REVIEW

Models of Vertebral Subluxation: A Review

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Abstract — Basic science and clinical models of the vertebral subluxation are reviewed. Neurobiological mechanisms associated with these models are described. Models reviewed include the subluxation complex model, subluxation degeneration, nerve compression, dysafferentation, the neurodystrophic model and segmental facilitation. Clinical models, including the segmental, postural, and tonal approaches are discussed.

Key words: Vertebral Subluxation, Models of Vertebral Subluxation, Subluxation Complex, Subluxation Degeneration, Nerve Compression, Dysafferentation, Segmental Facilitation.

Historical Considerations

The term "subluxation" has a long history in the healing arts literature. According to Haldeman[1] it was used at the time of Hippocrates, [2] while the earliest English definition is attributed to Randall Holme in 1688. Holme[3] defined subluxation as "a dislocation or putting out of joynt." Watkins[4] and Terrett[5] refer to a 1746 definition of the term. The matter is further complicated by the diverse array of alternative terms used to describe subluxations. Rome[6] listed 296 variations and synonyms used by medical, chiropractic, and other professions. Rome concluded the abstract of his paper by stating, "It is suggested that, with so many attempts to establish a term for such a clinical and biological finding, an entity of some significance must exist."

The possible neurological consequences of subluxation were described by Harrison in 1821, as quoted by Terrett[5]: "When any of the vertebrae become displaced or too prominent, the patient experiences inconvenience from a local derangement in the nerves of the part. He, in consequence, is tormented with a train of nervous symptoms, which are as obscure in their origin as they are stubborn in their nature..." Although medical authorities acknowledge that neurological complications may result from subluxation, [7] classical chiropractic definitions mandate the presence of a neurological component. D. D. Palmer and B. J. Palmer[8] defined subluxation as follows: "A (sub)luxation of a joint, to a Chiropractor, means pressure on nerves, abnormal functions creating a lesion in some portion of the body, either in its action, or makeup." According to Stephenson's 1927 text, [9] the following must occur for the term "vertebral subluxation" to be properly applied:

1. Loss of juxtaposition of a vertebra with the one above, the one below, or both.
2. Occlusion of an opening.
3. Nerve impingement.
4. Interference with the transmission of mental impulses.

As Lantz[10] noted, "Common to all concepts of subluxation are some form of kinesiologic dysfunction and some form of neurologic involvement."

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Component Models of Subluxation

Dishman[11] and Lantz[12-13] developed and popularized the five component model of the "vertebral subluxation complex" attributed to Faye. [10] However, the model was presented in a text by Flesia[14] dated 1982, while the Faye notes bear a 1983 date. The original model has five components:

1. Spinal kinesiopathology
2. Neuropathology
3. Myopathology
4. Histopathology
5. Biochemical changes.

The "vertebral subluxation complex" model includes tissue specific manifestations described by Herfert[15] which include:

1. Osseous component
2. Connective tissue involvement, including disc, other ligaments, fascia, and muscles
3. The neurological component, including nerve roots and spinal cord
4. Altered biomechanics
5. Advancing complications in the innervated tissues and/or the patient's symptoms. This is sometimes termed the "end tissue phenomenon" of the vertebral subluxation complex.

Lantz[10,16] has since revised and expanded the "vertebral subluxation complex" model to include nine components:

1. Kinesiology
2. Neurology
3. Myology
4. Connective tissue physiology
5. Angiology
6. Inflammatory response
7. Anatomy
8. Physiology
9. Biochemistry.

Lantz[10] summarized his objectives in expanding the model: "The VSC allows for every aspect of chiropractic clinical management to be integrated into a single conceptual model, a sort of 'unified field theory' of chiropractic...Each component can, in turn, be described in terms of precise details of anatomic, physiologic, and biochemical alterations inherent in subluxation degeneration and parallel changes involved in normalization of

structure and function through adjustive procedures." Whether this model will realize these objectives remains to be seen.

Subluxation Degeneration Model

Subluxation degeneration has been described as a progressive process associated with abnormal spinal mechanics. The degenerative changes are associated with various mechanisms of neurological dysfunction. [14] Progressive degeneration of the cervical spine is thought to begin with the intervertebral discs, progressing to changes in the cervical vertebrae and contiguous soft tissues. [16] Several early investigators explored the relationship of spinal degenerative disease to neurological compromise. In 1838, Key[17] described a case of cord pressure due to degenerative changes causing spinal canal stenosis. Bailey and Casamajor[18] reported that cord compression could result from spinal osteoarthritis. They suggested that disc thinning was the basic pathology underlying degenerative change. As early as 1926, Elliott[19] gave an account of how radicular symptoms could be caused by foraminal stenosis secondary to arthritic changes.

Several mechanisms have been suggested which may be operative in cervical spine degeneration. Resnick and Niwayama[20] used the term "intervertebral (osteo)chondrosis" to describe abnormalities which predominate in the nucleus pulposus. Osteoarthritis of the uncovertebral and zygapophyseal joints is another manifestation of cervical spine degeneration. Spondylosis is the term these authors applied to degenerative changes which occur as a result of enlarging annular defects which lead to disruption of the attachment sites of the disc to the vertebral body. This leads to the appearance of osteophytes. O'Connell[21] employed the term "spondylosis" in a broader context. Three lesions were described: disc protrusion into the intervertebral canal; primary spondylosis, characterized by degenerative changes between the vertebral bodies and zygapophyseal joints; and secondary spondylosis, associated with disc protrusion at a single spinal level.

In the lumbar spine, pathomechanics and torsional stress have been implicated as etiological factors in spinal degeneration. [22-23] It is likely that these factors are operative in the pathogenesis of cervical spine degeneration as well. Although it has been suggested that aging is responsible for degenerative changes in the spine, this appears to be an oversimplification. [24] For example, Lestini and Weisel[16] report that there is a high statistical correlation between disc degeneration and posterior osteophyte formation. Furthermore, it is noted that the incidence of degenerative changes varies from one segmental level to another. The C5/C6 level is most frequently involved, with C6/C7 being the level next most frequently affected. The C2/C3 level is the one least likely to exhibit degenerative changes. [25] Since the prevalence of cervical spine degenerative change is not uniform throughout the region, the hypothesis that degenerative change is associated with spinal pathomechanics deserves consideration.

Hadley[25] suggests that both aging and pathomechanics are operative in the pathogenesis of cervical spine degeneration. Age related disc degeneration causes hypermobilty, resulting in greater tractional forces on ligaments. This is said to result in the formation of reactive osteophytes. Trauma can result in local spondylotic changes. This is similar to MacNab's description of traction spur formation in the lumbar spine. [26]

Pesch et al. [27] measured the dimensions of the fifth, sixth, and seventh cervical vertebral bodies in 105 cadavers aged 16 to 91 years. Similar measurements were made on the third, fourth, and fifth lumbar vertebral bodies. The authors suggest that dynamic stressing of the cervical vertebral bodies leads laterally to friction between vertebral bodies at the uncovertebral joints, causing osteophytosis. Anteriorly, osteophytic formation is attributed weakness of the anterior longitudinal ligament, leading to anterior disc protrusion.

Neurological Consequences of Spinal Degeneration

Neurological manifestations of spinal degeneration may be due to a variety of mechanisms. These include:

1. *Cord compression*. Compression of the spinal cord may result from disc protrusion, ligamentum flavum hypertrophy/corrugation, or osteophytosis. Myelopathy may result in cord pressure and/or pressure which interferes with the arterial supply. [21, 28-30] Payne and Spillane[31] found that myelopathy was more likely to occur in persons with congenitally small spinal canals who subsequently develop spondylosis. Hayashi et al. [32] report that in the cervical region, dynamic canal stenosis occurs most commonly in the upper disc levels of C3/C4 and C4/C5.

2. *Nerve root compression*. Compromise of the nerve roots may develop following disc protrusion or osteophytosis. [33] Symptoms are related to the nerve root(s) involved.

3. *Local irritation*. This includes irritation of mechanoreceptive and nociceptive fibers within the intervertebral motion segments. MacNab[33] reports that arm pain may occur without evidence of root compression. The pain is attributed to cervical disc degeneration associated with segmental instability.

4. *Vertebral artery compromise.* MacNab[33] advises that osteophytes may cause vertebral artery compression. Furthermore, Smirnov[34] studied 145 patients with pathology of the cervical spine and cerebral symptoms. Fifty nine percent had vertebrobasilar circulatory disorders.

5. *Autonomic dysfunction*. Symptoms associated with the autonomic nervous system have been reported. The Barre'-Lieou syndrome includes blurred vision, tinnitus, vertigo, temporary deafness, and shoulder pain. This phenomenon occurs following some cervical injuries, and is also known as the posterior cervical syndrome. [35] Stimulation of sympathetic nerves has been implicated in the pathogenesis of this syndrome. [36] Another manifestation of autonomic involvement, reflex sympathetic dystrophy, results in shoulder and arm pain accompanied by trophic changes. [37]

Nerve Root Compression Model

Compression of spinal nerves has traditionally been proposed as a mechanism associated with spinal subluxation, [38] although attempts have been made to discredit the premise that subluxations cause nerve interference by mechanical compression. [39] Results of early animal studies of nerve compression reported that pressures ranging from 130 mm Hg to over 1000 mm Hg were required to produce a significant compression block. [40-42]

However, these older studies dealt with peripheral nerves, not spinal roots.

Sunderland and Bradley[43] reported that spinal roots may be more susceptible to mechanical effects because of their lack of the perineurium and funicular plexus formations present in peripheral nerves. However, few experimental studies involving compression of nerve roots were reported in the literature. [44] Those which were reported were criticized. [45]

In 1975, Sharpless[45] reported the results of a series of animal experiments to determine the susceptibility of spinal roots to compression block. These investigations were supported by the ICA and the ACA. The results were published in a monograph by the National Institutes of Health. Sharpless described his results as "astonishing" and "spectacular." According to the report, "A pressure of only 10 mm Hg produced a significant conduction block, the potential falling to 60% of its initial value in 15 minutes, and to half of its initial value in 30 minutes. After such a small compressive force is removed, nearly complete recovery occurs in 15 to 30 minutes. With higher levels of pressure, we have observed incomplete recovery after many hours of recording." Korr[46] listed factors which render nerve roots more vulnerable to mechanical effects than peripheral nerves:

1. Their location within the intervertebral foramen is in itself a great hazard.
2. Spinal roots lack the protection of epineurium and perineurium.
3. Since each root is dependent on a single radicular artery entering via the foramen, the margin of safety provided by collateral pathways is minimal.
4. Venous congestion may be more common in the roots because the radicular veins would probably be immediately compressed by any reduction in foraminal diameter. There is also the possibility of reflux from the segmental veins through pressure damaged valves; and venous congestion would have additional consequences because the swelling, being within the foramen, would contribute to compression of the other intraforaminal structures.
5. Circulation to the dorsal root ganglion is especially vulnerable.

Contemporary papers have been published concerning nerve root compression. In 1995, Konno et al. [47] reported results similar to those of Sharpless, noting that compression of the nerve roots of the cauda equina with as little as 10 mm Hg of pressure resulted in decreased action potentials. Rydevik[48] described other adverse effects of nerve root compression: "Venous blood flow to spinal roots was blocked with 5-10 mm Hg of pressure. The resultant retrograde venous stasis due to venous congestion is suggested as a significant cause of nerve root compression. Impairment of nutrient flow to spinal nerves is present with similar low pressure." Hause[49] observed that compressed nerve roots can exist without causing pain. Also described in the paper was a proposed mechanism of progression, where mechanical changes lead to circulatory changes, and inflammatogenic agents may result in chemical radiculitis. This may be followed by disturbed CSF flow, defective fibrinolysis and resulting cellular changes. The influence of the sympathetic system may result in synaptic sensitization of the CNS and peripheral nerves, creating a "vicious circle" resulting in radicular pain.

Kuslich, Ulstrom, and Michael[50] discussed the importance of mechanical compromise of nerve roots in the production of radicular symptoms. Their human surgical studies revealed that "Stimulation of compressed or stretched nerve roots consistently produced the same sciatic distribution of pain as the patient experienced preoperatively...we were never able to reproduce a patient's sciatica except by finding and stimulating a stretched, compressed, or swollen nerve root." The importance of asymptomatic lesions was reported by Wilberger and Pang[51] who followed 108 asymptomatic patients with evidence of herniated discs. They reported that within three years, 64% of these patients developed symptoms of lumbosacral radiculopathy. Schlegal et al., [52] Kirkaldy-Willis[53] and Manelfe[54] noted that subluxation of the facet joints may be associated with nerve root entrapment and spinal stenosis, particularly when degenerative disease is present. The degenerative changes are described as a progressive "cascade." Nerve root compression is one of many mechanisms of neural disruption which may be associated with vertebral subluxation. While some may criticize the "garden hose" model as being overly simplistic, the nerve root compression hypothesis is far from obsolete.

Dysafferentation Model

The neurological dysfunction associated with the vertebral subluxation may take other forms. The intervertebral motion segment is richly endowed by nociceptive and mechanoreceptive structures. As a consequence, biomechanical dysfunction may result in an alteration in normal nociception and/or mechanoreception. Aberrated afferent input to the CNS may lead to dysponesis. To use the contemporary jargon of the computer industry, "garbage in—garbage out." Appreciation of these processes begins with an understanding of the neuroanatomy of the tissues of the intervertebral motion segment.

Several papers have described the innervation of human cervical and lumbar intervertebral discs. Bogduk et al. [55] observed that the lumbar intervertebral discs are supplied by a variety of nerves. According to Bogduk, the sinuvertebral nerve supplies the posterior aspect of the disc and the posterior longitudinal ligament. The posterolateral aspects are innervated by adjacent ventral primary rami and from the grey rami communicantes. The lateral aspects of the disc are innervated by the rami communicantes. The anterior longitudinal ligament is innervated by recurrent branches of rami communicantes. Clinically, Bogduk[56] stated that intervertebral discs can be a source of pain without rupture or herniation. Torsional stress may result in circumferential tears in the innervated outer third of the annulus. Compression injuries may lead to internal disruption of the disc, resulting in mechanical or chemical stimulation of the nerve endings in the annulus.

Nakamura et al. [57] reported that the anterior portion of lumbar intervertebral discs is innervated by sympathetic fibres alone. Sympathetic afferents return through the sympathetic trunks and the rami communicantes and pass through the same dorsal horn as the somatosensory afferents. The posterior portion of

the disc is innervated by sinuvertebral nerves derived from the recurrent branch of the spinal nerve, or both the recurrent spinal nerve and sympathetic nerve. These authors observed that dual innervation exists in the intervertebral discs of the lumbar region, and that no other organs are known to have such dual innervation.

Bogduk et al. [58] examined the nerve supply to the cervical intervertebral discs. The sinuvertebral nerves were found to supply the disc at their level of entry as well as the disc above. Nerve fibers were found as deeply as the outer third of the annulus. Mendel[59] et al. stated that nerves were seen throughout the annulus. In addition, receptors resembling Pacinian corpuscles and Golgi tendon organs were seen in the posterolateral region of the disc. The authors conclude that human cervical intervertebral discs are supplied with both nerve fibers and mechanoreceptors.

Human cervical facet joints are also equipped with mechanoreceptors. McLain[60] found Type I, Type II, and Type III mechanoreceptors, as well as unencapsulated nerve endings in the cervical facet joints of normal subjects. The author stated, "The presence of mechanoreceptive and nociceptive nerve endings in cervical facet capsules proves that these tissues are monitored by the central nervous system and implies that neural input from the facets is important to proprioception and pain sensation in the cervical spine. Previous studies have suggested that protection muscular reflexes modulated by these types of mechanoreceptors are important in preventing joint instability and degeneration." Wyke[61-62] has described articular mechanoreceptors, and explored the clinical implications of dysafferentation in pain perception.

Besides the discs and articular capsules, mechanoreceptors and other neural tissues have been described in the ligaments attached to the spine. Jiang et al. [63] noted that Pacinian corpuscles were scattered randomly, close to blood vessels, whereas Ruffini corpuscles were seen in the periphery of human supraspinal and interspinal ligaments. Rhalmi et al. [64] found nerve fibers in the ligamentum flavum, the supraspinal ligament, and the lumbodorsal fascia.

Alterations in mechanoreceptor function may affect postural tone. Murphy[65] summarized the neurological pathways associated with the maintenance of background postural tone: "Weight bearing disc and mechanoreceptor functional integrity regulates and drives background postural neurologic information and function (muscular) through the unconscious mechanoreception anterior and posterior spinocerebellar tract, cerebellum, vestibular nuclei, descending medial longitudinal fusciculus (medial and lateral vestibulospinal tracts), regulatory anterior horn cell pathway." The anterior horn cells provide motor output which travels via motor nerves to muscle fibres.

Although stimulation of articular mechanoreceptors may exert an analgesic effect, use of manipulation for the episodic, symptomatic treatment of pain is not chiropractic. The authors of the remarkable book Segmental Neuropathy,[66] published in the 1960's by Canadian Memorial Chiropractic College, proposed the concept of a "neural image," dependent upon the integrity of neural receptors and afferent pathways. If afferent input is compromised, efferent response may be qualitatively and quantitatively compromised. Correcting the specific vertebral subluxation cause is paramount to restoring normal afferent input to the CNS, and allowing the body to correctly perceive itself and its environment.

Neurodystophic Model

The "neurodystrophic" model suggests that neural dysfunction is stressful to body tissues and that "lowered tissue resistance" can modulate specific and nonspecific immune responses and may alter the trophic function of the involved nerves. A growing number of investigators are exploring the common denominators in disease processes, and the role of the nervous, immune, and endocrine systems in pathogenesis. [67]

Korr[68] proposed that spinal "lesions" (analogous to the vertebral subluxation) are associated with exaggerated sympathetic activity as well as exaggerated paraspinal muscle tone. It is interesting that Korr, like D.D. Palmer, employed the term "tone" in reference to ambient nervous system activity. According to Korr, "High sympathetic tone may alter organ and tissue responses to hormones, infectious agents, and blood components." The mechanism postulated by Korr was one of segmental facilitation. Decreased thresholds in efferent neurons arising from the anterior and lateral horn cells are postulated to result in increased impulse traffic to the somatic and visceral structures innervated by the affected neurons.

More recently, other authors have explored the relationship of sympathetic activity to immune system function in greater depth. Murray et al. [69] examined the effect of sympathetic stimulation on the immune system. Sympathetic stimulation was induced in human volunteers by exhaustive exercise. They found that acute sympathetic stimulation leads to selective release of immunoregulatory cells into the circulation, with subsequent alterations in cellular immune function. These authors stated, "Growing evidence suggests that immune function is regulated in part by the sympathetic nervous system. Sympathetic nerve endings densely innervate lymphoid tissue such as the spleen, lymph nodes and thymus, and lymphoid cells have beta 2 andregenergic receptors." In their experiments, there was a sharp rise in T suppressor/cytotoxic cells and natural killer cells following sympathetic stimulation. However, only modest rises were seen in T helper and B cells. The cells most affected, the T suppressor/cytotoxic cells and the natural killer cells, are those with the largest density of beta receptors."

Felten et al. [70] reported that the neurotransmitter norepinephrine is present in postganglionic sympathetic fibers which innervate lymphoid organs and act on the spleen. Furthermore, there are available receptors on cells in the white pulp and the localized neurotransmitter terminal which directly contact T lymphocytes in the periarticular lymphatic sheath. The authors propose that norepinephrine in lymphoid organs fulfills the criteria for neurotransmission, and plays a significant role in the modulation of immune responses. They state, "Stressful conditions lead to altered measures of immune function, and altered susceptibility to a variety of diseases. Many stimuli, which primarily act on the central nervous system, can profoundly alter immune responses. The two routes available to the central nervous system for communication with peripheral organs are neuroendocrine channels and autonomic nerve channels." In a more recent paper, Felten's team[71] reviewed aspects of neural-immune signaling. Noradrenergic and

peptidergic nerve fibers abundantly innervate the parenchyma of both primary (bone marrow) and secondary (spleen, lymph nodes) lymphoid organs. Nerve fibers distribute within the parenchyma of these organs, as well as along smooth muscle compartments. Both noradrenaline and peptides such as substance P have been shown to fulfill the basic criteria for neurotransmission with lymphocytes, macrophages, and other immunocytes as targets. Denervation or pharmacological manipulation of these neurotransmitters can profoundly alter immunological reactivity at the individual cellular level, at the level of complex multicellular interactions (such as antibody response), and at the level of host responses to a disease-producing challenge."

The relationship between the nervous system and the immune system has attracted the attention of the popular press. An article in the New York Times[72] stated, "Scientists have found the first evidence of an anatomical connection between the nervous system and the immune system. Nerve cell endings in the skin and white blood cells of the immune system are in intimate contact, and chemicals secreted by the nerves can shut down immune system cells nearby." The New York Times author was describing the findings of a paper written by Hossi et al. [73]

Inflammatory disease is influenced by the nervous system. Undem[74] noted that nerve stimulation can affect the growth and function of inflammatory cells. Sternberg et al. [75] stated, "The central nervous system may coordinate both behavioral and immunologic adaptation during stressful situations. The pathophysiologic perturbation of this feedback loop, through various mechanisms, results in the development of inflammatory syndromes, such as rheumatoid arthritis, and behavioral syndromes, such as depression. Thus, diseases characterized by both inflammatory and emotional disturbances may derive from common alteration in specific central nervous system pathways. Fricchoine and Stefano[76] also reviewed what they termed the "neuroendocrine-neuroimmune stress response system." [9] Central nervous system influences on lymphocyte migration was addressed by Ottaway and Husband. [77] These authors suggested that "Many of the alterations in immunity resulting from CNS activity may be explained in terms of changes in lymphocyte migration patterns in response to endocrine signals, neural signals via neurotransmitter release, or direct contacts between nerves and cells of the immune system." Weihe and Krekel[78] observed that "peptides, being present in small-diameter nerve fibers, could exert an indirect immunoregulatory role by influencing vascular tone and/or permeability."

A very interesting hypothesis proposed by Grossman et al. [79] is that cells can learn to associate responsiveness to antigens and other immunoactive agents, with responsiveness to signals originating in the CNS delivered via neuroendocrine or autonomic nervous channels. They propose storage (memory) of stimuli in the immune system rather than in the brain. Just what does this mean to the chiropractor? Can spinal adjustments alter immune system activity? Brennan et al. [80] found that when a thoracic "manipulation" was applied, the response of polymorphonuclear neutrophils isolated from blood collected 15 minutes after the manipulation was significantly higher than blood collected 15 minutes before and 30 and 45 minutes after manipulation. A slight, but significant rise in substance P was also observed.

What are the clinical implications of the nervous system—immune system link? A small controlled study of HIV positive patients was conducted by Selano et al. [81] The effects of specific upper cervical adjustments on the immune system CD4 cell counts of HIV positive individuals was studied. Half the patients received atlas adjustments based upon Grostic upper cervical analysis. The other half received a placebo in the form of an inactive adjusting instrument applied to the mastoid bone. Over the six month period of the study, the control group experienced a 7.96% decrease in CD4 cell counts, while the adjusted group experienced a 48% increase in CD4 cell counts over the same period. Contemporary research is beginning to shed light on the neurobiological mechanisms which may explain the outstanding clinical results chiropractors have experienced when managing patients with infectious diseases. The popular press has been filled with stories describing the emergence of antibiotic resistant pathogens, and the futility of the long term strategy of developing new, stronger antibiotics. [82-83] As author Geoffrey Cowley observed, "Drug resistant microbes don't threaten us all equally. A healthy immune system easily repels most bacterial invaders, regardless of their susceptibility to drugs." [84] Maintaining a healthy immune system depends upon maintaining a healthy nervous system.

Clinical Applications

It is obvious that these neurobiological models are not mutually exclusive, and that any or all may be operative in a given patient. Clinical practice requires that theoretical models of nerve dysfunction be operationalized. This process has resulted in the development of clinical operational models. Selection of outcomes assessments is dependent upon the nature of the model employed by the practitioner.

Cooperstein[85] described two broad approaches to chiropractic technique, the segmental approach and the postural approach. Murphy[86] added a third, the tonal approach. These conceptual models determine the nature of the analytical procedures employed, the type of adjustments applied, and the criteria for determining the success or failure of a given intervention.

A summary of each follows:

1. *The segmental model*. Subluxation is described in terms of alterations in specific intervertebral motion segments. In segmental approaches, the involved motion segments may be identified by radiographic procedures which assess intersegmental disrelationships, or by clinical examination procedures such as motion palpation. Examples of segmental approaches are the Gonstead[87] and Diversified techniques. [88]

2. *Postural approaches.* In postural approaches, subluxation is seen as a postural distortion. Practitioners of postural approaches assess "global" subluxations using postural analysis and radiographic techniques which evaluate spinal curves and their relationship to the spine as a whole. Examples of techniques emphasizing a postural approach are Pettibon Spinal Biomechanics[89] and Applied Spinal Bioengineering. [90-91]

3. *Tonal approaches.* In 1910, D. D. Palmer[92] wrote, "Life is an expression of tone. Tone is the normal degree of nerve tension. Tone is expressed in function by normal elasticity,

strength, and excitability...the cause of disease is any variation in tone." Tonal approaches tend to view the spine and nervous system as a functional unit. Tonal approaches emphasize the importance of functional outcomes, and acknowledge that clinical objectives may be achieved using a variety of adjusting methods. Examples of tonal approaches include Network Spinal Analysis[92-93] and Torque-release Technique. [94]

Conclusion

In reviewing the preceeding basic science and clinical models of the subluxation, it may be seen that the wide diversity of techniques in chiropractic may use different methods, but generally share the common objective of correcting spinal nerve interference caused by vertebral subluxation. Commonality and accountabiliy may be achieved through the development of models which emphasize clinical outcomes, yet afford the practitioner flexibility in determing how those objectives are achieved. Such outcomes include, but are not limited to, evidence of functional integrity of the nervous system, and improvement in general health and quality of life indicators. Research resources should be directed toward the development of models and clinical strategies which result in more predictable and more efficient practice procedures.

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