



TMD News

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Temporomandibular Disorders/Orofacial Pain
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Central Sensitization Syndromes

Central sensitization (CS) ...

CS can be defined as *a state of the nervous system in which neurons activated by noxious sensory stimuli are sensitized by such stimuli and become hyperresponsive to all subsequent stimuli delivered to the neurons' receptive fields*. This generally occurs over time when pain stimuli are persistent and repetitive, becoming a frequent characteristic that distinguishes the transition from acute to chronic pain. The original source of the noxious stimuli may be healed in chronic pain syndromes and yet innocuous stimuli may still elicit the hyperresponse that is perceived in the brain as pain.

Central sensitization syndromes (CSS) ...

CSS comprise *a similar and overlapping group of syndromes without demonstrable structural pathology* and are bound by this common pathophysiologic mechanism of CS. A growing body of scientific literature identifies temporomandibular disorders as a member of that group, along with other familiar chronic syndromes such as **fibromyalgia, chronic fatigue syndrome, burning mouth syndrome, atypical odontalgia and myofascial pain syndrome**.

Identification of Central Sensitization ...

Identifying CS underlying the cluster of symptoms presented by our chronic TMD patients gives us insight into the interrelationships among these symptoms. This awareness permits an encompassing approach to diagnosis and treatment planning that is evidence-based and effective and fits within the biopsychosocial paradigm of practice. **Roberts (2011)** has reviewed the CSS literature and presented several characteristic sequelae of the pathophysiologic changes associated with CS. He relates these changes to the various clinical symptoms routinely seen in chronic pain patients, thus permitting **a means of identifying CS during patient assessment**.

Dr. Roberts summarizes the pathophysiology ...

“Vasal dysregulation reduces endorphin release and alters serotonin production and utilization. This produces altered accommodation of minimally painful events, and contributes to depression. **A sensitized trigeminal complex leads to lowered parasympathetic drive and increased sympathetic drive**. This contributes to dysfunctional sleep and anxiety via increased norepinephrine levels. Decreased medullary descending inhibition of nociception increases the effective peripheral input and is reflected in lowered pain thresholds, hyperalgesia, allodynia, and greater impact of peripheral sensitization. Hypoactivity of the HPA axis and autonomic nervous system alterations produce increased sympathetic tone, low vagal tone, and contribute to immune abnormalities. This contributes to the fatigue and malaise often associated with CSS.

“Utilizing the resultant depression, anxiety, hyperalgesia, allodynia, stress-related pain exacerbation, fatigue and poor sleep, we can establish a list of indicators that are highly suggestive of CS.”