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Research Article

Bruxism in Relation to White Sugar Consumption

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Abstract

Bruxism is considered a functional disorder characterized by grinding and/or clenching the teeth, which may be associated with stress, anxiety and some neurological conditions. This article aims to discuss bruxism and how sugar interferes with some neurochemical substances called catecholamines (dopamine being one of them).

Keywords: *Bruxism; Functional Disorder; Sugar*

Introduction

Bruxism, a word of Greek origin, has the free translation "to grind your teeth", these "habits" of grinding, clenching your teeth is nothing more than a parafunction and which is defined by Isrraela Macena (2019), dental surgeon, "They are habits with no specific natural function of the human being that place the mouth and the bones of the face in abnormal positions or overload them".

TMD (temporomandibular disorder) is totally associated with parafunctional activities, which would be part of non-functional activities, and having Bruxism as one of the most researched and thus developing several studies on more effective therapies for its control and treatment.

Nowadays it is possible to observe greater attention regarding the link between bruxism and the relationship with neurochemical substances, despite the fact that there are still few studies. It is in agreement that Dopamine ($C_8H_{11}NO_2$) corresponds to more than half of the catecholamine content. He thinks that it is properly related to the blocking of these repetitive and coordinated movements.

It is known that glucose, present in most foods rich in carbohydrates, is the primary source of energy for the brain. Which does not suggest that an extra consumption is beneficial. For proper functioning, brains also need a proper diet. As glucose is the main energy generator for the brain, too much sugar can put it into overload mode.

Studies point to the existence of a strong link between altered, parafunctional behaviors and poor emotional regulation with heavy sugar consumption.

Literary Review

According to Okeson [13], some studies reveal that a large portion of TMD symptoms is identified between the ages of 20 and 40 years and its etiology is complex and multifactorial. It is estimated that 85% to 90% of the population grind their teeth to some degree during their lives and only about 5% of these patients have bruxism as a clinical condition.

In 1999, Vanderas., *et al.* [22] investigated the correlation between the levels of catecholamines found in the urine and the development of bruxism in 314 children, of both sexes, aged between 6 and 8 years. The recognition was derived from the performance of clinical examination and anamnesis, using diagnostic criteria for bruxism the presence of wear on a permanent molar. Urine was collected for a period of 24 hours from each participant and later analyzed by chromatography, in order to assess the amount of catecholamines. No dietary restrictions are applied. In this way, bruxism was diagnosed in 129 of the 273 participants who completed the urine collection. The analysis showed that adrenaline, noradrenaline and dopamine have a significant influence on the probability of developing bruxism.

Regarding neurotransmitters and their relationship with bruxism, the work by Seraidarian [15] showed a numerically significant difference between the urinary levels of adrenaline, noradrenaline and dopamine measured in bruxism and non-bruxism subjects.

Such as Nascimento [9], which demonstrates through a study that there is a relationship between emotional stress and bruxism. Seraidarian [16], on the other hand, defined the changes in an individual's behavior through the influence of dopaminergic neurons, Machado [9], a set of neurons in the central nervous system responsible for synthesizing the dopamine neurotransmitter, which are determined by serotonergic neurons, which are responsible for by the synthesis of serotonin (a molecule responsible, among others, for inhibiting sleep, body temperature, appetite), which, in turn, interferes with the extrapyramidal system (these structures are involved in processes such as the modulation of motor control) where the dysfunction of its structure is associated with movement disorders. L-Dopa was the first neurochemical related to bruxism and is used in the treatment of Parkinson's disease, aiming to increase dopamine levels.

According to Aloé [1], there is no functional and anatomical condition in the central nervous system that can be detected as a determining factor that generates specific involuntary oromandobular movements. However, there is evidence of dopaminergic, noradrenergic and serotonergic neurotransmission in the genesis and modulation of bruxism [8].

For Guyton [7], dopamine can cause an increase in heart rate, nausea, increase in the tone of the suprahyoid muscles and the beginning of rhythmic masseter masticatory muscle activity and, consequently, teeth grinding.

Conclusion

Studies on catecholamines have shown an important correlation between neurochemical substances, sugar consumption and bruxism, confirming the hypothesis that excess sugar is involved in the etiology of this parafunction.

We can conclude that the generating factor of bruxism is still enigmatic and there are many factors that may be related to its triggering. Some studies show that certain medications can increase bruxism events, although it is still scarce, some studies show that there may be a genetic predisposition.

Some studies demonstrate that parafunction with the PSG exam and central neurotransmitters are indispensable. And the procedures involved in the cure of clinical problems linked to bruxism must be evaluated by a multidisciplinary team trained for this purpose.

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