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REVIEW ARTICLE

Sleep bruxism: an updated review of an old problem

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ABSTRACT

Objective To provide an update on what is known about bruxism and some of the major clinical highlights derived from new insights into this old problem in dentistry. **Materials and methods** A selective, non-systematic but critical review of the available scientific literature was performed. **Results** There are two main different types of bruxism, which are related to different circadian periods (sleep and awake bruxism) that may differ in terms of pathophysiology, but they share some common signs and symptoms. Approximately one out of 10 adult individuals may suffer from bruxism, but not all bruxers may need treatment. Bruxism is complicated to diagnose in the clinic and self-report of bruxism may not necessarily reflect the true presence of jaw muscle activity. Better understanding has been acquired of bruxism relationships with sleep stages, arousal responses and autonomic function with the help of polysomnography and controlled sleep studies. Meanwhile, there is still much more to learn about awake bruxism. With the available scientific knowledge it is possible to systematically assess the effects of bruxism and its potential risk factors for oral and general health. Moreover, we can be aware of the realistic possibilities to manage/treat the patient suffering from bruxism. **Conclusion** Bruxism is a parafunctional activity involving the masticatory muscles and probably it is as old as human mankind. Different ways have been proposed to define, diagnose, assess the impact and consequences, understand the pathophysiology and treat or manage bruxism. Despite the vast research efforts made in this field, there are still significant gaps in our knowledge.

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Introduction

Most of the patients suffering from sleep bruxism (SB) have found out that they grind or clench their teeth during sleep by the report from somebody who sleeps in the same room. This parafunctional activity not only bothers others with the unpleasant sound of the 'gnashing of teeth', but it may also be associated with several other problems. Dentists among other professionals can help to recognize this parafunctional activity, explain its characteristics and consequences to the patients and manage these problems. In this review we will try to help to organize and understand the available scientific knowledge related to this topic by answering the following questions:

What is bruxism and sleep bruxism?
How many people grind/clench their teeth?
How do we diagnose it?
Why do we grind/clench our teeth?
What problems can teeth grinding/clenching cause?
Can we treat/manage bruxism?

It should be mentioned that the purpose is not to provide a systematic review on bruxism and the reader is referred to some of the several recent publications.[1–6]

What is bruxism and sleep bruxism?

Bruxism is considered a parafunctional activity of the masticatory muscles that has been reported in the history since early times.[7] This oral parafunctional activity is known scientifically as bruxism that comes from the Greek word 'brygmos' (βρυγμός) and it means gnashing of the teeth. Marie and Pietkiewicz [8] were the first to describe bruxism in the scientific literature and defined it as 'La Bruxomanie'. A recent publication from an international expert group has discussed all the different definitions available.[9] They have proposed a new and simple definition: 'Bruxism is a *repetitive jaw-muscle activity* characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible'.[9] It is, indeed, challenging to define a grading and classification system for bruxism due to the different variables that have been proposed

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to be associated with bruxism (e.g. idiopathic and iatrogenic, sleep and awaken, painful or asymptomatic, EMG activity: phasic, tonic or mixed, etc.). Several classification systems and interactions of the different variables have been proposed. For example, bruxism could be sub-divided as primary idiopathic and secondary iatrogenic (e.g. as a manifestation of neurological disorders and psychiatric sleep disorders). In this review we will use the circadian rhythm to classify bruxism into awake bruxism (AB) and SB.[10] These two different circadian manifestations of bruxism may have a different pathophysiology.[4] From these two types of bruxism, SB is the one that has been most studied. SB is included in the International Classification of Sleep Disorders–Third Edition (ICSD-3) under the group of sleep-related movement disorders.[11] The sleep-related movement disorders are characterized by simple and stereotypic movements that disturb sleep while the patients may or may not be aware of such movements.[11]

How many people grind/clench their teeth?

Most people grind or clench their teeth once in a while. Scientific epidemiological studies have shown that interviews or questionnaires (self-report based method) asking: 'do you grind/clench your teeth during sleep?' will give results of ~10% of the adult population answering 'yes' to the option of 'frequent as three times a week'.[4,12,13] Epidemiological studies show that bruxism tends to decrease with age. Nevertheless, there is a big variability in the reports about bruxism in children (3.5–40.6%) due to the different age groups reported and the problems that may arise applying the self-report method to children/or their parents. As people become older (>65 years), the frequency of teeth grinding/clenching during sleep drops to 1.1%.[14] It is interesting to note that no difference has been found between men and women who grind/clench their teeth during sleep. It is, however, important to point out that these frequencies are based on information that people have provided and, clearly, there is significant uncertainty relative to the accuracy of such figures.

Recently an epidemiological study of bruxism has been published using the polysomnographic (PSG) method without video and audio recordings in which an overall prevalence of 7.4% has been reported.[15] This study also demonstrated the discrepancies between self-report and PSG methods to diagnose SB.[15]

How do we diagnose it?

The up-to-date gold standard for diagnosis of SB is a full PSG study including video and audio assessments and following the diagnostic criteria proposed by Lavigne and colleagues.[16,17] The PSG method assesses an exclusive type of muscle activity that characterizes bruxism. This characteristic muscle activity is well described in the scientific literature and it is known as 'rhythmic masticatory muscle activity' (RMMA) [16] (Figure 1). The PSG method has a sensitivity and specificity >80% to detect SB. Unfortunately, the PSG is a method that requires highly trained professionals to score and interpret the PSG data, as well as expensive and not easy to access equipment. All these factors limit its application to research purposes and complex patients.

Other diagnostic methods have been proposed and are used as an alternative to diagnose SB. These methods range from self-report (interview/questionnaires), clinical examination to the use of additional methods such as portable EMG devices.[18] All these methods have been questioned because they do have some limitations to assess bruxism.[19]

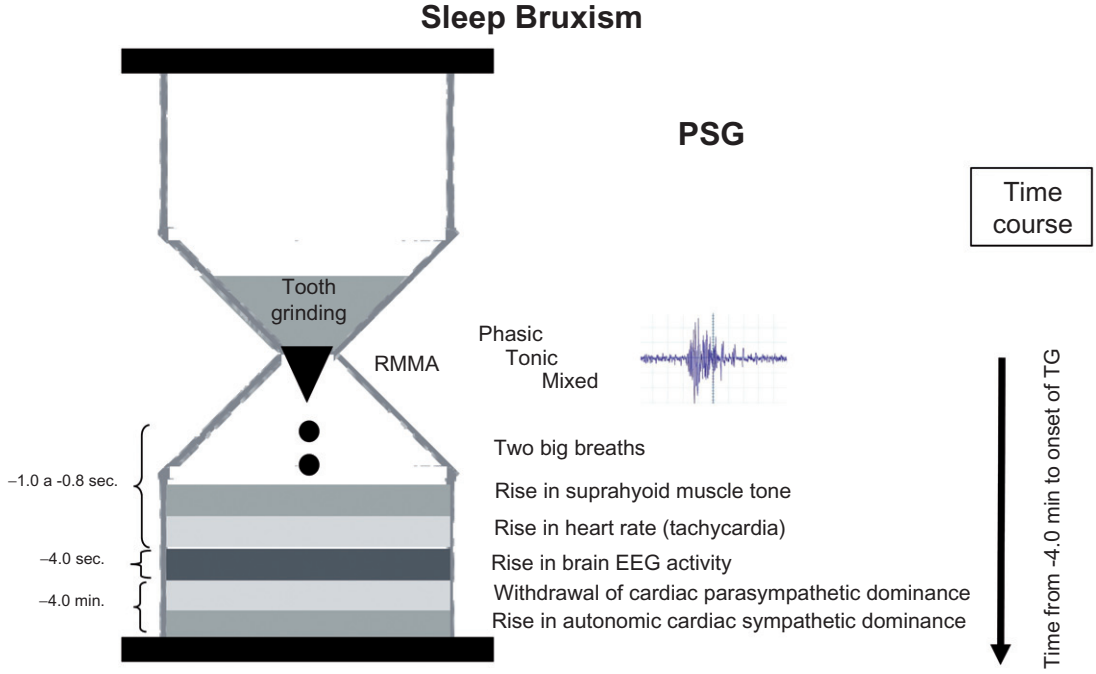

Figure 1. Time course of a tooth-grinding (TG) event in sleep bruxism. The figure illustrates the time course of the different biological events needed to occur before TG is assessed as rhythmic masticatory muscle activity (RMMA) following the 'gold standard' criteria for bruxism assessment in a polysomnographic (PSG) study. The RMMA could be phasic, tonic or mixed. This figure is adapted from Lavigne et al. [54].

Patients' interviews and questionnaires about their dental history can help dentists to be able to screen whether or not the patient is likely to grind/clench her/his teeth during sleep.[20] Using simple questions such as 'has your bed partner noticed that you clench/grind your teeth when you sleep?', 'do you wake up with tenderness/pain in your jaw muscles?' and 'do your teeth often hurt when you wake up?' may be useful. The next step after the interview/questionnaire would be a clinical examination searching for clinical signs or symptoms related to SB: jaw-muscle fatigue/pain upon awakening, masseter muscle hypertrophy on voluntary contraction,[12] visual evidence of moderate-to-severe hyperkeratosis of cheeks/lips/tongue, visual evidence of advanced tooth wear ($\geq$ grade 1c),[21,22] loss of cusp protection and frequent non-iatrogenic/non-material-related fractures and failures of teeth/restorations/implants. All these clinical signs assess the possible outcomes of the bruxism activity, but not the real bruxism event, i.e. the jaw muscle activity itself. It has to be considered that maybe some of these signs represent an old or accumulated lesion/effect that does not correspond to the actual status of the patient.

The use of additional methods to assess bruxism is an area that has been of interest for many researchers in the last decades.[18,19,23–32] These methods mainly focus on the ambulatory EMG assessment of the jaw-closing muscles. The key point of the validity of these methods has been built up on their correlation to the assessment of PSG-derived measures RMMA (gold standard). The application of these possible correlated assessments may become a real possibility to offer a more affordable and practical method to assess bruxism and increase the accuracy of the available clinical diagnostic process in dental practice.[33]

In addition to use in dental practice, the portable EMG activity assessment devices provide excellent opportunities for addressing various scientific questions, such as better and more precise assessment of how many people actually grind/clench their teeth too much during the day and/or at night.[18,23,31,32] Furthermore, the connection between attrition, implant problems and the occurrence of temporomandibular pain and headache may now be studied much more accurately. Nevertheless, a recent systematic review on portable devices to assess SB has concluded that the scientific evidence of the accuracy to diagnose bruxism with these types of devices is 'still scarce and not solid enough as a stand-alone diagnostic method'.[19]

The scarcity of reliable and valid diagnostic tools for bruxism inspired an international group of bruxism experts to propose a diagnostic grading system for clinical and research purposes.[9] This diagnostic grading system takes into account the circadian rhythm (SB and AB). The reliability of the diagnostic methods used to determine the existence of bruxism was used to sub-divide SB and AB. The experts proposed a 3-level grading system: 'possible', 'probable' and 'definite'. 'Possible AB or SB' should be based on 'self-report' using anamnestic methods or questionnaires. 'Probable AB or SB' will be the same conditions for 'possible' plus some positive finding during the clinical examination. 'Definite SB' will continue the grading system using the criteria for determining 'probable' plus a PSG study following the gold standard method indications [16] for SB [9] (Figure 2). Finally 'definite AB' will include self-report, clinical examination and an EMG recording, preferably combined with the so-called ecological momentary assessment methodology [9]. Future studies will be needed to operationalize these proposed criteria and demonstrate the usefulness of this approach.

Why do we grind/clench our teeth?

In order to be able to grind or clench the teeth together, the jaw closing muscles must be used (masseter, temporalis, internal pterygoid muscles). We now know that particular groups of motor neurons in the brain stem, constituting what is known as the central pattern generator, are responsible for rhythmic masticatory action and SB [12]. So the question is: 'Why does this central pattern generator click in when we sleep?' The current hypothesis is that several factors in the central nervous system may affect the central pattern generator, such as changes in the sleep pattern and in the parts of the brain that control sleep [12,33]. Microarousals appear to be crucial in the sequence of events leading to activation of the central pattern generator and jaw closing muscles. For example, elaborate studies on the cyclic alternating pattern (CAP) of sleep have indicated that sleep bruxers have subtle difference in the A3-phase, which are characterized by more pronounced arousal responses. Autonomous regulation of the heart rhythm may, furthermore, affect the central pattern generator, just as drugs affecting the central nervous system (such as anti-depressants, hypnotics and anxiolytics) may cause changes in the occurrence of SB. Genetic factors presumably also play a certain role and work is currently being done to identify genetic markers for pronounced SB.[34] Preliminary results also indicate that there is a very marked individual difference in the occurrence of teeth grinding/clenching.[34]

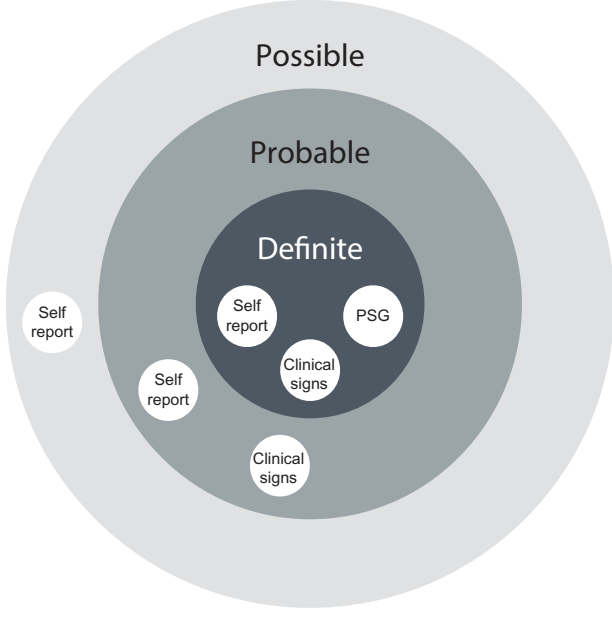

Figure 2. Proposed diagnostic grading system for sleep bruxism. The figure illustrates the criteria required in order to establish the grade of accuracy of the diagnostic in sleep bruxism. This figure is adapted from Lobbezoo et al. [9].

For example, one twin may be a teeth grinder/clencher, while the other twin is not.[34]

Whether or not stress has an influence on teeth grinding/clenching is a recurring question.[35] The data currently available indicate that stress does not significantly affect SB, but does, on the other hand, indicate that there is a strong connection between people's self-perception of stress and the occurrence of AB. Psychosocial factors may, thus, be partially responsible for more people 'grinding/clenching their teeth' during their waking hours.[35] Even though there is no scientific evidence, clinical experience with patients suggests that physical stress or considerable physical efforts may also cause people to grind/clench their teeth together. It suffices to think of pictures of athletes struggling to perform their utmost, with their teeth clamped together.

It is interesting to note that bruxism, apart from these 'parafunctions', apparently may also have a physiological function. For example, grinding/clenching the teeth causes increased activity in the mechanoreceptors located in the periodontal membrane, giving rise to reflex stimulation of saliva secretion and, hence, increase in the lubrication of the oral cavity and pharynx, which may be of advantage during sleep. SB is also associated with a slight protrusive movement of the lower jaw and the tongue musculature, which increases the pharyngeal volume and facilitates passage of air to the lungs.[12]

In summary, we are today able to say that it is important to differentiate between SB and AB. Extreme SB may be considered as a sleep-related movement disorder of the nature of periodical leg movements, while AB is to a large extent conditioned by stress and emotional factors. It should also be pointed out that the once widespread odontological assumption that (mal)-occlusion (such as supracontacts) could cause bruxism and that the grinding and equilibration of the teeth or occlusal rehabilitation could eliminate bruxism, is not supported by scientific documentation.[6,9] The focus, we might say, has shifted from the periphery (e.g. the teeth and their occlusion or craniofacial morphology) to the central nervous system.[5]

What problems can teeth grinding/clenching cause?

As described above, it is probably completely natural to grind/clench the teeth once in a while. However, bruxism may also constitute a risk for various problems of which dentists should be aware.

The most obvious consequence of bruxism is likely attrition of the teeth or occlusal reconstructions.[36,37] Bruxism may, therefore, become a problem, causing filling fractures and, in rare cases, even tooth fractures. There is today increased concern that bruxism is a risk factor in implant treatment, since the stress of an osseo-integrated implant has a markedly different effect on control of the jaw closing muscles.[38] Bruxism was once associated with progression of periodontal attachment loss, but this is not supported by scientific studies.[39] From the odontological point of view, it is important to record and document the degree of attrition, for which various scales may be used (Table I).

SB may also cause problems not only for people who grind/clench their teeth, but also for their bed partners, as mentioned earlier.[12] This is perhaps not a major problem, but it may well be irritating and may affect personal life.

Muscles may become too 'developed' due to bruxism. We do not know how much training (that is, teeth grinding or clenching) is needed to cause muscle hypertrophy, but it is common to hear patients reports claiming that particularly large-sized masseter muscles may be a consequence of years of bruxism. In such cases, attempts have been made to paralyse the muscle with the help of botulinum toxin injections and irreversible 'shrinking' (atrophy) of the muscle has been seen.[40]

Too much muscle action can be associated with tenderness or pain in the musculature. Self-reported bruxism has also been found to be a significant risk factor for painful temporomandibular disorder (TMD) and headache [41] It is important to point out that the relationship between bruxism and pain disorders is not 1:1, since extreme degrees of tooth attrition are also seen without any pain or tenderness in the jaw musculature.[42] The explanation is likely that there are considerable individual differences in the degree and intensity of muscle activity and of the feeling of pain and its control. Dentists must also be aware that pain is not merely pain, but that there are various forms/types of pain.[43,44] One pain form is characterized by the fact that it arises after strong muscle effort—known as post-exercise muscle soreness (PEMS)—which may include slight inflammatory changes in the musculature due to micro-ruptures of the muscle fibres.[45] SB may give rise to a form of PEMS characterized by tenderness/pain that is most intense in the morning and decreases during the day, whereas myofascial TMD pain may have a different pattern of pain intensity during the day.

In any case, there are several good reasons, summarized in Table 2, why dentist should be aware of bruxism.

Can we treat/manage bruxism?

The management of bruxism could be divided into three different approach areas: occlusal, cognitive/behavioural and pharmacological.[37] The occlusal approach is perhaps the most used and it could be sub-divided into 'non-reversible' and 'reversible'. The 'non-reversible' is usually chosen when it is thought that bruxism has an occlusal aetiology and by altering it the parafunction will be controlled. This approach will use methods that range from selective permanent occlusal adjustments, orthodontics and even prosthetics. The up-to-date knowledge does not support a relationship between bruxism and occlusion [5,46]; therefore, occlusal intervention as a treatment method for managing or curing bruxism is not justified [5,6].

Table 1. Example of graduated attrition scale [21,22].

0 = no enamel loss
1 = enamel loss, minimal surface change
2 = enamel loss with limited (< 1/3) dentine exposure
3 = enamel loss with extensive (> 1/3) dentine exposure
4 = complete enamel loss with exposure of pulp or secondary dentine

The 'reversible' occlusal approach is the most commonly used method by clinicians. Most clinicians following this approach will ask themselves the question: 'why not make a mouth guard?' Mouth guard mechanisms of actions have several hypotheses, but from all of them only one could be confirmed scientifically: mouth guards do work very well as tooth protectors [6], but mouth guards do not stop or cure bruxism [6]. This strongly indicates that bruxism is to a large extent regulated by the central nervous system and is not directly affected by the teeth (the peripheral factor) [5]. Scientific studies with PSG techniques have shown that muscle activity during sleep decreases as soon as a new mouth guard is used; however, after 2–4 weeks it returns to its original level[47] Various shapes of mouth guards do not seem to alter this effect. The most effective mouth guard, however, is a form of mandibular advancement device that keeps the lower jaw in a slightly anterior, displaced position. This type of guard may cause reactions from the jaw joints or musculature [48,49] and clinicians have reported occlusal changes as a result of the use of this device. Our own experiments with non-bruxers show that, in fact, EMG activity of the masticatory muscles is reduced, but this reduction is transient and will be back to normal levels after a short period (Figure 3).

Table 2. List of possible negative effects of bruxism. Note that not all effects are scientifically documented.

Tooth attrition
Occlusal changes and loss of vertical dimension of the face
Filling/restoration fractures
Tooth loosening
Implant loss
Tenderness/pain in teeth, jaw muscles
Headache
Disturbance of bed partner's sleep

At this time point there is no pharmacological protocol for the treatment or management of bruxism. Side-effects to different experimental pharmacological approaches for bruxism have been reported. Therefore, pharmacological treatment for bruxism is not a favourable option for most patients.[50] Some drugs that regulate blood pressure (such as clonidine) have a demonstrable inhibitory effect on jaw muscle activity during sleep, but other drugs, such as the selective serotonin re-uptake inhibitors (SSRI; anti-depressants) and dopamine antagonists (anti-psychotics) may actually increase bruxism.[50] The recent scientific literature has shown that this approach could be developed still because successful results with the use of Hydroxyzine to decrease bruxism in children have been reported. Hydroxyzine is usually used as an antihistamine, but it also has applications to decrease anxiety.[51]

Various forms of physiotherapy and more cognitive/behavioural treatment strategies are often used to deal with AB and SB. Hypnosis and acupuncture have also been proposed. Characteristically, the actual basis of bruxism—jaw muscle activity initiated from the central pattern generator—is not directly attacked by these treatments. Another option proposed for SB in this cognitive/behavioural approach is

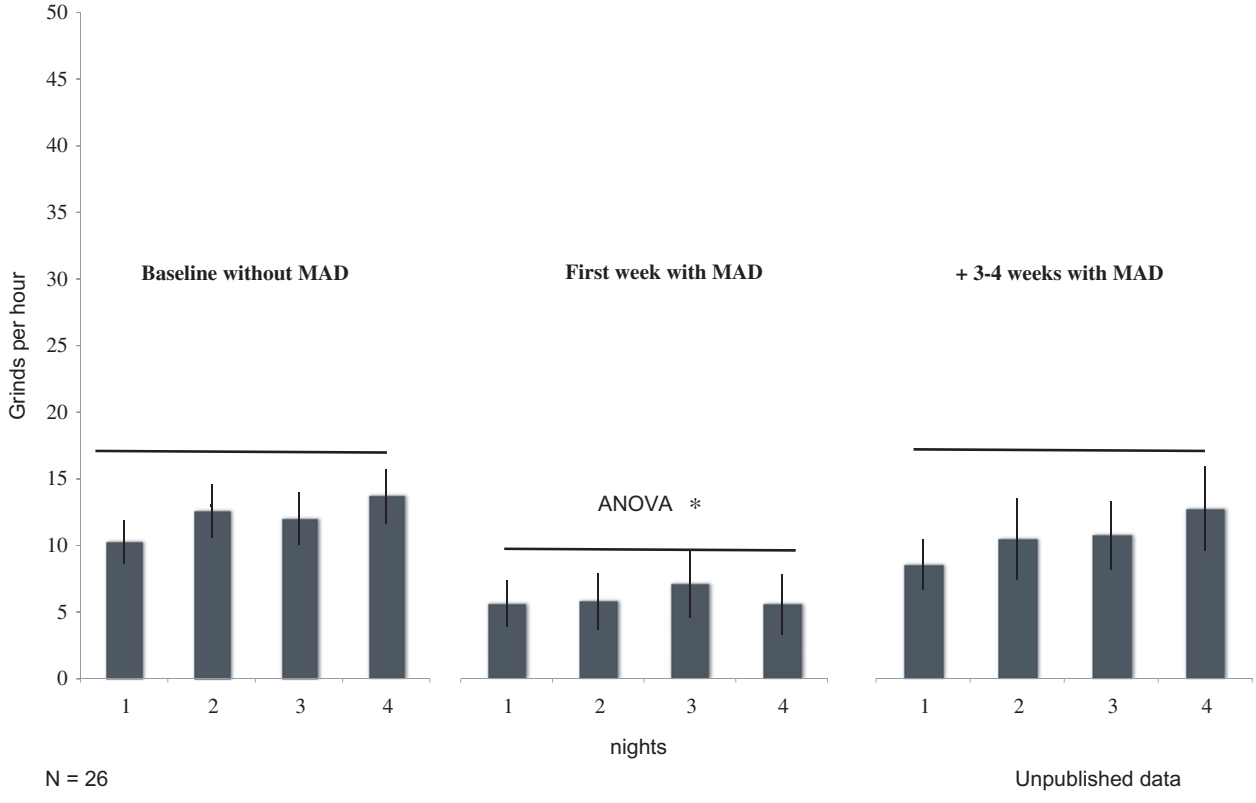

Figure 3. EMG activity (grinds/clench per hour ± SEM) of the masticatory muscles assessed during sleep with a portable EMG device. The graph shows the EMG activity before and during the use of a mandibular advancement device (MAD) in snorer patients ($n = 26$). ANOVA analyses show that there are significant differences in time (*) between weeks ($p < 0.01$). It can be appreciated that the EMG activity is decreased during the first week of use of the MAD and it returns to baseline levels after 3–4 months of continued use. There were no other statistically significant differences. Graph made from unpublished data.

'contingent electrical stimulation' (CES), which is a form of biofeedback that may induce a learning process while sleeping [52]. The muscle activity that triggers this electrical stimulation is based on measurement and assessment of the EMG signal from the temporalis muscle when the teeth are ground or clenched. The electrical stimulation causes a reflex inhibition of the muscle activity [32]. This reflex inhibition has been studied for decades and the neuronal circuits in the brain stem involved in this jaw muscle response have been specifically identified. In physiology, it is known as the 'exteroceptive suppression period'. Studies have shown that painless electrical stimulation of the various parts of the face triggers this reflex inhibition of the jaw-closing muscles [18,23]. CES may be, therefore, useful therapeutically to interrupt muscle activity. We have shown in a proof-of-concept study that CES causes an ~40% reduction in jaw muscle activity [23]. However, it remains unclear whether biofeedback is an effective method for treatment of SB [37] and/or if biofeedback therapy holds the potential for inducing long-term changes in behaviour that could include reduction or elimination of symptoms [1].

Recently published data from a pilot experiment using repeated transcranial magnetic stimulation (rTMS) aimed to investigate the effects of rTMS on the abnormal jaw muscle activities and jaw muscle soreness of patients with SB. The authors found out that the intensity of EMG was suppressed during and post rTMS compared to baseline and the ratings of soreness decreased compared with baseline values [53].

We can conclude based on the scientific knowledge available until now there is no definite treatment or cure for this parafunction. However, the dentists have some tools for managing the negative effects of bruxism. This parafunctional activity has a central nervous system aetiology that does not support local peripheral treatment/management/cure options (occlusal). Mouth guards may be used for the protection of teeth. Pharmacological approaches could perhaps be a future option, but, until now, it is still in early stages and so far is not readily available for the use by clinicians. Finally, the cognitive/behavioural approach has a great potential as an option and it is already available; nevertheless, up to this time point it has some limitations that still need further development.

In conclusion, the present selective, non-systematic but critical review has highlighted some of the most important findings in our understanding of bruxism. Clearly, there is a need for more research and developments of evidence-based guidelines for management protocols in clinical practice. We believe that, with the help of modern technology focused on personalized medicine, it will be possible to unveil and perhaps control better the pathophysiology of bruxism.

Declaration of interest

PS is a paid consultant for Sunstar, who manufactures Grindcare.

References

[1] Ilovar S, Zolger D, Castrillon EE, et al. Biofeedback for treatment of awake and sleep bruxism in adults: systematic review protocol. Syst Rev. 2014;3:42.

[2] Manfredini D, Restrepo C, Diaz-Serrano K, et al. Prevalence of sleep bruxism in children: a systematic review of the literature. J Oral Rehabil. 2013;40:631–642.

[3] Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010;109:e26–50.

[4] Manfredini D, Winocur E, Guarda-Nardini L, Lobbezoo F. Epidemiology of bruxism in adults: a systematic review of the literature. J Orofac Pain. 2013;27:99–110.

[5] Lobbezoo F, Ahlberg J, Manfredini D, Winocur E. Are bruxism and the bite causally related? J Oral Rehabil. 2012;39:489–501.

[6] List T, Axelsson S. Management of TMD: evidence from systematic reviews and meta-analyses. J Oral Rehabil. 2010;37:430–451.

[7] Basić V., Mehulić BruxismAn unsolved problem in dental medicine. Acta Stomat Croat. 2004;38:93–96.

[8] Marie MM, Pietkiewicz M. La bruxomanie. Rev Stomatol. 1907;107–116.

[9] Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: an international consensus. J Oral Rehabil. 2013;40:2–4.

[10] Reding GR, Rubright WC, Zimmerman SO. Incidence of Bruxism. J Dent Res. 1966;45:1198–204.

[11] American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Il: American Academy of Sleep Medicine; 2014.

[12] Lavigne GJ, Khoury S, Abe S, et al. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476–494.

[13] Svensson P, Lavigne GJ. Sleep Bruxism. In: Kushida C. (ed.) The Encyclopedia of Sleep. vol.4. Elsevier; 2013.

[14] Ohayon MM. Risk factors for sleep Bruxism in the general population. Chest. 2001;119:53–61.

[15] Maluly M, Andersen ML, Dal-Fabbro C, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. J Dent Res. 2013;92:S97–S103.

[16] Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res. 1996;75:546–552.

[17] Rompré PH, Daigle-Landry D, Guitard F, et al. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res. 2007;86:837–842.

[18] Jadidi F, Norregaard O, Baad-Hansen L, et al. Assessment of sleep parameters during contingent electrical stimulation in subjects with jaw muscle activity during sleep: a polysomnographic study. Eur J Oral Sci. 2011;119:211–218.

[19] Manfredini D, Ahlberg J, Castroflorio T, et al. Diagnostic accuracy of portable instrumental devices to measure sleep bruxism: a systematic literature review of polysomnographic studies. J Oral Rehabil. 2014;41:836–842.

[20] Koyano K, Tsukiyama Y, Ichiki R, Kuwata T. Assessment of bruxism in the clinic. J Oral Rehabil. 2008;35:495–508.

[21] Lobbezoo F, Naeije M. A reliability study of clinical tooth wear measurements. J Prosthet Dent. 2001;86:597–602.

[22] Wetselaar P, Lobbezoo F, Koutris M, et al. Reliability of an occlusal and nonocclusal tooth wear grading system: clinical use versus dental cast assessment. Int J Prosthodont. 2009;22:388–390.

[23] Jadidi F, Castrillon EE, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. J Oral Rehabil. 2008;35:171–183.

[24] Watanabe T, Baba K, Yamagata K, et al. A vibratory stimulation-based inhibition system for nocturnal bruxism: A clinical report. J Prosthet Dent. 2001;85:233–235.

[25] Baba K, Clark GT, Watanabe T, Ohyama T. Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain. 2003;17:58–64.

[26] Mizumori T, Inano S, Sumiya M, et al. Ambulatory bruxism recording system with sleep-stage analyzing function. J Prosthodont Res. 2009;53:150–154.

[27] Mainieri VC, Saueressig AC, Pattussi MP, et al. Validation of the Bitestrip versus polysomnography in the diagnosis of patients with a clinical history of sleep bruxism. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;113:612–617.

[28] Shochat T, Gavish A, Arons E, et al. Validation of the BiteStrip screener for sleep bruxism. Oral Surg Oral Med Oral Pathol Oral Radiol. 2007;104:e32–39.
[29] Castroflorio T, Deregibus A, Bargellini A, et al. Detection of sleep bruxism: comparison between an electromyographic and electrocardiographic portable holter and polysomnography. J Oral Rehabil. 2014;41:163–169.
[30] Yamaguchi T, Abe S, Rompré PH, et al. Comparison of ambulatory and polysomnographic recording of jaw muscle activity during sleep in normal subjects. J Oral Rehabil. 2012;39:2–10.
[31] Yachida W, Castrillon EE, Baad-Hansen L, et al. Craniofacial pain and jaw-muscle activity during sleep. J Dent Res. 2012;91:562–567.
[32] Jadidi F, Castrillon EE, Nielsen P, et al. Effect of contingent electrical stimulation on jaw muscle activity during sleep: a pilot study with a randomized controlled trial design. Acta Odontol Scand. 2013;71:1050–1062.
[33] Svensson P, Lavigne GJ. Sleep related bruxism In: Encyclopedia of Sleep. Kushida (CA); 2011.
[34] Lobbezoo F, Visscher CM, Ahlberg J, Manfredini D. Bruxism and genetics: a review of the literature. J Oral Rehabil. 2014;41:709–714.
[35] Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain. 2009;23:153–166.
[36] Johansson A, Johansson A-K, Omar R, Carlsson GE. Rehabilitation of the worn dentition. J Oral Rehabil. 2008;35:548–566.
[37] Lobbezoo F, van der Zaag J, van Selms MKA, et al. Principles for the management of bruxism. J Oral Rehabil. 2008;35:509–523.
[38] Manfredini D, Poggio CE, Lobbezoo F. Is Bruxism a Risk Factor for Dental Implants? A Systematic Review of the Literature. Clin Implant Dent Relat Res. 2012;16:460–469.
[39] Manfredini D, Ahlberg J, Mura R, Lobbezoo F. Bruxism is unlikely to cause damage to the periodontium: findings from a systematic literature assessment. J Periodontol. 2015;86:546–555.
[40] Long H, Liao Z, Wang Y, et al. Efficacy of botulinum toxins on bruxism: an evidence-based review. Int Dent J. 2012;62:1–5.
[41] Fernandes G, Franco AL, de Godoi Gonçalves DA, et al. Temporomandibular Disorders, Sleep Bruxism, and Primary Headaches Are Mutually Associated. J Orofac Pain. 2013;27:14–20.
[42] Svensson P, Jadidi F, Arima T, et al. Relationships between craniofacial pain and bruxism. J Oral Rehabil. 2008;35:524–47.
[43] De Laat AFC. Orofacial Pain: Taxonomy/Classification. Encyclopedia of Pain, Springer Berlin Heidelberg; 2013;2551–2553.
[44] Nixdorf DR, Drangsholt MT, Ettlin DA, et al. Classifying orofacial pains: a new proposal of taxonomy based on ontology. J Oral Rehabil. 2012;39:161–169.
[45] Svensson P. Pain mechanisms in myogenous temporomandibular disorders. Pain Forum. 1997;6:158–165.
[46] Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. 2001;28:1085–1091.
[47] Harada T, Ichiki R, Tsukiyama Y, Koyano K. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. J Oral Rehabil. 2006;33:482–488.
[48] Landry ML, Rompré PH, Manzini C, et al. Reduction of sleep bruxism using a mandibular advancement device: an experimental controlled study. Int J Prosthodont. 2006;19:549–556.
[49] Mainieri VC, Saueressig AC, Fagondes SC, et al. Analysis of the effects of a mandibular advancement device on sleep bruxism using polysomnography, the BiteStrip, the sleep assessment questionnaire, and occlusal force. Int J Prosthodont. 2014;27:119–126.
[50] Winocur E, Gavish A, Voikovitch M, et al. Drugs and bruxism: a critical review. J Orofac Pain. 2003;17:99–111.
[51] Ghanizadeh A, Zare S. A preliminary randomised double-blind placebo-controlled clinical trial of hydroxyzine for treating sleep bruxism in children. J Oral Rehabil. 2013;40:413–417.
[52] Arzi A, Shedlesky L, Ben-Shaul M, et al. Humans can learn new information during sleep. Nature Neuroscience. 2012;15:1460–1465.
[53] Zhou W, Fu H, Du Y, et al. Short-term effects of repetitive transcranial magnetic stimulation on sleep bruxism – a pilot study. Inter J Oral Science. 2015; (In press).
[54] Lavigne GJ, Cistulli P, Smith M. Sleep Medicine for Dentists: a Practical Overview. Quintessence Publishing; 2009.