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Bruxism an Issue Between the Myths and Facts

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Bruxismo, un problema entre los mitos y los hechos

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ABSTRACT: Is our goal in this paper to discuss the current concepts about bruxism, a topic that has been a matter of discussion on the dental field for many years. Recent International efforts have been made to challenge bruxism old definitions; this has derivate to a consensus and an actual new concept that defines bruxism as a behavior instead of a disorder. As a behavior, it is explained in this review how it can have negative health consequences, can be innocuous and how new research support that bruxism can even be a protective factor. Different etiological factors are reviewed in this paper as well the influence of external and internal mechanism related to medications, emotional stress, systemic factors, and potential pharmacological pathways. Moreover, it is briefly summarized the role of oral appliances on sleep bruxism. Finally, clinical considerations and recommendation for the dental professional regarding sings that should be notice during the exam are part of this overview.

KEYWORDS: Bruxism; Teeth clenching; Temporomandibular disorders; Oral parafunction.

RESUMEN: Es nuestro objetivo en este artículo discutir los conceptos actuales sobre el bruxismo, un tema que ha sido motivo de discusión en el campo odontológico durante muchos años. Se han hecho esfuerzos internacionales recientes para desafiar las viejas definiciones de bruxismo; esto ha derivado en un consenso y en la actualidad un nuevo concepto que define al bruxismo como un comportamiento en lugar de un trastorno. Como conducta, se explica en esta revisión cómo puede tener consecuencias negativas para la salud, puede ser inocuo y cómo nuevas investigaciones avalan que el bruxismo puede incluso ser un factor protector. En este artículo se exponen

diferentes factores etiológicos, así como la influencia de mecanismos externos e internos relacionados como: medicamentos, estrés emocional, factores sistémicos y posibles vías farmacológicas. Además, se resume brevemente el papel de los aparatos orales en el bruxismo del sueño. Finalmente, se presentan consideraciones clínicas y la recomendación para el profesional en odontología con respecto a los signos que deben ser observados durante el examen clínico son parte de esta revisión.

PALABRAS CLAVE: Bruxismo; Apretamiento dental; Desórdenes temporomandibulares; Parafunción oral.

Teeth clenching and grinding has been for years a topic of discussion inside the fields of dental and sleep medicine. This is mainly because of the fact that the dental community previously supported ideas based in what is nowadays called “the third pathway” and the myth that bruxism was the cause for temporomandibular disorders, ideas completely revoked by current scientific knowledge (1-4). Greene *et al.* brought up this concept, the third pathway, to explain how in different pain conditions the path for treatment follow two common methods: the conservative medical approach or irreversible methods, involving most of the time surgery; with both approaches having different applications and success on diverse conditions (3).

This so called “third pathway” is an idea born in the dental profession that believed that occlusal relationships, slides, interferences, and an inadequate temporomandibular joint position were the cause of bruxism and temporomandibular disorders. Time has passed, knowledge has grown and science has proven several times that

these suppositions should be abandoned (5-8). Nowadays it is evident that the dental profession has grown more closer than ever to the medical field, this is specially demonstrated in the specialties of Orofacial Pain and Oral Medicine. The foundation for these specialties rely on adequate care on an integrative diagnosis and human approach; which in the past years has led to new lines of research, specially between the relationship of conditions like obstructive sleep apnea, temporomandibular disorders and bruxism helping the dental community get a better understanding about these complex conditions (9).

Most recently the “International consensus on the assessment of bruxism” re-defined this condition (10), putting to rest the anterior definition in which bruxism was classified as a movement disorder and subclassified as a sleep movement disorder. Furthermore, this new definition separates bruxism into two different dimensions: sleep and awake bruxism. The current view of bruxism is challenged by the old definitions as can be observed on Table 1.

Table 1: Previous and current definitions on sleep bruxism.

Classic definition: movement disorder (11)	New definition (10): Behavior not a diagnosis
A diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth. In the absence of subjective awareness, past bruxism can be inferred from presence of clear wear facets that are not interpreted to be the result of masticatory function. Contemporary bruxism can be observed through sleep laboratory recordings. American Academy of Orofacial Pain 2008 Sleep-related bruxism is an oral activity characterized by grinding or clenching of the teeth during sleep, usually associated with sleep arousals. International Clasification of sleep disorders 2005.	<p>“Sleep bruxism is a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals”.</p> <p>“Awake bruxism is a masticatory muscle activity during wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals”. International consensus on assessment sleep bruxism 2018</p>

It is important to place emphasis that the current definition of bruxism classifies it as a behavior instead of a disorder. As a behavior, it means that bruxism can happen consciously or unconsciously, such as biting nails or lips, or daytime limb movements or tics. In addition, this explains there are different putative central mechanisms to describe this uncontrolled masticatory muscle activity. This narrative review provides the clinician with current concepts about etiology, consequences, and management of bruxism.

ETIOLOGY: IS THERE A SINGLE DIRECT CAUSE?

To date, not a single view exists about the etiology for bruxism and how the increasing muscle activity can become excessive. It is, however, well established that evidence based on literature has shown no relationship of peripheral factors such as “stable occlusion” or skeletal factors. This suggests that these involuntary movements are centrally regulated, probably in the brainstem and basal ganglia; structures in charge of controlling or inhibiting muscle movement for adequate muscle tone. Moreover, due to the great variability in subjects, it is impossible to establish a phenotype for severe bruxism. On the other hand, it has been

identified that excessive RMMA is more common in ages before 50, decreasing its prevalence after; suggesting the involvement of multiple environmental and personal factors playing a role on triggering this activity (12).

Current knowledge classifies bruxism as a common behavior, a situation that can take in to account three different directions: 1) Not a risk or protective factor, but a harmless behavior; 2) a risk factor when bruxism is associated with 1 or more negative health consequences; 3) a protective factor when bruxism is associated with 1 or more positive or protective results (1). The dental profession has well recognized the different clinical signs of destructive bruxism, so how can we consider this protective? Current research is pointing towards the fact that most sleep arousals (more than 80%) are ended by episodes of rhythmic masticatory muscle activity (RMMA), suggesting a role of this activity on increasing the airway patency and promoting salivation to prevent occurrence of apnea, an oxygen desaturation (11).

To better understand rhythmic masticatory muscle activity (RMMA), studies use the gold standard method for assessing sleep, which is all

night polysomnography examination. It has been observed that this RMMA is present on average 1-4 episode per hour in all people, thus is considered a physiologic muscle activity. Sleep bruxism is considered to be the intensification of frequency and strength of a normal orofacial activity during sleep and it is currently considered severe in cases when presenting episodes of more than 4 per hour (13).

Literature has reported the relationship of elevated catecholamines with the severity of bruxism, suggesting that an increased sympathetic tone that can be a triggering factor for RMMA. In addition, dopamine is supposed to have an inhibitory effect, whereas adrenaline and noradrenaline are well known as sympathetic activators. Moreover, some sleep modulators as serotonin, amino gamma butyric acid, cholecystokinin and orexin are also playing a role on modifying involuntary muscle activity (2,11,14).

It is hypothesized that anxiety and emotional stress increase the general arousal system, thus facilitating the presence of involuntary muscle activity on susceptible subjects. Some observations have shown that stimulants like caffeine, alcohol, illicit drugs, selective serotonin reuptake inhibitors and nicotine have a direct effect on bruxism, suggesting also the implication of cholinergic and adrenergic mechanisms (12). Interestingly in some patients, activity of the masticatory muscles during sleep increased after self-reported periods or days with emotional stressors. In addition, on self-reported anxiety test, patients with more RMMA scored higher than the normal controls (15,16).

More recent studies on bruxism have pointed out an association between sleep arousal and cardiac/motor activity while sleeping. Evidence supports that RMMA onset usually ends on sleep arousal which is characterized by a rise in autonomic sympathetic cardiac activity followed by

withdrawal precipitated by previously mentioned episodes of RMMA (17). *Lavigne et al.* pointed out that other factors should be considered for excessive masticatory muscle activity such as hypoxia, medications, sleep architecture, or sleep apnea (central or obstructive) because new research has pointed to bruxism as a putative protective factor. This is due to the fact that muscle activation produces an increased salivation, opening up the airway and stopping the sleep arousal on what is called altering cyclic patterns or in other words an transitory activation phase (1,12).

It is important to understand that both conditions, sleep and awake bruxism are complex trait phenomenon that are currently explained by multifactorial models that recognize specific factors that can influence masticatory muscle activity on different levels. Sleep bruxism is clearly centrally mediated and characterized by short episodes of clenching and teeth grinding reported for approximately 8 minutes per 6-9 hours of sleep (8,11). On the other hand, awake bruxism seems to be more related to psychosocial factors, an reflection of a non-conscious oral motor behavior characterized by trusting or bracing the mandible with associated sustained concentric masticatory muscle contraction (10).

PHARMACOLOGICAL TREATMENTS

Regarding medications, there are few studies implementing their usage specifically on bruxism. Considering the potential role of noradrenaline on bruxism, studies have tested the effect of propranolol and clonidine. Propranolol and other beta blockers, however, have not changed bruxism patterns. On the other hand, clonidine, an central acting alpha agonist, has shown a reduction in the autonomic cardiac sympathetic activation, which significantly reduced sleep bruxism, however was associated with severe hypotension in the morning (18,19).

Recently botulinum toxin has been used in bruxism, in cause due to its extensive usage and success on treating oromandibular dyskinesias, driving clinicians to off label usage of it to treat excessive masticatory muscle activity. To date studies do not support its usage for bruxism or RMMA, having shown after its application on the temporalis and masseter muscles a minimal reduction in muscle strength, meaning less intensity on movements but no effect on jaw muscle activity (20-22).

ORAL APPLIANCES

The most common method to prevent tooth damage related to sleep bruxism is the use of an intraoral appliance. Even though appliances are a conservative non-invasive treatment method; several considerations must be taken into account by the clinician before inserting and delivering one of these devices. It is strongly recommended to use a full mouth flat acrylic or thermoplastic device, because partial coverage can result in individual tooth movement, therefore, bite changes (7).

There are no clinical differences between a lower or upper appliance, or between hard acrylic or intermediate material. Patients tend to adjust easier to upper appliances but on patients with an amplified gag reflex a lower appliance is a better option. Hard acrylic appliances tend to last longer but are at times are difficult for some patients to adjust to due to it being so hard. A hard thermoplastic material is ideal due to its strength and ability to obtain a stable adequate bite position, and at the

same time being more comfortable for the patient to wear.

As previously mentioned, oral appliances are a straightforward method to prevent tooth damage and decrease the sounds related to teeth grinding, however, there is no evidence that these devices can reduce, alter, or stop rhythmic masticatory muscle activity. The majority of experimental or randomized trials using oral appliances demonstrate a short reduction on the frequency of RMMA but it tends to be transient. On the other hand, oral appliances used for the treatment of sleep apnea know as mandibular advanced devices have demonstrated a decrease of around 50% on RMMA when used in a protruded position (17).

The dental professional must always first and foremost have in mind the ancient concept “do no harm”. Based on this concept when treating a patient for bruxism an adequate diagnosis based on a human approach is necessary. A clinician must carry out a thorough interview and examination, focusing on the biopsychosocial situation of each patient.

When analyzing a patient with bruxism the clinical examination should include a complete dental and periodontal exam. It should also be noted the presence of abnormal tooth wear, masticatory muscle hypertrophy, and muscle tenderness (Table 2). It is also important to focus on signs and symptoms related to potential sleep and headache disorders, such as a decreased airway, a high body mass index, a high Mallampati index, a history of loud snoring or the report of severe daytime fatigue.

Table 2. Common Clinical consequences associated with sleep and awake bruxism (adapted from Balasubramaniam *et al*. 2014).

Dental and Periodontal	Temporomandibular
Severe occlusal and incisal wear	Masticatory muscle hypertrophy due to excessive forces.
Frequent failure of restorations	Masticatory muscle tenderness. Aggravating factor before temporomandibular pain.
Dental fractures and Tooth sensitivity	Limited range of mandibular motion.
Gingival recession (controversial)	Contributing factor for some headache disorders.

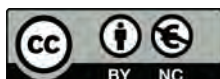
Clinical signs such as tooth wear or temporomandibular pain, are not always related to bruxism. In other words, dental wear facets could be indicative of past episodes of severe bruxism, acid reflux, other oral habits or even it could be related to some specific occlusal patterns. Emphasis should be made on the fact that the term temporomandibular disorders involve an umbrella of different diagnosis that requires a more extensive interview and clinical examination using preferably the Diagnostic Criteria for temporomandibular disorders. Therefore, when pain is presenting in the orofacial region an adequate diagnosis should be made and the clinician should try to avoid the terrible mistake of telling a patient that his or her pain is solely caused by “bruxism”. The state of art, in current scientific literature, does not support the use of a single treatment for bruxism instead the use of a multidisciplinary approach is recommended.

It is important to recall that the current definition treats bruxism as a behavior instead of a disorder, in otherwise healthy individuals. This major change in the definition helps to explain the fact that for some patients, this condition happens without consequences and for others it has severe implications on different areas of one’s health. Our responsibility as dental professional is to help to identify when this behavior is contributing to a negative health outcome or when it is raising a red flag for other life-threatening conditions such as obstructive sleep apnea.

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