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INVITED REVIEW

Bruxism: a summary of current knowledge on aetiology, assessment and management

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Abstract

Bruxism is a common condition that clinicians come across in both adult and children. Prevalence rates in adults range from 22% to 30% for awake bruxism (AB) and from 8% to 16% for sleep bruxism (SB), while in children they raise up to 40% for SB. Currently, bruxism is considered an 'umbrella term' for different jaw muscle activities, occurring during sleep and/or wakefulness. They have a different aetiology, but there is agreement on their central, not peripheral, origin. In otherwise healthy individuals, bruxism can be considered a muscle behaviour, which can be harmless or represent a risk and/or protective factor for clinical consequences, rather than being a disorder per se. Nonetheless, given the merging knowledge on the interaction with several associated factors and concurrent conditions, bruxism should be investigated for being a possible sign of an underlying primary condition. Consequently, treatment should be directed to the management of the possible clinical consequences and/or to the underlying primary conditions. It is generally based on the conservative strategies. The present manuscript summarises the available knowledge on bruxism aetiology, assessment and management for both SB and AB in adults and children, with focus on the future directions to implement the clinical relevance of bruxism researches.

Clinical relevance: A narrative overview summarising such a quickly evolving topic as bruxism may be useful to help clinicians understanding the complex relationship among bruxism, the possible underlying primary conditions, and the possible clinical consequences.

Introduction

Bruxism is a much-debated oral condition that interests several disciplines, such as dentistry, psychology, neurology and sleep medicine. Due to the constantly evolving knowledge and the different specialties involved in the study of bruxism, several definitions have been proposed over the past decades,[1,2] to the point that the need to find a common language emerged. After a first consensus paper dating back to 2013,[3] an international consensus meeting ('Assessment of bruxism status'), with bruxism experts from around the globe, took place in San Francisco, USA, in March 2017, prior to the 95th General Session & Exhibition of the International Association for Dental Research (IADR). The meeting led to an updated consensus paper, reporting the work in progress on the

development of bruxism knowledge.[4] As a first step, the experts provided separate definitions for sleep bruxism (SB) and awake bruxism (AB):

- Sleep bruxism is a masticatory muscle activity during sleep that is characterised as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals.
- Awake bruxism is a masticatory muscle activity during wakefulness that is characterised by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals.

These definitions implicitly suggest an ongoing paradigm shift. In particular, it must be underlined that both definitions begin with 'masticatory muscle activity' (MMA), a wording intended to emphasise that focus is put on motor phenomena, independently on any specific neurological correlates. This means that the definition of bruxism goes beyond the typical rhythmic masticatory muscles activity (RMMA) that has been associated with sleep arousals. Indeed, the clinical implications of bruxism are related to the role of different types of MMA during sleep and wakefulness as the source of potential consequences, if any. Besides, both definitions end with 'in otherwise healthy individuals'. This wording intends to point out that in individuals without any health concerns bruxism should not be considered a disorder (e.g. something that is always pathological or associated with negative clinical consequences), but rather a muscle behaviour that can have different aetiologies and that can be harmless, harmful or even protective with respect to several health outcomes.

Prevalence rates among adults range from 8% to 15% for SB and from 22% to 30% for AB; in younger populations, reported prevalence is higher (e.g. 40% to 50% of children and adolescents).[5–8] Nonetheless, in 2013, a systematic review on the prevalence of bruxism in adult populations cautioned about the interpretation and generalisation of findings due to the poor methodological quality of the reviewed literature, with particular regard to the amount of articles relying on single-item self-report to 'diagnose' bruxism.[5]

Current epidemiological knowledge is mostly related to SB. A large-scale polysomnography (PSG)-based epidemiological study[9] pointed out that the prevalence of SB was 5.5% when screened by questionnaires and confirmed by PSG, while it was 7.4% when PSG was used as an exclusive criterion for diagnosis regardless of the presence or absence of self-reported SB. As for AB, knowledge on its prevalence and natural course is poor, since data are available only from retrospective self-reports at single-observation points.[5] Such an approach may potentially lead to an imperfect estimate due to the absence of information on the frequency as well as to the patients' forced recall of their oral conditions during the time span covered by the report, which is usually very generic and refers to wide periods (e.g. days, weeks and months). To overcome this limitation, a recent investigation introduced the use of ecological strategies via smartphone to assess the frequency of AB behaviours.[10]

Within this framework, this manuscript will provide a narrative overview of available knowledge on bruxism aetiology, assessment and management. Some considerations on the difficulties to perform studies in children and the future directions of research will be also discussed.

Aetiology and pathophysiology

Current concepts on the aetiology of bruxism resemble the ongoing paradigm shift from peripheral to central regulation.[11–13] Part of bruxism activity is genetically determined, while an increase in bruxism activity may be associated with several potential risk factors and concurrent conditions.[14,15] In short, bruxism must be viewed as a muscle behaviour that reflects the presence of one or several underlying conditions or factors (i.e. 'a sign of something'). Different types of MMA may recognise different aetiology and be associated with different health outcomes, if any.[14,16]

Based on current knowledge, morphological factors (e.g. features of the facial skeleton and dental occlusion) are no longer considered important,[17] while increasing evidence suggests a role for a combination of several psychosocial, physiological/biological and exogenous factors.[18–26]

As for the psychosocial factors, stress sensitivity and anxiety have been associated with bruxism in several studies.[18–21] This relationship has been shown also by the presence of higher levels of urinary catecholamine in children and adults with bruxism.[27,28] In addition, having poor coping skills is a possible personality feature that has been associated with increased bruxism.[26]

The group of physiological/biological factors includes different neurochemicals and neurotransmitters that have been associated with sleep phenomena: dopamine reportedly has an inhibitory influence, whereas adrenaline and noradrenaline are

activators. Serotonin, amino gamma butyric acid, cholecystokinin and orexin are also considered RMMA modulators.[29–31] As discussed above, there is evidence for a genetic basis, but the inheritance model or genetic markers are unknown.[15]

Multiple exogenous factors can also influence bruxism. For instance, alcohol, smoking, caffeine, recreational substances and some drugs (e.g. selective serotonin reuptake inhibitors) may have an activating influence on SB.[32] Furthermore, bruxism is increased in the presence of concurrent conditions and disorders, such as Attention Deficit Hyperactivity Disorder (ADHD), Parkinson's disease,[33] Huntington's disease, dementia, epilepsy, gastroesophageal reflux and sleep disorders. For each condition, the interaction with bruxism is not fully elucidated yet.[34]

Thus, a multifactorial model is involved in the aetiology of bruxism, but it must be remarked that specific factors may have different relationships with the different types of MMA.[4] While SB features a combination of all bruxism activities (e.g. short- or long-lasting tonic clenching and phasic grinding, with or without teeth contact), AB is commonly characterised by teeth contacting habits or mandible bracing.[4,10] This means that purported aetiological factors may be also different with respect to the circadian manifestations of bruxism. While SB is centrally mediated, with a complex interaction of all factors influencing autonomic system function during sleep,[35–37] AB is mainly related to psychosocial factors.[18]

The study of bruxism pathophysiology also involves its relationship with the potential clinical implications. Pain in the jaw muscles or the temporomandibular joints (TMJ), prosthodontic complications and mechanical tooth wear are examples of potential negative outcomes due to bruxism.[2,38–41] On the other hand, it must be pointed out that all those conditions are multifactorial in origin. For instance, loss of hard dental tissue may be due to a combination of mechanical and/or chemical and intrinsic and/or extrinsic factors.[42] Evaluation of tooth wear is part of the clinical investigation in a bruxism diagnosis and there is sufficient evidence that bruxism can be a cause of tooth wear, even if tooth wear cannot be considered pathognomonic of bruxism.[3,4] Due to the multifactorial nature of tooth wear, diagnosis and treatment can be difficult, and a good clinical guideline is therefore essential, such as the recently described Tooth Wear Evaluation System (TWES).[42] Similarly, the relationship between bruxism and pain is controversial, with contrasting literature findings.[2] Indeed, while investigations on self-reported bruxism consistently found an association with pain, the few PSG and electromyography (EMG) studies did not replicate such findings. An explanation for the contrasting reports may be that PSG/EMG devices can only offer a count of SB episodes, without any information on the actual amount of muscle work or the behaviour during wakefulness. A possible confirmation of this hypothesis is obtained from a recent study[43] showing that patients with temporomandibular disorders (TMD)-related pain have elevated background levels of muscle activity during sleep, which may be an indicative of tonic, prolonged, low intensity mandible bracing that provokes exhaustion of muscle fibres and joint load. The amount of muscle work, in turn, is related to anxiety personality.[19] Based on these considerations, it is recommendable that future studies with a better discrimination between different bruxism activities are performed to get deeper into this issue.[16]

On the other hand, bruxism may even be associated to positive consequences. For example, in some patients, a certain amount of bruxism episodes occurs in correspondence with the end of respiratory arousals, possibly being instrumental to restore the patency of the upper airway while asleep.[30] The existence of an association between SB and Obstructive Sleep Apnoea (OSA) has been known for quite a while, but the mechanism underlying this association is still not entirely clear. A recent expert opinion paper[44] underlined the complexity of the SB–OSA relationship, with particular regard to the anatomical site of obstruction. In addition, gastroesophageal reflux occurs in patients with OSA and SB in 35% and 26% of cases respectively; in these patients, bruxism could reduce the risk of detrimental chemical tooth wear by increasing salivation.[1,45] In short, the interrelationship among bruxism, pain, tooth wear and concurrent sleep disorders is really complicated to evaluate at the individual level, especially considering that different health outcomes may co-occur.[46]

Bruxism assessment

With the aim of defining the advantages and limitations of the available diagnostic approaches, the international expert panel (see above) proposed a diagnostic grading for the operationalisation of bruxism diagnosis:

1. Possible sleep/awake bruxism is based on a positive self-report only.
2. Probable sleep/awake bruxism is based on a positive clinical inspection, with or without a positive self-report.

3. Definite sleep/awake bruxism is based on a positive instrumental assessment, with or without a positive self-report and/or a positive clinical inspection.[4]

It should be stressed, as reported by the authors, that for this recently introduced grading system, research is obviously needed to establish the reliability, validity and responsiveness to the change in this new system. In general terms, approaches for assessing bruxism can be distinguished as non-instrumental or instrumental. A combination of both approaches will likely emerge as the best available option.

Non-instrumental approaches

Non-instrumental approaches for assessing bruxism include self-report (questionnaires, oral history) and clinical examination, both for AB and SB.[4]

Self-report via structured questionnaires, interviews, and, more in general, self-reported measures may be useful to gather information on perceived bruxism activities and the possible associated factors. However, via self-report, the intensity and duration of specific masticatory muscle activity cannot be quantified easily.[47] One of the limitations is that the bruxism–psyche relationship could alter self-reporting, reflecting distress rather than masticatory muscle activity. The derived 'diagnosis' risks of having limited value because of its subjectivity, but it is nonetheless a basis for getting deeper into the diagnostic process.

For AB, the patients are asked to monitor their behaviour over a 1- or 2-week period after being informed of the possible conditions belonging to the spectrum of AB behaviours (i.e. clenching, bracing, thrusting, teeth contact habit). Such ecological momentary assessment (EMA) approach, also known as experience sampling methodology (ESM), improves the quality of data collection as it provides multiple time point reporting over an observation period.[48] Several studies[10,49–51] recommend the possible use of EMA strategies to report AB behaviours, to collect real-time report on specific oral conditions that are related to the spectrum of AB activities, while also allowing for the association of tooth contact habits with other conditions (i.e. masticatory muscle pain[52]).

Approaches for assessing SB also allow other options, since not only the patients themselves but also multiple informants can be interviewed, such as their bed partner or, in the case of children, their parents. The patient and partner are asked to monitor behaviour, preferably using a diary, concerning teeth grinding, teeth clenching and/or jaw bracing.

The clinical examination is divided into an extraoral evaluation and an intraoral inspection. The extraoral evaluation should assess the jaw muscles (e.g. evident muscle hypertrophy), the TMJ (e.g. disc position and joint degeneration), the presence of pain (e.g. teeth soreness and/or hypersensitivity, jaw muscle pain, TMJ pain, headache) and functional symptoms (e.g. difficulty to open the mouth wide on awakening).[4,53]

The intraoral inspection should comprehend a complete dental examination (e.g. tooth wear, tooth enamel chippings, cracks and fractures of natural teeth, restorations failure, periodontal ligament thickening) and an inspection of the cheek and tongue mucosa (e.g. linea alba, tongue scalloping, traumatic lesions).[53]

Instrumental approaches

Instrumental approaches for assessment are currently available for both circadian forms of bruxism.

Concerning AB, EMG recordings during wakefulness may theoretically provide measurements of AB, but such strategy is currently not easy to figure out due to the absence of dedicated devices on the market.[4]

To overcome these limitations, the use of the EMA principles has recently been maximised using smartphone applications, thanks to their user-friendly interface, thus opening up a new era for the EMA approach.[10,50,51] This data recording strategy, which has been created to collect real-time subjective information about jaw muscle activities at certain time points during wakefulness,[48] is useful both for research and clinical purposes. In the research setting, it allows gathering a huge amount of data on the epidemiology of different AB behaviours at the individual and population levels,[51] while in the clinical setting it helps patients to recognise their habits, monitor changes over time and implement corrective measures.[50]

As for SB, EMG recordings during sleep provide key evidence of motor activity and may also be complemented by other measures used in polysomnography, such as audio and/or video recordings.[4] Full PSG is of particular help to understand the neurophysiological correlates of SB events. Its use is not recommendable for routine cases due to the needed technical equipment, but it is fundamental when the presence of other sleep disorders (e.g. apnoea) is suspected. In

recent years, some EMG devices for in-home recordings emerged as a valid option for an easier approach to a definite diagnosis of the motor activity.[54–56] As an important note, given the progressive diffusion of portable EMG recordings devices, there is a need to define and standardise some technical and conceptual aspects. Issues of importance include the definition of EMG threshold above which a masticatory muscle activity is considered a SB event (e.g. percentage of the maximum voluntary contraction level; n times the relaxed baseline level; muscle activity level achieved during swallowing) and the choice of the EMG outcome measures. Classically, the number of EMG events exceeding an arbitrary threshold (as bursts, or clustered burst in episodes) is counted per hour of sleep to generate indexes. However, such data may only give a partial representation of the amount and pattern of muscle activity.[19,43,57] For a more comprehensive assessment, EMG outcome measures like power (area), peak amplitude and interval duration between activities could be included.[16] It would also be advisable to adopt measures that help distinguish clenching from grinding, although the practical and valid use of such outcomes needs to be confirmed.

Differential diagnosis

Table 1 reports a series of conditions for which a differential diagnosis with bruxism may be necessary.[58]

Management

Bruxism management must adhere to three basic principles:

- Bruxism may be a behaviour that does not mandate treatment[14,59];
- Indications to treat bruxism are mostly based on the presence of purported negative clinical consequences;
- Bruxism is always a sign of one or more underlying conditions. Thus, unless the specific cause is identified, treatment is oriented to the management of purported clinical consequences.[60]

In view of the above, from a clinical viewpoint, it is important that research efforts are directed towards the identification of treatment-demanding bruxism, with specific focus on the aetiology of the motor activities associated with clinical consequences. Current treatment approaches are mainly symptomatic strategies, and they aim to control and/or prevent the consequences of bruxism, especially as far as the stomatognathic system is concerned.[60,61] In general, evidence-based recommendations on bruxism management at the individual level are not yet available.

The authors of a recent qualitative systematic literature review on SB suggested that management should be based on common sense conservative approaches, referring to the so-called 'Multiple-P' approach as the standard of reference:

- Pep talk (counselling)
- Psychology (cognitive-behavioural strategies)
- Physiotherapy (exercises of the jaw muscles)
- Plates (oral appliances)
- Pills (drugs)[61]

Actually, such a 'Multiple-P' approach may be extended also to AB, with minor differences.

- **Pep talk**

Patients can play an active role in the self-care management of bruxism.[62,63] For this reason, it is important to explain them some concepts on bruxism pathophysiology and teach them some sleep hygiene instructions (e.g. reduction of caffeine, smoking and alcohol intake; avoidance of vigorous exercise or late-night working).

Concerning AB, patients should be informed that physiological conditions provide that tooth contact occurs only during chewing and swallowing, for a total of <17 min in 24 h.[64] Therefore, a conscious effort should be made to maintain a 'teeth apart' and 'relaxed jaw position' for the rest of the time. Given the importance of psychological factors, counselling should enhance stress coping skills and promote lifestyle changes.

- **Psychology**

The potential benefit of biofeedback (BF) and cognitive–behavioural treatment (CBT) to manage bruxism has always been advocated in the clinical setting, but recent studies do not seem to support their effectiveness.[8,65]

The most investigated cognitive–behavioural approach is BF, even if findings from the literature do not suggest real benefits on bruxism reduction,[65] with the possible exception of contingent electrical (CES).[8,60,66] These findings contrast with clinical perception of positive effects, which led Lobbezoo et al.[61] to include 'psychology' within the bruxism management strategies. To this purpose, it must be remarked that the recently introduced EMA approaches to bruxism assessment can also be used to introduce a habit reversal training, viz. ecological momentary intervention (EMI).[50] Consequently, even if not effective as stand-alone therapies, it is

Table 1 List of conditions for differential diagnosis with bruxism.

Condition	Definition	Differentiating signs/symptoms	Differentiating tests
Oromandibular dystonia	Oromandibular dystonia (OMD) is one of the many forms of dystonia, which is a group of conditions characterised by involuntary lasting severe muscle contractions, which lead to rhythmic and atypical movements in different parts of the body[107]	Slow, twisting, repetitive muscle spasms that affect the mandible, tongue and lips. Often associated with dystonia of the neck muscles (cervical dystonia/spasmodic torticollis), eyelids (blepharospasm) or larynx (spasmodic dysphonia). Sleep bruxism can also be present[58]	No differentiating testsClinical diagnosis[58]
Huntington's disease	Huntington disease (HD) is an autosomal dominant neurodegenerative disorder, characterised by affective, cognitive, behavioural and motor dysfunctions[108]	Hereditary neurodegenerative condition characterised by irregular, unpredictable choreatic body movements. Sleep bruxism may also be a featureNeurological evaluation identifies characteristic cognitive impairment (e.g. concentration impairment, task apathy and anxiety), behavioural features (e.g. irritability, impulsivity) and motor features (e.g. chorea, twitching/restlessness, bradykinesia/rigidity)[58]	Genetic testing confirms gene with an expanded trinucleotide CAG repeat (the mutant allele)[58]
Tourette's syndrome	Tourette syndrome (TS) is a complex disorder characterised by repetitive, sudden and involuntary movements or noises called tics[109]	Repetitive, irregular, stereotyped, suppressible movements (tics) of the eyes, face and neck. May occur during light non-rapid eye movement (non-REM) sleep, sleep stage shifts and micro-arousals and awakenings[58]	No differentiating testsClinical diagnosis[58]
Hemifacial spasms	Hemifacial spasm (HFS) is a neuromuscular disorder characterised by paroxysms of tonic or clonic contractions involving predominantly peri-ocular and perioral facial musculature[110]	Unilateral, non-epileptic twitches of the face also during sleep[58]	Needle EMG shows irregular, brief, high-frequency bursts (150-400 Hz) of motor unit potentials, which correlate with clinically observed facial movements[58]
Parkinson's disease	Parkinson disease (PD) is a progressive disorder of the nervous system. The disorder affects several regions of the brain, especially an area called the substantia nigra that controls balance and movement[111]	Multisystem neurological syndrome characterised by hypokinetic movements due to muscle stiffness and resting tremor. Caused by degeneration of the dopaminergic system. Swallowing difficulties and drooling may persist during sleep, whereas resting orolingual tremor is absent[58]	Dopaminergic agent trial shows improvement in symptoms[58]
Tardive dyskinesia	Tardive dyskinesia (TD) is a term historically used to refer to delayed and persistent abnormal movements caused by exposure to dopamine receptor blocking agents (DRBA). The typical pattern is a stereotyped combination of tongue twisting and protrusion, lip smacking and puckering, and chewing movements[112]	Neuroleptic-induced abnormal oromandibular movement disorder eventually associated with sleep bruxism. May feature any or all of movement of the lips and tongue (grimacing, smacking, pursing, sticking out the tongue), rapid blinking, impaired finger movement or 'fluttering', rapid movements of the arms, toe-tapping, moving the leg up and down, twisting and bending of the torso (in extreme cases)[58]	No differentiating testsClinical diagnosis[58]
REM-behaviour disorder	REM sleep behaviour disorder (RBD) is a parasomnia characterised by repeated episodes of sleep-related vocalisation and/or complex motor behaviours[113]	Acting out dramatic and/or violent dreams, so may involve limbs. Often involves grunting or shouting. Usually seen in men $\geq$ 60 years old[58]	Polysomnographic video recording shows increase in muscle tone associated with the EEG pattern of REM sleep (in contrast to the EEG pattern of REM sleep associated with an absence of muscle tone in healthy individuals). Video shows body movements coinciding with the EEG pattern of REM sleep[58]

recommendable that cognitive–behavioural approaches are included in any multimodal treatment protocol thanks to the favourable cost-to-benefit ratio.[60]

- **Physiotherapy**

Physiotherapy is an important treatment option in patients with pain and fatigue of the jaw muscles due to its twofold effect: it is effective in relieving pain and restoring muscle and joint mobilisation, and it also reinforces counselling or cognitive behavioural strategies.[67] Currently, a standard of reference physiotherapeutic regimen has not been established and the different protocols seem to have similar efficacy.[68]

- **Plates**

Oral appliances (OA) are commonly used, but evidence does not support a role in the long-term reduction of SB activity.[69–71]

Many types of OA are somehow reportedly effective to reduce SB activity at the short term.[72–74] This may suggest a potential 'novelty effect' associated with the use of an OA, which leads to a transient reduction in sleep-time MMA, possibly due to the need for re-organising motor unit recruitment. This hypothesis may find support in the observation that intermittent OA use is more effective than continued use.[75]

Clinically, OAs find indication also to prevent clinical consequences, such as in patients with severe and progressing tooth wear and/or repeated fractures or failures of dental restorations.[69] It is important that a full-arch appliance is provided, to avoid undesired changes in dental occlusion.[76,77] Considering the risk that OSA may be worsened with a stabilisation appliance, in patients with concur rent sleep-disordered breathing, prescription of appliances should be discussed with a sleep medicine specialist.[78–80]

Regarding AB, a 24-h use is not recommended because of the potential iatrogenic changes in the occlusal contact patterns and potential poor compliance by the patients. Nonetheless, as in the case of SB, the rationale for using them is to prevent damage from teeth contact, rather than a reduction in AB. Interestingly, OA could be used as part of a CBT regimen to teach patients to avoid any contacts and gain awareness of their AB behaviours.

- **Pills**

Although all the pharmacological approaches that have been investigated in the research setting (i.e. botulinum toxin, clonazepam, and clonidine) seem to reduce either the intensity or the number of SB with respect to placebo, drugs are not indicated as a first-step approach, due to the potential side effects associated with long-term use.[60,61,81]

More specifically, centrally acting drugs, such as the benzodiazepine clonazepam[82] and the anti-hypertension drug clonidine,[31,83] are both effective in reducing SB frequency. Botulinum toxin determines a reduced intensity of SB events, but does not affect their frequency, thus suggesting that it does not influence the genesis of SB episodes.[84–86] The low number of subjects included in the experimental protocols and cautionary considerations on the risk-to-benefit ratio suggest that clonazepam and clonidine should not be used for the long-term management of SB.[87] Amitriptyline, bromocriptine, levodopa, propranolol, and tryptophan have been tested, but due to lack of effect and/or potential severe side effects, they cannot be recommended for the management of SB.[22,23]

In general terms, given the emerging concepts that bruxism is not a disorder per se, it can be suggested that a better picture of the possible pharmacological management of SB will become progressively available with the advancement of knowledge on the aetiology and physiopathology.

As for AB, there is a paucity of data, since the only available investigation was directed to the short-term use of mild analgesics when severe jaw muscle pain is present.[58]

- **Cautionary statement**

Performing irreversible occlusal changes with the aim to reduce bruxism activities or to decrease pain symptoms in the jaw muscles and/or the TMJ is not recommendable,[8,17,61,88] in view of the fact that the association of bruxism with occlusal features is negligible, if at all present.[89,90]

Bruxism in children and adolescents

As in the case of the adult population, the reported prevalence of SB in children varies among different studies; indeed, all studies have internal validity problems, due to a SB 'diagnosis' being based on parental report.

Parental-reported tooth grinding, which may be viewed as a proxy of SB, is quite common in children, with prevalence up to about 40% when occasional tooth grinding is considered.[7,8] Prevalence data are variable: 3.5-8.5% in children aged under 5 years and <6% in children aged between 7 and 11 years.[7]

Only a few studies investigated the prevalence in adolescents: In a sample of Israeli adolescents, SB

and AB were reported by 9.2% and 19.2% of subjects, respectively, with no gender differences,[91] while a prevalence of 14.8% and 8.7% for SB and AB, respectively, has been reported in a sample of Dutch adolescents.[92]

Among the possible factors that are associated to SB in children, a recent review of the literature pointed out the role of concurrent sleep disturbances and second hand smoke.[93] Another review has also found an association with several other factors: male gender, genetics, anxiety, psychological reactions, some personality features (e.g. high sense of responsibility), restless sleep, sleeping with light on, noise in room, 'sleep hours $\leq$ 8 h', concurrent headaches, conduct problems, peer problems, emotional symptoms and mental health problems.[94] Primary researches are now focusing on the psychosocial environment of the child and the sleep architecture; a recent investigation showed that some sleep disorders and parasomnias are associated with parental-reported SB in children, while the influence of socio-economic layers on sleep behaviours is not relevant.[95] Similar results have been found in teenagers aged from 11 to 19 years: sleep disturbances (snoring in particular), headache, jaw muscle fatigue and tooth wear seem to be associated with SB.[96] Some studies showed that parental-reported bruxism could also be associated with perfectionism, aggressive behaviour, ADHD or antisocial behaviour[97] and unsteady family environment.[98]

For assessment purposes, despite the fact that PSG recordings represent the standard of reference to collect data on sleep correlates, parental-reported tooth grinding remains the most diffused option to perform studies on a large scale. The limitation of such an approach is quite intuitive. On the other hand, it must be borne in mind that children tend to be reluctant to enter sleep laboratories. Thus, the correlation of parental-reported tooth grinding with PSG findings is currently under study.[99,100] As for adults, current paradigm shift to the construct of bruxism as a muscle behaviour[4] suggests that the introduction of EMG measurement devices for home use could be recommended. While efforts are mainly directed to the study SB and its correlates, information on the frequency of AB and its associated factors in children is lacking. Thus, the use of EMA approaches may help getting deeper into this issue, at least in the adolescence age groups.

When parents complain of their child's sleep-time tooth grinding, they must be informed about the aetiology and pathophysiology. In case of severe tooth wear, and/or high frequency of parental-reported tooth grinding, special focus should be put on the search for comorbid conditions that may represent the actual health concern (e.g. respiratory disturbances, neurological disorders).

For management purposes, relaxation techniques may be the best option for young children (3 to 6 years), while oral appliances (e.g. mouth guards) are not recommended due to the ever-changing occlusal conditions.[101] Concerning pharmacological treatments, preliminary evidence indicates that hydroxyzine could be effective for parent-reported bruxism in children,[102] but its routine use is not recommendable for risk-to-benefit ratio. Based on the paucity of data, it is recommended that treatment indications and strategies are based on the identification of the underlying condition.[101,103,104]

Data on the natural course suggest that SB in children decreases progressively after the age of 9-10 years, and that most children do not keep on bruxing during adolescence and adulthood.[5,7] The fluctuation of bruxism over time[105] is influenced by the changes in the psychological or health conditions of the young patient; hence, the construction of a multidisciplinary framework is required to assess the course of bruxism in children and adolescences.[106]

Future directions

As pointed out in several sections of this manuscript, knowledge on bruxism is quickly evolving. The most important evolution regard the concept of bruxism itself, which is now viewed as an umbrella term for different jaw muscle activities, both during sleep and during wakefulness, that are not necessarily related to specific sleep correlates or with teeth contact.[4] This means that past literature will likely need a progressive re-evaluation as soon as new information on the different bruxism activities emerges. Within these premises, future studies are necessary to assess the prevalence of both SB and AB in adults and children, since most of the data available so far have been drawn from single-item, single-observation self- or parental report.

The concepts discussed in the 2018 consensus paper[4] will have an impact also on the assessment strategies. Indeed, there is a need to move on from the adoption of cut-off points to discriminate between bruxers and non-bruxers and embrace an evaluation based on the continuum of jaw muscles activities for SB or experienced behaviours for AB. Consequently, future studies should preferably be based on the measurement of the amount of

bruxism behaviour that increases (or reduces) the probability of any health outcomes.[4,16]

Finally, it is also necessary to develop an evaluation system based on a multidimensional assessment of the bruxism status and its aetiological factors. Such an evaluation system could be useful to define possible algorithms for clinical decision-making.

Conclusions

- Sleep and awake bruxism are masticatory muscle activities that occur during sleep and wakefulness respectively.
- In otherwise healthy individuals, bruxism should be considered a muscle behaviour that can be a risk and/or protective factor for clinical consequences, rather than a disorder per se.
- Different muscle activities included under the umbrella term 'bruxism' have likely different aetiology.
- In general terms, aetiology is mainly related to central factors rather than peripheral factors.
- To assess bruxism, both non-instrumental approaches and instrumental approaches can be used; a combination of both approaches may even emerge as the best available option, with special focus on the future design of multidimensional evaluation systems.
- Bruxism must be viewed as the sign of an underlying primary condition that may or may not require treatment. As such, treatment need is also based on the possible presence of clinical consequences. Discrimination between the causal treatment of any condition underlying bruxism and the management of bruxism consequences should be made, whenever possible.
- Management of bruxism and its consequences is generally provided with conservative strategies, viz. 'Multiple-P' approach.
- SB in children decreases progressively with age and, based on the natural course, the reference approach is based on observational and non-intervention strategies, unless it is the sign of treatment-demanding primary conditions.

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