The Causes of the Vertebral Subluxation Complex and Related Treatment Approaches

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Introduction

At one time or another, all chiropractors have been told that we address the cause of disease and disease. Indeed, many DCs presume that we do not treat symptoms or diseases, and that is what separates us from the medical profession. Current research in the basic and clinical sciences suggests that this presumption should be aborted. This paper will demonstrate that chiropractors do, in fact, treat a disease process known to us as the subluxation complex.

It is a well known fact that diseases are driven into existence by certain causative factors. Are all the causes for each disease completely understood and categorized at the present time? Of course not. However, researchers and clinicians accept the fact that reducing the incidence of disease depends largely upon eliminating the associated causative factors. This paper will discuss factors which cause the subluxation complex and lead us toward developing a rational treatment approach.

The subluxation complex is a disease

Certain DCs may find it offensive to read that subluxation is being defined as a disease. Some readers may go as far as to say that such an assertion is heresy. However, it must be understood that this conclusion, that subluxation is a disease, is not my opinion; rather, it is a fact that is asserted by our chiropractic colleges and associations.

Is it not a fact that all DCs describe subluxation as a disruption of spinal function that is characterized by certain pathological changes such as myopathology, histopathology, neuropathophysiology? The subluxation complex has been described as having five, eight, and now nine pathological components. Is it not true that all so-called straight and mixer DCs embrace one of these models which have been popularized by Faye, Flesia or Lantz? Absolutely, the answer is yes.

Medical and standard dictionaries discuss disease when they define "pathology" or "pathological." Examine any pathology book and you will see that each discusses a variety of diseases. Indeed, pathology refers to "the structural and functional manifestations of a disease."[1]

For some reason, the entire chiropractic profession defines subluxation as a pathological en-

tity/disease, and then a large percentage of the profession goes on to claim that we do not treat disease. This is an oxymoron. Clearly, the subluxation complex is a type of spinal disease, which chiropractors treat via the chiropractic adjustment.

What causes the subluxation complex?

In 1910, DD Palmer stated that disease is caused by trauma, toxins and autosuggestion.[2] Modern pathology texts, such as Robbins' *Pathological Basis of Disease*, provide a list of disease-causing agents which fall into one of the categories put forth by D.D. Palmer. At the present time, most chiropractic colleges teach, and have taught, that subluxations are caused by agents found in these three categories.

In a moment we will look at a process by which trauma, toxins and autosuggestion cause subluxation. But first, it is important to ask ourselves a question. Does the chiropractic adjustment eliminate a cause of disease or does it address the disease itself? In fact, the adjustment treats disease. This is because the adjustment does not and cannot eliminate trauma, toxins and autosuggestion. Thus, it is safe to say that chiropractors treat disease and not its cause. From this perspective, we are just like medical doctors.

Trauma, toxins, autosuggestion and the nervous system

Contemporary research defines how trauma, toxins (chemical irritants), and autosuggestion can enhance nociception and reduce mechanoreception. I propose that enhanced nociception can act as the primary drive that reduces joint mobility, and further suggest that such hypomobility will initiate and perpetuate the pathogenesis of the subluxation complex, i.e., the development of the local pathological and inflammatory components.

I have been told by many DCs that the information in the preceding paragraph is confusing. The confusion is based on the fact that very few practitioners understand the nature of nociception and mechanoreception, and then describe their relationship to the subluxation complex.

As we all know, sensory neurons are designed to receive and transmit stimuli, from outside the body, into the central nervous system. Sensory neurons are able to do this because they possess specialized "nerve endings" known as receptors. In actual fact, receptors are "nerve beginnings," because impulses in sensory neurons begin at the level of the receptor and end in the spinal cord.

The majority of well-respected, contemporary texts indicate that there are two main categories of sensory receptors, those being nociceptors and mechanoreceptors.[3,4,5,6] Such contemporary texts do not discuss the interoceptor, exteroceptor and proprioceptor system of classification. This appears to be a wise choice as *Gray's Anatomy*[7] goes as far as to state that the proprioceptor classification system is "rather arbitrary."

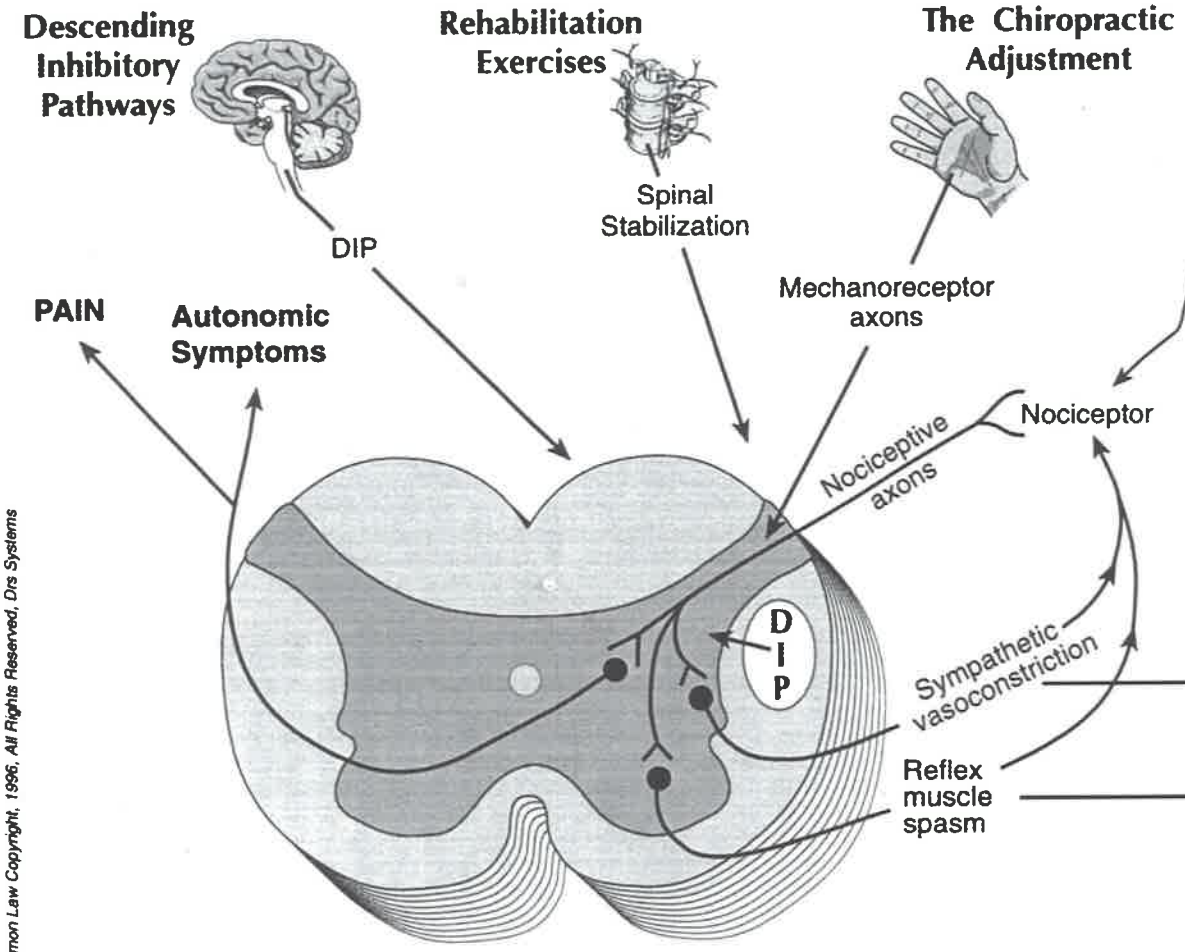
There are two main points that we must drive into our memory banks regarding nociceptors and mechanoreceptors. First, it must be understood that nociceptors and mechanoreceptors are located in skin, muscles, and joints [5], the precise tissues that chiropractors address. Second, DCs must understand that nociceptors and mechanoreceptors encode completely different types of stimuli. Whereas nociceptors encode noxious stimuli such as that associated with body movement and physical touch,[5] I must emphasize the fact that mechanoreceptors, or the so-called proprioceptors, do not encode noxious mechanical or chemical stimuli associated with tissue injury.[5,8,9,10]

Nociceptors

Any good physiology or pathology book will explain that tissue injury results in the release of a variety of chemical mediators, such as prostaglandin E-2, bradykinin, serotonin, and histamine. The same books will tell you that the chemical mediators serve two main functions, they stimulate nociceptors and act as the driving force behind the inflammatory process. Thus, bradykinin et al. can be referred to as the chemical mediators of inflammation and nociception. Nociceptors are also stimulated by the noxious mechanical irritation that occurs during tissue injury.

Nociceptors are classified as mechanical nociceptors, mechanothermal nociceptors and polymodal nociceptors, depending on the type of energy used to activate them in the nociceptive range. Polymodal nociceptors are activated by noxious mechanical and thermal stimulation, as well as the chemical mediators released from the injured tissues.[9] Generally speaking, mechanical and mechanothermal nociceptors are

CHIROPRACTIC & THE DORSAL HORN



NOCICEPTIVE IRRITANTS

- A. Mechanical (trauma, injury)
- B. Chemical
  1. Lactic acid
  2. Potassium ions
  3. Prostaglandin E-2
  4. Leukotriene B-4
  5. Glycosaminoglycans
  6. Histamine
  7. 5-hydroxytryptamine
  8. Bradykinin

Muscle spasm & vasoconstriction initiate & perpetuate the subluxation complex:

1. Kinesiopathology
2. Neuropathophysiology
3. Myopathology
4. Connective Tissue Pathology
5. Vascular Abnormalities
6. Inflammatory Response
7. Histopathology
8. Biochemical Abnormalities

associated with typeA-delta or Group III fibers, whereas polymodal nociceptors are associated with type C or Group IV fibers.

Nociceptors are located in nearly every single musculoskeletal, connective tissue, and vascular structure, save for the joint cartilage, synovial membranes, the nucleus pulposus and inner layers of the annulus fibrous.[11,12,13,14] Wyke provides the most vivid anatomical description of the nociceptive receptor system.[15] He describes interstitial nociceptors as "a continuous tridimensional plexus of unmyelinated nerve fibers that weaves (like chicken-wire) in all directions throughout the tissue." A similar plexus of unmyelinated nerve fibers are embedded in the adventitial sheath and encircle each blood vessel. From this description, we can envision the presence of an unending meshwork of nociceptors within nearly all spinal tissues.

The extent of nociceptive innervation has been described in muscles and joints. It is thought that, save for afferents from stretch receptors, 75% of the sensory innervation of skeletal muscles is supplied by nociceptors located in fascia, tendons, between muscle fibers, and in the walls of blood vessels.[16] In one study, which examined the medial articular nerve of the cat, it was demonstrated that 21% of the fibers were of the A-delta variety and 70% were C fibers. The posterior articular contained 14% A-delta and 60% C fibers.[8]

Based on the information presented thus far, it certainly appears that a significant percentage of our neurological focus should be directed at the nociceptive afferent system because it is the most abundant system found in the spinal structures to which we devote our lives. At this point, many readers may be wondering how nociceptors specifically relate to the subluxation complex. This relationship will be discussed after basics of mechanoreceptor function are described.

Mechanoreceptors

Recall that mechanoreceptors are located in skin, joint capsules, ligaments, and muscles. Examples of mechanoreceptors include Ruffini endings, Merkel cell complexes, Meissner's corpuscles, Pacinian corpuscles, muscle spindles, golgi tendon organs, and many others.[5] Mechanoreceptor afferents (A-beta fibers) influence the nervous system in many ways. For example, the spinal cord level, mechanoreceptive input can inhibit nociception,[17,18] reduce sympathetic hyperactivity[18,19] and reduce muscle spasm by inhibiting the activity of alpha- motoneurons.[18] Mechanoreceptive input can also influence brain function. Review any respectable neuroanatomy book and you will discover that mechanoreceptor afferents end in the brain stem, cerebellum, and cerebral cortex. Indeed, researchers have stated that mechanoreceptor stimulation results in proprioception[20] and suprasegmental motor control.[21] It should be quite obvious that mechanoreceptor input has beneficial neurological effects. This is good news for the chiropractic profession because many authors state that a major neurological effect of the chiropractic adjustment is the stimulation of mechanoreceptors.[22,23,24,25,26]

As mentioned earlier, mechanoreceptors are activated by mechanical stimuli in the non-noxious range, such as normal body movement and physical contact. Researchers explain that joint hypomobility depresses mechanoreceptor activity.[18,27,28,29] Thus, the presence of joint hypomobility, associated with the subluxation complex, most likely results in a reduction in mechanoreceptor activity. A common misconception is that the presence of the subluxation complex results in enhanced activity of spinal mechanoreceptors or so-called proprioceptors. This belief has led to terms such as "proprioceptive insult hypothesis" or "noxious proprioception" to describe the neurological effects of subluxation. It should be clear that these terms are wholly inaccurate because, as mentioned earlier, mechanoreceptors are physiologically incapable of being excited by noxious input.

Nociception, mechanoreception and the subluxation complex

Figure 1 illustrates how the basic sciences can be used to describe clinical chiropractic. Notice that nociceptors are irritated by mechanical (trauma) and chemical irritants (toxins). The associated nociceptive axons (A-delta and C fibers) enter the spinal cord and excite interneurons in the dorsal horn. Ultimately, this leads to the excitation of segmental preganglionic autonomic neurons and somatomotor neurons.[18] The end result can be local tissue vasoconstriction and muscle spasm. These two reflex effects can play

a role in reducing joint mobility.[30] Local nociceptors can be further irritated by the muscle spasm [31] and sympathetic discharge into the area of injury, [32,33,34] which may further stimulate the spasm and vasoconstriction. As the joint in question becomes hypomobile, it is likely that the various pathologic components of the subluxation complex (histopathology, inflammation, etc.) will become more pronounced and further irritate local nociceptors. See Liabenson [35] for a description of the pathological events that occur due to joint immobility.

It should now be clear that the subluxation complex is involved in a process that can mechanically and chemically activate nociceptors. At the same time, the joint hypomobility component (kinesiopathology) of the subluxation complex will reduce the activation of mechanoreceptors. Recall that some of the effects of mechanoreception include the inhibition of nociceptive-induced pain, muscle spasm, and sympathetic hyperactivity. Thus, a lack of mechanoreceptive input due to joint hypomobility may enhance the nociceptive processes described above.

Based on what has been described thus far, we can state with a reasonable degree of confidence that the subluxation complex is associated with abnormal afferent input; that is, enhanced nociception and reduced mechanoreception. I refer to this abnormal afferent input as dysafferentation.

Thus far, the topic of subluxation-induced symptoms has not been discussed. It must be understood that the segmental responses to nociceptive input, those being muscle spasm and vasoconstriction, are not necessarily associated with symptoms. This means that the subluxation complex, like other degenerative diseases, can develop without symptoms.

The types of symptoms that may develop due to the presence of the subluxation complex is largely dependent upon which supraspinal centers are particularly influenced by dysafferentation. For example, the most well-known supraspinal response to nociception is pain (see Figure 1). Recall that nociceptive input is conveyed along the anterolateral pathway to the limbic system where the hurt of pain is experienced.

In addition to pain, nociceptive input can result in a variety of autonomic and neuroendocrine responses (see Figure 1 where it says Autonomic Symptoms). It must be emphasized that the presence of autonomic and neuroendocrine responses is not dependent on the presence of pain.[36,39] Indeed, clinical research has actually demonstrated that, upon noxious irritation of paraspinal muscles, certain patients experience no pain; rather, they are overwhelmed by a distressing combination of nociceptive-induced autonomic concomitants.[38] Some autonomical concomitants include nausea, dizziness, and changes in heart rate, blood pressure and respiration.[38,39] Hyperventilation can even develop.[36] Such responses are thought to occur because nociceptive input can excite the autonomic centers in the hypothalamus and medullary reticular formation.[36]

It should be understood that pain can have catastrophic effects on the human body, which is why our primary goal should be to eliminate pain as quickly as possible. Fear, apprehension and anxiety, which are commonly induced by pain, are known to activate a variety of autonomic and neuroendocrine responses, such as increased blood pressure and the release of catecholamines and cortisol.[36] Research has demonstrated that hypercortisolemia can inhibit immune function, damage the GI tract, reduce tissue healing, and initiate catabolic processes in muscle and connective tissue.[37]

Based on what has been developed thus far, we can theorize that the subluxation complex develops in response to tissue injury and the related enhanced nociceptive input and reduced mechanoreceptive input (dysafferentation) which induce segmental myospasm and sympathetic hyperactivity. After the subluxation complex develops, it now becomes a source of dysafferent input. This is because the subluxation complex is a musculoskeletal lesion characterized by joint hypomobility, myopathology, inflammation, etc., which excites nociceptors and reduces mechanoreceptor activity. The dysafferent input that is associated with the subluxation complex can further enhance the segmental responses, and also result in pain and conceivably generate a variety of patient-specific suprasegmental symptoms and autonomic and neuroendocrine responses. It is very likely that such nociceptive-induced suprasegmental symptoms would respond to chiropractic care because the chiro-

practic adjustment improves musculoskeletal function and reduces nociception.[22,23,24,25,26,39,40]

See Nansel and Szlazak for a review of the suprasegmental responses to nociceptive input.[39] Save for this article, there has been no rational chiropractic research or reports that discuss these autonomic and neuroendocrine relationships to subluxation or the chiropractic adjustment. Nansel and Szlazak cite almost 300 articles that discuss a variety of unexpected symptoms, which mimic visceral disease, that arise due to nociceptive input from somatic tissues. Oddly enough, not one of these papers is discussed in the so-called Mercy or Wyndham Guidelines for chiropractic care, which demonstrates that both the so-called straights and mixers are equally to blame for limiting the scope of chiropractic care and poorly educating society about the benefits of chiropractic care.

Autosuggestion ("psychologic/mental irritation") and nociception

One of the original theories put forth by BJ Palmer indicates that subluxations inhibit the transmission of mental impulses from the brain to systemic tissues, which suggests that the mental impulses were flowing but could not reach the tissues due to the compromising nature of subluxation. BJ Palmer also maintained that the neurological dysfunction associated with subluxation could only affect the motor system. He asserted that the sensory system could not be affected by subluxation because innate always receives the appropriate sensory information. Palmer maintained that a subluxation blocks the transmission of motor impulses at the level of the intervertebral foramen.

The information presented in this paper clearly indicates that BJ Palmer's theory about subluxation and the motor system was incorrect. It is, in fact, the afferent system which is affected by subluxation, resulting in enhanced nociceptive input and reduced mechanoreceptive input, which together enhance segmental motor output. Present day chiropractors need to accept these facts of physiology.

It appears that BJ Palmer's belief in the "constant flow of mental impulses," is also incorrect. We must realize that the precise nature of these "mental impulses" was never characterized by Palmer or his present day advocates. To this day, no one has ever described the neuroanatomical origin of these mental impulses or the neuroanatomical pathways through which they supposedly flow. This is not to suggest that thoughts in our brain do not descend into the spinal cord. However, it is apparent that Palmer's "mental impulse" theory lacks supportive physiological evidence.

At the present time there is sufficient evidence which demonstrates the fact that our mental state (autosuggestion) can influence nociceptive activity at the level of the dorsal horn. Powerful descending inhibitory pathways are known to emanate from the brain stem, particularly from the medullary serotonergic nucleus raphe magnus and various catecholamine nuclei located in the pons, and travel down the dorsolateral funiculus to reach the dorsal horn. The activity of these pathways results in the inhibition of nociception. Conversely, a reduction of descending inhibitory activity results in a disinhibition, or a net enhancement, or nociceptive input. It is known that mentally engaging attitudes, or mental fitness, can facilitate the activation of these antinociceptive pathways. Several authors have discussed these relationships,[41,42,43,44] which help to form a scientific basis for explaining how our mental state can help promote the development of the subluxation complex.

This descending inhibitory system is fairly well characterized at this time, and it is known for sure that these particular descending "impulses" do not just "flow constantly" by themselves. Indeed, we must exert a mental effort in order to activate the descending inhibitory impulses.

As you can see, B.J. Palmer was on to something; the scientific knowledge of his time was insufficient to help provide an accurate explanation. Mental impulses do flow down the cord but in a fashion that is even more significant than B.J. may have thought.

A recent clinically-oriented paper suggests psychologic/mental distress can enhance nociception.[45] A population of 1638 subjects without back pain were followed to determine the relationship between psychologic distress and low back pain. The results indicate that symptoms of psychologic distress can predict the onset of new episodes of back pain. The authors state that psychologic factors are involved in 16% of new episodes of low back pain in the general population.

Conclusion

The information presented in this paper explained how trauma, toxins and autosuggestion can enhance nociception which can, in turn, promote the development of the subluxation complex and a wide variety of symptoms and metabolic problems. Based on this information, methods to inhibit nociception should be an integral component of a treatment program designed to reduce subluxation. Research suggests that the chiropractic adjustment, motivational training, proper nutrition, [46,47] exercise [48] can all help to reduce nociceptive input. ◆

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