

# A Contemporary View Of Subluxation That Is Consistent With The Founder's Views: A Commentary

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## ABSTRACT

The vertebral subluxation remains a topic of debate within the chiropractic profession. Unfortunately, little chiropractic-driven research has been done to elucidate the precise pathophysiological character of vertebral subluxation. We are left to consider various models of subluxation that, while put forth by chiropractors, are based largely on basic science research that has been done by scientists outside the chiropractic profession. That this statement is one of fact, is represented in Kent's review of subluxation models in this journal (1996), in which virtually none of the cited research used to support the various models of vertebral subluxation was performed by chiropractors. This

is true of any paper written about models of vertebral subluxation. While this is not inherently problematic, it does illustrates that the chiropractic profession needs to develop and perform subluxation research, and also demands that we be consistent with our application of basic science research when we use it to describe potential models of vertebral subluxation. This commentary will offer a contemporary view of subluxation based on physiology and pathophysiology, which happens to be consistent with the writings of DD Palmer.

**Key Words:** *Subluxation, subluxation complex, nociception, inflammation, pathology*

## Introduction

It is popular for chiropractors to discuss models of subluxation. For example, Leach<sup>1</sup> and Kent<sup>2</sup> have described various models of subluxation including segmental dysfunction, neurocompression, the vertebral subluxation complex, component models and neurodystrophy. These models have found their origin with various chiropractors or researchers, such as BJ Palmer, Homewood<sup>3</sup>, Faye<sup>4</sup>, and Korr<sup>5</sup>.

While models are valuable in that they provide us with mental constructs with which we can conceptualize and explain subluxation, such models cannot be viewed as rigged categories into which we must fit our patients. For example, a patient presents with severe disc degeneration, facet hypertrophy, loss of sensation and reflexes, and radicular pain. It would be unreasonable to suggest that this patient's primary problem stems from the neurodystrophic model of subluxation. Clearly, this patient suffers with a neurocompressive model, and must be cared for appropriately. Chiropractic care may restore such patients to full function<sup>6</sup>; however, we all know that some of these patients do not respond to chiropractic care and must be referred for surgery.

Another patient presents with diffuse pain in the back that often extends into the thigh, leg, and sometimes the foot. This

patient has no disc degeneration, no facet hypertrophy, and all neurological and orthopedic tests are normal. Radiographs are unremarkable, such that one would not even remotely hypothesize that this patient would suffer with back pain, if examination of the x-rays were the sole diagnostic criteria. Clearly, this patient could not possibly have a neurocompressive subluxation, as no such symptoms or signs exist. This patient's pain is nociceptive or somatic in nature. The pain referral is also nociceptive or somatic in nature; sometimes called sclerotogenous or sclerotomal pain. What model of subluxation fits this patient?

A third patient presents without pain, yet complains of nagging malaise, a tendency to develop numerous colds per year, and recently discovered that he has slight hypertension. After a handful of adjustments, this patient is relieved of his complaints. What model of subluxation would fit this individual? In order to understand the nature of subluxation in the last two patients, we need to consider pathophysiological mechanisms related to the spine; and it was DD Palmer who set us on the right course about 100 years ago.

## Trauma, toxins, autosuggestion and subluxation

In 1982, I completed my first Chiropractic Principles class, at New York Chiropractic College, in which we were taught that trauma, toxins, and autosuggestion are the cause of subluxation. We were told that DD Palmer was the author of this statement.

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It was several years later that I acquired Palmer's 1910 text entitled *The Chiropractor's Adjuster*<sup>7</sup>, in which he stated: "the determining cause of disease are traumatism, poison, and auto-suggestion," which could also be stated as trauma, toxins, and autosuggestion are the cause of disease. I searched and searched, but no where in DD's text did it state that trauma, toxins, and autosuggestion cause subluxation or dis-ease, which came to me as somewhat of a surprise. This led me to question whether or not subluxation might be a disease, which of course, was contrary to what I had been taught, i.e., that subluxation was the cause of dis-ease and ultimately disease.

Then I examined the commonly accepted descriptions of the subluxation complex put forth by Faye<sup>4</sup> and Lantz<sup>8</sup> that were embraced by nearly all chiropractors, which characterize subluxation by tissue pathology (i.e., disease), such as myopathology, kinesiopathology, and histopathology. Whether we as a profession realized it or not, when we embraced the Faye and Lantz models, we all embraced subluxation as a pathology, or disease.

By definition, pathology begins in response to cell or tissue injury and is reflected as an alteration in tissue function that occurs long before symptoms appear<sup>9,10</sup>. When left unchecked, a subclinical pathology will develop into an overt pathology. Thus, anytime we have a shift away from normal physiological function, we have disease. The first edition of *Pathologic Basis of Disease*<sup>9</sup>, published in 1974, stated it quite clearly:

"All injuries, whether mild or lethal, ultimately occur at a biochemical level beyond our present range of detection. For this reason, it has not been possible to determine the precise biochemical site of action of injurious agents or the extent of cellular injury compatible with reversibility or irreversibility. Four intracellular systems are thought to be particularly vulnerable: (1) aerobic respiration involving oxidative phosphorylation and production of ATP, (2) synthesis of enzymatic and structural proteins, (3) maintenance of the integrity of cellular membranes on which the ionic and osmotic homeostasis of the cell and its organelles are dependent, and (4) preservation of the integrity of the cell's genetic apparatus."

These sentiments have been echoed through every edition of *Robbins Pathologic Basis of Disease*, including the current edition<sup>10</sup>, which suggests that we must learn to think biochemically when considering a so-called structural pathology such as subluxation. Regarding overt structural pathologies, Robbins originally stated that, "morphologic changes become apparent only after some critical biochemical system within the cell has been deranged for some time"<sup>9</sup>. In other words, biochemical dysfunction will lead to pathological changes in a given structure, such as disc degeneration or muscle atrophy, which are considered to be part of the subluxation complex. Several common biochemical themes are important in the mediation of cell injury and cell death, i.e., pathology, whatever the inciting agent. These include ATP depletion, free radicals, intracellular calcium and loss of calcium homeostasis, defects in membrane permeability, and irreversible mitochondrial damage<sup>10</sup>, none of which can be consciously felt or scientifically detected when they first begin, and this is why every chronic disease begins asymptotically.

Most individuals do not realize they have heart disease or cancer until it is too late. The same holds true for subluxation, such that altered spinal function can exist for years before it

becomes symptomatic, and even when symptoms develop they are not consistent from person to person. For example, one may develop neck pain, another one gets headaches, someone else gets nausea, and another dizziness<sup>11</sup>.

In summary, we should consider that trauma or microtrauma to spinal tissues (joint, muscle, and/or disc) results in the release of chemical mediators of inflammation and nociception, such as prostaglandin E2, thromboxane A2, leukotriene B4, histamine, serotonin, interleukin-1, interleukin-6, interleukin-8, and tumor necrosis factor<sup>12,13</sup>. In the case of herniated discs, macrophages will infiltrate the area and release the same mediators of inflammation. Additionally, cells of the disc itself, including histiocytes, fibroblasts, endothelial cells and chondrocytes will also produce such mediators<sup>13</sup>.

These mediators stimulate nociceptors that reside in spinal tissues, which then result in the generation of action potentials in the nociceptive fibers that enter the dorsal horn of the spinal cord. Nociceptive bombardment into the cord has many potential outcomes. Segmental responses include excitation of alpha-motoneurons and gamma-motoneurons, as well as preganglionic sympathetic fibers, which can result in alterations of muscle tone, mobility, vascular changes, and inflammatory activity<sup>11</sup>. Over time these alterations of normal function lead to the development of kinesiopathology, myopathology, histopathology, etc., which we refer to as the subluxation complex, and which consist of the objective findings we associate with subluxation.

### How should we view subluxation?

Our view of subluxation should depend, in part, on patient history and physical findings. What happened to the patient? Did he suffer a neurocompressive injury or a musculoskeletal injury? What are the signs and symptoms? These factors give insight to the nature of the spinal problem or which so-called model of subluxation that may be present. It all depends on the patient, not on our personal preference toward a given model of subluxation.

Neurocompression is known to be an extremely rare clinical occurrence; perhaps 1% of patients under the age of 65 suffer with neurocompressive lesions<sup>14</sup>. Accordingly, most patients are not plagued by neurocompression and instead suffer from an injured and dysfunctional musculoskeletal system; that is, injury to spinal joints, ligaments, muscles, and disc, and the various pathological changes that we chiropractors call the subluxation complex.

We should consider viewing these pathological spinal changes as a consistent outcome of spinal injury. It is well known that spinal muscles atrophy, connective tissues degenerate, and chronic inflammation develops after trauma and due to sedentary living<sup>8,14-22</sup>. We chiropractors call this disease "subluxation," which means far more to us than the term "back pain." That is to say, subluxation is not synonymous with back pain, and this should be emphasized.

Trauma is the initiator and the chemical mediators (toxins) drive the inflammatory and nociceptive processes, which is consistent with DD Palmer's 100 year-old description. Consider that Palmer stated that disease may be associated with either an increase or a decrease in the momentum of impulses<sup>7</sup>.

It is known that the chemical mediators of inflammation and nociception dramatically increase the impulses in nociceptive neurons<sup>23</sup>. For example, the resting discharge of neurons exiting the cat knee joint is 1,800 impulses in a 30 second interval, which increases to 11,000 impulses during inflammation. During motion, some 4,400 impulses discharge in 30 seconds, whereas with inflammation, some 31,000 impulses occur, reflecting a 7-fold increase. When individual afferents have been studied during normal and inflamed conditions, it was determined that there can be as much as a 100-fold increase in nociceptive impulses with inflammation<sup>23</sup>. When it comes to human spinal joints, McLain found mechanoreceptors in only 17 of 21 cervical spinal joint capsules. In the 17 joint capsules only 35 mechanoreceptors were found, but numerous nociceptors were present in all 21 specimens<sup>24</sup>, reflecting the abundance of nociceptors in human joints that will respond dramatically to spinal joint injury. Clearly, spinal injury and inflammation will result in a significant increase in the momentum of nociceptive impulses, which is commonly experienced as pain.

Back pain is only one potential manifestation of subluxation due to such extraordinary increases in nociceptive impulses, and this point needs to be emphasized. Some patients may not suffer at all; some may experience moderate pain, while others may experience severe pain. Certain patients will experience no pain at all and instead, suffer from a variety of visceral symptoms. Consider the fact that nociceptive input from the subluxation complex can stimulate the vegetative center in the brainstem and hypothalamus and induce profound neuroendocrine and immune responses<sup>11,25-27</sup>, such that the term “visceral disease mimicry syndromes” has been used to describe how spinal subluxation can generate symptoms that are often indistinguishable from true visceral disease<sup>27</sup>.

It is likely that numerous individuals are suffering with “visceral disease mimicry syndromes” from which they will not find adequate relief unless they seek out the aide of a chiropractor. We need to better educate ourselves about these visceral manifestations and appropriately educate the public.

Clearly, we as chiropractors must reduce subluxation with adjustments as they can perceivably cause a host of maladies and reduce one’s ability to express their fullest potential. However, if we want to be thorough and serve our patients at the highest level, we should also consider addressing the causes of subluxation as well.

### **The cause of subluxation**

We need to examine the possibility that reduction of subluxation with an adjustment may be akin to a dentist reducing a cavity. The reduction of the cavity only addresses the acute cause of the pain and suffering, it does not address the underlying cause of the cavity, i.e., an unhealthy body due to poor dental hygiene and a sugar-rich diet.

Similarly, the surgical excision of a tumor only removes the tumor; it does nothing for the sick body from which the tumor developed, i.e., the cause of the tumor is not addressed by surgery or radiation therapy. In other words, if cancer patients do not change their lifestyle, particularly related to diet, their chance

of recurrence is greater. This same line of thinking applies equally to chiropractic care.

While an adjustment may reduce a subluxation and its subsequent nociceptive induction of a wide variety of symptoms, we should consider that we are then left with the same unhealthy body that promoted the development of subluxation. Lifestyle changes are of paramount importance to reduce and prevent subluxation. We should not mislead our patients into thinking they are healthy because they are pain-free or because we perceive that they have been rendered subluxation-free.

Thus, we should urge our patients to be wary of the causes of subluxation, and educate them about the simple preventive procedures that are available. This is not at all inconsistent with DD Palmer’s thinking, as he made an effort to be consistent with the science of his time. He made himself quite clear when he stated:

“There are two classes of Chiropractors, those who desire to know all they can of physiology, pathology, neurology and anatomy, and those who have an aversion for intelligence, do not want to take effect into consideration, depending only upon an examination of the spinous processes.”<sup>7</sup>

Trauma is a fact of life, and a deconditioned spine is predisposed to injury and subluxation<sup>22,28</sup>. It is now a known fact that spinal muscle atrophy and associated fatty infiltration occurs in both symptomatic and asymptomatic individuals<sup>29,30</sup>. If this was known in DD Palmer’s day, he would have embraced the need for spinal exercises, as only exercise can reverse muscle atrophy and render the spine stable. Appropriate spinal stabilization exercises, postural training, and balance exercises have been outlined by several chiropractors for the purpose of reconditioning spinal muscles to help prevent spinal injury and subluxation<sup>31-34</sup>.

We all know that lack of exercise and various dietary factors can promote cancer and heart disease. Accordingly, we would look askance at the surgeon who removes a tumor or performs angioplasty and says nothing to the patient about regular cardiovascular exercise or adopting appropriate dietary modifications to prevent the recurrence of cancer or heart disease. Should we not hold ourselves to the same standard?

It is well-known that omega-6 fatty acids promote the production of inflammatory and fibroplastic mediators (toxins when produced in excess) that drive both cancer and heart disease, while omega-3 fatty acids inhibit their production and prevent these diseases<sup>35,36</sup>. Foods rich in omega-6 fatty acids (grains, grain products, seeds, and seed oils) should be avoided and replaced by omega-3 fatty acid-rich and flavonoid-rich foods including fish, omega-3 eggs, green vegetables, and fruit. These same recommendations apply to reducing and preventing subluxation, as the same mediators that drive cancer, heart disease, and Alzheimer’s disease are known to promote the development of subluxation<sup>37,38</sup>.

Is it not our duty to make such simple health-promoting recommendations to our patients? Should we rely on other professionals to make appropriate exercise and nutritional recommendations to help correct and prevent subluxation? Clearly, this does not make sense and is also inconsistent with Faye’s original model for correcting the subluxation complex<sup>4</sup>, and also inconsistent with The Palmer Tenets<sup>39</sup>, which is a current guid-

ing document for the first chiropractic college and is reflective of the views of the Founder of Chiropractic.

## Conclusion

The study of pathology has advanced significantly since the time of DD Palmer. The first issue of Robbin's Pathologic Basis of Disease was published in 1974<sup>9</sup>, and in 1999, the 6th edition was published<sup>10</sup>, reflecting numerous advances in knowledge that have occurred in a mere 30 years. The chiropractic profession began over 100 years ago, and Palmer published *The Chiropractor's Adjustor* some 93 years ago. Our knowledge of pathology has changed drastically since Palmer's day. He clearly made an effort to be up-to-date with the sciences during his time, and there is no reason to suspect that would be different today.

The pathological process begins slowly and subclinically, with trauma, toxins and autosuggestion as the promoters. It is well known that degenerative and inflammatory processes can rage on for years in a subclinical state. When this pathological process occurs in the spine and reaches a clinical or symptomatic threshold, patients will present to our offices. In effect, we tell patients that we use the term "subluxation complex" to describe this pathological manifestation in the spine that involves kinesiotherapy, neuropathophysiology, myopathy, histopathology, and biochemical abnormalities/inflammation.

The correction of subluxation should be multifactorial and based on causation. It is likely that the adjustment will influence kinesiotherapy, neuropathophysiology and histopathology; stabilization exercises will influence myopathy and histopathology; and nutrition will address biochemical abnormalities/inflammation and histopathology. Most of our colleges now teach such an approach to patient care, although it is not consistently applied, and in many cases, never applied to the correction of subluxation. This needs to change and needs to be reflected in research efforts at our colleges.

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