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Oral Parafunction - Aetiology, Implications and Relation to Orthodontic Treatment

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Abstract

Oral parafunction can be defined as an extra-functional action of certain components of the stomatognathic system. The automation of this kind of occurrence that persists in the form of a reflex arc is a denominated habit. The oral parafunctional habits are described as the action of clenching or grinding teeth (bruxism), among others. This work approached bruxism due to its clinical importance. To evaluate the predisposing factors to the development of oral parafunction, the orthodontist should have updated knowledge of the whole process of the phenomenon of bruxism. The purposes of this chapter were about the comprehension of the neurophysiology of bruxism and also about the capacity of structural adaptation of the components of the stomatognathic system, the analysis of its aetiological factors, as well as its implications on the structures of the masticatory system, and the verification of the relation between bruxism and the orthodontic treatment. In conclusion, the nature of that oral habit is multifactorial, which implies extrafunctional demand of neurophysiological mechanisms, whose effects are installed from the rupture of the structural limit of the adaptive capacity of the stomatognathic system, peculiar to each individual. The performance of orthodontic treatment is not related to the development of bruxism.

Keywords: bruxism, parafunction, neurophysiology, orthodontic treatment, stomatognathic system

1. Introduction

Parafunction is any disorder in the action of a particular organ or organ system, often characterised by an overactivity of the physiological action associated with a normal function.

In a dental approach, oral parafunction can be defined as an extra-functional action of certain components of the stomatognathic system. In order to understand it, a more complex scope is necessary, reaching the physiological and neurophysiological action of the entire tract of the stomatognathic system involved, as well as the implications of any disturbances within the normal function, always taking into consideration the aspects of the structural and functional adaptations to which the human organism is subject, within certain limits.

The automation of this type of occurrence that persists in the form of a reflex arc is called a habit. Ferreira [1] defined habit as a lasting disposition acquired by frequent repetition of an act, use and custom.

Oral parafunctional habits are described as grinding or clenching teeth (bruxism), nail biting, finger sucking, chewing objects, abnormal craniocervical-facial posture, among others.

In an orthodontic approach, bruxism should be considered due to the clinical importance of its deleterious effects on the dento-orofacial architecture and the awareness of its increasing prevalence observed in individuals seeking orthodontic treatment.

Its aetiology is quite diverse [2], but it is closely related to the central nervous system (CNS) stimulus and its neurotransmissive mechanisms, as well as psychological-emotional aspects.

The parafunctional action is due to the frequent repetition of a specific function over a prolonged period, which may lead to anatomical alterations [2]. Its implications are several and depend directly on the organic reaction, which is individual and particular.

A clinical significance of the investigation of bruxism lies in its considerable role in the aetiology of pain and temporomandibular dysfunction (TMD) [3–5]. In addition, the increasing prevalence of this parafunctional habit in the population and the deleterious effect it causes to the stomatognathic system represent a strong justification for the current approach [6].

2. Aetiology of parafunction

There is controversy regarding the nature of the aetiology of oral parafunction, that is, whether it is multifactorial or of single origin [6, 7].

However, at present, there is a tendency to agree with a strong psychic-emotional participation and, more recently, for the contribution of the use of certain types of drugs in the aetiopathogenesis of bruxism.

It is understood that psychic-emotional situations such as depression and anxiety modify the perception and the tolerance of the individual in face of physical symptoms and situations triggering stress [8]. Considering the constant state of alert imposed by the mere participation in the current society [9], the professional needs to be aware of the degree of involvement of the aetiology, consequences and implications of the mechanism of oral parafunction in the human organism and, above all, in the individual who will undergo orthodontic treatment.

Regarding the prevalence of bruxism, it is believed that when it occurs in the absence of signs and symptoms resulting from this habit that can occur in an episodic and transient way, it is high. As the effects that are characterised by the collapse of the component structures of the stomatognathic system lead to the search for treatment, the prevalence in a population group does not appear significant [3]. In short, as long as bruxism is performed on a level of subconsciously controlled reflex, this habit is not perceived by the individual, except when it begins to draw attention through its signs and symptoms.

3. Neurophysiological aspects of oral parafunction

All the functions exerted by the masticatory complex depend basically on the mechanism of muscular contraction that is nothing more than a response to a stimulus.

In order to study the neurophysiology of the process involved in parafunction, it is necessary to understand the integration between the main functional and anatomical components of the stomatognathic system and the CNS.

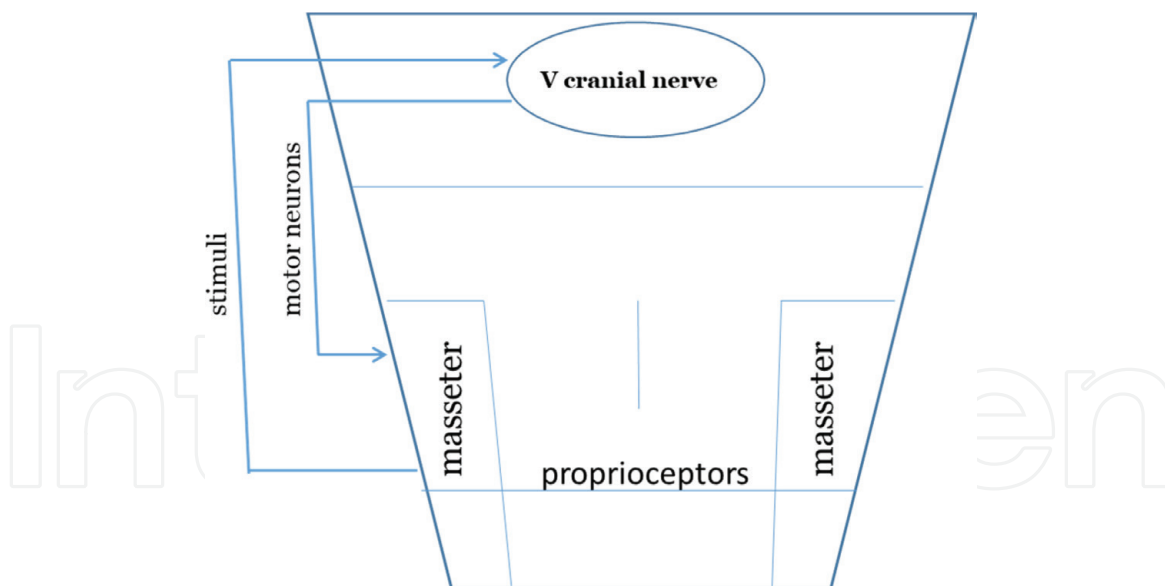


Figure 1.
Reflex action.

The grinding and clenching of teeth are mainly conditioned by the mechanism of muscle contraction. The extra-functional muscular demand caused by bruxism emits stimuli through neuromuscular spindles contained in the intimacy of the muscle fibres that travel through the afferent pathways to the mesencephalic nucleus of the V cranial nerve (trigeminal). From this mesencephalic nucleus, through specific secondary neurons, messages are sent to the trigeminal motor nucleus, located just below, which will send messages via motor neurons to the muscle from which the stimulus started, producing the contraction of its fibres (**Figure 1**) [2].

Although the information is sent to the CNS, the response is independent of the will and normally occurs without the influence of higher centres. This process is known as reflex action [10].

This reflex action occurs through the mechanism called proprioception, which is the term applied to the ability to perceive sensations originating in one's own body. It is a phenomenon linked to cognitive systems of the brain, including memory. The receptors responsible for this function are proprioceptors, which provide information about the position and movement of the mandible and associated oral structures [10].

The parafunctional behaviour is closely related to certain types of stimuli originating in the CNS. These stimuli provoke several organic reactions; among them, the increase of neuromuscular motor excitations, which, in the condition of constant and uninterrupted hyperactivity, is classified as parafunction [11].

4. Organic structural adaptive capacity

All structures of the stomatognathic system, after growth has ceased, are always in the process of transformation and adaptation in conformity with their physiological functional demand, thus conferring a dynamic quality to these structures.

Among the structures of the stomatognathic system is the temporomandibular joint (TMJ). It is subject to functional adaptive changes due to discrepancies that exist between the positions of maximum dental intercuspation and centric relation of the mandible head. These constant TMJ accommodation processes testify to the remarkable adaptability of this joint to adapt to the conflict of these discrepancies, giving the TMJ the characteristic of performing its function in a condition of continuous displacement. No other junction of the human organism has this characteristic [12].

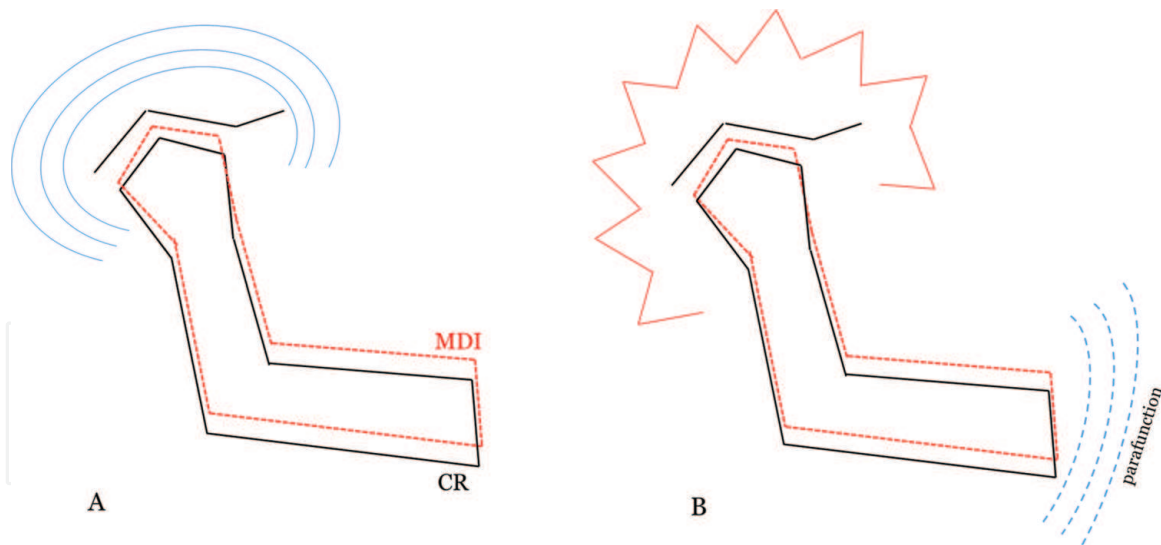


Figure 2.

TMJ accommodation process (A); after the limit of its adaptive capacity has expired, collapses can lead to unpleasant signs and symptoms (B).

Thus, it is understood that the organism, even in the presence of non-physiological situations, exhibits a degree of tolerance in which it has a limiting structural adaptive capacity. That is, after the limit of its adaptive capacity has expired, collapses or even changes in the involved structures can lead to unpleasant signs and symptoms. This is what happens in the mechanism of parafunction (**Figure 2**).

Studies have shown that the bite force performed in episodes of nocturnal bruxism showed an amplitude that exceeded the force that occurred voluntarily during wakefulness and could reach a frequency of four times more [11, 13]. The nocturnal dental contacts during the parafunctional activity can reach a frequency between 15 and 45 minutes of contact between the teeth of the bruxism patients, obtaining, then, an average of 11.4 minutes in these individuals whereas in people without the habit, the average was 3.1 minutes. Thus, it is inferred that the horizontal forces performed in parafunction may have potential to promote adaptations that, associated with other predisposing factors present, determine lesions, since they have a potential much greater than the forces exerted in the physiological function [14].

What modulates the muscular forces and the duration of dental contacts in both the physiological masticatory function and the parafunction is the mechanism of neurological proprioception present in the masticatory system.

There is no functional and anatomical structure in the CNS identifiable as a specific generator of involuntary oromandibular movements [15]. What can be concluded, from the knowledge of the phenomenon of organic adaptability, is that parafunction is a disease that is perceived upon the installation of signs or symptoms related to rupture of the structural limit of the adaptive capacity of the stomatognathic system, particular of each individual.

5. Classification of the oral parafunction

Bruxism can be classified according to severity of symptoms, aetiopathogenesis and clinical manifestations.

The severity of the symptoms depends on the deleterious occurrences on the masticatory system, which present a degree of variability among individuals [6]. This individual variability is conditioned by the organic response, adaptability and predisposition factors represented by the presence of certain deficiencies of the components of the stomatognathic system.

1. Primary
Peripheral: occlusal factors (Ex.: Malocclusion); central: CNS aminergic imbalance (Ex.: Use of SSRIs)
2. Secondary
Associated with drugs or other substances (Ex.: Parkinson's disease treatment); associated with sleep disorders (Ex.: Obstructive sleep apnea), neurological disorders (Ex: Akathisia disorder), psychiatric disorders and other diseases

Adapted from Aloé [15].

Table 1.
Classification of bruxism according to its aetiopathogeny.

From another point of view, bruxism can be distinguished according to its aetiopathogenesis, which will lead it basically to two categories: primary and secondary. The primary is the aetiology of bruxism conditioned to peripheral factors due to malocclusion and central factors resulting from neurotransmission disorders [16]. In the secondary category, parafunctional habits associated with clinical, neurological or psychiatric disorders are included, as well as those related to iatrogenic factors, such as in the use or withdrawal of substances or medications, and those resulting from sleep disorders [15] (**Table 1**).

The clinical manifestations represent the signs that denounce the presence of bruxism or its existence for some period, such as the facets of wear and morphology of the dental arches, among others. It is thus perceived that its classification covers the entire process involved in its mechanism, from aetiology to the expression of signs and symptoms.

6. Aetiological considerations of oral parafunction

Bruxism can be considered as a behavioural disorder when considering the prevalence of emotional aetiology. However, when aetiological factors related to organic alterations, such as those occurring in sleep disorders, are considered, it consists of a derangement of the central nervous system [3].

In an analysis of the contribution of peripheral and central factors in the aetiology of bruxism, Lobbezoo and Naeije [17] concluded that there is strong evidence that the role of occlusal features and other morphological factors is small or even null. There is also evidence that disturbances in the central dopaminergic system are implicated in the aetiology of bruxism. In addition, the role of other aetiological factors such as smoking, alcohol, disease, trauma, heredity, stress and other psychological factors is probably lesser than assumed so far. In short, it can be said that bruxism has central and not peripheral mediation [17].

The theory of the combination of peripheral (occlusal) and emotional factors in the aetiology of bruxism is advocated by several authors [6, 14, 17]. Moreover, some drugs such as amphetamines, alcohol, and also sleep disorders, CNS disorders and hereditary factors may be related to the onset of bruxism [6].

Based on these considerations, it is inferred that the multifactorial nature of the aetiology of bruxism can be enumerated in a way to clarify, briefly, each of the main causes of this parafunctional habit increasingly investigated by several research fronts, according to their interests and lines of conduct.

6.1 Emotional factor

Several psychological conditions, such as stress, anxiety or aggressiveness, have been associated with the presence of oral parafunctional habits, which are also

recognised as stereotyped movements of the masticatory muscles [18]. An emotional aetiological component, such as psychological stress, may play an important role [19–21], but not all those who grind teeth present emotional problems [21].

The relationship between degrees of dysfunctions of the masticatory system, presence of parafunctional habit and anxiety can be analysed by means of trait-state anxiety scales. Anxiety trait refers to personality trait; anxiety state is defined as a transient emotional state. Thus, there is a positive correlation between the degree of myofascial pain dysfunction (MPD) and the presence of bruxism with the level of anxiety in both scales of trait and state, that is, both an anxious personality and a transient state of anxiety may be directly proportional to the bruxism event and degrees of MPD [22].

Stress is a reaction of the organism to situations of danger that, at first, does not produce deleterious implications. What causes the installation of serious organic complications is uninterrupted stress [11].

In a stress condition, stimuli are routed to the hypothalamus, which in turn activates the pituitary gland, which will sensitise the adrenal gland. The observed reactions can be described as increased blood pressure, gastric problems, insomnia, hair loss and heavy and involuntary isometric contractions of the masticatory muscles. Even though the masticatory muscles are classified as voluntary, their stress contractions may become involuntary through CNS-induced stimuli [11]. Another modification is the creation of additional muscle activity without altering the performance of tasks. These additional activities are considered to be nervous habits, such as bruxism [10].

It is interesting to note that the hypothalamus, in addition to producing the hormone corticotropin to induce the pituitary gland, also activates the cognitive systems of the brain, including memory, to evaluate the stimulus. If the situation that initiated the stimulus poses no danger, the hypothalamus suspends the whole process (Figure 3). For this interpretation, it is worth remembering that the body reacts to stress by increasing its metabolism in order to adapt to the new demands. This leads to the decrease in the organic adaptation threshold. This structural tolerance, which is the point at which collapse begins, is an individual characteristic and directly dependent on personality factors. Thus, what differentiates the stressful manifestations in individuals, in general, are these personality factors as radical positions in relation to the facts, competitiveness and the need to dominate situations [11].

For this reason, the individual evaluation becomes essential in the approach of the emotional effects on the parafunction. However, emotional conditions are difficult to become operational in a research because there are many individual

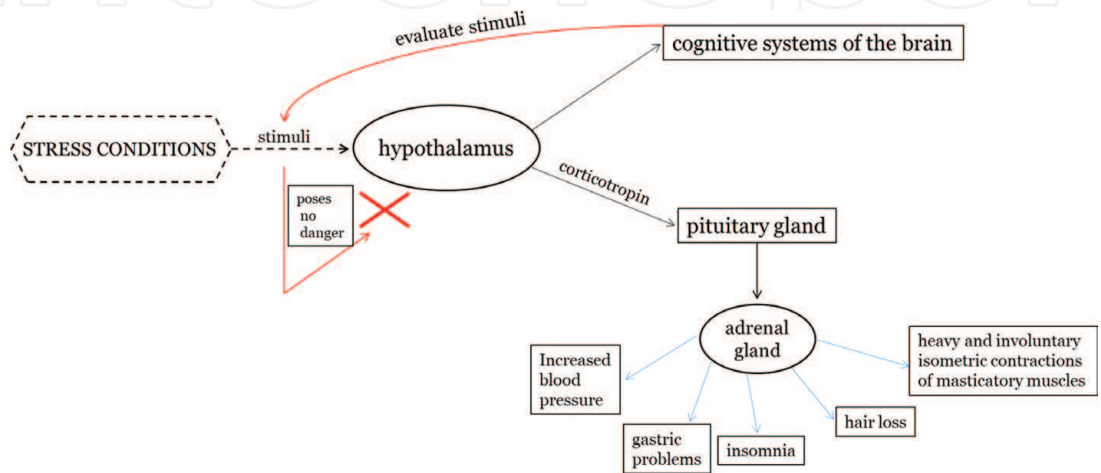


Figure 3.
Stress conditions.

differences in the way the personality and the presence of bruxism are investigated [17]. Thus, it is believed that the participation of emotional factors in the aetiology of this habit may be less than what was previously attributed.

In a study about the relationship between stress level and bruxism using tricyclic antidepressants (amitriptyline) to control pain and stress, there were positive results, that is, the level of stress associated with bruxism was reduced in low doses of this medication [23]. Thus, as previously mentioned, emotional stress has a more significant influence on bruxism than the occlusal factor [7].

Another expression of oral parafunction characterised by grinding or clenching of teeth in an altered emotional condition may attenuate the effects of stress or anxiety on the organism, since in this condition, there are central neurotransmissive changes that can be considered as a strategy to support or even deal with stress and thus minimise its effects on the body [18].

The lack of awareness of the complications that can arise from stressful situations, the lack of perception of the slow and gradual installation of the organic implications of constant stress and the alienation before the management of these situations for a better coexistence converge to the inevitable condition of veritable collapse of important organic structures, such as the stomatognathic system, which can sometimes become irreversible.

6.2 Factors associated with medications and neural pathways

There is evidence that neurochemical factors may be related to the aetiology of bruxism [6]. The acute exposure to stress determines the production of various neurotransmitters such as noradrenaline, dopamine and serotonin [18]. Thus, the involvement of central dopamine in the aetiopathogenesis of parafunctional movements [18] confers the participation of dopaminergic and serotonergic neurotransmission in the genesis and modulation of bruxism [16].

The corpus striatum has the highest concentration of dopamine and is considered the most relevant brain region in the mediation of oral stereotyped movements. Studies in rodents have shown that administration of high doses of dopaminergic agonists (apomorphine) induced oral stereotyped movements such as those occurring in bruxism [18].

Bruxism is primarily a CNS phenomenon, common to all people. In this way, the intensity of the central process responsible for bruxism could be a persistent feature of a person who presents it in childhood and continues to present it in adulthood [24].

Most brain functions are the result of converged actions of various neurotransmitters. The neurotransmitter responsible for inhibiting spontaneous movements of the masticatory muscles and maintaining its tonus is dopamine [16].

Serotonin-concentrating drugs such as selective serotonin reuptake inhibitors (SSRIs) also alter the level of dopamine in the mesocortical tract and frontal cortex [15–17, 25, 26]. This effect can be explained by the effect of these drugs on the dopaminergic system [16, 17]. It is believed that serotonin exerts modulatory influence on dopamine, which is the main neurotransmitter in muscle activity [17].

There are two hypotheses compatible with these changes: hyperdopaminergic and hypodopaminergic. The first may be related to the chronic use of antidopaminergic drugs, such as in the treatment of Parkinson's disease, which causes hypersensitivity of the dopaminergic receptors and may lead to teeth grinding. The second is implicit in the mechanism that occurs with the use of SSRIs: this drug induces the increase of serotonergic transmission that will cause a dopaminergic reduction due to the heteroreceptive serotonin binding on the dopamine receptors of the dopaminergic neurons. In this fashion, there will be a decrease in dopamine binding to its receptors leading to motor disinhibition by the prefrontal cortex and

resulting in bruxism. Thus, disordered movements frequently result from elevation of serotonin levels by SSRIs, reducing dopaminergic activity in both the mesocortical and nigrostriatal tracts.

Nevertheless, recent studies indicate that evidence on the manifestation and establishment of drug influences, such as that of the dopamine agonist (pramipexole), on the parafunctional activities of bruxism is inconsistent [27, 28].

There are reports of bruxism induced by other antidepressant medications (venlafaxine, citalopram and SSRIs) that can be controlled with buspirone and gabapentin [15, 16, 29–32]. That is, the use of buspirone can eliminate the bruxism induced by these medicaments due to the restoration of the motor modulation.

Bruxism can be an acute reaction of induction by drugs that increase the level of synaptic serotonin and represent mainly a variation of akathisia. Thus, the diagnosis of bruxism induced by antidepressants may be controversial because of its vague symptoms such as bitemporal headaches, masseter spasm or mandibular pain in addition to classic teeth grinding findings. Therefore, because of its masked presence, bruxism may be much more common than reported [30].

6.3 Occlusal factors

There is controversy regarding the relationship between malocclusion and muscle hyperactivity present in parafunction [11].

What makes it difficult to establish the cause and effect of relationship between these factors is the knowledge of the phenomenon of organic, structural and functional adaptability [11]. As a result of this phenomenon, the divergence of the responses in the individuals is defined as a function of the degree of possible alterations provoked by the muscular hyperactivities for non-functional purposes.

Some authors [33–37] considered that local mechanical factors, especially dental malocclusion, would play a major role in the aetiology of bruxism.

A statistical study performed by Olkinuora [38] found no correlation between the incidence of malocclusion and bruxism.

An analysis of the literature revealed that most of the controversies regarding the role of occlusion in the aetiology of parafunction were derived from inconsistently oriented studies, thus not leading to scientifically defined results. This is due to the difficulty of some researchers to clinically define and stabilise a TMJ position in which the condyles could operate in harmony with the occlusal surfaces of the teeth, preventing a logical and scientific analysis [2].

Stimuli caused by occlusal alterations can generate motor reflex responses by altering the mandibular position and affecting muscle tone [11]. What is understood is that, depending on occlusal interference, its type and location in the occlusal anatomy of the tooth, there may be displacement of the mandible in its closing or eccentric movements, producing unbalanced muscle forces. These mandibular slides, in addition to moving the condyles out of their positions of musculoskeletal stability, may require muscle hyperactivity, altering their tonicity. This may lead to dysfunctions in the stomatognathic system characterised by MPD and orofacial pain involving the whole musculature of the masticatory apparatus [33, 39].

The excess of fatigue and subsequent pain resulting from the sustained contraction of the muscles in the parafunctional activity decreases the threshold of excitability of the neurons of the reflex centre initiating the feedback mechanism. This vicious cycle of the perpetuating increase in muscle tension related to dysfunctional disorders of the teeth, periodontium, TMJ, and masticatory muscles is the basis of bruxism [33].

Based on this reasoning, one could theorise the intrinsic participation of the occlusal factor as a trigger for the parafunctional mechanism. However, it is essential to infer that there will be imbalance of the involved structures if the limit of the

organic structural tolerance is broken upon the participation of other supporting aetiological factors, among them the emotional one.

Historically, discrepancies between centric relation and maximum habitual intercuspation and poor tooth positioning were labelled as the most common causes and perpetuating factors of bruxism [17].

Yet, some authors have confirmed the opinion that no occlusal factor alone represents greater importance in the development of TMD and bruxism [40]. Others [6, 33, 36], in contrast, considered the interaction of occlusal and emotional factors as a cause of bruxism.

However, it is believed that there is no scientific evidence that malocclusion or interceptive contact between opposing teeth can initiate or maintain bruxism [3, 14, 32, 41, 42].

In the past, authors such as Ramfjord [33] believed that bruxism should be an instrument with which the individual attempted to eliminate occlusal interference that would cause reflex excitation of the masticatory muscles through stimulation of periodontal mechanoreceptors. However, it is now known that the stimulation of the mechanoreceptors of interceptive dental contacts has a more reduction than increase effect in muscle activity [34, 4–43].

Recently, some studies have demonstrated that the elimination of interferences in occlusion had no influence on parafunctional activity [17, 43–45].

Moreover, not every bruxism patient necessarily presents occlusal interference and those who present it do not always develop the parafunctional habit [17, 35].

The literature points to several controversial theories and opinions regarding the true role of occlusion in the aetiology of bruxism. However, at present, the association between occlusal disharmony and the triggering of bruxism has been debated and challenged, as well as a specific relation between principles of ideal occlusion and absence of bruxism.

What should be considered relevant in relation to the occlusal arrangement during the bruxism mechanism is the distribution of the forces resulting from this activity for non-functional purposes on the teeth and support structures. In a situation of occlusal disharmony, these forces can generate deleterious effects on important components of the masticatory system.

6.4 Other aetiological factors

Other sources in the aetiopathogenesis of oral parafunction are also considered to be factors that trigger bruxism, but no less important in a therapeutic approach [43, 46] such as the aforementioned emotional factors, those associated with medications and neural pathways and occlusal. These are: sleep disorders, genetic factors, use of stimulant and alcoholic beverages and smoking.

6.4.1 Sleep disorders

With regard to sleep disorders, it is understood that during the sleepy state, the use of the masticatory muscles in non-functional activities during the rhythmic attrition of the teeth is characterised as a parafunctional behaviour, that is, it has no functional purpose [3], and thus is believed to be more related to changes in emotional stress levels and sleep stages when neuromuscular protection mechanisms appear to be absent resulting in less influence on muscle activity for non-functional purposes [10, 35, 41, 47].

Night bruxism is common in the general population and represents the third most frequent sleep disorder [21].

As a result of this high prevalence of parafunctional sleep habit, sleep physiology has been studied extensively in order to search for possible causes of this disorder.

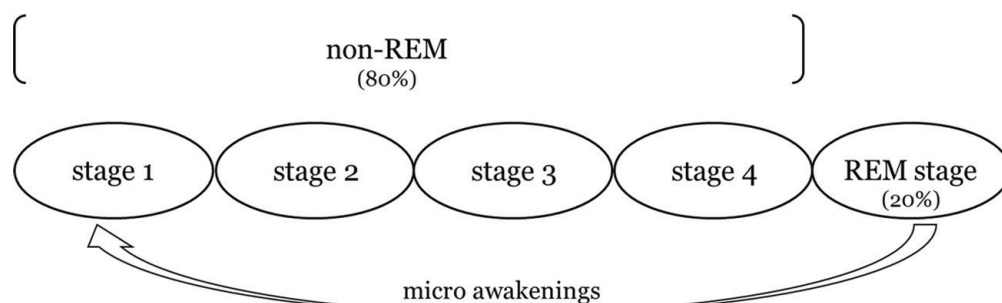


Figure 4.
Adult's sleep period.

The sleep process can be described basically as two types of mental activity: a mild one, which is divided into stages 1 and 2, and a deep one, which is divided into stages 3 and 4. The sequence of these two types of mental activity is called the non-REM phase and represents 80% of an adult's sleep period. The other 20% corresponds to the phase called REM, which is characterised mainly by rapid eye movements. In this way, the sleep cycle is initiated by stages 1 and 2 (superficial), passing to stage 3 (deep) and to stage 4 (even deeper), when it reaches the REM stage. From this REM period, there is a return to the more superficial stage when the so-called micro-awakenings occur. And so the cycle restarts (**Figure 4**) [10].

There is an understanding that nighttime bruxism occurs normally in the non-REM phase, especially in stage 2 and during changes in sleep stages. Nonetheless, there is also another consensus that bruxism may occur during REM sleep and, in this case, is more frequently associated with facial and dental pain [21, 48].

Following this same line of reasoning, there is the theory that parafunctional habit can start at any stage of sleep, but never in its REM stage [36, 49].

Patients with obstructive sleep apnoea, snorers, who present moderate daytime sleepiness, alcoholics, caffeine users, smokers, and those with a highly stressful and anxious life are considered to be at high risk for nighttime bruxism [21].

However, clinical findings showed that there were no significant differences in sleep microstructure in patients with parafunctional habits when compared with individuals considered normal [50].

There are divergences in the literature about which stage of sleep bruxism occurs, such as the observation that bruxism is a relatively nonspecific disturbance of the awakening mechanism, that is, from the passage from sleep to wakefulness. Thus, it can occur as an awakening reaction from any stage [36, 37]. In any case, bruxism is considered a parasomnia, that is, it is a sleep disorder with a high degree of impairment of the structures of the masticatory system, in view of the deficiency of mechanoreceptor mechanisms during the sleep period.

In general, some of the main symptoms that affect people who grind or clench their teeth during sleep are pain in their own teeth, face, head and sensation of muscle fatigue in the areas corresponding to the masseter and temporal muscles that occur in the morning, after awakening and relieving after a few hours [51].

6.4.2 Genetic factors

There are reports of a significant connection of a particular genetic contribution to the pathophysiology of bruxism [21, 38], as well as to the statistically high frequency of the same wear pattern, which reinforces the hypothesis that hereditary factors are important in the genesis and pattern of bruxism and seems to influence the central trigger of this habit [38, 52, 53].

However, in order to establish a pattern of inheritance of the mechanism of bruxism, studies are required encompassing several generations and chromosome identification [54].

6.4.3 Stimulant beverages, tobacco and alcohol

Smoking may increase episodes of tooth grinding during sleep [21]. Both tobacco and stimulant beverages are excitatory substances in the CNS. This means that they may contribute to increase the effect of the stress mechanism, since they present diverse neuromuscular, cardiovascular, and respiratory repercussions. Among stimulating beverages, coffee, tea, chocolate, and cola-based soft drinks may be mentioned [11].

Nicotine also stimulates central dopaminergic activity, which may explain the findings that smokers report bruxism almost twice as often as non-smokers [17, 50, 55]. Alcohol can also induce bruxism [17, 56, 57].

The search for an understanding of the aetiopathogenic basis of bruxism results in diverse and controversial opinions of authors who have studied the subject.

However, it is an almost always present opinion that the aetiological principle of bruxism involves at least two triggering agents and is therefore considered an aetiology of a multifactorial nature.

7. Implications of the mechanism of oral parafunction

Generally, the parafunctional activity for an extended period may imply damage to the structures of the stomatognathic system due to excessive forces applied to the components of this system, which often exceeds the structural tolerance limit of certain structures, as already described.

When the result of the combination of occlusal changes and stress is greater than the body's ability to adapt, muscular hyperactivity is increased, generating intense forces that can reach the structures of the masticatory apparatus, causing collapse [11], that is, this tolerance can be described as a critical level of tolerance to the increased forces generated by the muscular hyperactivity of the components of the masticatory system [10].

Okeson [10] elaborated an equation to demonstrate the aetiology and effect of this muscular hyperactivity :

Malocclusion + Emotional Stress > Physiological Tolerance →
Increased muscle hyperactivity > Structural tolerance → Collapse

This statement reinforces the idea that the response to these situations is individual. The type of reaction is directly related to factors such as particular predisposition to periodontal and dental problems, muscle changes and TMJ. Each one will cause symptomatology originating from its respective deficiency and will give rise to implications on the stomatognathic system, which can also be called biomechanically induced dental diseases. The latter affect at least 75% of the adult population. These data confirm that more teeth are lost due to these diseases than to the effects of caries [12, 58, 59].

There are indications that it is rare to find an individual who presents a condylar and dental relationship with complete balance and harmony. Likewise, an in-depth examination of adults reveals that it is equally rare to find one who has no signs of biomechanically induced dental disease that can be proven to result from grinding and clenching teeth [12].

Following this reasoning, the diagnosis of the presence of the parafunctional habit should be made as early as possible, since in most cases the bruxism patients only suspect its existence when they present damages in the structures of the stomatognathic apparatus, often in a very advanced stage such as dental wear, tooth or restoration fractures, dental hypersensitivity, masticatory muscle discomfort or TMJ pain and in muscles involved in mastication [54].

McCoy [60] used the term dental compression syndrome (DCS) to refer to bruxism and other parafunctional habits such as nail biting. The grinding and clenching of the teeth produce fatigue on the teeth and supporting tissues causing damage to the dental and bone structures and soft and articular tissues. Thus, the understanding of the relationship between DCS and TMD should be further investigated since the effects caused by DCS lead to it becoming an important contributing factor in the onset of TMD and injuries on the teeth [45, 60]. However, there is evidence that clenching teeth (centric bruxism) is more damaging to the masticatory system than grinding teeth (eccentric bruxism) [61].

Among the clinical findings that characterise bruxism, there is a greater predominance of myofascial pain affecting mainly the masseter and the anterior temporalis muscle, followed by dental problems and in the periodontium of support, limitation of the mandibular movements and muscular hypertrophies [62].

The recognition by dentists that bruxism rarely occurs alone, that is, it is always associated with other symptoms, becomes an approach of fundamental importance [63].

What will determine the type of deleterious effects (signs and symptoms) on the stomatognathic system due to the prolonged action of parafunction is the structural tolerance of each component of this system. The potential sites of collapse are the muscles, the TMJs, the supporting structures of the teeth and the teeth themselves.

There is a strong consensus that bruxism is a significant contributing factor in the cause of TMD, including masticatory muscle disorders and joint pain [4–6, 10, 54], such as osteoarthritis, capsulitis, synovitis, disc adhesions and joint pain, in addition to muscle pain [6].

Among the neuromuscular effects that fit the TMDs, one may mention muscle hypertonicity, muscular hypertrophy, movement limitation, myositis and spontaneous myalgia, and myofascial pain. The most frequent symptoms are pain affecting the masseter and anterior temporalis muscles [62, 64].



Figure 5.
Main masticatory muscles involved in bruxism: masseter and temporal.

One can also attribute as implications of bruxism the increase in muscle tone and resistance to manipulation of the mandible, in addition to compensatory hypertrophy, muscular fatigue sensation and pain to the palpation of the mastication muscles [36].

7.1 Most frequent symptoms of TMDs

Once the imbalance of the stomatognathic system is established due to oral parafunction, the main symptoms are limitation of physiological activity, noises and pain in TMJ, muscular pain, and limitation and deviation of mandibular movements [65]. However, the mandibular elevating masticatory muscles, especially the masseter and temporal, are the most affected structures in TMDs due to bruxism (Figure 5) [65, 66].

TMDs may also be associated with anatomical, physiological and psychological factors, as well as headaches and neck and ear pains [65–67].

8. Changes in morphological craniofacial architecture

Research on the relationship between form (or structure) and function of the components of the masticatory system is of extreme scientific interest because it can elucidate how structures develop beyond their genetic component.

In the case of the extra-functional demand of bruxism, can it exert influence on the craniofacial structures of the individuals who are growing or even those who have passed this stage?

In this context, the craniofacial morphology could perhaps represent one of the signs of some relevance for the clinician to search for. However, the literature shows different opinions, even going to the controversies on this subject.

In 1994, Menapace et al. [68] mentioned that some authors advocated the hypothesis that bruxism directs towards a particular craniofacial morphology and that other researchers, on the contrary, believed that a certain craniofacial morphology may predispose to the parafunctional mechanism. However, when investigating the type of craniofacial and dental morphology between individuals with and without bruxism, these authors did not find significant differences between the two groups.

In a recent study, it was observed that some adult individuals, that is, after the period of facial growth and development, may develop anterior open bite due to episodes of parafunctional oral activity [69].

In another study on the relationship between craniofacial morphology and bruxism, Young et al. [70] found a statistically significant difference in the bizygomatic and cranial widths when they compared bruxism patients with those who did not present the habit. They associated this finding with the theory that the increase in non-functional demand that occurs in bruxism can result in broader or wider skull-and-mouth traits as prescribed by functional matrix theory. Perhaps, the result of these studies indicated a greater functional effect of the masseter and temporal muscles on the skeletal traits of bruxism patients. However, these authors also affirmed that this evidence is based on an indirect rather than a direct premise, since independent variables reported in bruxism were evaluated, such as muscle strength, tooth wear, number and density of muscle fibres and occlusion.

Other data found in research focused on the relationship between tooth wear occurring in bruxism, and the morphology of craniofacial structures was more rectangular maxillary arch in combination with anterior mandibular rotation, diminished anterior facial height and greater bimaxillary interincisal angle [17, 71].

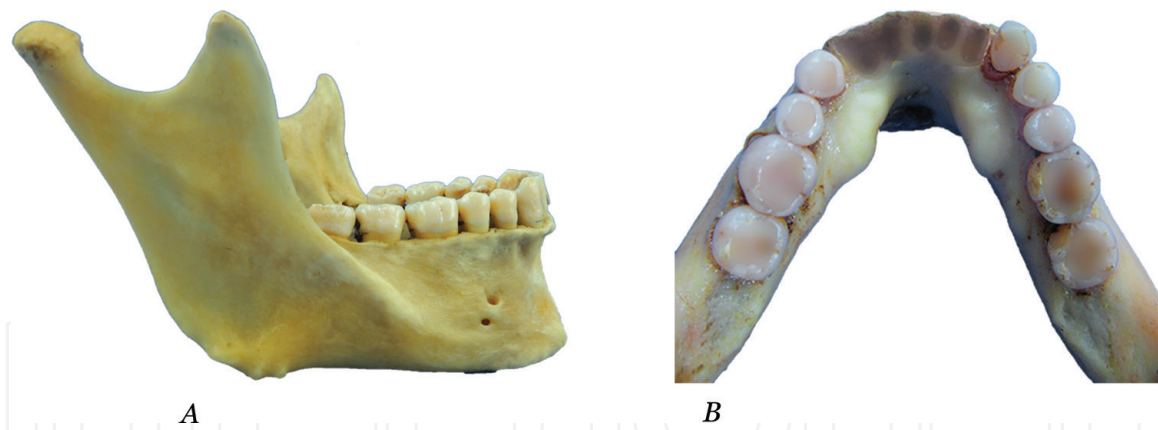


Figure 6.
Prominence of mandibular angle (A); mandibular torus on lingual surface (B).

Other authors [17, 70, 72] believe that parafunctional habit does not imply alteration of facial morphology, since dentoalveolar development should compensate for tooth wear related to bruxism and, therefore, deny a possible effect of reducing facial anterior height in bearers of this habit. Thus, they demonstrated that there is maintenance of the overbite due to the extrusion of anterior teeth so that the contact with its antagonists remains.

Otherwise, alterations of some facial structures can take place, like the prominence of mandibular angle (masseter insertion) and mandibular torus on the lingual surface (**Figure 6**) [73].

From the above, it is observed that there is no consensus on the actual association between craniofacial morphology and the presence of bruxism, and this probable clinical sign consequent to the parafunctional habit should be carefully examined.

9. Therapeutic strategies to control bruxism

Considering the multifactorial aetiological nature of oral parafunction, as well as the different forms of involvement on the dentofacial structures involved, once it is perceived that when the structural limit of the adaptive capacity of the stomatognathic system, which is individual, is reached, the therapeutic approach becomes complex and at the same time palliative. It can be said that bruxism can be controlled by means of different therapeutic procedures on the signs and symptoms resulting from its installation, which is slow and gradual.

The proposed therapeutic strategies can be listed as psychological approach, guidelines on sleep hygiene measures combined with relaxation methods, use of interocclusal devices, prescription of medication for pain control, electrical stimulation, physiotherapy, acupuncture and, more recently, use of botulinum toxin to control the contraction of the main muscles involved in the parafunction, decreasing muscular strength and pain [66, 74–76].

There are currently questions about the influence of the use of invisible orthodontic aligners on the effects of bruxism with regard to parafunctional muscle activity during sleep and, consequently, related symptoms, such as pain. These devices are compared with those used in the traditional therapy of nocturnal bruxism control: hard-resin appliances. The latter provide a decrease in the force of muscle contraction, thus relieving pain symptoms and protecting teeth and structures of the temporomandibular joint. There is no scientific evidence that invisible orthodontic aligners can exert some effect on parafunctional muscle activity during sleep, nor on related symptoms [77].

10. Relation to orthodontic treatment

It is relevant to investigate the real relationship between orthodontic treatment and parafunctional habit, since it is not uncommon for an individual to seek correction of malocclusion in order to solve problems caused by bruxism or even to intercept its own mechanism.

On the other hand, in some cases it may develop the habit of grinding or clenching the teeth during or after orthodontic treatment.

What, then, is the association between orthodontic treatment and bruxism? Is orthodontic treatment responsible for TMD signs and symptoms resulting from parafunction? Can orthodontic therapy be considered as a triggering factor of the parafunctional mechanism? How to evaluate, predict and verify the presence of predisposing factors to parafunction in an individual who will undergo orthodontic treatment?

These issues have been raised, both by individuals who will undergo orthodontic treatment and by professionals themselves, because of a growing prevalence of bruxism currently observed.

To evaluate the factors predisposing to the development of parafunction, the orthodontist must have updated knowledge of the whole process of the phenomenon of bruxism. This encompasses from the neurophysiological criteria of the whole mechanism (CNS control) to the problems related to the aetiology and its implications.

The diversity of factors capable of deflagrating, maintaining or aggravating the mechanism of bruxism and its sequelae requires the orthodontist to know and understand the mechanism of this parafunctional habit in order to have success and clarity in clinical behaviour in relation to individuals who have or who develop it before, during or after orthodontic treatment.

A longitudinal study by Knight et al. [8] for a period of 20 years in individuals receiving orthodontic treatment showed that they presented signs of bruxism characterised by wear in the mixed dentition that remained with the same pattern in the permanent dentition. They inferred that this parafunctional habit may represent a persistent characteristic in the childhood and in the adult phase of the same person, thus not having a direct relation between orthodontic treatment and bruxism.

In another study, Egermark et al. [40] investigated the risk of developing bruxism and TMD in a group of 402 subjects, some of whom received orthodontic treatment while others represented the control group. The results reinforced the opinion that orthodontic treatment did not present a high risk of developing TMD and bruxism in the long term when compared to those who did not undergo orthodontic intervention to correct malocclusion.

In contrast, there are reports that bruxism and some signs and symptoms of TMD have decreased during the active phase of orthodontic treatment [78].

A possible explanation for the association between these two entities is based on the understanding of the neurophysiology of the CNS involved in its processes.

According to Okeson [10], the constant change of the dental positions in the orthodontic conduct results in altered peripheral sensorial stimuli causing a decrease of CNS activity. That is, occlusal contacts generate peripheral (outside the CNS) stimuli, which present an inhibitory effect of muscle activity through the nociceptive reflex mechanism. In a different way, bruxism seems to be conditioned to the CNS, whose stimulus has an excitatory effect on the muscles.

Thus, it can be inferred that the muscular activities related to functional (occlusion) and parafunctional (bruxism) stimuli are different, as they result in controlled and voluntary movements and uncontrolled and involuntary movements, respectively. From this reasoning, it can be deduced that changes in dental contacts have little effect on bruxism, that is, as the teeth move in orthodontic treatment,

they produce constant peripheral stimuli that act to inhibit parafunctional activity, which is mediated mainly by the CNS. However, once the orthodontic movements have settled, if the main aetiological factor persists, bruxism may restart [10]. Likewise, the effect of therapy using rigid plaques that are constantly adjusted to control the habit and protect the structures associated with the parafunctional act, such as teeth, neuromuscular structures and TMJ, is understood.

This hypothesis is contradictory in relation to the authors who defended the occlusion and its variations as one of the main triggers of bruxism [33]. On the other hand, it supports the results of studies of other researchers who found a reduction in parafunctional muscle activity after the inclusion of deflective occlusal contacts in a given population sample [79].

At present, there is a concern to investigate the real participation of orthodontic treatment in the development of one of the consequences of bruxism represented by TMD.

Studies have demonstrated that parafunctional habits can act as triggers for TMD, that is, they are conditioned by the contributing factors of these disorders, as revealed by Conti [4] in a cross-sectional evaluation of the relationship between TMD signs and symptoms and orthodontic treatment.

Other authors have claimed that parafunctional habits do not represent consistent factors in the induction of muscle or joint pathologies [80].

In this way, one cannot neglect the fact that the uncertainty caused by the lack of scientific evidence based on subsidies of greater content around the true involvement of orthodontics on the parafunctional mechanism converges to a more prudent conduct in relation to orthodontic procedures in patients with bruxism.

Clinical examination, diagnosis and discernment to know the timing to initiate or continue orthodontic treatment in patients who are included in this context constitute adequate procedures to conduct the orthodontic intervention.

Some professionals postpone the control and treatment of bruxism only after removal of the fixed appliance, even though this may result in excessive tooth wear. Others advocate some type of control for the consequences of bruxism during orthodontic treatment, such as the use of occlusal plaques [81].

In a longitudinal follow-up of a sample composed of 58 TMD patients, of whom 45 were submitted to orthodontic treatment, Imai et al. [82] suggested that TMD-related symptoms did not recur in individuals receiving orthodontic treatment due, probably, to the reestablishment of the functional balance of the masticatory system.

These data are consistent with the idea that a healthy occlusal condition is essential for the entire dynamics of the stomatognathic system. Otherwise, along with other triggering factors, a situation of occlusal disharmony can act as an aggravating factor of the deleterious phenomena originated by bruxism.

From this, studies have shown that the relationship between tooth movement and the aetiological mechanism of parafunctional habits is inconsistent.

Therefore, the researched literature did not provide scientific support for the hypothesis of a direct association between bruxism and orthodontic treatment, which reinforces the need for further research on this subject.

11. Conclusions

This chapter dealt with aspects related to the aetiology of oral parafunction, as well as related neurophysiology, organic structural adaptive capacity, some clinical implications such as the main structures of the stomatognathic system involved consequent to the TMDs, alterations in the craniofacial morphological architecture, some therapeutic conducts and, finally, the association between orthodontic treatment and bruxism.

The aetiology of bruxism is multifactorial. The deleterious events on the cranio-orofacial architecture come from the rupture of the structural limit of the adaptive capacity of the stomatognathic system as a consequence of the oral parafunction, and it is particular of each individual. However, the facial structures that are most affected by bruxism are the mandibular elevating masticatory muscles: masseter and temporal. The therapeutic conducts used to control bruxism should be directed in accordance with the prevalence of each symptomatology, as well as according to the specific aetiology. Finally, orthodontic treatment cannot be considered as an aetiological factor of bruxism, since occlusal interferences are no longer accepted as the main aetiological factor in this occurrence. Following this reasoning, it can be concluded that the performance of orthodontic treatment is not related to the presence of signs and symptoms of temporomandibular disorders (TMD), since the degree of TMD may be associated with the presence of parafunctional habits (such as bruxism and clenching of teeth) and emotional tension.

Thus, knowledge and mastery of signs and symptoms of bruxism, in addition to contributing factors in its aetiology, as well as knowledge about the different therapeutic approaches, become fundamental requirements, within a more current perspective, in the practice of orthodontics.

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References

- [1] Ferreira ABH. Dicionário Aurélio básico da língua portuguesa. São Paulo: Nova Fronteira; 1994-1995. p. 687
- [2] Paiva HJ. Oclusão: Noções e Conceitos Básicos. São Paulo: Santos; 1997
- [3] Leles CR, Melo M. Bruxismo e apertamento dental—Uma conduta clínica racional. *Revista Odontológica do Brasil-Central*. 1995;5(15):22-26
- [4] Conti ACCF. Avaliação transversal da relação entre sinais e sintomas das disfunções têmporo-mandibulares e o tratamento ortodôntico [Dissertação de Mestrado]. Bauru: Faculdade de Odontologia da USP; 2000
- [5] Rodrigues L, Lemos JBD, Tokura M, Luz JGC. Frequência de hábitos parafuncionais e suas manifestações clínicas em pacientes com disfunções da articulação temporomandibular. *Revista de Odontologia da Universidade Cidade de São Paulo*. 2001;13(2):113-123
- [6] Uetanabara R, Mazzetto MO. Bruxism: A current view. *Revista de Odontologia da Universidade Cidade de São Paulo*. 2000;12(2):163-169
- [7] Pavarina AC, Bussadori CMC, Alencar FGP Jr. Aspectos dos hábitos parafuncionais de interesse para o clínico geral. *Journal Brasileiro de Odontologia Clínica*. 1999;3(13):86-90
- [8] Knight DJ, Leroux BG, Zhu C, Almond J, Ramsay DS. A longitudinal study of tooth wear in orthodontically treated patients. *American Journal of Orthodontics and Dentofacial Orthopedics*. 1997;112(2):194-202. DOI: 10.1016/S0889-5406(97)70246-4
- [9] Pinheiro M. Era da Ansiedade. Estado de Minas, Belo Horizonte, Feminino & Masculino; 2002. p. 8
- [10] Okeson JP. Fundamentos de Oclusão e Desordens Têmporo-Mandibulares. Trad. de Milton Edson Miranda. 2nd ed. São Paulo: Artes Médicas; 1992
- [11] Maciel RN, Gomes SS. Oclusão e ATM: Procedimentos Clínicos. 2nd ed. São Paulo: Santos; 1996
- [12] Simon J. Biomechanically-induced dental disease. *General Dentistry*. 2000;48(5):598-605
- [13] Nishigawa K, Bando E, Nakano N. Quantitative study of bite force during sleep association bruxism. *Journal of Oral Rehabilitation*. 2001;28(5):485-491. DOI: 10.1046/j.1365-2842.2001.00692.x
- [14] Kydd WL, Daly C. Duration of nocturnal tooth contacts during bruxing. *The Journal of Prosthetic Dentistry*. 1985;53(5):717-721. DOI: 10.1016/0022-3913(85)90031-9
- [15] Aloé F, Gonçalves LR, Azevedo A, Barbosa RC. Sleep bruxism. *Reviews Neuroscience*. 2003;11(1):4-17
- [16] Bostwick JM, Jaffee MS. Buspirone as an antidote to SSRI-induced bruxism in 4 cases. *The Journal of Clinical Psychiatry*. 1999;60(12):857-860
- [17] Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *Journal of Oral Rehabilitation*. 2001;28(12):1085-1089. DOI: 10.1046/j.1365-2842.2001.00839.x
- [18] Gómez FM, Giralt MT, Sainz B, Arrue A, Prieto M, García-Vallejo P. A possible attenuation of stress-induced increases in striatal dopamine metabolism by the expression of non-functional masticatory in the rat. *European Journal of Oral Sciences*. 1999;107(6):461-467. DOI: 10.1046/j.0909-8836.1999.eos107607.x
- [19] Arman K, Petruninaitė A, Grigalaukienė R. Stress experience

and effect on self-perceived oral health status among high school students. *Stomatologija*. 2016;**18**(3):75-79

[20] Alves AC, Alchieri JC, Barbosa GAS. Bruxism. Masticatory implications and anxiety. *Acta Odontológica Latinoamericana*. 2013;**26**(1):15-22

[21] Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. *Chest*. 2001;**119**(1):53-61. DOI: 10.1378/chest.119.1.53

[22] Alencar Junior FGP. Fatores psicológicos nas disfunções craniomandibulares: estudo da relação entre graus de disfunção e escalas de ansiedade traço-estado [Dissertação de Mestrado]. Bauru: Faculdade de Odontologia da USP; 1997

[23] Raigrodski AJ, Mohamed SE, Gardiner DM. The effect of amitriptyline on pain intensity and perception of stress in bruxers. *Journal of Prosthodontics*. 2001;**10**(2):73-77. DOI: 10.1111/j.1532-849X.2001.00073.x

[24] Seligman DA, Pullinger AG, Solber WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion and TMJ symptomatology. *Journal of Dental Research*. 1988;**67**(10):1323-1333. DOI: 10.1177/00220345880670101601

[25] Baldessarini RJ, Marsh E. Fluoxetine and side effects. *Archives of General Psychiatry*. 1990;**47**(2):191-192. DOI: 10.1001/archpsyc.1990.01810140091015

[26] Possidente E, Nardi AE, Figueira I, Mendlowicz M, Marques C, Versiani M. SSRI-associated bruxism: Case report of 4 patients. *Jornal Brasileiro de Psiquiatria*. 1997;**46**(5):285-288

[27] Macedo CR, Macedo EC, Torloni MR, Silva AB, Prado GF. Pharmacotherapy for sleep bruxism. *Cochrane Database of Systematic*

Reviews. 2014;**23**(10):CD005578. DOI: 10.1002/14651858.CD005578.pub2

[28] Cahlin BJ, Hedner J, Dahlström L. A randomised, open-label, crossover study of the dopamine agonist, pramipexole, in patients with sleep bruxism. *Journal of Sleep Research*. 2017;**26**(1):64-72. DOI: 10.1111/jsr.12440

[29] Ellison JM, Stanziani P. SSRI—Associated nocturnal bruxism in four patients. *The Journal of Clinical Psychiatry*. 1993;**54**(11):432-434

[30] Jaffee MS, Bostwick JM. Buspirone as an antidote to venlafaxine-induced bruxism. *Psychosomatics*. 2000;**41**(6):535-536. DOI: 10.1176/appi.psy.41.6.535

[31] Wise MEJ. Citalopram-induced bruxism. *The British Journal of Psychiatry*. 2001;**178**:182. DOI: 10.1192/bjp.178.2.182

[32] Brown ES, Hong SC. Antidepressant-induced bruxism successfully treated with gabapentin. *Journal of the American Dental Association*. 1999;**130**(10):1467-1469. DOI: 10.14219/jada.archive.1999.0057

[33] Ramfjord SP. Bruxism, a clinical and electromyographic study. *Journal of the American Dental Association*. 1961;**62**:21-44. DOI: 10.14219/jada.archive.1961.0002

[34] Strother EW, Mitchell GE. Bruxism: A review and a case report. *The Journal of Dental Medicine*. 1954;**9**:189

[35] Wigdorowicz-Makowerowa N, Godzki C, Panek H, Maslanka T, Plonka K, Palacha A. Epidemiological study on prevalence and etiology of functional disturbances of the masticatory system. *The Journal of Prosthetic Dentistry*. 1979;**41**(1):76. DOI: 10.1016/0022-3913(79)90361-5

[36] Reimão R, Lefèvre AB. Prevalence of nocturnal bruxism in childhood.

Revista do Hospital das Clínicas
Universidade de São Paulo Hospital das
Clínicas. 1982;**37**(5):228-232

[37] Funato M, Ono Y, Baba K, Kudo Y. Evaluation of the non-functional tooth contact in patients with temporomandibular disorders by using newly developed electronic system. *Journal of Oral Rehabilitation*. 2014;**41**(3):170-176. DOI: 10.1111/joor.12129

[38] Olkinuora M. Bruxism: A review of the literature on and a discussion of studies of bruxism and its psychogenesis and some new psychological hypotheses. *Suomen Hammaslääkäriseuran Toimituksia*. 1969;**65**(6):312-324

[39] Rosara JV, Barbosa TS, Dias IOV, Kobayashi FY, Costa YM, Gavião MBD, et al. Effect of interocclusal appliance on bite force, sleep quality, salivary cortisol levels and signs and symptoms of temporomandibular dysfunction in adults with sleep bruxism. *Archives of Oral Biology*. 2017;**82**:62-70. DOI: 10.1016/j.archoralbio.2017.05.018

[40] Egermark I, Magnusson T, Carlsson GE. A 20-year follow-up of signs and symptoms of temporomandibular disorders and malocclusions in subjects with and without orthodontic treatment in childhood. *The Angle Orthodontist*. 2003;**73**(2):109-115. DOI: 10.1043/0003-3219(2003)73<109:AYFO SA>2.0.CO;2

[41] Clarke NG. Occlusion and myofascial pain dysfunction: Is there a relationship? *Journal of the American Dental Association*. 1982;**104**:443-446. DOI: 10.14219/jada.archive.1982.0233

[42] Vanderas AP, Manetas KJ. Relationship between malocclusion and bruxism in children and adolescents: A review. *Pediatric Dentistry*. 1995;**17**(1):7-12

[43] Dalewski B, Chrusciel-Nogalska M, Frazczak B. Occlusal splint versus modified nociceptive trigeminal inhibition splint in bruxism therapy: A randomized, controlled trial using surface electromyography. *Australian Dental Journal*. 2015;**60**(4):445-454. DOI: 10.1111/adj.12259

[44] Kardachi BJR, Bailey JO, Ash MM. A comparison of biofeedback and occlusal adjustment on bruxism. *Journal of Periodontology*. 1978;**49**:367. DOI: 10.1902/jop.1978.49.7.367

[45] Bailey JO, Rugh JD. Effect of occlusal adjustment on bruxism as monitored by nocturnal EMG recordings. *Journal of Dental Research*. 1980;**59**:317. DOI: 10.1016/0022-3913(81)90207-9

[46] Kuhn M, Türp JC. Risk factors for bruxism: A review of the literature from 2007 to 2016. *Swiss Dental Journal*. 2018;**128**(2):118-124

[47] Shinkai RSA, Santos LM, Silva FA, Santos MN. Prevalence of nocturnal bruxism in 2-11-year-old children. *Revista de Odontologia da Universidade Cidade de São Paulo*. 1998;**12**(1):29-37. DOI: 10.1590/S0103-06631998000100006

[48] Carra MC, Macaluso GM, Rompré PH, Huynh N, Parrino L, Terzano MG, et al. Clonidine has a paradoxical effect on cyclic arousal and sleep bruxism during NREM sleep. *Sleep*. 2010;**33**(12):1711-1716. DOI: 10.1093/sleep/33.12.1711

[49] Satoh T, Harada Y. Electrophysiological study on tooth-grinding during sleep. *Electroencephalography and Clinical Neurophysiology*. 1973;**35**(3):267-275. DOI: 10.1016/0013-4694(73)90238-1

[50] Lavigne GJ, Rompré PH, Guitard F, Sessle BJ, Kato T, Montplaisir JY. Lower number of K-complexes and K-alphas in sleep bruxism: A controlled quantitative

study. *Clinical Neurophysiology*. 2002;**113**(5):686-693. DOI: 10.1016/S1388-2457(02)00037-8

[51] Silva SR. Bruxismo. *Revista da Associação Paulista de Cirurgiões Dentistas*. 2003;**57**(6):409-417

[52] Abe K, Shimakawa M. Genetic and developmental aspects of sleep talking and teeth-grinding. *Acta Paedopsychiatrica*. 1966;**33**(11):339-344

[53] Lindqvist B. Bruxism in twins. *Acta Odontologica Scandinavica*. 1974;**32**(3):177-187. DOI: 10.3109/00016357409002546

[54] Kato T, Thie NMR, Montplaisir JY, Lavigne GJ. Bruxism and orofacial movements during sleep. *Dental Clinics of North America*. 2001;**45**(4):657-684

[55] Madrid G, Madrid S, Vranesh JG, Hicks RA. Cigarette smoking and bruxism. *Perceptual and Motor Skills*. 1998;**87**(3 Pt 1):898. DOI: 10.2466/pms.1998.87.3.898

[56] Lavigne GJ, Lobbezbo F, Rompré PH, Nielsen TA, Montplaisir JY. Cigarette smoking as a risk or exacerbating factor for restless legs syndrome and sleep bruxism. *Sleep*. 1997;**20**(4):290-293. DOI: 10.1093/sleep/20.4.290

[57] Hartman E. Bruxism. In: Kryger MH, Roth T, Dement WC, editors. *Principles and Practice of Sleep Medicine*. 2nd ed. Philadelphia: W. B. Saunders; 1994. pp. 598-601

[58] Alharby A, Alzayer H, Almahlawi A, Alrashidi Y, Azhar S, Sheikho M, et al. Parafunctional behaviors and its effect on dental bridges. *Journal of Clinical Medical Research*. 2018;**10**(2):73-76. DOI: 10.14740/jocmr3304w

[59] Kawakami S, Kumazaki Y, Manda Y, Oki K, Minagi S. Specific diurnal EMG activity pattern observed in occlusal collapse patients: Relationship between diurnal bruxism and tooth loss

progression. *PLoS One*. 2014;**9**(7):e101882. DOI: 10.1371/journal.pone.0101882

[60] McCoy G. Dental compression syndrome: A new look at an old disease. *The Journal of Oral Implantology*. 1999;**25**(1):35-49. DOI: 10.1563/1548-1336(1999)025<0035:DCS ANL>2.3.CO;2

[61] Kampe T, Tagdae T, Bader G, Edman G, Karlsson S. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. *Journal of Oral Rehabilitation*. 1997;**24**(8):581-587. DOI: 10.1111/j.1365-2842.1997.tb00377.x

[62] Fernandes RSM, Mazzetto MO. Achados clínicos que caracterizam o bruxismo em pacientes com queixas de desordens crânio-mandibulares. In: *Anais*. Vol. 21. Ribeirão Preto: FORP-USP; 1999. p. 52

[63] Muzyka BC. Sleep bruxism—New findings. *Practical Procedures & Aesthetic Dentistry*. 2001;**13**(3):190

[64] Arita CA, Carvalho PCL, Silva MAMR, Bataglioni C, Chaguri NA, Nunes LJ. Alterações provocadas pelo bruxismo. *RGO*. 1990;**38**(4):257-261

[65] Garip H, Tufekcioglu S, Kaya E. Changes in the temporomandibular joint disc and temporal and masseter muscles secondary to bruxism in Turkish patients. *Saudi Medical Journal*. 2018;**39**(1):81-85. DOI: 10.15537/smj.2018.1.20873

[66] Asutay F, Atalay Y, Asutay H, Acar AH. The evaluation of the clinical effects of botulinum toxin on nocturnal bruxism. *Pain Research & Management*. 2017;**2017**:6264146. DOI: 10.1155/2017/6264146

[67] Lund JP et al. *Dor Orofacial: Da Ciência Básica à Conduta Clínica*. São Paulo: Quintessence; 2010. p. 300

[68] Menapace SE, Rinchuse DJ, Zullo T, Pierce CJ, Shnorhokian H. The

dentofacial morphology of bruxers versus non-bruxers. *The Angle Orthodontist*. 1994;**64**(1):43-52. DOI: 10.1043/0003-3219(1994)064<0043:TDMOBV>2.0.CO;2

[69] Broberg K, Lindskog-Stokland B, Mejersjö C. Anterior bite opening in adulthood. *The Open Dentistry Journal*. 2017;**11**:628-635. DOI: 10.2174/1874210601711010628

[70] Young DV, Rinchuse DJ, Pierce CJ, Zullo T. The craniofacial morphology of bruxers versus nonbruxers. *The Angle Orthodontist*. 1999;**69**(1):14-18. DOI: 10.1043/0003-3219(1999)069<0014:TCMOBV>2.3.CO;2

[71] Waltimo A, Nyström M, Könönen M. Bite force and dentofacial morphology in men with severe dental attrition. *Scandinavian Journal of Dental Research*. 1994;**102**(2):92. DOI: 10.1111/j.1600-0722.1994.tb01161.x

[72] Crothers A, Sandham A. Vertical height differences in subjects with severe dental wear. *European Journal of Orthodontics*. 1993;**15**(6):519-525. DOI: 10.1093/ejo/15.6.519

[73] Clifford T, Lamey PJ, Fartash L. Mandibular tori, migraine and temporomandibular disorders. *British Dental Journal*. 1996;**180**(10):382-384. DOI: 10.1038/sj.bdj.4809094

[74] Jadhao VA, Lokhande N, Habbu SG, Sewane S, Dongare S, Goyal N. Efficacy of botulinum toxin in treating mofascial pain and occlusal force characteristics of masticatory muscles in bruxism. *Indian Journal of Dental Research*; **2017****28**(5):493-497. DOI: 10.4103/ijdr.IJDR_125_17

[75] Salgueiro MDCC, Bortoletto CC, Horliana ACR, Mota ACC, Motta LJ, Motta PB, et al. Evaluation of muscle activity, bite force and salivary cortisol in children with bruxism before and after low level laser applied to acupoints: Study protocol for a

randomised controlled trial. *BMC Complementary and Alternative Medicine*. 2017;**17**(1):391. DOI: 10.1186/s12906-017-1905-y

[76] Kesikburun S, Alaca R, Aras B, Tuğcu I, Tan AK. Botulinum toxin injection for bruxism associated with brain injury: Case report. *Journal of Rehabilitation Research and Development*. 2014;**51**(4):661-664. DOI: 10.1682/JRRD.2013.10.0218

[77] Manfredini D, Lombardo L, Vigiani L, Arreghini A, Siciliani G. Effects of invisible orthodontic retainers on masticatory muscles activity during sleep: A controlled trial. *Progress in Orthodontics*. 2018;**19**(1):24. DOI: 10.1186/s40510-018-0228-y

[78] Egermark I, Rönnerman A. Temporomandibular disorders in the active phase of orthodontic treatment. *Journal of Oral Rehabilitation*. 1995;**22**(8):613-618. DOI: 10.1111/j.1365-2842.1995.tb01058.x

[79] Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. *The Journal of Prosthetic Dentistry*. 1984;**51**(4):548-553. DOI: 10.1016/0022-3913(84)90312-3

[80] Barne A, Sbordone L, Ramaglia L. Craniomandibular disorders and orthodontic treatment need in children. *Journal of Oral Rehabilitation*. 1997;**24**(1):2-7. DOI: 10.1111/j.1365-2842.1997.tb00252.x

[81] Sullivan TC. A new occlusal splint for treating bruxism and TMD during orthodontic therapy. *Journal of Clinical Orthodontics*. 2001;**35**(3):142-144

[82] Imai T, Okamoto T, Kaneko T, Umeda K, Yamamoto T, Nakamura S. Long-term follow-up of clinical symptoms in TMD patients who underwent occlusal reconstruction by orthodontic treatment. *European Journal of Orthodontics*. 2000;**22**(1):61-67