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## **How Does Subluxation Affect the Nervous System?**

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Our colleges, associations and research institutions may be barking up the wrong tree and leading us down the road to Never Never Land.

### **Introduction**

Research performed in the first half of this century demonstrated that extremity pain could be induced by injecting hypertonic saline into paraspinal muscles and interspinous ligaments.<sup>1,2</sup> For readers who are unfamiliar with this work, it is known that the injection of hypertonic saline into musculoskeletal structures causes a significant amount of pain and discomfort. Presumably, the reason for the pain response is that hypertonic saline acts to depolarize local tissue nociceptors.

In 1976, Drs. Vert Mooney and James Robertson set out to confirm the earlier research on referred pain and discussed their findings in a well-known paper, "The Facet Syndrome."<sup>3</sup> Their attention was directed toward the facet joints rather than spinal muscles and ligaments. The subjects in this study included five normal individuals and 15 patients with low back pain. To make a semi-long story short, Mooney and Robertson discovered that, indeed, injecting hypertonic saline into facet joints resulted in local and referred pain. They also discovered that, "slightly increasing the volume of injection would consistently increase the amount of pain radiation."

For those who are unfamiliar with Drs. Mooney and Robertson, it should be understood that both are medical doctors. Therefore, the treatment methods to which they are most familiar include surgery, medications and facet injections. In the clinical application section of "The Facet Syndrome," the authors described their research trial that involved 100 patients who had chronic low back pain and/or lower extremity pain for at least six months. Each of the 100 patients received facet injections, consisting of a steroid and a local anesthetic. Many patients experienced a significant reduction in symptoms which lasted for varying lengths of time.

My reason for describing this study is that I have a couple of questions for our colleges and national associations, all of whom are involved in research. Why is it that none of our researchers have yet to duplicate the Mooney and Robertson study? Why is it that so few ICA, ACA and independent doctors and students are unaware of this area of investigation that began in the 1930s?

### **What Is So Significant about These Studies?**

I have asked many researchers and so-called leaders of our profession about why these studies which focused on irritating posterior spinal structures are rarely, if ever, discussed. I am not exaggerating. As an example, a new book that is devoted to the topic of subluxation does not describe these studies at all.<sup>4</sup>

We must all realize that the posterior structures described in these studies are the same ones to which chiropractors focus their attention. Thus, it would make sense that we do similar studies that involve the noxious irritation of the same spinal structures.

Is it not true that chiropractors maintain that the subluxation complex irritates the nervous system? Do we not constantly suggest to the world that optimal spinal function is critically important for overall body health? Why then, do we not study the effects of noxious irritation of posterior spinal structures and assess the types of symptoms that may develop? Maybe we would discover that back pain and many other symptoms develop as a consequence of inducing nociceptive input from spinal structures.

In 1954, Dr. Bernard Feinstein and his colleagues repeated the work of Kellgren and were amazed by the severity of the pain and the bizarre symptoms that developed after noxious spinal irritation. As of yet, no one in chiropractic has attempted to duplicate Feinstein's work. This is not surprising because the great majority of DCs have never heard of the Feinstein paper. I recently asked one of our illustrious chiropractic leaders if he was aware of a landmark paper written in 1954 which demonstrated the neurologic impact of noxious irritation of the spine. He told me that he was only two years old at the time and consequently, there was no way that he would be aware of such a study. I found this excuse very amusing because, in 1954, I had yet to be born. In fact, my parents had yet to meet.

Feinstein et al., injected six percent hypertonic saline into the interspinous tissues of 82 subjects, 75 of whom were medical students. The results were rather dramatic. Before discussing the results, we must realize that the dorsal ramus is the nerve that innervates the interspinous tissues. Thus, we can conclude that the symptoms developed due to excitation of nociceptive afferents within the dorsal ramus. None of the

symptoms had anything to do with the ventral ramus. I state the obvious here because so many DCs are enamored by the ventral ramus and think it is the key to understanding subluxation and the chiropractic adjustment.

Feinstein et al., discovered that pain was initially felt at the sight of injection. Most subjects described the local pain as deep and aching. Doesn't this sound like the complaints you hear from your patients? The only difference is that your patients have not received hypertonic saline injections; rather, they suffer from a pathological changes of the spine, which we call the subluxation complex.

Feinstein et al., also discovered that interspinous tissue irritation resulted in the spasm of certain muscles. "For example, an injection at the level of the sixth cervical segment was fairly consistently accompanied by spasm of the supraspinatus, infraspinatus, or biceps."

Feinstein's subjects also experienced pain that extended into the extremities. Neck injections caused arm pain and low back injections caused leg pain. Consider the implications of this finding. Noxious irritation of dorsal ramus-innervated structures results in pain that extends into the extremities. Clearly, neurocompression at the IVF is not required to induce pain referral into the extremities. Clearly, the statement that "we should avoid adjusting patients with leg pain" needs to be carefully reassessed and restated.

To demonstrate that the ventral ramus is not necessarily involved in the generation of referred pain, Feinstein et al., performed a complete left brachial plexus block on one subject. The result was complete motor and sensory paralysis for over 30 minutes. Pain could not be induced by irritating the left arm itself. Hypertonic saline was then injected into the interspinous tissues on left side between the spinous processes of the sixth and seventh cervical vertebrae. The result was local pain and pain in the left forearm. Clearly, noxious irritation of posterior spinal structures can have rather dramatic effects. However, it should be understood that the "dramatic effects" extend well beyond pain syndromes associated with the musculoskeletal system.

Feinstein et al., explain interspinous injections often resulted in a variety of bizarre autonomic reactions. The autonomic concomitants included pallor, sweating, bradycardia, fall in blood pressure, subjective "faintness" and nausea. We are told that, "these features were not proportional to the severity of the pain or to the extent of radiation; on the contrary, they seemed to dominate the experience of subjects who complained little of pain but who were overwhelmed by this distressing complex of symptoms." Feinstein et

al., found that the autonomic concomitants occurred most frequently with injections in the thoracic region. However, it should be understood that Campbell and Parsons, who investigated head pain, found that autonomic concomitants were a common occurrence after injection of hypertonic saline into cervical spine structures.<sup>6</sup>

DCs frequently find that seemingly bizarre visceral symptoms often disappear after chiropractic care. More than likely such bizarre symptoms are nothing more than nociceptive-induced autonomic concomitants. A recent paper by Nansel and Szlazak list numerous symptoms which can be induced by nociceptive input from the spine.<sup>7</sup>

### **Clinical Chiropractic**

Feinstein et al., discovered that, "the volume [of hypertonic saline] injected accounts not only for the intensity of the ensuing pain but also for the extent of its referral."<sup>5</sup> This suggests that not all subluxations will ignite the same symptomatic picture because the degree of tissue disruption and chemical mediator release will be different in each patient. What does this mean exactly?

In real-life situations, nociceptors are not irritated by injections of hypertonic saline; rather nociceptors are irritated by mechanical and chemical irritants. The chemical irritants include substances such as bradykinin, serotonin, potassium ions, prostaglandin E-2, and histamine.<sup>8</sup> These and other chemical irritants are released after tissue injury,<sup>9</sup> and the chemical irritants are responsible for exciting nociceptors,<sup>8</sup> and for driving the inflammatory process.<sup>9</sup> Naturally, each patient is unique and the degree of chemical mediator release will vary and therefore, the nociceptive processes and related symptoms will vary.

Before going any further, I need to make it clear that if the chiropractic profession is to understand and utilize the work of Mooney, Kellgren, Feinstein et al., and Campbell, chiropractors must fully understand the topic of nociception, and simultaneously place neurocompression on the back burner. This is not my opinion; rather, it is a physiological fact that we need to embrace. Consider the following information about nociception.

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.<sup>10</sup> Generally speaking, mechanical and mechanothermal nociceptors are

associated with type A-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 the inner layers of the annulus fibrosus.<sup>11,12,13,14</sup> Wyke provides the most vivid anatomical description of the nociceptive receptor system.<sup>15</sup> 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 percent 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.<sup>16</sup> In one study, which examined the medial articular nerve of the cat, it was demonstrated that 21 percent of the fibers were of the A-delta variety and 70 percent were C fibers. In the posterior articular nerve, 14 percent were A-delta fibers and 60 percent were C fibers.<sup>17</sup>

Clearly, from a clinical and research perspective, we are being urged to focus on nociception. We can no longer avoid it.

Consider some of the nociceptive processes chiropractors encounter on a daily basis. It is well known that nociceptors themselves and nociceptive activities in the spinal cord can become sensitized. Researchers in the field of nociception use the terms peripheral sensitization and central sensitization [See Woolf for a concise explanation].<sup>18</sup>

Under normal circumstances, nociceptor thresholds are very high; they only respond to noxious stimuli. However, the presence of chemical irritants, which are released after tissue injury, can lower nociceptor thresholds. Peripheral sensitization refers to the lowering of a normally high nociceptor threshold. Now innocuous stimuli, such as turning the head, bending over, and gentle palpation can activate nociceptors. How many times during the course of a day do you palpate a patient and find that gentle palpation results pain? It happens all the time. In the field of nociception, term "allodynia" is used to describe pain that is produced by normally painless stimuli.<sup>19</sup>

Central sensitization is more difficult to describe than peripheral sensitization. First, it should be understood that central facilitation and wind-up are generally considered to be synonyms of central sensitization. It is now believe that neuronal plasticity is the process which creates central sensitization. In brief, through a complex series of biochemical and genetic processes, nociceptive afferents (particularly C fibers) induce a modification in the central processing of both nociceptive and mechanoreceptive input. The end result is an amplification or an increase in the excitability of dorsal horn neurons, such that the experience of pain can be magnified by noxious and innocuous stimuli.

It is very likely that the nociceptive processes described above are the main cause of back pain and the bizarre autonomic concomitants which are routinely seen in chiropractic practice. It is also very likely that the injection of hypertonic saline mimics the nociceptor irritation induced by the subluxation complex.

### **What Should We Do?**

We need to focus on nociception and realize that the pathological components of the subluxation complex are responsible for depolarizing nociceptors. We also need to repeat the studies of Kellgren and Feinstein. I am sure that students and doctors would be happy to serve as experimental subjects.

Interested chiropractors and chiropractic students need to petition our colleges and research organizations to initiate these studies. At the present time, we have task forces on Medicare, managed care, government relations and nearly every other topic. The only task force we do not have is a task force on physiology and pathophysiology of spinal function and dysfunction. Isn't it about time we started one?

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