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The Mechanism of the Chiropractic Spinal Adjustment/Manipulation: The Neurological Effect Part 3 of a 5 Part Series

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The Mechanism of the Chiropractic Spinal Adjustment/Manipulation: Bio-Neuro-Mechanical Effect

Part 3 of a 5 Part Series

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A report on the scientific literature

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In part 1 of this series, we discussed the osseous mechanisms of the chiropractic spinal adjustment (CSA) and in part 2 we discussed the mechanical and neurological functions of connective tissue. It is in this connective tissue as well as in other neurological components located in the osseous structure of the spine that the primary effector structures of a CSA are to be found. To fully understand the bio-neuro-mechanical mechanism of the CSA, we must explore the mechanical aspect of the chiropractic adjustment, what effect it has on the neurological effector organs, how the spine and brain are inter-related and finally, how the muscles and ligaments (intervertebral discs) working in tandem effectuate homeostasis.

HISTORICAL REPORTING

Kent (1996) reported:

Dishman and Lantz developed and popularized the five component model of the “vertebral subluxation complex” attributed to Faye. However, the model was presented in a text by Flesia 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 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 has since revised and expanded the "vertebral sub-luxation 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 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... (p.1)

However, like many theories, these concepts have proven close to accurate and this report of the literature, although not designed to prove or disprove the Vertebral Subluxation Complex, validated many of the previous "beliefs" based upon contemporary findings in the literature and personal clinical experience, which along with patient expectations, are the three key components to evidence-based medicine.

CONTEMPORARY FINDINGS

In Part 1, we discussed specific biomechanical references in modern literature.

Evans (2002) reported:

...on flexion of the lumbar spine, the inferior articular process of a zygapophyseal joint moves upward, taking a meniscoid with it. On attempted extension, the inferior articular process returns toward its neutral position, but instead of re-entering the joint cavity, the meniscoid impacts against the edge of the articular cartilage and buckles, forming a space-occupying "lesion" under the capsule: a meniscoid entrapment...A large number of type III and type IV nerve fibers (nociceptors) have been observed within capsules of zygapophyseal joints. Pain occurs as distension of the joint capsule provides a sufficient stimulus for these nociceptors to depolarize. Muscle spasm would then occur to prevent impaction of the meniscoid. (p. 252-253)

This verifies that with a vertebra out of position, there is a negative neurological sequella that causes a "cascade effect" bio-neuro-mechanically. Historically, this has been objectively identified and in chiropractic practices called a vertebral subluxation. This nomenclature has been accepted federally by the U.S. Department of Health and Human Services and by the Centers for Medicare and Medicaid Services as an identifiable lesion, for which the chiropractic profession has specific training in its diagnosis and management.

To further clarify the modern literature, Panjabi (2006) stated:

The spinal column has two functions: *structural* and *transducer*. The structural function provides stiffness to the spine. The transducer function provides the information needed to precisely characterize the spinal posture, vertebral motions, spinal loads etc. to the neuromuscular control unit via innumerable mechanoreceptors present in the spinal column ligaments, facet capsules and the disc annulus. These mechanical transducers provide information to the neuromuscular control unit which helps to generate muscular spinal stability via the spinal muscle system and neuromuscular control unit. (p. 669)

Panjabi (2006) reported:

1. Single trauma or cumulative microtrauma causes *subfailure injury* of the spinal ligaments and injury to the mechanoreceptors [and nociceptors] embedded in the ligaments.
2. When the injured spine performs a task or it is challenged by an external load, the transducer signals generated by the mechanoreceptors [and nociceptors] are corrupted.
3. Neuromuscular control unit has difficulty in interpreting the corrupted transducer signals because there is spatial and temporal mismatch between the normally expected and the corrupted signals received.
4. The muscle response pattern generated by the neuromuscular control unit is corrupted, affecting the spatial and temporal coordination and activation of each spinal muscle.
5. The corrupted muscle response pattern leads to corrupted feedback to the control unit via tendon organs of muscles and injured mechanoreceptors [and nociceptors], further corrupting the muscle response pattern.
6. The corrupted muscle response pattern produces high stresses and strains in spinal components leading to further subfailure injury of the spinal ligaments, mechanoreceptors and muscles, and overload of facet joints.
7. The abnormal stresses and strains produce inflammation of spinal tissues, which have abundant supply of nociceptive sensors and neural structures. (p. 669-670)

This indicates that once there is a bio-neuro-mechanical lesion (aka vertebral subluxation), there is a “negative cascade” both structurally (biomechanically) and neurologically in the body’s attempt to create homeostasis. However, should the cause of the lesion not be “fixed,” the entire system will perpetually fail. Over time, due to the Piezoelectric effect and Wolff’s Law of remodeling, the skeletal structure is now permanently altered. Therefore, treatment goals then switch from curative to simply management and is a long-term process.

In part 2, we discussed subfailure, and will examine it again as explained by Solomnow (2009).

Solomonow (2009):

Inflammatory response in ligaments is initiated whenever the tissue is subjected to stresses which exceed its routine limits at a given time. For example, a sub-injury/failure load, well within the physiological limits of a ligament when applied to the ligament by an individual who does not do that type of physical activity routinely. (p. 143)

Jaumard, Welch and Winkelstein (2011) reported:

In the capsular ligament under stretch, the collagen fiber structure and the nerve endings embedded in that network and cells (fibroblasts, macrophages) are all distorted and activated. Accordingly, capsular deformations of certain magnitudes can trigger a wide range of neuronal and inflammatory responses...Although most of the proprioceptive and nociceptive afferents have a low-strain threshold (~10%) for activation, a few receptors have a high-strain threshold (42%) for signal generation via neural discharge. In addition, capsular strains greater than 47% activate nociceptors with pain signals transmitted directly to the central nervous system. Among both the low- and high-strain threshold neural receptors in the capsular ligament a few sustain their firing even after the stretching of the capsular ligament is released. This persistent afterdischarge evident for strains above 45% constitutes a peripheral sensitization that may lead to central sensitization with long-term effects in some cases. (p. 12)

The cascade effect works in 2 directions, one to create a bio-neuro-mechanically failed spinal system and one to correct a bio-neuro-mechanically failed system.

Pickar (2002) reported:

The mechanical force introduced into the during a spinal manipulation (CSA) may directly alter segmental biomechanics by releasing trapped meniscoids, releasing adhesions or by reducing distortion of the annulus fibrosis. (p. 359)

This fact verifies that there is an osseous-neurological component that exists with the nociceptors at the facet level.

Pickar (2002) also stated:

In addition, the mechanical thrust could either stimulate or silence nonnociceptive, mechanosensitive receptive nerve endings in paraspinal tissue, including skin, muscle, tendons, ligaments, facet joints and intervertebral disc. (p. 359)

CENTRAL NERVOUS SYSTEM MODULATION

When discussing central nervous system activity as a direct sequella to a CSA, we must divide our reporting into 2 components, reflexively at the area being adjusted and through higher cortical responses. When discussing local reflexive activity, we must also determine if it is critical to adjust the specific segment in question or if the adjustment will elicit neurological and end organ (muscle) responses to help create a compensatory action for the offending lesion.

Reed and Pickar (2015) reported in an animal study:

First, during clinically relevant spinal manipulative thrust durations (≤ 150 ms), unilateral intervertebral joint fixation significantly decreases paraspinal muscle spindle response compared with non-fixated conditions. Second and perhaps more importantly, this study shows that while L₄ muscle spindle response decreases with L₄ HVLA-SM, 60%-80% of an L₆ HVLA-SM muscle spindle response is still elicited from an HVLA-SM delivered 2 segments away in both the absence and presence of intervertebral joint fixation. These findings may have clinical implications concerning specific (targeted) versus nonspecific (nontargeted) HVLA-SM. (p. E755-E756)

Reed and Pickar (2015) also reported:

The finding that nontarget HVLA-SM delivered 2 segments away elicited significantly less but yet a substantial percentage (60%–80%) of the neural response elicited during target HVLA-SM may have important clinical implications with regard to HVLA-SM thrust accuracy/specificity requirements. It may explain how target vs non-target site manual therapy interventions can show similar clinical efficacy. In a recent study using the same model as the current study, the increase in L₆ muscle spindle response caused by an HVLA-SM is not different between 3 anatomical thrust contact sites (spinous process, lamina, and mammillary body) on the target L₆ vertebra but is significantly less when the contact site is located 1 segment caudal at L₇...The current study confirms that a nontarget HVLA-SM compared with a target HVLA-SM decreases spindle response but adds the caveat that a substantial percentage (60%–80%) of afferent response can be elicited from an HVLA-SM delivered 2 segments away irrespective of the absence or presence of intervertebral fixation. (p. E756)

Coronado, Gay, Bialosky, Carnaby, Bishop and George (2012) reported that:

Reductions in pain sensitivity, or hypoalgesia, following SMT [spinal manipulative therapy or the chiropractic adjustment] may be indicative of a mechanism related to the modulation of afferent input or central nervous system processing of pain...The authors theorized the observed effect related to modulation of pain primarily at the level of the spinal cord since 1.) these changes were seen within lumbar innervated areas and not cervical innervated areas and 2.) the findings were specific to a measure of pain sensitivity (temporal summation of pain), and not other measures of pain sensitivity, suggesting an effect related to attenuation of dorsal horn excitability and not a generalized change in pain sensitivity. (p. 752)

These findings indicate that a chiropractic spinal adjustment affects the central nervous system specifically at the interneuron level in the dorsal horn. This is part of the cascade effect of the CSA where we now have objectively identified the mechanism of the central nervous system stimulation and its effects.

Gay, Robinson, George, Perlstein and Bishop (2014)

...pain-free volunteers processed thermal stimuli applied to the hand before and after thoracic spinal manipulation (a form of MT [Manual Therapy]). What they found was, after thoracic manipulation, several brain regions demonstrated a reduction in peak BOLD [blood-oxygen-level dependent] activity. Those regions included the cingulate, insular, motor, amygdala and somatosensory cortices, and the PAG [periaqueductal gray regions].

The purpose of this study was to investigate the changes in FC [functional changes] between brain regions that process and modulate the pain experience after MT [manual therapy]. The primary outcome was to measure the immediate change in FC across brain regions involved in processing and modulating the pain experience and identify if there were reductions in experimentally induced myalgia and changes in local and remote pressure pain sensitivity. (p. 61)

Therefore, a thoracic CSA adjustment produced direct and measurable effects on the central nervous system across multiple regions, specifically the cingulate cortex, insular cortex, motor cortex, amygdala cortex, somatosensory cortex and periaqueductal gray matter. This could only occur if “higher centers,” also known as the central nervous system, were affected.

Gay, Robinson, George, Perlstein and Bishop (2014) went on to report:

Within the brain, the pain experience is subserved by an extended network of brain regions including the thalamus (THA), primary and secondary somatosensory, cingulate, and insular cortices. Collectively, these regions are referred to as the *pain processing network* (PPN) and encode the sensory discriminate and cognitive and emotional components of the pain experience. Perception of pain is dependent not merely on the neural activity within the PPN [pain processing network] but also on the flexible interactions of this network with other functional systems, including the descending pain modulatory system. (p. 617)

Daligadu, Haavik, Yelder, Baarbe, and Murphy (2013) reported that:

Numerous studies indicate that significant cortical plastic changes are present in various musculoskeletal pain syndromes. In particular, altered feed-forward postural adjustments have been demonstrated in a variety of musculoskeletal conditions including anterior knee pain, low back pain and idiopathic neck pain. Furthermore, alterations in trunk muscle recruitment patterns have been observed in patients with mechanical low back pain. (p. 527)

This concludes that there are observable changes in the function of the central nervous system seen in patients with musculoskeletal conditions and chronic pain. Chiropractors have observed this clinically and it demonstrates the necessity for chiropractic care for both short and long-term management of biomechanical spinal conditions.

CONCLUSION

Although there is significantly more research verifying what occurs with a CSA, the above outlines the basics of how the adjustment works both biomechanically and neurologically from the connective tissue and peripheral nerves to the central nervous system both at the cord level and higher cortical regions. The final question is one of public safety.

Based on their study on 6,669,603 subjects after the unqualified subjects had been removed, Whedon, Mackenzie, Phillips, and Lurie (2015) concluded, “No mechanism by which SM [spinal manipulation] induces injury into normal healthy tissues has been identified” (p. 265).

Part 4 will be the evidence of subluxation degeneration and the literature verifying the mechanisms. Part 5, the final part of our series, will be an in-depth contemporary comparative analysis of the chiropractic spinal adjustment vs. physical therapy joint mobilization.

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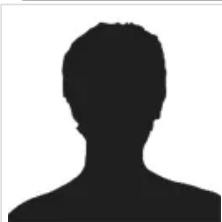
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