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# Common therapeutic approaches in sleep and awake bruxism — an overview

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## ABSTRACT

Bruxism, a common medical condition characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible, can occur during sleep, when it is known as sleep bruxism (SB), or during wakefulness, when it is known as awake bruxism (AB).

Although bruxism often causes headaches, temporomandibular joint pain, masticatory muscle pain, mechanical tooth wear, prosthodontic complications and cracked teeth, there is still not enough data to define and support a standardised approach to its treatment.

The aim of this review was to present the pathophysiology, consequences, types and treatment methods of bruxism in order to increase readers' knowledge of this topic. Differences between awake and nocturnal bruxism are included, as well as risk factors and indicators visible during the clinical examination of affected patients. Among the causes we consider are genetics, stress, oral parafunctions and changes in the Central Nervous System (CNS). Potential and common methods of treatment are presented, along with suggested guidelines that should be followed when determining an appropriate treatment method. We draw attention to the notably dynamic development of bruxism in today's society and the importance of informational and preventive projects, especially those targeted at high-risk patients as well as those targeted at specialists, in order to better tackle the bruxism 'epidemic'.

**Key words:** bruxism, temporomandibular disorder (TMD), chewing muscle pain, tension headaches

## Introduction

For years, bruxism has been considered to be a synonym for teeth grinding and it was unclear whether it was a pathological behaviour or a disorder. In 2018, a consensus was reached, defining bruxism as a repetitive activity in the masticatory muscles characterised by clenching or grinding of the teeth and/or stiffening or thrusting of the mandible causing forceful contact between the biting surfaces of maxillary and mandibular teeth [1].

It can occur during sleep (SB, sleep bruxism, nocturnal bruxism) or while awake (AB, awake bruxism, diurnal bruxism) [2]. SB is the masticatory muscles activity happening during sleep, especially in the N1 sleep phase [3], including brief daytime naps, and can be rhythmic (phased) or non-rhythmic (tonic). This is neither a motor disorder nor a sleep disorder encountered in the healthy population. SB is associated with headache upon awakening [4], and some studies have pointed to its slight effect on insomnia [5]. AB is described as an activity of the masticatory muscles during wakefulness characterised by repeated or prolonged contact of the opposing teeth and/or stiffening or thrusting of the mandible, and is not a motor disorder seen in otherwise healthy populations [1].

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Although it has never been proven that bruxism affects quality of life [6, 7], there are patients seeking medical help daily from dentists around the world. It is a behaviour affecting 8–31.4% [2, 8] of the population, and is more usually encountered in people under the age of 40 and in females [9].

## Aetiology

Researchers have suggested that there may be some degree of inherited susceptibility towards SB symptoms progression. It has been reported that 21–50% of people with SB have a close family member who had SB in childhood, suggesting the involvement of genetic factors [10, 11].

With that in mind, Rintakoski et al. [12] tried looking for any genetic or environmental factors that may play a role in the phenotypic variance of bruxism. They demonstrated that there is a sex-independent genetic component that occurs more frequently in phenotypic SB variations than in other TMD. Based on that, they reported the heredity of bruxism, although they emphasised that the emancipation is dependent on age and environment, and so for example the older a pair of twins, the greater the differences that are observed between them. Thus, although the genetic factor has been found, the entire mechanism behind it remains unknown. Similarly, Maciejewska-Szaniec et al. have reported that polymorphic variants in genes related to stress coping are associated with AB, although they cautioned that their research should serve only as a spur to conduct further analyses on a bigger sample group, in order to draw truly objective conclusions [13].

On the other hand, one of the most commonly used explanations of causes of bruxism is stress [5, 14, 15]. We can clearly observe an increasing tendency of patients in the COVID-19 pandemic period to look at articles on the topic "teeth grinding at night" on the internet [16, 17]. Increased stress during the pandemic resulted in an elevated number of bruxism episodes in society. That, combined with more difficult access to doctors in the same period resulted in people looking for explanations for their symptoms on the internet, leading to growing public awareness [18, 19]. Recently, researchers have focused more on younger people such as college students, noting a significant increase in the occurrence of bruxism in this studied group [20].

This serves to highlight the presence of a correlation between age and incidents of bruxism as epidemiologically variable, which indicates the need for local research to isolate vulnerable groups and implement appropriate preventive measures.

## Pathophysiology

Bodily functions disturbed by bruxism can include, among other things, increased activity of the masticatory muscles, masticatory muscle hypertrophy (especially of the masseter), scalloped or burning tongue, linea alba on the cheeks along the bite line, tooth tissue damage (enamel cracks, abfractions, excessive tooth wear beyond what is expected with age) [9, 21, 22], repeated damage to prosthetic restorations, disturbances of the amount and composition of secreted saliva, an often severe manifestation of craniofacial pain, stiffness of the TMJ [23, 24], gingival fluid secretion increase [25], bone exostoses, and periodontal leases (increased tooth mobility, gingival recessions) [21–23, 26, 27].

Yet, in most cases, the origin of symptoms is unknown. The vast majority of researchers agree that the direct trigger of bruxism symptoms comes from the central nervous system (CNS) [28]. It has been proved in metanalysis that bruxism seems to be associated with distinct differences in the neural pathways related to the control of the jaw-closing muscles [29]. Disturbances in the concentration of catecholamines, especially dopamine, which affect the mandibular motor dysfunctions, are thought to be of great importance in the mechanism of bruxism [30].

The first evidence of such a correlation came from a case report in which a patient suffering from Parkinson's syndrome was treated for grinding his teeth with L-3,4-dihydroxyphenylalanine (L-DOPA), a dopamine precursor. In a series of controlled studies in young, healthy SB patients, L-DOPA has been shown to cause a slight but noticeable reduction in episodes of SB compared to a placebo group, while bromocriptine, a dopamine antagonist, has been shown not to affect expected rhythmic activity in the masticatory muscles [31, 32]. Given the presumed role of norepinephrine in bruxism, experimental trials have been conducted with propranolol and clonidine [33, 34]. Propranolol, a non-selective beta-blocker, has not caused a significant reduction in SB, but clonidine, an alpha-agonist acting on the CNS, has significantly reduced SB score when compared to a placebo. This effect was partially related to the concomitant reduction in cardiovascular sympathetic dominance preceding the rhythmic activity of the masticatory muscles. Simultaneously, fluctuations in the level of dopamine and disorders of its receptors in the brain are often associated with chronic stress [14, 15, 35]. The pathophysiological mechanism in which stress affects the occurrence of bruxism has been explained by evidence that individuals with increased levels of neuroticism and anxiety disorders tend to release their emotional tension by engaging in activities related to bruxism [36]. Increased concentration of stress hormones can also be noted in many chronic diseases. Psychosocial factors such as state anxiety and trait anxiety as well as alexithymia are also among the reported causes of the occurrence and maintenance of AB [14].

Bruxism-related TMD can be explained by the craniofacial anatomy. As there is a fixed relationship between the maxillary teeth and the base of the skull, and at the same time the mandibular teeth have a set position in relation to the TMJ, this means that misaligned contact of the upper and lower occlusal surfaces may have a direct impact on the position and motor function of the mandible head in the TMJ [26]. In acute and subacute TMD, significantly greater weakening and thinning of the mandibular neck ultrastructure have been observed, along

with more disorders than in an asymptomatic control group. It has been shown that mandibular neck weakness and craniofacial symptoms are more common in women with bruxism than in a control group [8]. Clear indications of bruxism have also been observed in children with asthma and sleep apnoea [37, 38]. Moreover, there are scientific reports indicating the possibility of diagnosing psychological abuse at an early age, based on the severity of bruxism in children. This subject certainly requires further research, as alternative methods of diagnosing various types of violence are exceptionally important for the mental health of the very young [39, 40]. It is also possible that drugs such as dopamine antagonists, dopamine agonists, tricyclic antidepressants, and selective serotonin inhibitors, as well as alcohol, cocaine and amphetamines, can contribute to the progression of bruxism symptoms. Studies have shown that reducing the dose clearly reduces the symptoms [20, 22, 41].

Dystonias and other muscular problems, as well as developmental disorders including various types of autism and neurological diseases that may have environmental and traumatic causes, can further be exemplified as bruxism-related conditions [22].

### Diagnosis

Due to the variety of symptoms and the overlaps with other conditions, a bruxism diagnosis requires careful evaluation that includes questionnaires, records of past bruxism episodes, and thorough examination. To confirm the diagnosis of bruxism, there are two different methods: a non-instrumental method based on the patient's description of symptoms, questionnaires, medical history, and clinical examinations [21, 25, 42–44]; and an instrumental method using electromyography (EMG) and polysomnography, showing muscle activity during sleep and the lack of associated epileptic activity [1].

EMG recording of masticatory muscle activity is considered to be an objective criterion and together with polysomnography should be considered the gold standard in SB diagnosis [1, 2, 9, 45]. Interestingly, in 3.75% of patients with bruxism, EMG has shown epilepsy activity, and in a polysomnography study, a higher percentage of patients woke up after falling asleep than in the control group [46]. Standardised diagnostic criteria for temporomandibular joint dysfunction (DC/TMD) are also helpful in setting a diagnosis [1, 2, 47]. The minimum diagnostic criteria for SB, according to the International Classification of Sleep Disorders, Revised (ICSD-R), include symptoms of clenching and grinding of the teeth while sleeping, and at least one additional symptom such as excessive tooth wear, tooth grinding heard by other household members, or excessive tension in the facial and neck muscles [26].

Every dentist should be able to distinguish between the tooth wear caused by bruxism and that caused by malocclusion, especially because evaluation scales helpful in reliable assessment are widely available [21]. There are three different levels of probability that a patient suffers from bruxism, depending on the following diagnostic media. The diagnosis basing on the symptoms reported by the patient as "positive self-report" indicates "possible bruxism", while "probable bruxism" is based on clinical examinations with/without a positive patient interview, and "certain bruxism" is determined by instrumental tests with/without a positive history and/or a positive clinical examination [1, 47].

- In relation to the TMD diagnostic criteria, bruxism is determined using Axis II and the Oral Behaviour Checklist (OBC). Axis II is related to an assessment of the psychological and physical dimension of pain, including pain intensity, pain-related disability, and the presence of depression and non-specific symptoms, based on the patient's opinion [47]. An awake bruxism diagnosis lacks objective tests, as it is mostly based on medical interview consisting of a detailed history of tics and parafunction and visual observation of the patient's behaviour [48].
- An easy way to initially differentiate between sleep and awake bruxism symptoms is to observe the severity of them during the day: symptoms gradually decrease as the day progresses in SB, in contrast to awake bruxism symptoms, which gradually increase throughout the day [49].

## Therapy for bruxism

The treatment of bruxism may include various therapy options, e.g.:

- educational therapy for a patient about harmful habits;
- biofeedback;
- muscle relaxation exercises;
- occlusal splint therapy;
- short-term medications;
- botulinum toxin injections;
- psychotherapy (stress reduction, lifestyle change, hypnosis);
- electric method;
- correction of short-circuit disturbances.

### Educational therapy

The purpose of this is to make the patient aware of the existence of parafunction and its harmfulness, as well as to interrupt pathological reactions. The patient is recommended to conduct regular physical activity, maintain muscle and joint balance (correct posture), to avoid gum chewing, to not clench their teeth, and to maintain correct nasal breathing with the correct position of the tongue.

He/she is also advised to use physical self-regulatory techniques. The goal of this method is to produce physiological changes that will reduce pain, fatigue and physical overload [50]. However, only a small number of well-designed studies into the effectiveness of Cognitive Behavioural Therapy (CBT) are available at the moment, meaning that further exploration is needed in order to unequivocally state that CBT has a positive effect in SB management [51].

## Biofeedback therapy

This technique is based on the premise that patients with bruxism can consciously 'unlearn' their pathological behaviour [52]. It uses positive feedback as a teaching strategy focused on lowering patients' stress levers, potentially ensuing in long-term behavioural changes aiming at the reduction or elimination of symptoms.

This is, however, a controversial method of remedying bruxism, as studies have shown that significant results have been achieved with AB but not yet with SB [1, 45, 53]. Furthermore, proper sleep hygiene (i.e. relaxation before bedtime, caffeine reduction, etc.) is recommended for SB control, although the therapeutic effect has not been proven [54]. Mobile phone applications such as BruxApp are available to download and may be helpful in tracking occurrences of AB as well as control attempts. They should make patients aware of the scale of the problem along with helping to cut down the number of episodes of AB [55–57]. The effectiveness of biofeedback therapy in studies is in general highly rated, but the patient often needs to carry a portable device that's either continuously monitoring muscle tension or frequently reminds the carrier to consciously relax their masseters throughout the day [51].

## Muscle relaxation and posture exercises

The proper mandibular resting position is as follows: lips together, teeth having contact only when swallowing. To balance the overactivity of the masticatory muscles and stretch them, muscle relaxation exercises are recommended. These exercises are also aimed at teaching the patient the correct resting position of the tongue, the correct path of nasal breathing, and how to avoid parafunction. For example, myorelaxation exercises have been used with good results in our clinic. A single cycle of relaxation exercises includes five steps:

I. Setting the mandible in a relaxed position
II. Protrusive movement to reach 'tete-a-tete' teeth contact
III. Opening mouth
IV. Closing mouth until tete-a-tete contact
V. Returning jaw to starting position.

Exercises are recommended three times a day, with 20 repetitions each time in a half-sitting position with supported head, straight and uncrossed legs. Patients should exercise daily for a month, freely but without force, roughly at the speed of their heartbeat [52].

## Occlusal splint therapy

To prevent negative consequences of SB, stabilising and repositioning splints can be used [58–60]. These splints eliminate occlusal obstacles, relax the chewing and neck muscles by passive stretching, improve the occlusive and neuromuscular stabilisation, and reposition the mandibular heads and articular discs.

Gomes at el. [61] showed that the use of splint therapy in the treatment of SB improves quality of life. They showed that the use of occlusal splints creates a biomechanical balance between the physiological load and that generated by stress. Occlusal splints can lead to the stabilisation of bruxism by reducing deformities and deviations in the temporomandibular joint, reducing the load on the joint [62]. Different types of splints vary slightly in their effectiveness, but due to the low level of commitment needed from the patient, the relatively low price, and noticeable pain reduction, they are often the chosen option. We must underline nevertheless that most splints only mechanically minimise tooth destruction, and so do not affect bruxism itself. Additionally, in the case of SB, they have to be worn at night, which for some patients is a significant inconvenience [51].

## Short-term medications

Analgesics, sedatives, anxiolytics, antidepressants, and muscle relaxants can be used in the pharmacotherapy of bruxism [63], whereby the NSAIDs are recommended to use for pain relief [64]. In order to reduce the increased skeletal muscle tone, patients are administered tolperisone (e.g. mydocalm 50 mg 3 x 1). For anxiolytic and sedative treatment, most often 25 mg of hydroxyzine 1 hour before bedtime is prescribed [26]. However, recent studies have shown that there is insufficient evidence-based data to draw definite conclusions concerning medications attenuating SB and/or AB [65].

## Botulinum toxin

Botulinum toxin (BTX) has been widely used both in the treatment of various diseases and in aesthetic medicine. It is a strong neurotoxin — an exotoxin produced by strictly anaerobic bacteria (bacilli called *Clostridium botulinum*, and a few other representatives of the genus *Clostridium*). Type A botulinum toxin (BTX-A) is the most potent toxin known [66]. It binds permanently to the neuromuscular plate, and paralyses neuromuscular conduction by fragmenting the SNAP-25 protein (synaptosomal protein) necessary in the process of acetylcholine release from presynaptic terminals. After intramuscular injection, there is initially rapid binding to specific, high affinity cell surface receptors for the toxin, followed by the toxin being transported across the cell membrane by receptor-mediated endocytosis, and eventually the toxin is released into the cytosol. This process is accompanied by a progressive inhibition of acetylcholine release. The results begin to be noticeable after 2–3 days; the maximum effect occurs around 2–6 weeks after the injection and lasts 2-4 months. BTX-A indirectly reduces the bite force (i.e. the force generated by the masticatory muscles measured at the contact point of antagonist teeth during biting) by 20–40% in the masseter muscle [67–70]. Therapeutically, very low doses are used, resulting in the effect of botulinum toxin being limited to the area of administration.

Recently, there has been an increase in the number of cases treated with botulinum toxin type A [71–74]. Alonso-Navarro et al. [75] found BTX-A to be a safe and effective treatment for patients with severe bruxism. They administered BTX-A into

both the masseter and temporal muscles, using a final dose of 25–40 IU per muscle. The treatment effect lasted between 13 and 26 weeks. Fernandez-Nunez et al. [76] reported that an injection of BTX-A can reduce the frequency of bruxism episodes, lower the level of pain, and weaken the bite strength.

Kwon et al. [69,70] have recommended the administration of BTX-A at a dose of 25–30 BU in the masseter per side, at three injection sites, and in the temporalis muscle recommended a dose of 15–20 BU per side at 2–3 sites. It is advised to aspirate each time in order to avoid intravenous administration. Jost [77] recommended in his book a dose of 50–100 U in the masseter per side at three injection sites, and in the temporalis muscle a dose of 50–100 U per side at 3–4 sites as well. Monroy et al. [78] have stated that treatment of bruxism with botulinum toxin can cause dysphagia, slight pain at the injection site, and temporary excessive salivation, but that these symptoms appeared only in patients who received a dose above 100 IU or who had other systemic diseases present. It is believed that doses of below 100 IU of BTX-A administered to the masseter and temporalis muscles are safe, and that their use is practicable in day-to-day clinical practice. Although BTX-A is a very good agent in the therapy of bruxism, whether the events of bruxism would recur or rebound after botulinum toxin injection needs more follow-up clinical evidence [79]. Furthermore, Yurttutan et al. have reported that relaxation splints may not be needed in patients treated with BTX-A [73].

### Psychological care

Psychological factors vastly influence the aetiology and treatment of bruxism [14, 36, 39, 80–82]. Zhang et al. [26] showed that the maximum bite force decreased in an experimental group (administered with BTX-A) and in control and placebo groups as well. The lack of differences between the control group and the placebo group may indicate a substantial role of psychological intervention in the treatment of bruxism. Psychological counselling, self-suggestion, hippotherapy or psychotherapy, relaxation techniques and stress management are all endorsed in the treatment of bruxism.

The most recent reports have emphasised the importance of proper sleep hygiene, additionally recommending meditating or listening to relaxing music while falling asleep [83].

### Biostimulation

Electro-galvanic stimulation relaxing the muscles is currently used as a therapy for bruxism. Despite its simplicity, this is not a widespread method, and there are still only a few publications regarding its efficiency [84]. Another inexpensive new method is photobiomodulation with light-emitting diode (LED) [85], which has been used, with very promising results, in the treatment of fibromyalgia [86]. Despite little research, we can already find optimistic results reporting biostimulation to have a magnifying effect on other forms of treatment, for example in patients with splints having their trigger points irradiated [87].

### Correction of short-circuit disturbances

In some cases, correction of malocclusion is sufficient. Improvements are achieved by integrated dental work including orthodontic, prosthetic and conservative treatment. This might be explained by the fact that all occlusal obstacles recognised by the stomatognathic system will result in pulp inflammation and, in the longer term, remodelling of first the alveolus, and later the entire temporomandibular joint, which will burden the masticatory muscles. Obtaining evenly distributed occlusal forces on the surfaces of the teeth will allow harmonious work of the muscles and, as the outcome, their relaxation [57, 88, 89]. However, opinions remain divided. Some scientists consider the above claims to be true, while others highlight the fact that in order to properly remodel patients' occlusion, a dentist has to interfere, even minimally, with already worn teeth. Both parties agree though that the problem does not start with the teeth, but rather with the nerves and muscles [49].

Future bruxism research should focus on a better understanding of the pathophysiology of the condition and the impact of its negative and positive clinical factors. Nevertheless, establishing reliable guidelines for a correct diagnosis will be challenging. A '4A' principle has been suggested: accurate, applicable, affordable and accessible [1]. Additionally, an irrefutable relationship between bruxism, TMD and neck pain is still poorly understood, and calls for further analysis [1, 90].

And beyond that, the current definition of SB seems to ignore the fact that the evidence is insufficient and inconclusive in determining how the condition alters sleep quality and how it is related to other sleep disorders. Therefore, changing the definition status without further exploration of these aspects is simply wrong [91].

## Clinical implications/bruxism as potential manifestation of neurological disorder

TMD caused by bruxism may manifest as otalgia [92]. Based on self-reports, bruxism may also be accompanied by sleep problems and their awake consequences such as: difficulties initiating sleep (DIS), disrupted sleep (DS), early morning awakenings (EMA), non-restorative sleep (NRS), tiredness, and sleep deprivation (SLD) [93]. An association between self-reported bruxism and increased nightmares has also been found [94].

All of the above points to the conclusion that bruxism might be a symptom of some neurological disorders. Among these, we can mention obstructive sleep apnoea (OSA) (33.3–53.7%); somnologists have been encouraged to consider SB as a comorbid condition with OSA [95]. Lavigne et al. [96] have pointed to the frequent coexistence of SB and Restless Leg Syndrome (RLS); they report, based on a questionnaire, a prevalence of SB of 17.3% of adults with RLS, and of 14.5% of those reporting unpleasant leg sensations at night. 73.7% of patients with sleep-related gastroesophageal reflux disease have been diagnosed with probable SB (by self-report and clinical inspection) [97].

There are also reports showing a correlation between SB, REM behavioural disorder and Parkinson's Disease, although a full explanation is lacking [95]. In medical interview analysis of patients with epilepsy, 23.7% of them reported SB, compared to 5.4% of individuals from the control group [95]. In one article [97] a report of the occurrence of possible SB in patients with nightmares was stated, and furthermore in another questionnaire study [98] the prevalence of SB in sleepwalkers and sleeptalkers was reported to be higher than in the control group.

Symptoms such as headache, pain and stiffness in the muscles and joints of the head and neck, difficulty in opening and closing the mouth, and tinnitus, are thought to be associated with SB, while AB is associated with joint sounds and depressive personality disorder [2, 8, 9, 22].

The above consequences of bruxism can be divided into three groups:

- negative, e.g. chewing muscle pain, pathological tooth wear, prosthetic complications [41, 50, 99, 100]
- positive, e.g. termination of an episode of sleep apnoea, reducing risk of chemical clash by increased salivation in patients with gastro-oesophageal reflux disease [1, 25]
- neutral, where bruxism is seen as a harmless habit that does not affect masticatory system [1].

## Takeaway message

As bruxism is multi-factorial and complex, an interdisciplinary approach in diagnosis and treatment is highly recommended. Although easier access to a wider range of information has increased the awareness of patients suffering from bruxism [17], they often only start to seek medical advice when the symptoms are already advanced. Thus, the importance of awareness campaigns and the encouragement of earlier check-ups must be underlined [80, 98].

An equally important aspect is broadening medical professionals' knowledge of bruxism, which will help earlier diagnosis and earlier adoption of appropriate treatment. This whole topic is a very complex one, and deserves further multidimensional research.

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## References

1. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: report of a work in progress. J Oral Rehabil. 2018; 45(11): 837–844, doi: 10.1111/joor.12663, indexed in Pubmed: 29926505.
2. Manfredini D, Winocur E, Guarda-Nardini L, et al. Epidemiology of bruxism in adults: a systematic review of the literature. J Orofac Pain. 2013; 27(2): 99–110, doi: 10.11607/jop.921, indexed in Pubmed: 23630682.
3. Toyota R, Fukui KI, Kamimura M, et al. Sleep stage-dependent changes in tonic masseter and cortical activities in young subjects with primary sleep bruxism. Sleep. 2022; 45(4): zsab207, doi: 10.1093/sleep/zsab207, indexed in Pubmed: 34383078.
4. Vieira KRM, Folchini CM, Heyde MD, et al. Wake-up headache is associated with sleep bruxism. Headache. 2020; 60(5): 974–980, doi: 10.1111/head.13816, indexed in Pubmed: 32323305.
5. Chattrattrai T, Blanken TF, Lobbezoo F, et al. A network analysis of self-reported sleep bruxism in the Netherlands sleep registry: its associations with insomnia and several demographic, psychological, and life-style factors. Sleep Med. 2022; 93: 63–70, doi: 10.1016/j.sleep.2022.03.018, indexed in Pubmed: 35429746.
6. Castelo P, Barbosa T, Gavião M. Quality of life evaluation of children with sleep bruxism. BMC Oral Health. 2010; 10: 16, doi: 10.1186/1472-6831-10-16, indexed in Pubmed: 20546581.
7. Machado NA, Costa YM, Quevedo HM, et al. The association of self-reported awake bruxism with anxiety, depression, pain threshold at pressure, pain vigilance, and quality of life in patients undergoing orthodontic treatment. J Appl Oral Sci. 2020; 28: e20190407, doi: 10.1590/1678-2019-0407, indexed in Pubmed: 32236355.
8. Piekartz Hv, Rösner C, Batz A, et al. Bruxism, temporomandibular dysfunction and cervical impairments in females - Results from an observational study. Musculoskelet Sci Pract. 2020; 45: 102073, doi: 10.1016/j.msksp.2019.102073, indexed in Pubmed: 31678819.
9. van Selms MKA, Visscher CM, Naeije M, et al. Bruxism and associated factors among Dutch adolescents. Community Dent Oral Epidemiol. 2013; 41(4): 353–363, doi: 10.1111/cdoe.12017, indexed in Pubmed: 23121154.
10. Caivano T, Felipe-Spada N, Roldán-Cubero J, et al. Influence of genetics and biopsychosocial aspects as etiologic factors of bruxism. Cranio. 2021; 39(3): 183–185, doi: 10.1080/08869634.2021.1904181, indexed in Pubmed: 33853510.
11. Heyat MdB, Akhtar F, Khan MH, et al. Detection, treatment planning, and genetic predisposition of bruxism: a systematic mapping process and network visualization technique. CNS Neurol Disord Drug Targets. 2021; 20(8): 755–775, doi: 10.2174/1871527319666201110124954, indexed in Pubmed: 33172381.
12. Rintakoski K, Hublin C, Lobbezoo F, et al. Genetic factors account for half of the phenotypic variance in liability to sleep-related bruxism in young adults: a nationwide Finnish twin cohort study. Twin Res Hum Genet. 2012; 15(6): 714–719, doi: 10.1017/thg.2012.54, indexed in Pubmed: 22953759.
13. Maciejewska-Szaniec Z, Kaczmarek-Ryś M, Hryhorowicz S, et al. Polymorphic variants in genes related to stress coping are associated with the awake bruxism. BMC Oral Health. 2021; 21(1): 496, doi: 10.1186/s12903-021-01844-1, indexed in Pubmed: 34610834.
14. Przystańska A, Jasielska A, Ziarko M, et al. Psychosocial predictors of bruxism. Biomed Res Int. 2019; 2019: 2069716, doi: 10.1155/2019/2069716, indexed in Pubmed: 31737656.
15. Dahoun T, Nour MM, McCutcheon RA, et al. The relationship between childhood trauma, dopamine release and dexamphetamine-induced positive psychotic symptoms: a [C]-(+)-PHNO PET study. Transl Psychiatry. 2019; 9(1): 287, doi: 10.1038/s41398-019-0627-y, indexed in Pubmed: 31712556.
16. Almeida-Leite CM, Stuginski-Barbosa J, Conti PC. How psychosocial and economic impacts of COVID-19 pandemic can interfere on bruxism and temporomandibular disorders? J Appl Oral Sci. 2020; 28: e20200263, doi: 10.1590/1678-7757-2020-0263, indexed in Pubmed: 32401942.
17. Kardeş E, Kardeş S. Google searches for bruxism, teeth grinding, and teeth clenching during the COVID-19 pandemic. J Orofac Orthop.

2022; 83(6): 1–6, doi: 10.1007/s00056-021-00315-0, indexed in Pubmed: 34185102.
18. Wetselaar P, Vermaire EJH, Lobbezoo F, et al. The prevalence of awake bruxism and sleep bruxism in the Dutch adult population. J Oral Rehabil. 2019; 46(7): 617–623, doi: 10.1111/joor.12787, indexed in Pubmed: 30830687.
19. Winocur-Arias O, Winocur E, Shalev-Antsel T, et al. Painful temporomandibular disorders, bruxism and oral parafunctions before and during the COVID-19 pandemic era: a sex comparison among dental patients. J Clin Med. 2022; 11(3), doi: 10.3390/jcm11030589, indexed in Pubmed: 35160041.
20. Quadri MF, Mahnashi A, Al Almutahhir A, et al. Association of awake bruxism with khat, coffee, tobacco, and stress among jazan university students. Int J Dent. 2015; 2015: 842096, doi: 10.1155/2015/842096, indexed in Pubmed: 26491448.
21. Koyano K, Tsukiyama Y, Ichiki R, et al. Assessment of bruxism in the clinic. J Oral Rehabil. 2008; 35(7): 495–508, doi: 10.1111/j.1365-2842.2008.01880.x, indexed in Pubmed: 18557916.
22. Goldstein RE, Auclair Clark W. The clinical management of awake bruxism. J Am Dent Assoc. 2017; 148(6): 387–391, doi: 10.1016/j.adaj.2017.03.005, indexed in Pubmed: 28550845.
23. Svensson P, Jadidi F, Arima T, et al. Relationships between craniofacial pain and bruxism. J Oral Rehabil. 2008; 35(7): 524–547, doi: 10.1111/j.1365-2842.2008.01852.x, indexed in Pubmed: 18557918.
24. Carra MC, Huynh N, Lavigne G. Sleep bruxism: a comprehensive overview for the dental clinician interested in sleep medicine. Dent Clin North Am. 2012; 56(2): 387–413, doi: 10.1016/j.cden.2012.01.003, indexed in Pubmed: 22480810.
25. Nijakowski K, Ortarzewska M, Morawska A, et al. Bruxism influence on volume and interleukin-1β concentration of gingival crevicular fluid: a preliminary study. Appl Sci. 2022; 12(4): 2089, doi: 10.3390/app12042089.
26. Murali RV, Rangarajan P, Mounissamy A. Bruxism: conceptual discussion and review. J Pharm Bioallied Sci. 2015; 7(Suppl 1): S265–S270, doi: 10.4103/0975-7406.155948, indexed in Pubmed: 26015729.
27. Hoz-Aizpurua JLd, Diaz-Alonso E, LaTouche-Arbizu R, et al. Sleep bruxism. Conceptual review and update. Medicina Oral Patología Oral y Cirugia Bucal. 2011; 16(2): e231–e238, doi: 10.4317/medoral.16.e231, indexed in Pubmed: 21196839.
28. Klasser GD, Rei N, Lavigne GJ. Sleep bruxism etiology: the evolution of a changing paradigm. J Can Dent Assoc. 2015; 81: f2, indexed in Pubmed: 25633110.
29. Boscato N, Exposto F, Nascimento GG, et al. Is bruxism associated with changes in neural pathways? A systematic review and meta-analysis of clinical studies using neurophysiological techniques. Brain Imaging Behav. 2022; 16(5): 2268–2280, doi: 10.1007/s11682-021-00601-w, indexed in Pubmed: 35088353.
30. Mascaro MB, Bittencourt JC, Casatti CA, et al. Alternative pathways for catecholamine action in oral motor control. Neurosci Lett. 2005; 386(1): 34–39, doi: 10.1016/j.neulet.2005.05.062, indexed in Pubmed: 15978723.
31. Lavigne GJ, Soucy JP, Lobbezoo F, et al. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. Clin Neuropharmacol. 2001; 24(3): 145–149, doi: 10.1097/00002826-200105000-00005, indexed in Pubmed: 11391125.
32. Lobbezoo F, Lavigne GJ, Tanguay R, et al. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord. 1997; 12(1): 73–78, doi: 10.1002/mds.870120113, indexed in Pubmed: 8990057.
33. Huynh N, Kato T, Rompré PH, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res. 2006; 15(3): 339–346, doi: 10.1111/j.1365-2869.2006.00536.x, indexed in Pubmed: 16911037.
34. Huynh N, Lavigne GJ, Lanfranchi PA, et al. The effect of 2 sympatholytic medications - propranolol and clonidine - on sleep bruxism: experimental randomized controlled studies. Sleep. 2006; 29(3): 307–316, doi: 10.1093/sleep/29.3.307, indexed in Pubmed: 16553016.
35. D'Ambrosio E, Dahoun T, Pardiñas AF, et al. The effect of a genetic variant at the schizophrenia associated AS3MT/BORCS7 locus on striatal dopamine function: A PET imaging study. Psychiatry Res Neuroimaging. 2019; 291: 34–41, doi: 10.1016/j.pscychresns.2019.07.005, indexed in Pubmed: 31386983.
36. Serra-Negra JM, Lobbezoo F, Martins CC, et al. Prevalence of sleep bruxism and awake bruxism in different chronotype profiles: Hypothesis of an association. Med Hypotheses. 2017; 101: 55–58, doi: 10.1016/j.mehy.2017.01.024, indexed in Pubmed: 28351492.
37. Amato JN, Tuon RA, Castelo PM, et al. Assessment of sleep bruxism, orthodontic treatment need, orofacial dysfunctions and salivary biomarkers in asthmatic children. Arch Oral Biol. 2015; 60(5): 698–705, doi: 10.1016/j.archoralbio.2015.02.011, indexed in Pubmed: 25757147.
38. Buske-Kirschbaum A, von Auer K, Krieger S, et al. Blunted cortisol responses to psychosocial stress in asthmatic children: a general feature of atopic disease? Psychosom Med. 2003; 65(5): 806–810, doi: 10.1097/01.psy.0000095916.25975.4f, indexed in Pubmed: 14508024.
39. Serra-Negra JM, Pordeus IA, Corrêa-Faria P, et al. Is there an association between verbal school bullying and possible sleep bruxism in adolescents? J Oral Rehabil. 2017; 44(5): 347–353, doi: 10.1111/joor.12496, indexed in Pubmed: 28214362.
40. Fulgencio LB, Corrêa-Faria P, Lage CF, et al. Diagnosis of sleep bruxism can assist in the detection of cases of verbal school bullying and measure the life satisfaction of adolescents. Int J Paediatr Dent. 2017; 27(4): 293–301, doi: 10.1111/ipd.12264, indexed in Pubmed: 27598528.
41. Buescher JJ. Temporomandibular joint disorders. Am Fam Physician. 2007; 76(10): 1477–1482, indexed in Pubmed: 18052012.
42. Leal TR, de Lima LC, Neves ÉT, et al. Factors associated with awake bruxism according to perceptions of parents/guardians and self-reports of children. Int J Paediatr Dent. 2022; 32(1): 22–30, doi: 10.1111/ipd.12786, indexed in Pubmed: 33730404.
43. Botelho J, Machado V, Proença L, et al. Relationship between self-reported bruxism and periodontal status: Findings from a cross-sectional study. J Periodontol. 2019 [Epub ahead of print], doi: 10.1002/JPER.19-0364, indexed in Pubmed: 31850520.
44. Mizutani S, Ekuni D, Tomofuji T, et al. Factors related to the formation of buccal mucosa ridging in university students. Acta Odontol Scand. 2014; 72(1): 58–63, doi: 10.3109/00016357.2013.797102, indexed in Pubmed: 23692316.
45. Ilovar S, Zolger D, Castrillon E, et al. Biofeedback for treatment of awake and sleep bruxism in adults: systematic review protocol. Syst Rev. 2014; 3: 42, doi: 10.1186/2046-4053-3-42, indexed in Pubmed: 24886985.
46. Shadimetova NM, Saidkhodzhayeva SN. Neurophysiological characteristics of sleep bruxism in children. World Bull Soc Sci. 2022; 9: 47–50.
47. Schiffman E, Ohrbach R, Truelove E, et al. International RDC/TMD Consortium Network, International association for Dental Research, Orofacial Pain Special Interest Group, International Association for

the Study of Pain. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the international RDC/TMD consortium network* and orofacial pain special interest group†. J Oral Facial Pain Headache. 2014; 28(1): 6–27, doi: 10.11607/jop.1151, indexed in Pubmed: 24482784.
48. Lavigne GJ, Khoury S, Abe S, et al. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008; 35(7): 476–494, doi: 10.1111/j.1365-2842.2008.01881.x, indexed in Pubmed: 18557915.
49. Shetty S, Pitti V, Satish Babu CL, et al. Bruxism: a literature review. J Indian Prosthodont Soc. 2010; 10(3): 141–148, doi: 10.1007/s13191-011-0041-5, indexed in Pubmed: 21886404.
50. Carlson CR, Bertrand PM, Ehrlich AD, et al. Physical self-regulation training for the management of temporomandibular disorders. J Orofac Pain. 2001; 15(1): 47–55, indexed in Pubmed: 11889647.
51. Minakuchi H, Fujisawa M, Abe Y, et al. Managements of sleep bruxism in adult: a systematic review. Jpn Dent Sci Rev. 2022; 58: 124–136, doi: 10.1016/j.jdsr.2022.02.004, indexed in Pubmed: 35356038.
52. Amorim CSM, Espirito Santo AS, Sommer M, et al. Effect of physical therapy in bruxism treatment: a systematic review. J Manipulative Physiol Ther. 2018; 41(5): 389–404, doi: 10.1016/j.jmpt.2017.10.014, indexed in Pubmed: 30041736.
53. Wang LF, Long Hu, Deng M, et al. Biofeedback treatment for sleep bruxism: a systematic review. Sleep Breath. 2014; 18(2): 235–242, doi: 10.1007/s11325-013-0871-y, indexed in Pubmed: 23756884.
54. Mesko ME, Hutton B, Skupien JA, et al. Therapies for bruxism: a systematic review and network meta-analysis (protocol). Syst Rev. 2017; 6(1): 4, doi: 10.1186/s13643-016-0397-z, indexed in Pubmed: 28086992.
55. Manfredini D, Bracci A, Djukic G. BruxApp: the ecological momentary assessment of awake bruxism. Minerva Stomatol. 2016; 65(4): 252–5, indexed in Pubmed: 27374364.
56. Osiewicz MA, Lobbezoo F, Bracci A, et al. Ecological momentary assessment and intervention principles for the study of awake bruxism behaviors, part 2: development of a smartphone application for a multicenter investigation and chronological translation for the polish version. Front Neurol. 2019; 10: 170, doi: 10.3389/fneur.2019.00170, indexed in Pubmed: 30890999.
57. Colonna A, Lombardo L, Siciliani G, et al. Smartphone-based application for EMA assessment of awake bruxism: compliance evaluation in a sample of healthy young adults. Clin Oral Investig. 2020; 24(4): 1395–1400, doi: 10.1007/s00784-019-03098-2, indexed in Pubmed: 31646395.
58. Jokubauskas L, Baltrušaitytė A, Pileičikienė G. Oral appliances for managing sleep bruxism in adults: a systematic review from 2007 to 2017. J Oral Rehabil. 2018; 45(1): 81–95, doi: 10.1111/joor.12558, indexed in Pubmed: 28859236.
59. Hardy RS, Bonsor SJ. The efficacy of occlusal splints in the treatment of bruxism: a systematic review. J Dent. 2021; 108: 103621, doi: 10.1016/j.jdent.2021.103621, indexed in Pubmed: 33652054.
60. van der Zaag J, Lobbezoo F, Wicks DJ, et al. Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain. 2005; 19(2): 151–158, indexed in Pubmed: 15895838.
61. Gomes CA, El-Hage Y, Amaral AP, et al. Effects of massage therapy and occlusal splint usage on quality of life and pain in individuals with sleep bruxism: a randomized controlled trial. J Jpn Phys Ther Assoc. 2015; 18(1): 1–6, doi: 10.1298/jjpta.Vol18_001, indexed in Pubmed: 26733760.
62. Gholampour S, Gholampour H, Khanmohammadi H. Finite element analysis of occlusal splint therapy in patients with bruxism. BMC Oral Health. 2019; 19(1): 205, doi: 10.1186/s12903-019-0897-z, indexed in Pubmed: 31484524.
63. Barth SW, Lehner MD, Dietz GPH, et al. Pharmacologic treatments in preclinical tinnitus models with special focus on Ginkgo biloba leaf extract EGb 761®. Mol Cell Neurosci. 2021; 116: 103669, doi: 10.1016/j.mcn.2021.103669, indexed in Pubmed: 34560255.
64. Kulkarni S, Thambar S, Arora H. Evaluating the effectiveness of non-steroidal anti-inflammatory drug(s) for relief of pain associated with temporomandibular joint disorders: A systematic review. Clin Exp Dent Res. 2020; 6(1): 134–146, doi: 10.1002/cre2.241, indexed in Pubmed: 32067407.
65. de Baat C, Verhoeff M, Ahlberg J, et al. i in. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. J Oral Rehabil. 2021; 48(3): 343–354, doi: 10.1111/joor.13061, indexed in Pubmed: 32716523.
66. Rasetti-Escargueil C, Popoff MR. Antibodies and vaccines against botulinum toxins: available measures and novel approaches. Toxins (Basel). 2019; 11(9): 528, doi: 10.3390/toxins11090528, indexed in Pubmed: 31547338.
67. Botzenhart UU, Gerlach R, Gredes T, et al. Expression rate of myogenic regulatory factors and muscle growth factor after botulinum toxin A injection in the right masseter muscle of dystrophin deficient (mdx) mice. Adv Clin Exp Med. 2019; 28(1): 11–18, doi: 10.17219/acem/76263, indexed in Pubmed: 30085421.
68. Shim YJ, Lee HJ, Park KJ, et al. Botulinum toxin therapy for managing sleep bruxism: a randomized and placebo-controlled trial. Toxins (Basel). 2020; 12(3): 168, doi: 10.3390/toxins12030168, indexed in Pubmed: 32182879.
69. Kwon KH, Shin KSu, Yeon SH, et al. Application of botulinum toxin in maxillofacial field: part I. Bruxism and square jaw. Maxillofac Plast Reconstr Surg. 2019; 41(1): 38, doi: 10.1186/s40902-019-0218-0, indexed in Pubmed: 31649901.
70. Kwon KH, Shin KSu, Yeon SH, et al. Application of botulinum toxin in maxillofacial field: part III. ancillary treatment for maxillofacial surgery and summary. Maxillofac Plast Reconstr Surg. 2019; 41(1): 45, doi: 10.1186/s40902-019-0226-0, indexed in Pubmed: 31709199.
71. Pirazzini M, Rossetto O, Eleopra R, et al. Botulinum neurotoxins: biology, pharmacology, and toxicology. Pharmacol Rev. 2017; 69(2): 200–235, doi: 10.1124/pr.116.012658, indexed in Pubmed: 28356439.
72. Ågren M, Sahin C, Pettersson M. The effect of botulinum toxin injections on bruxism: a systematic review. J Oral Rehabil. 2020; 47(3): 395–402, doi: 10.1111/joor.12914, indexed in Pubmed: 31769044.
73. Yurttutan ME, Tütüncüler Sancak K, Tüzüner AM. Which treatment is effective for bruxism: occlusal splints or botulinum toxin? J Oral Maxillofac Surg. 2019; 77(12): 2431–2438, doi: 10.1016/j.joms.2019.06.005, indexed in Pubmed: 31302066.
74. Fernández-Núñez T, Amghar-Maach S, Gay-Escoda C. Efficacy of botulinum toxin in the treatment of bruxism: systematic review. Med Oral Patol Oral Cir Bucal. 2019; 24(4): e416–e424, doi: 10.4317/medoral.22923, indexed in Pubmed: 31246937.
75. Alonso-Navarro H, Jiménez-Jiménez FJ, Plaza-Nieto JF, et al. Treatment of severe bruxism with botulinum toxin type A. Rev Neurol. 2011; 53(2): 73–76, indexed in Pubmed: 21720976.
76. Fernández-Núñez T, Amghar-Maach S, Gay-Escoda C. Efficacy of botulinum toxin in the treatment of bruxism: systematic review. Med Oral Patol Oral Cir Bucal. 2019; 24(4): e416–e424, doi: 10.4317/medoral.22923, indexed in Pubmed: 31246937.
77. Jost W. Atlas of Botulinum Toxin Injection. Quintessence Publishing, Batavia 2019.

78. Monroy PG, da Fonseca MA. The use of botulinum toxin-a in the treatment of severe bruxism in a patient with autism: a case report. Spec Care Dentist. 2006; 26(1): 37–39, doi: 10.1111/j.1754-4505.2006.tb01508.x, indexed in Pubmed: 16703933.
79. Cheng Y, Yuan L, Ma Li, et al. Efficacy of botulinum - a for nocturnal bruxism pain and the occurrence of bruxism events: a meta-analysis and systematic review. Br J Oral Maxillofac Surg. 2022; 60(2): 174–182, doi: 10.1016/j.bjoms.2021.03.005, indexed in Pubmed: 34955330.
80. Fluerașu MI, Bocșan IC, Țig IA, et al. The epidemiology of bruxism in relation to psychological factors. Int J Environ Res Public Health. 2022; 19(2): 691, doi: 10.3390/ijerph19020691, indexed in Pubmed: 35055514.
81. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain. 2009; 23(2): 153–166, indexed in Pubmed: 19492540.
82. Duarte J, Souza JF, Cavalcante-Leão B, et al. Association of possible sleep bruxism with daytime oral habits and sleep behavior in schoolchildren. Cranio. 2021; 39(5): 372–378, doi: 10.1080/08869634.2019.1661113, indexed in Pubmed: 31483213.
83. Imbriglio TV, Moayedi M, Freeman BV, et al. Music modulates awake bruxism in chronic painful temporomandibular disorders. Headache. 2020; 60(10): 2389–2405, doi: 10.1111/head.13971, indexed in Pubmed: 32997813.
84. Lal SJ, Weber, DDS KK. Bruxism Management. [Updated 2022 Oct 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls 2022.
85. Kobayashi FY, Castelo PM, Gonçalves ML, et al. Evaluation of the effectiveness of infrared light-emitting diode photobiomodulation in children with sleep bruxism: Study protocol for randomized clinical trial. Medicine (Baltimore). 2019; 98(38): e17193, doi: 10.1097/MD.0000000000017193, indexed in Pubmed: 31567965.
86. da Silva MM, Albertini R, Leal-Junior EC, et al. Effects of exercise training and photobiomodulation therapy (EXTRAPHOTO) on pain in women with fibromyalgia and temporomandibular disorder: study protocol for a randomized controlled trial. Trials. 2015; 16: 252, doi: 10.1186/s13063-015-0765-3, indexed in Pubmed: 26040789.
87. Demirkol N, Sari F, Bulbul M, et al. Effectiveness of occlusal splints and low-level laser therapy on myofascial pain. Lasers Med Sci. 2015; 30(3): 1007–1012, doi: 10.1007/s10103-014-1522-7, indexed in Pubmed: 24504660.
88. Ribeiro-Lages MB, Martins ML, Magno MB, et al. Is there association between dental malocclusion and bruxism? A systematic review and meta-analysis. J Oral Rehabil. 2020; 47(10): 1304–1318, doi: 10.1111/joor.12971, indexed in Pubmed: 32246486.
89. Ghafournia M, Hajenourozali Tehrani M. Relationship between bruxism and malocclusion among preschool children in isfahan. J Dent Res Dent Clin Dent Prospects. 2012; 6(4): 138–142, doi: 10.5681/joddd.2012.028, indexed in Pubmed: 23277860.
90. Lobbezoo F, van der Zaag J, van Selms MKA, et al. Principles for the management of bruxism. J Oral Rehabil. 2008; 35(7): 509–523, doi: 10.1111/j.1365-2842.2008.01853.x, indexed in Pubmed: 18557917.
91. Hublin C, Kaprio J, Partinen M, et al. Parasomnias: co-occurrence and genetics. Psychiatr Genet. 2001; 11(2): 65–70, doi: 10.1097/00041444-200106000-00002, indexed in Pubmed: 11525419.
92. Bashir A, Jawa D, Somani R, et al. All about bruxism - the teeth grinding. J Adv Med Dent Sci Res. 2021; 9(5): 9–23, doi: 10.21276/jamdsr.
93. Ahlberg K, Jahkola A, Savolainen A, et al. Associations of reported bruxism with insomnia and insufficient sleep symptoms among media personnel with or without irregular shift work. Head Face Med. 2008; 4: 4, doi: 10.1186/1746-160X-4-4, indexed in Pubmed: 18307774.
94. Shokry SM, El Wakeel EE, Al-Maflehi N, et al. Association between self-reported bruxism and sleeping patterns among dental students in saudi arabia: a cross-sectional study. Int J Dent. 2016; 2016: 4327081, doi: 10.1155/2016/4327081, indexed in Pubmed: 27034672.
95. Kuang B, Li D, Lobbezoo F, et al. Associations between sleep bruxism and other sleep-related disorders in adults: a systematic review. Sleep Med. 2022; 89: 31–47, doi: 10.1016/j.sleep.2021.11.008, indexed in Pubmed: 34879286.
96. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep. 1994; 17(8): 739–743, indexed in Pubmed: 7701186.
97. Li Y, Yu F, Niu L, et al. Associations among bruxism, gastroesophageal reflux disease, and tooth wear. J Clin Med. 2018; 7(11): 417, doi: 10.3390/jcm7110417, indexed in Pubmed: 30404150.
98. Serra-Negra JM, Lobbezoo F, Correa-Faria P, et al. Relationship of self-reported sleep bruxism and awake bruxism with chronotype profiles in Italian dental students. Cranio. 2019; 37(3): 147–152, doi: 10.1080/08869634.2018.1431600, indexed in Pubmed: 29376478.
99. Erden S. Sleep-related bruxism response to melatonin treatment. J Child Adolesc Psychopharmacol. 2020; 30(3): 201, doi: 10.1089/cap.2019.0143, indexed in Pubmed: 31800301.
100. Câmara-Souza MB, Figueredo OMC, Rodrigues Garcia RCM. Tongue force, oral health-related quality of life, and sleep index after bruxism management with intraoral devices. J Prosthet Dent. 2020; 124(4): 454–460, doi: 10.1016/j.prosdent.2019.07.017, indexed in Pubmed: 31831164.