

## REPORT

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# CENTRAL SENSITIZATION AND CENTRAL PAIN: HUGE DIFFERENCE

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Some 15 years ago, central sensitization was a term I first started seeing when I was editor of Practical Pain Management. It was defined as experiencing a pain level above what was expected from the pain of arthritis, fibromyalgia, neuropathy, or other peripheral (outside the brain) painful condition. When it was present, it was an indication to more aggressively treat the pain condition with opioids and/or other measures. Unfortunately, this simple, well-meaning term has been transformed by some unscrupulous practitioners to label pain patients with “central sensitization” to imply they don’t need opioids or other treatment.

Also, some time ago central sensitization became synonymous with the term “hyperalgesia” meaning the patient was overreacting or feeling too much pain for their condition. What’s more, opioids were supposedly the cause of hyperalgesia, so they need to be stopped. Let’s be very clear. Neither “central sensitization” nor “hyperalgesia” are bona fide medical conditions. A medical condition is one in which there is a common set of symptoms and physical findings, and the condition can be confirmed by a diagnostic test such as an MRI or blood test. Central sensitization and hyperalgesia are bogus medical conditions that can’t be objectively identified, quantified, or diagnosed. They are simply terms that sound scientific and authoritarian but in reality, they have become fraudulent terms to justify withholding opioids and other treatments. It’s time patients, families, and physicians reject these terms and the medical practitioners who use them.

The term “central sensitization” is not to be confused with the term “central” pain.<sup>5-8</sup> This is a serious condition that more likely than not requires opioids along with great care and concern on the part of the medical practitioner.

The term “central pain” dates back to the emergence of pain after a stroke. The stroke wiped out and destroyed brain tissue that contained opioid receptors and the normal biologic apparatus which shuts down and relieves pain. Opioid drugs sometimes in high or unusual formulations are required for post-stroke (central) pain. One especially severe post-stroke, (central pain) is known as the Dejerine-Roussy Syndrome” as it involves the thalamus. Although strokes initiated the term “central pain,” it soon became appropriate and relevant to include brain tumors, hydrocephalus, and scarring from meningitis cases since these conditions wiped out some brain tissue and caused pain.

In recent times central pain has come to include those pain patients who have developed neuroinflammation and tissue destruction in the brain concomitantly with a peripheral pain problem that may involve joints, muscles, nerves, or spine.<sup>5,6</sup> It is interesting to note that central pain in the past was often called “secondary” pain as it tends to occur after someone has developed a peripheral pain condition. The first investigator to elucidate peripheral pain conditions with brain tissue destruction was Apkarian in 2004.<sup>1</sup> He and his colleagues found decreased prefrontal gray matter deficiencies in the brain scans of persons with chronic back pain. Since that time a plethora of brain scan and glial cell studies have found that persons with a peripheral pain condition may be associated with brain inflammation involving glial cells and tissue destruction akin to that which occurs after a stroke.<sup>5-8</sup>

Bona fide central pain is clinically typical and obvious. It is characterized by constant (24/7) pain and high pulse rates, hypertension, episodes of excess sweating, and cold hands and feet

(Raynaud's type phenomenon). Opioids including long-acting opioids may be required to control bonafide central pain. Also, central pain has what is called descending pain which requires dopamine stimulating drugs besides opioids to adequately control it.<sup>3</sup> The cause of central pain that accompanies or follows the development of a peripheral pain condition is now believed to be related to an autoimmune process and/or viral reactivation, especially Epstein-Barr.<sup>2-4</sup>

### Summary

Central sensitization and hyperalgesia are not bonafide medical conditions. To use these bogus labels to justify the withholding of medications is unscientific, fraudulent, and inhumane. These terms and the practitioners who use them should be summarily rejected. Central pain is a serious condition characterized by severe constant pain which often requires opioids for control. It is now believed to be due to an autoimmune-inflammatory process and possibly viral reactivation.

### References:

1. Apkarian AV, Sosa Y, Sont S, et al. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *J Neurosci* 2004;24:10410-10415.
2. Jakhmola J, Sho HM. Glial cell response to Epstein-Barr virus infection: a plausible contribution to virus-associated inflammatory reaction in the brain. *Virology* 2021;559:182-195.
3. Kwan M, Alkin M, Duenes H, et al. The role of descending inhibitory pathways on chronic pain modulation and clinical implications. *Pain Pract* 2014;14:656-667.
4. Li YL, Wang JY, Liu K, et al. Epstein-Barr virus to be a trigger of autoimmune glial fibrillary acidic protein astrocytopathy. *CNS Neurosci Ther* 2023;29:4139-4146.
5. Milligan ED, Watkin LA. Pathological and protective roles of glia in chronic pain. *Nat Rev Neurosci* 2009;10:23-36.
6. Rodriguez-Roecke R, Niemeier A, Ihler K, et al. Brain gray matter decrease in chronic pain is the consequence and not the cause of pain. *J Neurosci* 2009;29:13746-13750.
7. Tsuda M, Boggs S, Salter MW, et al. Microglia and intractable chronic pain. *Glia* 2012;61:55-61.
8. Watkins LR, Maier SF. Beyond neurons: evidence that immune and glial cells contribute to pathologic pain states. *Physiol Rev* 2002;82:98-1011.