

## **Clinical Excellence -- A Concept That May Be in Jeopardy of Being Replaced by Clinical Complacency, the Maintenance of the Status Quo!**

Let me give you a very good example of what I mean by the title of this article. If you were asked to describe the method by which dysfunction in the lumbosacral junction and/or sacroiliac joints could cause pain and facilitation (sympathetic hyperactivity and reflex muscle spasm) of spinal segments in the lower thoracic or upper lumbar regions, could you do it? If you can, then congratulations; if you cannot, then you are in need of upgrading. For whatever reason the diagnosis of thoracolumbar junction (TL) syndrome has become very popular with new graduates. Although I agree it can be possible, I wonder how many are diagnoses that demonstrate a lack of understanding of the normal anatomical pathways of our nervous systems?

What is the point, you might be thinking, and how is this relevant to my method of practice? No matter what technique you practice the normal anatomical pathways are there: it is the same for Gonstead, SOT, or Pettibon practitioners as it is for SBP, Diversified, NUCCA, Activator or any of the other 100 different types of chiropractic practitioners. The normals are just that, normals and it behooves one to be aware of the normal function of the musculoskeletal system as well as the nervous system.

Imagine yourself in a court of law trying to explain the bone-out-of-place and the big black shoe on the green garden hose, when talking about the patients T-L junction pains, when in reality it would be so nice to tell everyone present about the fact that noxious stimuli in the sacroiliac or lumbosacral areas ascends in the sympathetic trunk, utilizing the white rami, to gain access to the spinal nerve and DRG. The lowest white rami available just happens to be at the level of the TL junction. What does this mean? It means that increased and unopposed nociceptive input, as a result of spinal or sacroiliac joint subluxation/dysfunction (a loss of large diameter afferent input or dysafferentation) can cause sympathetic hyperactivity, reflex muscle spasm and pain at the TL junction area. The treatment is to be given to the cause, and in this case, the cause is not the TL junction.

Let us look at the reasons for muscle spasm and subsequent joint fixation after this situation has occurred. There are three sources of innervation to the lumbosacral/sacroiliac region: (1) the dorsal rami of spinal nerves, (2) sinuvertebral nerve; and (3) the somatosympathetic nerves. Each of these nerves are responsible for a pain presentation that is specific to the nerve in question. Above was described the somatosympathetic nerves. Although the lesion was in the pelvis, the pain and reflex muscle spasm was in the TL junction area. The dorsal rami, after leaving the IVF, wraps around the facet joint situated immediately caudal. The dorsal rami divides into three branches: the lateral branch, which supplies the lateral compartment and the iliocostalis muscle; the intermediate branch, which supplies the intermediate compartment and the longissimus muscle; and the medial branch, which supplies the multifidus muscle, the ligaments (interspinous, ligamentum flavum and the facet joint), and the intrinsic muscles (interspinalis, intertransversarii) of the lumbar and sacral segments. It is worth mentioning that the dorsal and ventral rami of the 5th lumbar and the sacral nerve roots

supply the sacroiliac joint capsule.

This little known fact is very important to chiropractic. An increase in nociception of the small diameter fibers of the dorsal ramus will result in the perception of pain, however, the overlap in innervation (ventral ramus) can cause a "sciatic" type of pain. Note that the pain perception (an illusion) is of ventral ramus distribution, and that the treatment is to be directed at the dorsal ramus. Chiropractors treat the dorsal ramus by adjusting the spine. The sinuvertebral nerve comes off prior to the division of the spinal nerve and re-enters the IVF to supply the PLL, periosteum of the posterior surface of the vertebral body, the outer layers of the annular fibers, and the anterior surface of the spinal dura. As well, the nerve can travel up or down two to three segments and can cross the midline to supply the contralateral side. This crossover will lead to the signs of increased nociception (sympathetic hyperactivity). If you treat the area of pain you may be treating the wrong side.

Just to complicate matters more, it is also worth noting that in some cases the sinuvertebral nerve crosses over in the spinal canal and exits from the contralateral IVF to supply the aforementioned structures. The sinuvertebral nerve supplies no muscles, so that weakness or wasting of muscles cannot be attributed to the sinuvertebral nerve.

Just how important is this to you and your everyday practice? Consider the above structures that are supplied by the sinuvertebral nerve, and then picture the subluxation and think about it. The sinuvertebral nerve can refer pain a number of segments up or down from the lesion site, and refer the pain to the side opposite, or in the case of the crossover sinuvertebral nerve, it could be referred pain bilaterally and three segments away from the cause. So if you treat the site of pain you are very wrong and your patients deserve better.

A number of years ago I heard Dr. Karl Lewit speak and I will always remember his words when he said: "Remember that the site of the pain is almost never the site of the subluxation." I agree with Dr. Lewit and I hope you do as well.

Two terms that seem to be used with haphazard spontaneity are sclerotogenous (referred pain), and dermatogenous (radicular pain). Sclerotogenous or referred pain occurs when tissues of mesodermal origin (muscles, ligaments, periosteum, joint capsule, and annular fibers) are exposed to noxious stimuli, such as excessive stretch or compensatory aberrant actions that cause torque moments. When subjected to these noxious stimuli, a deep, ill defined, dull aching type of discomfort is noted and, when in the lumbosacral region, may be referred into the lumbosacral junction, the sacroiliac joints, or the legs and/or buttocks. This pattern of referral is known as a sclerotome, which has the same embryonic origin as the mesodermal tissues being stimulated.

Dermatogenous or radicular pain (the term radicular refers to a radical or root) is superficial or cutaneous, clearly localized by the patient, sharp or lancinating and is restricted to the dermatome in question. It is of significance that an inflamed nerve root from whatever source is much more susceptible to compression than a normal root, and that the extent or excursion of the pain was a function of the resultant pressures generated. The concept of mechanical tension and the inflammatory response causing pain are well documented and very well accepted, however the concept of compression from herniated discs or bony stenosis not causing pain is difficult for a lot of people to accept. I would refer those people to study current literature on sensitization, hyperalgesia, and the inflammatory process as a cause of the pain.

The year 1997 will bring on more HMO and PPO types of health care concepts and agencies, and with

this comes clinical competency and accountability. MPI has made a commitment to the pursuit of clinical excellence in postgraduate chiropractic education, regardless of the type of chiropractic you choose to practice. An old friend within the profession once told me that the science of chiropractic is the same for all of us. Let's make 1997 the year to update and upgrade our own personal data basis and get sick people better everywhere because we know what we are doing.

*Keith Innes, DC*  
*310-1920 Ellesmere Rd.*  
*Toronto, Ontario*  
*Canada M1H2V*

JANUARY 1997