

Central Hypersensitivity

Why do many whiplash injured patients report so much pain in the absence of objective findings? This is a question that has been asked by doctors, lawyers and judges since the beginning of whiplash history. Some people will tell you that our scientific methods are lacking in sensitivity to identify the tissue damage. Others might say that the reported pain levels are motivated by the attempts to obtain disability benefits or financial settlement. But what if the people would not financially gain from the reports of pain? What if the only benefit of reporting pain was to find a solution to the problem and get better?

Michael Curatolo, MD, Ph.D. from the Department of Anesthesiology in Bern Switzerland addressed the issue of pain reporting at the 2005 IWTA. His presentation was titled **Central Hypersensitivity in Chronic Pain after Whiplash**. His presentation reviewed the current literature surrounding hypersensitivity as a central phenomenon. In other words, while the pain experienced from patients in the first several months post injury comes directly from tissue damage, the long term pain reported is the result of abnormal processing of pain signals from the spinal cord, brain stem and brain. This has been hypothesized as originating through several neurologic pathways.

Dr. Curatolo noted that the scientific literature consistently shows increased sensitivity after stimulation of healthy tissues in whiplash patients. He cited specific papers, including his own, that have produced objective evidence for the existence of spinal cord hypersensitivity. His conclusion was that *“Central hypersensitivity may explain exaggerated pain in the presence of minimal nociceptive input arising from minimally damaged tissues, detected or not by the available diagnostic methods”*. In other words, whiplash injured patients have lowered pain thresholds and are less tolerant to “normal” pain producing stimuli.

How does this really work? This is believed to be a multi-dimensional phenomenon. On one hand, the ascending spinal pathways carrying nociceptive (pain) signals to the brain become conditioned to carrying the pain to the brain. Neurologic pathways have the property of neuroplasticity. In other words, they can redefine their function based upon the training they receive. This neuroplasticity is demonstrated by stroke patients who train different parts of their brain to do functions that the damaged part of the brain did before the stroke. In this case, with a constant stimulation of pain, the neuroplastic changes result in the neurologic pathways being activated **without** requiring the stimulus from the original damaged tissue.

Once the pain is centralized at the spinal cord level, the pain is then modulated at the brain stem and Thalamus. This pain modulation also seems to undergo neuroplastic changes which results in amplification of the sensory (pain) signals which result in even greater reports of pain by patients. To complicate this further, the descending pathways are also modulated abnormally such that the inhibitory



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signals are disrupted. The result is a magnified pain which is self initiating and in a “loop”, absent the tissue damage.

OK. I got carried away and far too technical. Here’s the point. When the injured patients reach a chronic stage (over 3-6 months) and are still showing significant symptoms, there may be a real pain source beyond the original tissues damaged. Doctors and lawyers alike need to appreciate the possibility that the pain has centralized and that this may become a lifelong disability for the patient. It’s not fair or supported by the literature that a person is a “magnifier” or a “malingerer” just because their pain continues. In fact, I would agree that the original pain and suffering, complicated by the resulting lower pain threshold, will result in greater loss of function. In fact, the chronic pain patient may even develop psychological problems if they are not trained in coping techniques so that they can still enjoy what remains of the quality of their life.

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