

Beware of Central Sensitization!

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We all know that acute pain is an essential physiological defense/warning system of potential danger from the environment or within the body. However, it may subsequently develop into ongoing chronic pain with pain hypersensitivity, including allodynia and hyperalgesia, even after the initial acute pain stimulus has been removed [1]. What causes this? Clifford Woolf [2], who is often considered the “grandfather” of the construct of central sensitization (CS), initially introduced this phenomenon in 1983. He suggested that neuroplasticity in the central nervous system can cause a great enhancement of pain. CS mechanisms have been reported in a variety of studies with both animals and humans [1,3,4]. As further recently highlighted by Woolf [5] “...a prominent feature and sometimes prime driver of the pain is an altered modulation of pain processing within the central nervous system, an amplification consequent and increased excitability and/or reduced inhibition in specific neural networks, which constitute the phenomenon of central sensitization. Recognition of the importance of central sensitization for the maintenance and manifestation of clinical pain states has led to an appreciation that such pain typically is an expression of an altered/disease state of nociception circuits in the central nervous system, one triggered by but not necessarily sustained by peripheral injury, rather than only a symptom of some ongoing peripheral pathology, and treatment needs to be targeted accordingly” (p.1).

This CS construct has been proposed to be potentially the “root cause” of a variety of pain-related illnesses that have no apparent underlying tissue pathology, such as fibromyalgia, temporomandibular joint disorders, and irritable bowel syndrome, just to name a few [3]. Many of these disorders have a strong comorbidity with one another, and have comparable symptoms (e.g. sleep problems, fatigue, cognitive processing problems, etc.). Mohammad Yunus first introduced the term central sensitivity syndromes (CSSs) to describe these inter-related disorders, which are presumed to have CS as a common etiology [6,7].

Research on CS with human subjects often uses quantitative sensory testing (QST) protocols to assess perceptual responses to systematically-applied sensory stimuli, in order to evaluate somatosensory function or dysfunction [8]. Because this methodology is not practical for most healthcare providers, classification systems for identifying CS have been proposed [9,10]. According to these classification systems, diffuse pain patterns, involving allodynia and/or hyperalgesia, which are disproportionate to the nature and extent of an injury or pathology, and are associated with maladaptive psychosocial factors (including negative emotions, poor self-efficacy, maladaptive belief systems, and excessive pain behaviors), are likely related to CS. The Central Sensitization Inventory (CSI) has also been proposed as a valuable component for classifying and assessing other CS-related symptoms [9].

Mayer and colleagues [11] developed the CSI, as a means of providing a clinically-useful measure to screen for the possible presence of CS or CSS in patients. It includes 25-items, with a total score range from “0” to “100,” and with a 40-point cut-off score. It has become very popular around the world, and has already been translated into many languages (e.g. Brazilian Portuguese, Dutch, French, Gujarati, Japanese, Serbian, etc.; [12]). Multiple language versions of the CSI can be found at: www.pridedallas.com/questionnaires. A recent systematic review of 14 CSI studies, all of which were determined to have good-to-excellent quality of evidence, concluded that the CSI generates

psychometrically-sound, reliable, and valid data to quantify the severity of CS-related symptoms [13]. Moreover, Cuesta-Vargas., *et al.* [12] have confirmed the reliability of the CSI in a large pooled multi-country sample of 1,987 subjects. Thus, we now have a readily-available self-report measure of CS-related symptoms (the CSI) which can be the first-step in the assessment of the potential presence of CS in patients with an “unusual array” of symptoms that do not appear to be associated and without a specific disease, with a known underlying pathophysiology [14].

In conclusion, CS-related pain can occur with no objective tissue damage, peripheral pathology, or nociceptive stimuli [1]. When no apparent cause for pain can be identified, physicians and other healthcare providers may have a tendency to interpret patients with CS-related pain, and other symptoms, as neurotics, malingerers, or somatizers [15,16]. It is our hope that our new understanding of CS-related pain mechanisms will shed new light and hope for this difficult patient population.

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