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ETIOLOGICAL CONSIDERATIONS IN BRUXISM

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Abstract

The etiology of bruxism is controversial, many factors being implicated, like occlusion, psycho-behavioral factors, and genetic factors. The aim of the present review was to systematically assess the literature and identify main theories regarding the etiology of bruxism. Data extraction was carried out according to the standard Cochrane systematic review methodology. The following databases were searched: PubMed, Google Scholar, Medline and the Google library. The primary outcome was bruxism etiology. Screening of eligible studies and data extraction were conducted independently and in duplicate. The references were analyzed by two reviewers using the same search strategy and the same inclusion criteria were applied to the selected studies. Query terms used were „bruxism", „etiology" and „mechanism". Among the 95 related articles that were critically assessed, 31 were included in the critical appraisal. There is convincing evidence that the etiology of bruxism is various, involving local, systemic and psycho-behavioral factors.

Key words: bruxism etiology, psycho-behavioral factors

1.INTRODUCTION

Bruxism is a term generally used to define daytime and night time parafunctional activities of the masticatory system, which includes strained jaw and teeth grinding friction associated with tooth wear, myalgia of the masticatory muscles, temporomandibular joint disorders and morning fatigue.

Hippocrates, quoted by Rozencweig (1), pointed out that "dental wear is soul's clutter". This aphorism shows the dimension of this condition, which is outside the oro-dental sphere.

In 1907, Karolyi then Marie and Pietkiewicz, used the term "bruxomania", considering that "dental wear brings together at the same time the damages of the central nervous system"(2).

The term bruxism was first used in the literature in 1931 by Frohman for "non-functional grinding and rubbing the teeth" (3).

Over time there had been an ongoing concern for establishing a complete and comprehensive definition, related to the clinical manifestations of bruxism to explain at the same time the etiopathogenic mechanisms involved in the production and maintenance of bruxism.

Lavigne et al. (4) performed several studies on bruxism and concluded that the definition has evolved from the first considerations which were mainly referring to dental contacts and muscle contractions to considerations that relate to behavioral aspects and in particular the knowledge of sleep problems.

The aim of the present review was to systematically assess the literature and identify main theories regarding etiology of bruxism.

2. METHODS

In the literature, more than 400 articles on bruxism are available.

Data Sources: Data extraction was carried out according to the standard Cochrane systematic review methodology. The following databases were searched: PubMed, Google Scholar, Medline and the Google library. Case reports, reports with reviews and systematic review articles written in English were included.

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Data Selection: The primary outcome was bruxism etiology.

Data Extraction: Screening of eligible studies and data extraction were conducted independently and in duplicate. The references were analyzed by two reviewers using the same search strategy and the same inclusion criteria were applied to the selected studies. Query terms used were „bruxism", „etiology", and „mechanism". Among the 95 related articles that were critically assessed, 31 were included in the critical appraisal.

3. DATA SYNTHESIS

Internationally, the recent data published in the literature, shows that there is a consensus regarding a various etiologic involvement (5) in the pathogenic mechanisms of bruxism.

The first opinions on the etiology of bruxism were considered to be the dental bite and the pathology of muscle contractions. Behavioural factors and in particular, aspects related to sleep, were also included as etiological factors (6).

Attanasio R. (7), Lobbezoo et al (8), and Nascimento et al. (9) showed that the etiology of sleep bruxism involved local factors, systemic factors, psychological factors and hereditary factors.

3.1. THE HYPOTHESIS OF OCCLUSAL ETIOLOGY

Regarding local occlusal etiology of bruxism there were different opinions over time. If in 1966 Ramfjord et al. (10) believed that occlusal factors, particularly occlusal interference, would have an important role in the determination of bruxism, in 1984 Rugh et al. (11) proved, by creating experimental occlusal interference, that the role of occlusal disharmony is secondary to bruxism, since correcting occlusal interference did not lead to the disappearance of bruxism. The same situation occurred in patients with complete edentulism, which in the dentate period had bruxism. After wearing dentures, the bruxism reappeared.

However, the first affirmation on the role of occlusal interference in bruxism was based on the fact that occlusal interference suppression in patients with bruxism produced an improvement in symptoms. This is evident in current dental practice. Ramfjord (10) called centric bruxism the frequent jaw clenching. The author argued that when clenching teeth were accompanied by grinding, this occurred in the central occlusal area, in the absence of occlusal interference even in the presence of a stable occlusion, with slight slip of the teeth, of the mandible from centric relation to maximum intercuspidation.

Occlusal contacts during sleep, specific to bruxism, could be interrupted by swallowing, and muscle forces that appear during bruxism might exceed those of mastication. During sleep grinding, electromyographic bursts of the masseter muscle were observed mainly with mediotrusive mandibular movement from the canine edge-to-edge position. According to Minagi et al (12) muscular dynamics during sleep are unique compared to that during voluntary clenching, and exert a greater mechanical load to the balancing side temporomandibular joint.

In 2001, Rosales et al. (13) showed that the relationship between occlusal disorders and bruxism was not very consistent. Also, in a review in 2012, Lobezzo et al (14) concluded that to date, there is no evidence whatsoever for a causal relationship between bruxism and the bite.

3.2. MUSCLE ETIOLOGY HYPOTHESIS

There are authors who associated muscle pathology and bruxism. Hellmann et al (15) argued that anterior and posterior neck muscles co-contract during jaw clenching, their findings supporting the assumption of a relationship between jaw clenching and the activity of the neck muscles investigated.

3.3. THE HYPOTHESIS OF PSYCHO-BEHAVIORAL ETIOLOGY

The psycho-behavioural factors whose influence on bruxism etiopathology is accepted by the majority of the specialists are: stress, anger, fear, repressed aggressiveness etc. During the evolution of research, which had the goal of establishing the etiopathology of bruxism, an important moment was considered to be the one when stress was regarded as a decisive factor.

Rugh and Solberg (16,17) demonstrated the increase in intensity of bruxism episodes together with the increase of stress level. Kato (18) took into consideration the cognitive - behavioural factors such as stress, personality and anxiety in the etiology of bruxism and considered that patients with bruxism presented an anxious personality and that the dominant of their personality represents the reaching / fulfillment of personal goals.

Okeson (19) showed that patients with bruxism had a greater emotional stability, were more meticulous and got better learning results. Lavigne (20) showed that bruxism was connected to anxiety and was secondary to micro excitations during sleep (the increase of the cortical activity and cardiac frequency) followed by the grinding of teeth. Lavigne (21) pointed out that nocturnal bruxism must be differentiated from diurnal bruxism, the latter being linked to the organism's reaction to stress or anxiety and being manifested like a contraction tic of the mobilizing muscles of the mandible.

3.3.1. BRUXISM AS A SLEEP DISORDER

Sleep is an active state which takes 30% of our time, and is part of our vital behavior being essential to the survival and life quality of any individual. Sleep is made up of a succession of repeated stages which can be pointed out through EEG, EKG, EMG and eye movements. Specialists described two types of sleep REM (Rapid Eye Movement) and NREM (Non Rapid Eye Movement). There are several stages described: in NREM there are stages 1 and 2 corresponding to light sleep as well as stages 3 and 4 corresponding

to deep sleep, and in REM there is the paradoxical sleep which includes the dreaming period.

These stages of sleep alternate during a period of approximately 90 minutes on the average and repeat themselves four or five times.

The idea that bruxism was produced during paradoxical sleep has been present, but it seems that bruxism might also be present during stages 1 and 2 of the NREM sleep. These periods were associated with episodes of micro wakening, body movements and temporary acceleration of the cardiac rhythm (6).

Nascimento underlined the fact that nocturnal bruxism was found in all the stages of sleep but more often in stages 1 and 2 (9).

Kato (22) did polysomnographic recordings in which he specified the events which took place in stage 2 of sleep in normal subjects with bruxism. In the second stage the increase of cardiac frequency has been noticed through the intensification of the autonomous cardiac system, and during the last stage rhythmic activity of the masticatory muscles (ARMM) was observed. The authors have ascertained that in normal subjects the endogenous micro excitations appear approximately four seconds before ARMM while in bruxism they appear 10-60 seconds before. The increase of cardiac frequency, in normal subjects, appears at the beginning of an ARMM episode, while in patients with bruxism a gradual increase of cardiac frequency appears before the beginning of ARMM and an acceleration of the cardiac frequency is detected at the beginning of the bruxism episode.

It is still unclear why the ARMM is three times more frequent and 30% more ample in bruxers than in patients without bruxism (20). Hence the hypothesis that bruxism is a parasomnia.

In 2005, the American Academy of Sleep Medicine published the International Classification of Sleep Disorders 2nd ed. Westchester, showing that „nocturnal bruxism is defined as a disorder of the stereotypical movements during sleep and is characterized by the grinding of teeth or/and the clenching of teeth." According to this classification nocturnal bruxism is a sleep disorder, being included in the parasomnias (23).

3.3.2. THE ROLE OF THE CHEMICAL MEDIATORS IN BRUXISM

During recent years, at Lavigne's insistences, the research paths have lead towards neuropsychology in order to explain the mechanisms involved in the apparition and maintenance of bruxism, by invoking the role of some neuromediators such as dopamine and serotonin. Dopamine and serotonin are neurotransmitters which ensure the communication between neurons. Dopamine is involved in lust, pleasure and movement. Its deficit is met in Parkinson disease which is accompanied by a deficit of movement and in schizophrenia. Serotonin has a role in adjusting sleep, appetite and humour. Its deficit is met in case of anxious states and depressions (4,24).

The role of dopamine, as a causing factor of bruxism, is that of dopaminergic psycho stimulus (the same as amphetamines), worsening the bruxism episodes. The dopaminergic system has been placed in an important position in the regulation of stereotypical movements and in control of motion problems during sleep (4,24).

Yet, the voices announcing that dopamine plays a key role in bruxism etiology, are today more temperate. The selective inhibitors for the reuptake of serotonin have a direct influence on the dopaminergic system. Lobezzo et al. (25,26) stated that dopamine did not have an essential role in producing bruxism, as the selective inhibitors for the serotonin reuptake receptors had a direct influence on the dopaminergic system. These serotonin inhibitors are represented by antidepressants currently prescribed and which, used for a long time, can maintain or induce bruxism. In spite of these, the authors consider that bruxism can be adjusted at the central nervous system level and not at the peripheral one.

3.4. GENETIC ETIOLOGY HYPOTHESIS

The genetic etiology hypothesis (27) was advanced, but the transmission mechanism could not be demonstrated. Clinically, bruxism occurrences have been observed in patients belonging to the same families (parents, children or brothers). The original hypothesis about the fact that nocturnal bruxism may be associated with a familial predisposition was supported by studies on twins (27). Obviously, these observations cannot be considered as the results of a research process.

In a case-control study, Abe et al (28) investigated the association of genetic, psychological and behavioural factors with sleep bruxism in a Japanese population. Their analysis revealed that only the C allele carrier of the HTR2A single nucleotide polymorphism rs6313 (102C>T) was significantly associated with an increased risk of sleep bruxism (odds ratio = 4.250, 95% confidence interval: 1.599-11.297, p = 0.004), suggesting a possible genetic contribution to the etiology of sleep bruxism.

3.5. OTHER FACTORS POSSIBLY INVOLVED IN BRUXISM'S ETIOLOGY

In 2003, Winocur et al. (29) published a study showing the correlations between the consumption of alcohol, tobacco, drugs and pills and bruxism occurrences. In 2006, Lobezzo et al (8) showed that bruxism might be a brain injury consequence and might be associated with some psychiatric or neurological diseases. Also, bruxism was linked to the use of amphetamines, levodopa, phenothiazines and alcohol. Lavigne et al. (21) stated that the ARMM and nocturnal bruxism episodes were influenced by an increase in the electrical activity of the brain and by the stimulation of the ascending reticular system, which increased the activity of the motor neuronal network and of the cardiac autonomic system.

According to Behr et al (30), theories on factors causing bruxism are a matter of controversy in the

current literature, two main etiological models being the most important. The first one were peripheral local morphological disorders, such as malocclusion. 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 circuit processing of the basal ganglia is supposed to be responsible for muscle hyperactivity during nocturnal dyskinesia such as bruxism.

In Romania, the recent most important views on bruxism considered particularly the stress and occlusal interferences in the etiology of bruxism, but affirmed that, until now, there could not exist a clearly established direct causal link between a specific etiologic factor and bruxism (31). Just like the occlusal trauma, it is sure that only one etiologic factor cannot be incriminated in the etiology of bruxism.

The evidence of this finding is that, to date, there is not a single therapeutic method to obtain the removal or improvement of bruxism; there are always more associated therapeutic procedures (31).

4. CONCLUSIONS

* Bruxism is a dental disorder that deeply alters the dento-maxillary system's normal functionality.

* The etiology of bruxism is varied, involving local, systemic and psycho-behavioural factors.

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Questions

Regarding the etiology of bruxism

- ❑ a. Is unifactorial
- ❑ b. Behavioral factors are not involved
- ❑ c. The original definition refers mainly to dental contacts and muscle contractions
- ❑ d. Sleep problems are not a part of the etiology of bruxism

Which of the following are not involved in bruxism research:

- ❑ a. Electromyography
- ❑ b. Electroencephalography
- ❑ c. Telemetry
- ❑ d. Radiography

Which of following are not psycho-behavioral factors:

- ❑ a. Stress
- ❑ b. Anger
- ❑ c. Repressed aggressiveness
- ❑ d. Hunger

Regarding chemical mediators in bruxism:

- ❑ a. neurotransmitters do not ensure the communication between neurons
- ❑ b. Dopamine is not involved in lust and movement
- ❑ c. Serotonin does not have a role in adjusting sleep
- ❑ d. Selective serotonin inhibitors did not present the expected results