of acupuncture, these symptoms were eliminated in 27.8% of patients compared to 17.3% in the sham group. The positive response was sustained at twelve weeks. (193, 194, 195, 196)

Conditions of vertebrogenic origin

There is a plethora of vertebrogenic conditions recorded and considered to be of biomechanically disturbed segmental origin, that is, subluxation-related. Such a designation implies that a physical approach to restoration as an option to pharmaceutical administrations which may suppress symptoms without addressing the physical or biomechanical aspects. (70, 107, 108, 151, 197, 198)

An indication of the vertebrogenic principles associated with a range of conditions subject to similar pathoneurophysiological mechanisms of spinal segmental origin can be noted. Apart from biomechanical disturbance, such conditions as spondylosis may physically contribute to functional and neural disturbances. The most common and apparent of these conditions would be subluxation-related cervicogenic headaches. The range of other symptoms of spinal origin includes:-

Cervicogenic

- Cervicogenic dizziness, (199)
- Cervicogenic dysphagia, (46, 98)
- Cervicogenic dysphonia, (47, 200, 201)
- Cervicogenic exophthalmos, (202)
- Cervicogenic headache, (203)
- Cervicogenic migraine, (204)
- Cervicogenic otoocular syndrome, (205)
- Cervicogenic oesophageal dysfunction, (206)
- Cervicogenic tinnitus, (207)
- Cervicogenic vertigo, (208)
- Cervicogenic vestibule-ocular and post-concussion disorders. (209)

Vertebrogenic

- Vertebrogenic cardialgia, (210)
- Vertebrogenic chest pain, (211)
- Vertebrogenic dizziness, (212)
- Vertebrogenic dysphagia, (213)
- Vertebrogenic headache, (214)
- Vertebrogenic pseudoangina pectoris, (211)
- Vertebrogenic cervicoencephalic, (215)
- Vertebrogenic thoracoalgia, (216)
- Vertebrogenic vestibular dysfunction. (217)

Spondylogenic (Conditions primarily from spondylosis and anomalies)

- Spondylogenic esophageal spasms, (218)
- Spondylogenic visceropathies, (219)
- Spondylogenic hypotonic functional disturbances of the intestinal tract. (220)

Without using chiropractic terminology, Passatore and Roatta discuss a number of conditions that may be associated with whiplash. In particular they note mechanical derangements (subluxations) and signs of autonomic dysfunction including cardiac syndrome and irritable bowel syndrome. (221) Srinivasan and Greenbaum outline the abstruse character and diagnostic challenge of abdominal-wall pain of somatic origin. (222)

Discussion

Estimates have indicated that up to 25% of the population experience functional dyspepsia. Its prevalence presents a variety of signs and symptoms, many of which appear to be neurologically mediated. (223, 224, 225, 226)

In recognising the basis for science, empiricism is proposed by *Encyclopaedia Britannica* as '*the view that all concepts originate in experience, that all concepts are about or applicable to things that can be experienced, or that all rationally acceptable beliefs or propositions are justifiable or knowable only through experience*.' *Wikipedia* states that empiricism is '*of central importance to the sciences*.' We offer that in a pragmatic sense, without empirical evidence, clinical efficacy and daily clinical practice would be severely inhibited. (227, 228)

As this form of dyspepsia is designated as one of the so-called functional conditions, a reasonable part of evaluating the condition should be based on empirical evidence. This cause and effect observation would be similar to the previously noted pharmaceutical model.

Sympathetic nervous system - thoracic spine factor

Subluxation disturbances of mid-dorsal (thoracic) spinal segments have been identified as having an influence on dyspepsia through the sympathetic nervous system. (103, 131, 144, 229)

Hypertonic skeletal muscles, especially the intrinsic muscles at a functionally disturbed segmental level can indicate a sign of vertebral subluxation in an MSK (musculoskeletal) condition. Palpatory signs of structural and functional segmental changes, as well as tenderness of the mid-thoracic spine can also provide some of the indications of segmental disturbance. It is suggested here that in a vertebrogenic form of dyspepsia, the cardiac and/or the pyloric sphincter may become hypertonic causing a stenosis producing one of the visceral symptoms in gastric dysfunction. We hypothesise that a hypotonic sphincter may also develop depending on the vertebrogenic parasympathetic level of involvement. (46, 79, 98) A similar precept would apply to the smooth muscle layer of the tunica media in the gastrointestinal wall moderating motility. (28, 51, 52, 141, 230, 231, 232, 233)

In 2016, Turco et al identified two sub-types of functional dyspepsia although there was a percentage of overlap as well. They concluded with a suggestion that there was a '*common pathophysiological mechanism*' involved with that condition. (234)

The sympathetic subluxation model regarding FD was firmly recognised by in a *BMJ* Case Report of two cases by Garo-Falides and Wainwright in 2016. They went further to seemingly admonish medical colleagues when they highlighted the importance of considering thoracic spine dysfunction in cases of idiopathic abdominal pain. The title of their paper essentially infers that the medical appreciation of vertebrogenic subluxation disorders is quiescent. It is suggested that it may be politically, but not scientifically difficult, for political medicine to suddenly adopt this vertebrogenic model. (79)

The spinal role in vertebrogenic dyspepsia appears similar to initiating cases of vertebrogenic headaches, intercostal neuralgia, or sciatica. Gastric divisions of mid-dorsal spinal nerves from the sympathetic trunk reach their target via branches of the greater splanchnic nerve plexus. It is not conceivable that a sensory neural disturbance in the form noxious reflexes of vertebral origin would discriminate between spinal nerves only activating the posterior musculoskeletal branch and nociceptor reflexes, only to avoid its anterior visceral branch. Studies of this efferent activation are supported by the extensive neurology studies by Sato et al. (40,179,182,188,191)

Parasympathetic - cervical spine - *vagus* factor

A number of studies recognise that subluxated cervical segments may influence the parasympathetic nervous system, particularly the vagus. (46, 118, 138, 166, 189, 190)

On a medical website, Hauser notes the relationship between cervical spine instability, digestion, the vagus nerve and their association to sphincter function and a range of other digestive symptoms. He adds that '*The vagus nerve is responsible for managing our intestinal activity as well as managing the sphincter muscles in the gastrointestinal tract.*' The intervention he employs is prolotherapy – the injection of primarily d-glucose in order to stimulate regeneration of damaged tendons and ligaments in the cervical spine. (235)

An association of functional dyspepsia with the vagus nerve which has also been known appropriately in this instance as the pneumogastric nerve indicating a further possible cervicogenic association with dyspepsia. (138, 236, 237, 238, 239, 240, 241, 242, 243, 245, 246, 247)

Vaňásková et al note the involvement of the vagus in their reports on the association between the cervical spine, manipulation, dysphagia, oesophageal motility, and other gastrointestinal conditions. (46)

Physiologically, Sato and colleagues also study the effects of activation of the somato-vagal reflexes in rat subjects in relation to gastric innervation. They noted the sympathetic and parasympathetic activation with somatic sensory stimulation. (248)

In a somatovisceral trial by Diego et al in 2005, vagal influence on gastric motility was monitored by electrogastrogram readings (EGG) on 48 massaged preterm neonates. A notable increase in gastric motility was attributed to stimulated vagal activity. (249)

Summary

A reasonable neurophysiological rationale for FD has been considered together with pragmatic clinical evidence from six professions. It is suggested that this evidence may provide a judicious account for the apparent patient demand based on experiences of positive clinical outcomes.

It is suggested that there is a similar if not stronger level of supporting evidence for the manipulative management of a range of vertebrogenic gastrointestinal conditions than for that justifying the capricious nature of conventional pharmaceutical treatment of FD. In 2019, Masuy substantiated this claim when he stated that, '*To date, only limited treatment options are available and conflicting results in terms of efficacy have been reported. Consequently, nonpharmacological treatment options are increasingly being explored for functional dyspepsia.*' It appears to be an uneven field or double standard if the basis expressed by Masuy et al did not apply to alternative therapies, namely those that have demonstrated acceptable degrees of safe, positive outcomes. (70)

Evidence was also offered as to the role of the cervical spine through the parasympathetic vagus nerve, and the mid-dorsal sympathetic nerve supply as part of vertebrogenic subluxation models for the enigmatic FD.

While most of the supporting evidence cited originated from medical sources, no evidence was found to contradict the physiology or the efficacy of chiropractic care for subluxation-related vertebrogenic functional dyspepsia.

Conclusion

The evidence supports the rationale for a vertebrogenic, subluxation related aetiology as one of the possible models for ameliorating functional dyspepsia. As such, a manipulative correction of associated segment(s) involved would seem justified as one of the options available for patients. As with the criteria for conventional measures, a trial period of manipulative care would seem appropriate.

The authors respectfully suggest that as conventional care and the *Rome V model* (121) continues without recognition of somato-sensory-autonomic vertebrogenic-neural hypothesis, vertebrogenic dyspepsia may remain an enigma

Abdominal pain ' *... may be due to a "storm" of some kind which, arising in the irritated spinal segment, runs out along the autonomic nerves to upset the functions of the bowel.*' (250)

Peter Rome
DC (ret), FICC
cadaps@bigpond.net.au

John D Waterhouse
DC, FACC
Private practice, Melbourne

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References

1. Sato A. Somatovisceral reflexes. J Manipulative Physiol Ther. 1995;18(9):597-602.
2. Newman C. The inaugural essay on dyspepsia.[Tentamen medicum inaugurale dyspepsia.] 1773. https://wellcomecollection.org/works/qu7jecvw/items?canvas=1
3. Hicks GH, [Dissertatio medica inauguralis de dyspepsia] 1814. https://wellcomecollection.org/works/d7c25bu9.
4. Hutchinson R. A discussion on the treatment of functional dyspepsia. Proc Royal Society Med. 1908;1(The PharmacolSect):3-7. [One of 6 by various authors]
5. Hutchinson R. Remarks on the rational treatment of functional dyspepsia. Br Med J. 1908;1(2467):850-2 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2045783/?page=5
6. Hunt RH, Fallone C, van Zanten SV, et al. Etiology of dyspepsia: implications for empirical therapy. Can J Gastroenterol. 2002;16(9):635-41.
7. Tack J, Kindt S. Pathogenesis and therapy for idiopathic dyspepsia. Curr Gastroenterol Rep. 2005;7(6):437-44
8. Talley NJ, Goodsall T, Potter M. Functional dyspepsia. Aust Prescr. 2017;40:209-13. https://www.nps.org.au/australian-prescriber/articles/functional-dyspepsia
9. Oustamanolakis P, Tack J. Dyspepsia organic verses functional. J Clin Gastroenterology. 2012;46(3):175-90
10. Angus K, Asgharifar S, Gieberzon B. What effect does chiropractic treatment have on gastrointestinal GI disorders: a narrative review of the literature. J Canad Chiropr Assoc. 2015;59(2):122-133.
11. Fedorchuk C, St Bernard A. Case study, Improvement in gastroesophageal reflux disease following chiropractic care and the ALCAT procedure. Ann Vert Sub Res, 2011; Online access only p44-50.
12. Hains G, Hains F, Descarreaux M. Gastroesophageal reflex disease, spinal manipulative therapy and ischemic compression: a preliminary study. (62 subjects.) J Am Chiropr Assoc. 2007;44(1):7-19.
13. Hein T. Some effects of chiropractic manipulation on reflux oesophagitis: a case report. Br J Chiropr. 1999 Aug;3(3):59-61.
14. Iyer MM, Skokos E, Piombo D. Chiropractic management using multimodal therapies on 2 pediatric patients with constipation. J Chiropr Med.2017;16(4):340-45.

15. Jackson SB. Gastroesophageal reflex disease. Topics Clin Chiropr 1996;2(1):24-9

16. Leach RA. (ed) Gastrointestinal disorders. In: The chiropractic Theories. Principles and clinical application. 3. Baltimore. Williams & Wilkins.1994;165-6

17. Madill J. Improvement of GERD following chiropractic care: A case study and selective review of literature [case report]. Ann Vert Sublux Res. 2016 Summer;2016(3):Online access only p 53-60. https://vertebralsubluxation.sharepoint.com/Pages/2016_1438_GERD.aspx

18. Young K. Chiropractic. In Mullin GE, Singh M, Parian A. Integrative gastroenterology 2nd edn. 2020. https://oxfordmedicine.com/view/10.1093/med/9780190933043.001.0001/med-9780190933043-chapter-10

19. Young M, McCarthy P, King S. Chiropractic management of chronic heartburn, dyspepsia and reflux in chronically symptomatic adults [Chiropractic Evidence 2011, The College of Chiropractors, London, 19 January 2011; meeting abstract]. Clin Chiropr. 2011;14(2):66-67. http://dx.doi.org/doi:10.1016/j.clch.2011.02.007

20. Young MF, McCarthy PW, King SJ. Chiropractic manual intervention in chronic adult dyspepsia: A pilot study. Clin Chiropr. 2009 Mar;12(1):28-34. http://dx.doi.org/doi:10.1016/j.clch.2009.04.002

21. Mirocha NJ, Parker JD. Successful treatment of refractory functional dyspepsia with osteopathic manipulative treatment. Osteop Family Physician. 2012;4:193-6.

22. Morris HD, Dickey J: Management of peptic ulcer disease using osteopathic manipulation. AAO J 2007;17(1):26-29.

23. Branyon B: Healing hands: using osteopathic manipulative treatment to address visceral structures through somatovisceral reflexes, a case study. AAO J 2008;18(4):29-31

24. Smilowicz A. An osteopathic approach to gastrointestinal disease: somatic clues for diagnosis and clinical challenges associated with Helicobacter pylori antibiotic resistance. J Am Osteopath Assoc. 2013; 113(5):404-16.

25. Elsing C, Böhm M. Functional dyspepsia: alternative therapy by osteopathy. Internat Academy Osteop. 2020; https://www2.osteopathie.eu/en/publications/abstract-master-thesis/functional-dyspepsia-alternative-therapy-osteopathy

26. Nazareth D, Orchard D. An osteopathic approach to functional digestive disorders. In: Mullin GE, Singh M, Parian A. Integrative gastroenterology 2nd edn. 2020. https://oxfordmedicine.com/view/10.1093/med/9780190933043.001.0001/med-9780190933043-chapter-9

27. Bolen B, Burakoff R. All about all of the sphincters in your body. https://www.verywellhealth.com/what-is-a-sphincter-1945068. 2021. Extracted 18 Sept 2021.

28. Asamo H, Tomita T, Nakamura K, et al. Prevalence of gastric motility disorders in patients with functional dyspepsia. J Neurogastroenterol Motil. 2017;23(3):392-99.

29. Mayo Clinic Staff. Gastroparesis. https://www.mayoclinic.org/diseases-conditions/gastroparesis/symptoms-causes/syc-20355787 Extracted 18 Sept 2021

30. Schafer RC. Dyspepsia. In: Chiropractic physical and spinal diagnosis. First edn. Oklahoma City. Associated Chiropractic Academic Press. 1980;II-42.

31. Mathias, J. R., & Clench, M. H. Neuromuscular diseases of the gastrointestinal tract. Specific disorders that often get a nonspecific diagnosis. Postgraduate Medicine. 1995;97(3):95-108.

32. Functional dyspepsia. Mayo Clinic. https://www.mayoclinic.org/diseases-conditions/functional-dyspepsia/symptoms-causes/syc-20375709#:~:text=Functional%20dyspepsia%20(dis%2DPEP%2D,and%20symptoms%20are%20mostly%20intermittent. 2018-2021.

33. Madisch A, Andersen V, Enck P, et al. The diagnosis and treatment of functional dyspepsia. Dtsch Arztebl Int. 2018;115(13):222-232

34. Yarandi SS, Christie J. Functional dyspepsia in review: pathophysiology and challenges in the diagnosis and management die to coexisting gastroesophageal reflux disease and irritable bowel syndrome. Gastroent Res Prac. 2013; ID 351086, https://doi.org/10.1155/2013/351086

35. Camilleri M. Functional gastrointestinal disorders: Novel insights and treatments. Medscape General Medicine, 1999;1(3). https://www.medscape.com/viewarticle/717346_7.

36. Mosby's Dictionary of Medicine, Nursing & Health Professions. 10th edition. St Louis. Elsevier eBook. 2017, p 318.

37. Haavik H, Niazi IK, Holt K, Murphy B. Effects of 12 weeks of chiropractic care on central integration of dual somatosensory input in chronic pain patients: a preliminary study. J Manipulative Physiol Ther. 2017;40(3):127-38.

38. Haavik-Taylor H, Murphy B. The effects of spinal manipulation on central integration of dual somatosensory input observed after motor training: a crossover study. J Manipualtive Physiol Ther. 2010;33(4):261-72.

39. Rome PL, Waterhouse JD. Neurodynamics of vertebrogenic somatosensory activation and autonomic reflexes – a review: Part 11 – the vertebral (somatic) autonomic influence upon other organs and functions. Aust-Pacific Chiropr J. 2021;1.4. URL apcj.net/papers-issue-2-4/#RomeWaterhouseInfluence

40. Sato A, Sato Y, Schmidt RF. The impact of somatosensory input on autonomic functions. In: Blaustein MP, Grunicke H, Pette D, et al. Reviews of physiology, biochemistry and pharmacolosy. Berlin. Springer.1997.

41. Haavik-Taylor H, Murphy B. Cervical spine manipulation alters sensorimotor integration: a somatosensory evoked potential study. Clin Neurophysiol 2007;118(2):391-402.

42. Collins KF, Pfleger B. The neurophysiological evaluation of the subluxation complex: documenting the neurological component with somatosensory evoked potentials. Chiropr Research J. 1994;3(1):1-4.

43. Kent C. Proposed neurobiological processes associated with models of vertebral subluxation: dysafferentation, dyskinesia, dysponesis, dysautonomia, neuroplasticity,and ephaptic transmission. Arch Neurol Neurosci.2019;3(1). ANN.MS.ID.000555. DOI: 10.33552/ANN.2019.03.000555.

44. Seaman DR, Winterstein JF. Dysafferentation: A novel term to describe the neuropathophysiological effects of joint complex dysfunction. A look at likely mechanisms of symptom generation. J Manipulative Physiol Ther 1999;22(1):45-48.

45. Battakova S, Shraimanov B. Neurophysiological changes in the afferent somatosensory system indices in the case of vertebrogenic spine pathology in miners. Int J Occup Med Environ Health. 2013;26(2):230-4

46. Vaňásková E, Hep A, Vižď A J, Tosnerová V. Swallowing disorders related to vertebrogenic dysfunction. Ceska a Slovenska Neurologie a Neurochirurgie 2007;70(6):692-696. (English abstract) https://www.csnn.eu/en/journals/czech-and-slovak-neurology-and-neurosurgery/2007-6-1/swallowing-disorders-related-to-vertebrogenic-dysfunctions-52591

47. Roy N. Functional dysphonia. Curr Opin Otolaryngol Head Neck Surg. 2003;11(3):144-8.

48. Masarsky CS, Weber M. Somatic dyspnea and the orthopedics of respiration. Chiropr Tech 1991;3(1):26-29.

49. Functional dyspepsia. Mayo Clinic. https://www.mayoclinic.org/diseases-conditions/functional-dyspepsia/symptoms-causes/syc-20375709#:~:text=Functional%20dyspepsia%20(dis%2DPEP%2D,and%20symptoms%20are%20mostly%20intermittent. 2018-2021.

50. Bharucha, A. E. & Camilleri, M. Gastrointestinal dysmotility and sphincter dysfunction, In: Neurological Therapeutics: Principles and practice, Noseworthy, J. H. London: Taylor & Francis, 2003;3060-3064. (Cited by Vanaskova)

51. Sato A, Sato Y, Shimada Y, Changes in gastric motility produced by nociceptive stimulation of the skin in rats. Brain Res. 1975a;87:151-9.

52. 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-rellex-inlluence-upon-smo/

53. Kumar S, Khannar R, Hajra B, Bose J. Psychiatric disorder in essential dyspepsia. Internat J Psychiatr Clin Prac. 1998;2(1):41-45.

54. Talley NJ, Phillips SF. Non-ulcer dyspepsia: potential causes and pathophysiology. Ann. Int. Med. 1988;108(6):865-79.

55. Pubmed Timeline: https://pubmed.ncbi.nlm.nih.gov/?term=%22functional+dyspepsia%22&schema=alltitle

56. Pubmed Timeline: https://pubmed.ncbi.nlm.nih.gov/?db=PubMed&orig_db=PubMed&term=%22Functional+dyspepsia%22

57. Hijazi LS, Zahra F, Yarrarapu SNS, Mead T. Functional Asplenism. 2021 Aug 10. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.

58. Friedman O, Cekic E, Gunel C. Functional rhinoplasty. Facial Plast Surg ClinNorth Am. 2017;25(2):195-199.

59. Shin JE, Park KS. Nam K. [Chronic functional constipation]. Korean J Gastroenterol. 2019;73(2):92-98.

60. Tack J. Functional diarrhea. Gastoenterol Clin North Am, 2012;41(3):629-37.

61. Schmerler DA, Espay AJ. Functional dystonia. Handb Clin Neurol. 2016;139:235-245.

62. Popa SL, Chiarioni G, Golea DL, Dumitrascu DL. Functional emesis. J Gastrointestin Liver Dis. 2019;28(3):319-325

63. Marcoloni E, Tolchin B. Sunctional seizures, Emerg Med Clin North Am. 2021;39(1):123-132.

64. Schwingenshuh P, Deuschi G. Functional tremor. Handb Clin Neurol. 2016;139:229-233.

65. Thenganatt MA, Jankovic J. Psychogenic (functional) parkinsonism. Handb Clin Neurol. 2016;139:259-262.

66. Chavoustle CT, Silver N Functional dyspepsia causes and treatment. Healthline. Sept 28, 2018. https://www.healthline.com/health/functional-dyspepsia.

67. Beckić S. [Functional bloating, constipation and diarrhea]. Acta Med Croatica. 2015;69(4):253-62

68. Sanmiguel CP, Soffer EE. Constipation caused by functional outlet obstruction. Curr Gastroenterol Rep. 2003;5(5):414-8.

69. Tally NJ, Goodsall T, Potter M. Functional dyspepsia. Aust Prescr. 2017;40:209-13.

70. Masuy I, van Oudenhove L, Tack J. Review article: treatment options for functional dyspepsia. Aliment Pharmacol Ther. 2019;49(9):1134-72.

71. Rangan V. Functional dyspepsia: causes, treatments, and new direction. https://www.health.harvard.edu/blog/functional-dyspepsia-causes-treatments-and-new-directions-2020070620505. July 6, 2020.

72. Ford AC, Mahadeva S, Carbone F, et al. Functional dyspepsia. Lancet. 2020;396(19263):1689-1702.

73. Mounsey A, Barzin A, Rietz A. Functional dyspepsia: evaluation and management. Am Fam Physician. 2020;101(2):84-88.

74. Christy A. Update on indigestion. Med Clin North Am. 2021;105(1):19-30.

75. Farré R, van Heel H, Vanuytsel T, et al. In functional dyspepsia, hypersensitivity to postprandial distention correlates with meal-related symptom severity. Gastroenterol. 2013;145:566-73

76. Longstreth GF, Lacy BE. Approach to the adult with dyspepsia. UpToDate. Dec 2019. https://www.uptodate.com/contents/approach-to-the-adult-with-dyspepsia#:~:text=Dyspepsia

77. Functional Dyspepsia. https://www.mayoclinic.org/diseases-conditions/functional-dyspepsia/symptoms-causes/syc-20375709. (Current) Extracted Sept 9 2021

78. Functional dyspepsia. GI Society. https://badgut.org/information-centre/a-z-digestive-topics/functional-dyspepsia/. Extracted 13 Sept 2021.

79. Garo-Falides B, Wainwright TW. Pseudoappendicitis: abdominal pain arising from thoracic spine dysfunction – a forgotten entity and a reminder of an important clinical lesson. BMJ Case Rep. 2016. Doi: 10.1136/bcr-2016-216490.

80. Ernst E. Chiropractic treatment for gastrointestinal problems: a systematic review of clinical trials. Can J Gastroenterol. 2011;25(1):39-40.

81. Dyer B. What is functional overlay? https://www.blbchronicpain.co.uk/news/what-is-functional-overlay/

82. Bromberg W. Functional overlay: an illegitimate diagnosis? West J Med. 1979;130(6):561-65.

83. Kurlansik SL, Maffei MS. Somatic symptom disorder. Am Family Phys. 2016;93(1):49-54.

84. Sarno JE. Psychogenic backache: the missing dimension. J Fam Pract. 1974;1(2):8-12.

85. Cardin F. Zorzi M, Bovo E, et al. Effect of implementation of a dyspepsia and helicobacter pylori eradication guideline in primary care. Digestion. 2005;72(1):1-7.

86. Nwokediuko SC. Is functional dyspepsia idiopathic? 2012: DOI: 10.5772/5660 https://www.intechopen.com/chapters/45693

87. Biedermann H (Ed). Manual therapy in children. Edinburgh. Churchill Livingstone. 2004:195,295-7

88. Cailliet R. Subluxations of the cervical spine including the 'whiplash' syndrome. In: Neck and arm pain. Philadelphia: FA Davis Co. 1967:35-39.

89. Lewit K. Vertebrovisceral correlations. In Manipulative therapy: musculoskeletal medicine. Oxford, UK: Butterworth-Heinemann; 2009;281-287.

90. Maigne R. Orthopaedic Medicine: A new approach to vertebral manipulation. Thomas, Illinois. 1972:164,192-209:

91. Schmorl G, Junghanns H. Inefficient motor segment (Intervertebral Insufficiency). In: The human spine in health and disease. New York. Grune & Stratton.1971:213-229.

92. Sato S, Sato Y, Schmidt RF. (40 – pps 168, 180, 185)

93. Sato S, Sato Y, Schmidt RF. (40 – pps 2-3)

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

95. Battakova S, Shraimanov B. Neurophysiological changes in the afferent somatosensory system indices in the case of vertebrogenic spine pathology in miners. Int J Occup Med Environ Health. 2013;26(2):230-4

96. The Free Dictionary by Farlex. https://medical-dictionary.thefreedictionary.com/somatovisceral

97. Pikalov A, Kharin V. Use of spinal manipulative therapy in the treatment of duodenal ulcer: a pilot study. J Manipulative Physiol Ther 1994;17(5):310-3

98. Vaňásková E, Dolina J, Hep A. Swallowing disorders related to vertebrogenic dysfunctions. In: Brzozowski T (Ed). New advances in the basic and clinical gastroenterology. Chapter 8. 2012:175-184. https://www.intechopen.com/books/new-advances-in-the-basic-and-clinical-gastroenterology/swallowing-disorders-related-to-vertebrogenic-dysfunctions

99. Vaňásková E, Hep A, Lewit K, et al. Cervical dysfunction and disturbed oesophageal motility – scintigraphic assessment. J Orthop Med. 2001;23(1):9-11. https://doi.org/10.1080/1355297X.2001.11736122.

100. Biedermann H. Manual therapy in children: proposals for an etiological model. J Manipulative Physiol Ther. 2005;28(3):e1-e15

101. Hyland ME, Biedermann H. Colic. Chapter 22. Complexity theory and its implications for manual therapy. In Manual therapy in children. Edinburgh. Churchill livingstone.2004;295-297.

102. Rome P. Waterhouse J, Maginness G, Ebrall P. Medical management of literature. J Contemp Chiropr. 2019;2:54-69.

103. Klougart N, Nilsson N, Jacobsen J. Infantile colic treated by chiropractors - A prospective study of 316 cases. J Manipulative Physiol Ther 1989;12(4):281-288.

104. Goldstein M, Ed. The Research Status of Spinal Manipulative Therapy. US Department of Health, Education and Welfare. National Institute of Health. NINCDS Monograph No 15. Bethesda, Maryland. 1975:221.

105. Vernon H, Gatterman MI. Cervicogenic headaches. In: GattermanMI. Foundations of chiropractic subluxation. 2nd ed. St Louis. Elsevier Mosby..2005:376.

106. Bryner P. Symmetry: a premise of chiropractic diagnosis and therapy. J Aust Chiropr Assoc. 1986;16(3):91-4.

107. Jänig W, Häbler H-J. 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.

108. Shaballot N, Aloumar A, Manual J, May M, Beissner F. Segmental signs and spontaneous pain in acute visceral disease – lateralisation and bodily patterns. MedRχiv. BMJ. 2020;July 24. (Preprint –https://www.medrxiv.org/content/10.1101/2020.07.23.20160598v1

109. Welsh 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. doi: 10.1016/j.jcm.2008.04.001.

110. Rome P. Waterhouse JD. Differentiating chiropractic articular adjustments from manipulation: The terms 'adjustment' and 'manipulation'; Part 2 of a series.. Asia-Pacific Chiropr J. 2021;1.3. URL.

111. URL https://icdlist.com/icd-10/M54.89

112. Vertebrogenic algic syndrome. In: Rovenský J., Payer J. (eds) Dictionary of Rheumatology. Springer, Vienna. 2009. https://doi.org/10.1007/978-3-211-79280-3_1157

113. Vertebrogenic pain syndrome: disease bioinformatics. https://www.novusbio.com/diseases/vertebrogenic-pain-syndrome

114. Nathan H. Osteophytes of the spine compressing the sympathetic trunk and splanchnic nerves in the thorax. Spine. 1987l;12(6):527-32.

115. Rychliková E, Lewit K. [Vertebrogenic dysfunction and reflex changes in gastric and duodenal ulcer in adolescents.] Vniřní Lèkařstvi. 1976;22:326-335. (Cited in: Lewit K. Manipulative therapy in rehabilitation of the locomotor system. 3rd edn. Oxford. Butterworth Heinemann. 1999:332.)

116. Lewit K, Rychliková E. Reflex and vertebrogenic disturbances in peptic ulcer. In: Functional pathology of the Motor System. Rehabilitácia. 1975. Suppl .10-11:116. (Cited in: Lewit K. Manipulative therapy in rehabilitation of the locomotor system. 3rd edn. Oxford. Butterworth Heinemann. 1999:326.)

117. Rome PL, Waterhouse JD. Neurodynamics of vertebrogenic somatosensory activation and autonomic reflexes – a review: Part 11 – the vertebral (somatic) autonomic influence upon other organs and functions. Asian Pacific Chiropr J. URL apcj.net/papers-issue-2-4/#RomeWaterhouseInfluence

118. Filippkin MA, Akberov RF, Vasenin BN. [Functional disorders of the digestive tract in children with vertebrogenic diseases.] Vestnik rentgenologii i radiologii. 1995:19-22.

119. Stanghellini V, Poluzzi E, De Ponti F, et al. Idiopathic dyspepsia. Curr Treat Options Gastroenterol. 2005;8(2):175-183.

120. Chiropractic Library Consortium. Index to Chiropractic Literature. URL https://www.chiroindex.org/?advanced_search=1#results

121. Rome Foundation. URL https://theromefoundation.org/

122. Tack J. Why is functional dyspepsia the new hot topic? Rome III, Rome IV. Rome Foundation. https://theromefoundation.org/why-is-functional-dyspepsia-the-new-hot-topic/. Downloaded Sept 17,.2021

123. Ashby EC. Abdominal pain of spinal origin. Annals Royal Col Surgeons Engl. 1977;59;242-246.

124. Eulitt HK, Giannakakis V. Resolution of chronic constipation, fecal incontinence, and abdominal pain in an 8 year old child following chiropractic care: a case study and selective review of the literature. Ann Vert Sublux Res. 2014;1:1-7.

125. Fallon JM. Chiropractic manipulation in the treatment of costovertebral joint dysfunction with resultant intercostal neuralgia during pregnancy. J Neuromusculoskelet Syst. 1996;4(2):73-75.

126. Sweetster S. Abdominal wall pain: a common clinical problem. Gastroenterol Dis. 2019;94(2):347-55.

127. Swoboda B, Oman RE. Paediatric chiropractic care of a patient with recurrent abdominal pain (case report). Chiropr J Aust. 2008;38(3):92-6.

128. Van Loon M. Intertcostal neuralgia during pregnancy and chiropractic care. (Case report). J Clin Chiropr Pediatr. 2010;11(2):780-2.

129. Moses S. Dyspepsia red flags. Family Practice Notebook. August 2021. https://fpnotebook.com/gi/exam/DyspsRdFlgs.htm

130. Gotfried J. Dyspepsia. MSD Manual. March, 2020. https://www.msdmanuals.com/en-au/professional/gastrointestinal-disorders/symptoms-of-gastrointestinal-disorders/dyspepsia.

131. Beattie JW, Dyspeptic backache. Ann Rheum Dis. 1953;12(3):223-6.

132. Sato A, Sato Y, Schmidt RF. (40 – pp166-189)

133. Sato A: The modulation of visceral functions by somatic afferent activity. Jpn J Physiol. 1987;37:1-17.

134. Sato A, Sato Y, Schmidt RF. (40 – pp 297)

135. Sato A, Sato Y, Shimada Y, Changes in vesical function produced by nociceptive stimulation of the skin in rats. Brain Res. 1975b;94:465-474.

136. Sato A, Sato Y, Schmidt RF. (40 – pp 186)

137. Sato Y, Terui N. Changes in duodenal motility produced by noxious mechanical stimulation of the skin of rats. Neurosci Letters. 1976;2:189-193.

138. Song X-H, Xu X-X, Ding L-W, et al. A preliminary study of neck-stomach syndrome. World J Gastroenteral. 2007;13(18):2575-80.

139. Li H, Buisman- Pijlman FT, Nunez-Salces M, et al. Chromic stress induces hypersensitivity of murine gastric vagal afferents. Neurogastroenterology Motility. 2019;31(12):e13669.

140. Kametani H, Sato A, Simpson A. Neural mechanisms of reflex facilitation and inhibition of gastric motility to stimulation of various areas in rats. J Physiology. 1979;294:407-18.

141. Piché M, Watanabe N, Hotta H. Regulation of gastric motility and blood flow during acute nociceptive stimulation of the paraspinal muscles in urethane-anaesthetised rats. J Physiol Sciences. 2014;64:37-46

142. Bortolotti M, Bolondi L, Santi V, Brunelli F, Barbara L. Patterns of gastric emptying in dysmotility-like dyspepsia. Scand J Gastrol 1995;30(5):408-410.

143. Coffin B, Azpiroz F, Guarner F, Malagelda J-R. Selective gastric hypersensitivity and reflex hyporeactivity in functional dyspepsia. Gastroenterol 1994;107(5):1345-1351.

144. Budgell B, Suzuki A. Inhibition of gastric motility by noxious chemical stimulation of interspinous tissues in the rat. J Auton Nerv Syst. 2000;80(3):162-8.

145. Budgell B, Hotta H, Sato A. Reflex responses of bladder motility after stimulation of interspinous tissues in the anesthetized rat. J Manipulative Physiol Ther. 1998;21(9):593-9.

146. Gutzeit K. Das neurovaskulare problem in der Ätiologie und Pathogenese von Ulcus peptic und Gastritis (Enteritis).(The neurovascular problem in the etiology and pathogenesis of peptic ulcer and gastro-enteritis.) Münchener medizinische Wochenschrift 1951;93:47-49. (Cited in Weiant CW, Goldschmidt S. Medicine and chiropractic. Self Published, New York. 1966:120.)

147. Gutzeit K. [Peptic ulcer I. Incidence, pathogenesis, etiology, therapy.] Medizinische 1954;4:113-114. (German) (Extract)

148. Gutzeit K. [Peptic ulcer II. Incidence, pathogenesis,etiology, therapy.] Medizinische 1954;6:179-182. (German) (Extract)

149. Kamieth H. [Pathogenic importance of the thoracic portion of the vertebral column.] Arch. Orthop Unfallchir 1958;49(6):585-606. (German) (Pubmed extract)

150. Kameith H. [Diseases of the internal organs from the chiropractic point of view, represented by peptic ulcer.] Medizinische. 1957;25(46):1708-1715. (German) (PMID 13492964)

151. Krag E. Other causes of dyspepsia - especially abdominal pain of spinal origin. Scand J Gastroenterol Suppl 1982;79:32-37.

152. Qu LX. [Relationship between irritable bowel syndrome and unstable thoracolumbar vertebrae]. Zhongguo Gu Shang. 2009 Jun;22(6):456-7.

153. Rychlíková E. [Pain in the gall bladder region due to vertebrogenic disturbance.] Schmerzen im Gallenblasenbereich auf Grund vertebragener Störungen. Deutsches Gesundheitswesn. 1974;29:2092. (Cited in: Lewit K. Manipulative therapy in rehabilitation of the locomotor system. 3rd edn. Butterworth Heinemann, Oxford. 1999:285.)

154. Heidari Z, Keshteli AH, Feizi A, et al. Somatic complaints are significantly associated with chronic uninvestigated dyspepsia and its symptoms: a large cross-sectional population based study. J Neurogastroenteral Motil 2017;23(1):80-91. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5216638/

155. Michailov MK, Akberov RF.[X-ray symptomatology and differential diagnosis of functional obstruction of the digestive tract in children induced by birth injuries of the spine and spinal cord.] Radiol Diagn (Berl). 1989;30(6):669-74. (PubMed abstract)

156. Manabat ML, Cagir B. Intestinal motility disorders. Medscape. https://emedicine.medscape.com/article/179937-overview. Sept 16, 2020.

157. Silber W. Diabetes and oesophageal dysfunction. BMJ. 1969;3(5672):688-90. https://www.jstor.org/stable/20377783.

158. El-Hak NAG, Mostafa M, Hamid HA, Haleem M. Hypertensive lower oesophageal sphincter (HLES): prevalence, symptoms and effect of pneumatic balloon dilation. Saudi J Gastroenter. 2006;12(2):77-82.

159. Johns Hopkins Medicine. Swallowing disorders. https://www.hopkinsmedicine.org/gastroenterology_hepatology/_pdfs/esophagus_stomach/swallowing_disorders.pdf.

160. Hammad WAE, Pseudo-angina syndrome a prospective diagnostic study. Al-Ashar Assiut Medical J. 2015;13(1):Suppl 2:9-12

161. Pickar JG. Neurophysiological effects of spinal manipulation. Spine J. 2002;2(5):357-377.

162. Budgell B. Reflex effects of subluxation: the autonomic nervous system. J Manipulative Physiol Ther. 2000;23(2):104-6

163. Budgell B. Autonomic responses to spinal pain. Rigakuryoko Kagaku. 2000;15(3):81-7. https://www.jstage.jst.go.jp/article/rika1996/15/3/15_3_81/_pdf/-char/ja

164. Bolton P, Budgell B. Viscera; responses to spinal manipulation. J Electrom Kinesiol. 2012;22(5):777-784.

165. Budgell B, Sato A. Modulation of autonomic function by somatic nociceptive inputs. Chapt 29. In: Progress in brain research. Amsterdam: Elsevier; 1996;113:525-39.

166. Wiles MR. Observations on the effects of upper cervical manipulations on the electrogastrogram: a preliminary report. J Manipulative Physiol Ther. 1980;3(4):226-9.

167. McDowall D, Emmanuel E, Grace S, Chaseling M. Tone as a health concept. Complement Ther Clin Prac. 2017;29:27-34.

168. Texas Medical Institute. https://www.texasmedicalinstitute.com/about/

169. Can a chiropractor help with GERD? Texas Medical Institute. March 6, 2020. https://www.texasmedicalinstitute.com/can-a-chiropractor-help-with-gerd/

170. Bryner P, Staerker PG. Indigestion and heartburn: A descriptive study of prevalence in persons seeking care from chiropractors. J Manipulative Physiol Ther 1996;19(5):317-323.

171. DeBoer KF, Schultz M, McKnight ME. Acute effects of spinal manipulation on gastrointestinal myoelectric activity in conscious rabbits. Man Med 1988;3:85-94.

172. Young M, McCarthy P, King S. Chiropractic management of chronic heartburn, dyspepsia and reflux in chronically symptomatic adults [Chiropractic Evidence 2011, The College of Chiropractors, London, 19 January 2011; meeting abstract]. Clin Chiropr. 2011 Jun;14(2):66-67. http://dx.doi.org/doi:10.1016/j.clch.2011.02.007

173. Annis RS, Kumbhare DA, Parkinson WL. Functional dyspepsia: can chiropractic help?. J Am Chiropr Assoc. 2001 Apr;38(4):34-36.

174. Hein T. Some effects of chiropractic manipulation on reflux oesophagitis: a case report. Br J Chiropr. 1999 Aug;3(3):59-61.

175. Iyer MM, Skokos E, Piombo D. Chiropractic management using multimodal therapies on 2 pediatric patients with constipation. J Chiropr Med.2017;16(4):340-45.

176. Fedorchuk C, St Bernard A. Case study, Improvement in gastroesophageal reflux disease following chiropractic care and the ALCAT procedure. Ann Vert Sub Res, 2011; Online access only p44-50.

177. Jackson SB. Gastroesophageal reflux disease. Topics Clin Chiropr 1996;2(1):24-9

178. Madill J. Improvement of GERD following chiropractic care: A case study and selective review of literature [case report]. Ann Vert Sublux Res. 2016 Summer;2016(3):Online access only p 53-60. https://vertebralsubluxation.sharepoint.com/Pages/2016_1438_GERD.aspx

179. Rome PL. Anterior T6 subluxation syndrome: Neurospinal dysfunction within a vertebral subluxation complex. Chiropr J Aust. 2000 Dec;30(4):127-37.

180. Young M, McCarthy P, King S. Effect of dietary and lifestyle advice in addition to spinal manipulation for adult patients with chronic symptoms of heartburn, dyspepsia or reflux [Chiropractic Evidence 2011, The College of Chiropractors, London, 19 January 2011; meeting abstract]. Clin Chiropr. 2011 Jun;14(2):80-81. ICLID: 21820 URL: http://dx.doi.org/doi:10.1016/j.clch.2011.02.012.

181. Bull P, Love Z. Management of dyspepsia: A chiropractic perspective. Chiropr J Aust. 2003 Jun;33(2):57-63.

182. Mirocha NJ, Parker JD. Successful treatment of refractory functional dyspepsia with osteopathic manipulative treatment. Osteop Family Physician. 2012;4:193-6.

183. Morris HD, Dickey J: Management of peptic ulcer disease using osteopathic manipulation. AAO J 17:26-29, 2007 11.

184. Branyon B: Healing hands: using osteopathic manipulative treatment to address visceral structures through somatovisceral reflexes, a case study. AAO J 18:29-31, 2008

185. Smilowicz A. An osteopathic approach to gastrointestinal disease: somatic clues for diagnosis and clinical challenges associated with Helicobacter pylori antibiotic resistance. J Am Osteopath Assoc. 2013; 113(5):404-16.

186. Elsing C, Böhm M. Functional dyspepsia: alternative therapy by osteopathy. Internat Academy Osteop. 2020; https://www2.osteopathie.eu/en/publications/abstract-master-thesis/functional-dyspepsia-alternative-therapy-osteopathy

187. Nazareth D, Orchard D. An osteopathic approach to functional digestive disorders. In: Mullin GE, Singh M, Parian A. Integrative gastroenterology 2nd edn. 2020. https://oxfordmedicine.com/view/10.1093/med/9780190933043.001.0001/med-9780190933043-chapter-9

188. Grieve GP. Thoracic joint problems and simulated visceral disease. In: Grieve GP. Modern manual therapy of the vertebral column, London: Churchill Livingstone. 1986:390.

189. Bogduk N, Valencia F. Innervation and pain patterns of the thoracic spine. In: Grant R, Physical therapy of the cervical and thoracic spine. New York. Churchill Livingstone. 1988:34.

190. McNair JFS. Maitland G. Manipulative therapy techniques in the management of some thoracic syndromes. In: Grant R, Physical therapy of the cervical and thoracic spine. New York. Churchill Livingstone. 1988:2433-69.

191. Azizi S, Rezasoltani Z, Najafi S, et al. The relationship between dorsal spine dysfunction and gastrointestinal pain and the efficacy of manipulation on them,. Phys Med Rehab Electrodiag. 2020;2(1):2-6

192. Maler MM. Overview of veterinary chiropractic and its use in pediatric exotic patients. Vet Clin Exot Anim. 2012;15:299-310. https://www.vetexotic.theclinics.com/article/S1094-9194(12)00008-4/pdf

193. Ma Q. Somato-autonomic reflexes of acupuncture. Med Acupunct. 2020;32(6):362-66

194. Yang J-W, Wang L-Q, Zou X, et al. Effect of acupuncture for postprandial distress syndrome: a randomised clinical trial. Ann Intern Med. 2020;172(12):777-85.

195. Zhu Jm Guo Y, Liu S, et al. Acupuncture for the treatment of gastro-oesophageal reflux disease: a systematic review and meta-analysis. Acupunct Medicine. 2017;35(5):316-23.

196. Sato A, Sato Y, Suzuki A, Uchida S. Neural mechanisms of the reflex inhibition of gastric motility elicited by acupuncture-like stimulation in anesthetised rats. Neurosci Res. 1993;18(1):53-62.

197. Aarimaa H. Panel discussion. In: Krag E. Other causes of dyspepsia – especially abdominal pain of spinal origin. Scand J Gastroenterol Suppl. 1982;79:32-37.

198. Hobbs SF, Chandler MJ, Bolser DC, Foreman RD. Segmental organisation of visceral and somatic input onto C3-T6 spinothalamic tract cells of the monkey. J Neurophysiol. 1992;68(5):1574-88.

199. Chu ECP, Chin WL, Bhaumik A. Cervicogenic dizziness. Oxf Med Case reports. 2019;2019(11):476-478.

200. Hülsa M. [Cervical dysphonia) Folia Phoniatr (Basel). 1991;43(4):181-96. (Pubmed extract)

201. Jankovic J, Leder S, Warner D, Schwartz K. Cervical dystonia: clinical findings and associated movement disorders. Neurology. 1991;41(7):088-91

202. Wu C-M, Liao H-E, Hsu S-W, Lan S-J. Cervicogenic exophthalmos: possible etiology and pathogenesis. World J Clin Cases. 2020;8(2):318-324

203. Fredriksen TA, Antonaci F, Sjaastad O. Cervicogenic headache: too important to be left un-diagnosed. J Headache Pain. 2015;16(6): https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4417107/

204. 2022 ICD-10-CM Diafgnosis Code 644.86 Cervicogenic migraine. ICD10 Data.com. https://www.icd10data.com/ICD10CM/Codes/G00-G99/G40-G47/G44-/G44.86

205. Franz B, Altidis P, Altidis B, Collis-Brown G. The cervicogenic otoocular syndrome: a suspected forerunner of Ménière's disease. Internat Tinnitis J. 1999;5(2):125-130.

206. McCulloch TM, Jaffe D. Head and neck disorders affecting swallowing. Part 1. GI Motility Online. 2006;doi:10.1038.gimo36. https://www.nature.com/gimo/contents/pt1/full/gimo36.html

207. Oostendorp RA, Bakker I, Elvers H, et al. Cervicogenic somatosensory tinnitus: an indication for manual therapy? Part I: Theoretical concept. Man ther. 2016;23:120-3.

208. Hain TC. Cervicogenic cause of vertigo. Curr Opin Neurol. 2015;28(1):69-73.

209. Ellis MJ, Leddy JJ, Willer B. Physiological, vestibulo-ocular and cervicogenic post-concussion disorders: an evidence-based classification system with directions for treatment. Brain Inj, 2015;29(2):238-48.

210. Shakhnazarov AB. [Vertebrogenic cardialgia syndrome]. Vrach Delo. 1977;3:84-5.

211. Grgić V.[Vertebrogenic chest pain – "Pseudoangina pectoris": etiopathogenesis, clinical manifestations, diagnosis, differential diagnosis, and therapy]. Lijec Vjesn. 2007;129 (1-2), 20-25

212. Reicke N. [Vertebrogenic dizziness, etiology and differential diagnosis]. Fortschr Med. 1978;96:1895-902.

213. Andersen PM, Fagerlund M. Vertebrogenic dysphagia and gait disturbance mimicking motor neuron disease. J Neurol Neurosurg Psychiatry. 2000;69(4):560-1

214. Pesl J. [Vertebrogenic headache in childhood]. Cesk Pediatr. 1976;31(11):635-7

215. Fengler H, Schulze KJ, Kleditzsch J, Opitz JU. [Vertebrogenic cervicoencephalic syndrome.] Z Arztl Fortbild (Jena). 1986;80(21):877-81.

216. Kondziella W, [The vertebrogenic factor and thoracalgia.] Schmerz. 1995;9(1):34-8.

217. Likhachev SA, Borisenko AV. [The dynamics of vertebrogenic vestibular dysfunction under the influence of manual therapy]. Lik Sprava. 1994;7-8:84-8.

218. Brugsch T. [Spondylogenic esophageal spasms.] Z Gesamte Inn Med.1953;8(15):659-70

219. Simonenko VB, Davydov OV. [Spondylogenic visceropathies.] Klin Med (Mosk). 2010;88(3):59-62

220. Sielaff HJ. Neurovegative organisation through spondylopathies. [Neurovegative organ irritationen durch spondylopathien.] Acta Radiol. 1973;1:778. Cited by Schmorl G, Junghanns H. The human spine in health and disease. 2nd edn.New York. Grune & Stratton, 219.

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

222. Srinivasan R, Greenbaum DS. Chronic abdominal wall pain: a frequently overlooked problem. Practical approach to diagnosis and management. Am J Gastroenterol 2002;97:824–830.

223. Camilleri M. Functional gastrointestinal disorders: Novel insights and treatments. Medscape Gastroenterology, 1999. Medscape Inc. http://psychiatry.medscape.com/Medscape/gastro/journal/1999/v01…/pnt-mge6911.cami.htm.

224. Mimidis K, Tack J. Pathogenesis of dyspepsia. Dig Dis. 2008;26(3):194-202.

225. Home P, Reducing neuroinflammation: how the vagus might be key. Australian Spinal Research. https://spinalresearch.com.au/reducing-neuroinflammation-how-the-vagus-might-be-key/

226. Marriott CL, Winarski KD, Crisp CA. Resolution of vomiting in a 9-month-old following chiropractic care (case report). J Clin Chiropr Pediatr. 2011;12(1):865-8.

227. https://www.britannica.com/topic/empiricism

228. https://en.wikipedia.org/wiki/Empirical_evidence

229. Sweidan MJ. A pilot study to determine the preliminary effects of spinal manipulative therapy on functional dyspepsia. Master's Dissertation. Durban University of Technology. 2015.

230. Jänig W. Integration of neural and non-neural signals influencing blood vessels. In: The integrative action of the autonomic nervous system. Neurobiology and homeostasis. Cambridge: Cambridge Press. 2006:273-276.

231. Budgell BS, Sato A. Somatoautonomic reflex regulation of sciatic nerve blood flow. J Neuromuscular Syst 1994;2:170-177

232. Adams-Ray J. Studies on a cutaneous pallor reflex in the fourth cervical segment in cardiac pain. Acta Medica Scand 1953;146(6): URL https://onlinelibrary.wiley.com/doi/abs/10.1111/j.0954-6820.1953.tb10259.x

233. Amatuzzi F, Gervazoni B de L, De Silva ML, et al. Acute and time-course effects of osteopathic manipulative treatment on vascular and autonomic function in patterns with heart failure: A randomised trial. J Manipulative Physiol Ther. 2021;44(6):455-66.

234. Turco R, Russo M, Martinelli M, et al. Do distinct functional dyspepsia subtypes exits in children? J Pediatr Gastroenterol Nutr. 2016;62(3):387-

235. Hauser R. Cervical spine instability and digestive disorders: indigestion and irritable bowel syndrome caused by cervical spondylosis. https://www.caringmedical.com/prolotherapy-news/gerd-neck/. June 17, 2021.

236. Mercovich AP. Resolution of chronic constipation and dysautonomia in a three-year-old female following chiropractic care. A case report and review of the literature. J Pediatr Matern Fam Health – Chiropr. 2019;100-104.

237. Marriott CL, Winarski KD, Crisp CA. Resolution of vomiting in a 9-month-old following chiropractic care. J Clin Chiropr Pediatr. 2011;12(1):865-8.

238. Cordner ZA, Li Q, Liu L, et al. Vagal gut-brain signalling mediates amygaloid plasticity, affect, and pain in a functional dyspepsia model. JCI Insight. 2021. Mar 22;6(6):e144046. doi: 10.1172/jci.insight.144046.

239. Tominaga K, Fujikawa Y, Tsumoto C, et al. Disorder if autonomic nervous system and its vulnerability to external stimulation in functional dyspepsia. J Clin Biochem Nutr. 2016;58(2):161-5

240. Guo W-J, Yao S-K, Zhang Y-L, et al. Impaired vagal activity to meal in patients with functional dyspepsia and delayed gastric emptying. J Int Med Res. 2018;46(2):792-801.

241. Zhu Y, Xu F, Lu D, eta l Transcutaneous auricular vagal nerve stimulation improves functional dyspepsia by enhancing vagal efferent activity. Am J Physiol Gastrointest Liver Physiol. 2021;320(5):G700-G711.

242. Holtman G, Goebell H, Jockenhoevel F, Talley NJ. Altered vagal and intestinal mechanosensory function in chronic unexplained dyspepsia. Gut 1998;42:501-6.

243. Travagli RA, Anselmi L. Vagal neurocircuity and its influence on gastric motility. Nat Rev Gastroenterol Hepatol. 2016;13(7):389-401.

244. Browning KN, Travagli RA. Plasticity of vagal brainstem circuits in the control of gastric function. Neurogastroenterol Motil. 2010;22(11):1154-63.

245. Liu H, Yu B, Zhang M, et al. Treatment of diabetic gastroparesis by complementary and alternative medicines. Medicines (Basel) 2015;2(3):212-9

246. Neuromodulation of the brain-gut axis by transcutaneous vagal nerve stimulation in functional dyspepsia. National Institute of Health. Massachusetts. https://grantome.com/grant/NIH/R21-DK116029-01. Downloaded Oct 2, 2021

247. Holtman G, Goebell H, Jockenhoevel F, Talley NJ. Altered vagal and intestinal mechanosensory function in chronic unexplained dyspepsia. Gut. 1998;42(4):501-6.

248. Sato A, Sato Y, Schmidt RF. (40 – pp183,185-6)

249. Diego MA, Field T, Hernandez-Reif M. Vagal activity, gastric motility and weight gain in massaged preterm neonates. J Pediatr. 2005:147(1):50-55.

250. Alvarez WC. Nervousness indigestion and pain. New York. Paul B Hoeber Inc. 1943:213.