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Temporomandibular Joint Pain Treatment and Practices

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Disorders of the Temporomandibular Joint (TMJ) have become more prevalent in patients seeking care for joint, muscle, and associated headache pain. Currently, clinicians have limited treatment options to treat most cases of TMJ pain, given the diffuse nature of the reported symptoms. In some cases, radiographic diagnoses can identify structural displacement or damage to the articular disk in relation to the adjacent condyle. However, observing and recording pain behavior in the patient following multiple jaw movements and digital palpitations to the musculature of the maxillofacial region is primarily used to diagnose the majority of TMJ pain cases. Recent studies provide illumination to the physiological etiologies that may contribute to TMJ pain.

TMJ pain is believed by many top precipitate from joint inflammation, either by overuse, of age, which is almost twice the prevalence in men [1]. Several theories have received significant support in recent studies. Estradiol has been shown to significantly sensitize pain responses in TMJ disorder model rodents [2], as they have in other pain behavior models [3]. Prevalent theory intimates that hormonal activation of nuclear receptors stimulates increased transcription, and subsequent increased protein to support an increase in afferent sensitivity to a pain inducing stimulus. Opponents of this theory indict plasma membrane bound estrogen receptors capable of activating intraneuronal signaling mechanisms within minutes of activation to post translational reduce depolarizing ion channel thresholds, also resulting in increased afferent sensitivity. Ofcourse, for either of these theories to be proven, afferent innervation must first be demonstrated in the tissue surrounding the TMJ. There are several schools of thought that speak to the roles of afferent and efferent neuronal innervations of the TMJ. Milam's group reports that increased capsular pressure precipitates hypoxic reperfusion injury, resulting in the accumulation of inflammatory mediators capable of sensitizing primary afferents that innervate the region [4]. However, Bereiter's work suggests that too few afferents actually innervate the TMJ tissue to account for the intensity of TMJ pain, and that increased pain sensitivity is significantly modified by efferent influences [5]. This finding would be strongly supported by those clinicians who observe a higher correlation between stress and TMJ pain in patients. Still, others suggest that overuse of facial and masticatory muscles can stimulate glutamate accumulation, there by resulting in lower mechanical thresholds for small fiber activation and transient pain in experimental models [6]. Although multiple theories may all play important roles in the generation of TMJ pain, most clinicians agree on treatments for pain amelioration. Treatments for TMJ pain can become surgical in nature when structural deformities are detected and require immediate repair to restore joint function. However, most clinicians begin TMJ pain treatment plans with pharmacotherapy including Non Steroidal Anti Inflammatory Drugs (NSAIDs), muscle relaxants, and when necessary, narcotic analgesics. Often times, clinicians also prescribe splint therapy to their patients, utilizing a mold fitted acrylic mouthpiece worn between the upper and lower teeth to improve jaw alignment and reduce unwanted muscle movement, including clenching of the jaw. A recent study has demonstrated this therapy to significantly improve jaw function and reduce TMJ pain in treated patients [7]. In some cases, psychological modifications including relaxation techniques to reduce stress are prescribed as well, bolstering support for Bereiter's etiological theory mentioned above. Taken together, studies indicate that there could be multiple causes to TMJ pain. However, time tested treatment plans agree that non invasive approaches can significantly improve TMJ pain for the majority of afflicted patients.

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