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The two main theories on dental bruxism.

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Source

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Abstract

Bruxism is characterized by non-functional contact of mandibular and maxillary teeth resulting in clenching or grating of teeth. Theories on factors causing bruxism are a matter of controversy in current literature. The dental profession has predominantly viewed peripheral local morphological disorders, such as malocclusion, as the cause of clenching and gnashing. This etiological model is based on the theory that occlusal maladjustment results in reduced masticatory muscle tone. In the absence of occlusal equilibration, motor neuron activity of masticatory muscles is triggered by periodontal receptors. The second theory assumes that central disturbances in the area of the basal ganglia are the main cause of bruxism. An imbalance in the circuit processing of the basal ganglia is supposed to be responsible for muscle hyperactivity during nocturnal dyskinesia such as bruxism. Some authors assume that bruxism constitutes sleep-related parafunctional activity (parasomnia). A recent model, which may explain the potential imbalance of the basal ganglia, is neuroplasticity. Neural plasticity is based on the ability of synapses to change the way they work. Activation of neural plasticity can change the relationship between inhibitory and excitatory neurons. It seems obvious that bruxism is not a symptom specific to just one disease. Many forms (and causes) of bruxism may exist simultaneously, as, for example, peripheral or central forms.