Bruxism in children: What do we know? Narrative Review of the current evidence

INTRODUCTION

Bruxism is defined as a “repetitive Masticatory Muscles’ (MMs) activity characterised by the clenching or grinding of teeth and/or bracing or thrusting of the mandible” [Lobbezoo et al., 2018]. Such definition has been recently introduced by an experts’ consensus due to the need to properly described a common and much debated condition. Dental problems, oro-facial pain and dysfunctions, neurological disorders and obstructive sleep apnea are all conditions related to Bruxism [Manfredini et al., 2011; Svensson et al., 2008; Lavigne et al., 2008]. Therefore, an increasing interest has been raised around bruxism in order to identify risk factors and to try to standardise diagnosis and management. First of all, two distinct circadian manifestations of bruxism exist: Sleep Bruxism (SB) and Awake Bruxism (AB). SB represents a phasic or tonic MMs’ activity during sleep, while AB and is a MMs’ activity during wakefulness characterised by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible [Lobbezoo et al., 2018]. However, both types can no longer be categorically defined movement disorders in all individuals [Lobbezoo et al., 2018]. Unfortunately, up to now authors have focused their attention on bruxism mainly in adult population and lack of evidence still exists about paediatric bruxism. Furthermore, SB in children has been investigated deeper than AB and thus even more scarcity of data persists about this latter. Additionally, available studies adopt different diagnostic strategies and investigate populations with different characteristics. It can be argued that accurate estimations of bruxism’s features are complicated in children at the community level. Therefore, there is the need to summarise the available knowledge. Within this framework, the aim of the current manuscript is to overview the literature on paediatric bruxism and to try to outline the state of art about this condition.

MATERIALS AND METHODS

The current narrative review overviews the characteristics of both AB and SB in children. For this purpose, a search in PubMed has been performed using as key terms “bruxism”, “children”, “sleep”, “tooth wear”, “headaches”, “temporomandibular disorders”. Only full-length original publications dealing with human subjects with bruxism since 1990 have been taken into consideration. Case reports/series, commentaries and editorials have been excluded. A comprehensive and narrative investigation has been conducted throughout epidemiology, aetiology and pathophysiology, signs and symptoms, diagnosis and management of both AB and SB.
Epidemiology

Notwithstanding the prevalence’s rates of bruxism in children have been the target of several studies, a clear consensus is still nowadays complicated to assess. Manfredini et al. [2013] reported a wide range of variability in paediatric bruxism, from 5% to 40.6%. Similar results were found by Machado et al. [2014] in another systematic review, with a peak up to 50%. The variability of parameters used to assess paediatric bruxism contributes to the high variability in its prevalence. Indeed, instrumental and non-instrumental diagnostic approaches recognize different cut-offs. The former are recommended as more specific and sensitive, but the latters are clearly simpler although susceptible to different interpretations and evaluations. As reported by Simola P et al., [2010] bruxism prevalence notably changes when frequency of events is investigated. Additionally, as most studies are based on parental reports, parents’ education in recognizing clenching and grinding, and their frequency, of their children may be another significant variable [Lemos Alves et al., 2019].

Speculations about the role of age in bruxism’s prevalence is still debated and have not yet found strong consensus. Nevertheless, an inverse relationship between age and bruxism seemed to emerge [Manfredini et al., 2013]. Such observation may support the common belief that bruxism tends to diminish spontaneously with age in children. On the other hand, the pathological role of bruxism in children should be carefully investigated since proper MMs’ activity play a key role in facial growth and development [Storari et al., 2021]. Carra et al. [2011] further confirmed such results regarding SB, albeit, interestingly, found that AB increases among adolescents. Such evidence may support the role of stress, social impositions and fear of failure imposed by the modern society in the genesis of AB. Furthermore, it could be also hypothesised that the decrease in SB prevalence with age is due to less frequent parents’ visits to children bedroom during the night. Different interpretations in when bruxism is physiological and pathological may represent other confounding factors in estimating bruxism prevalence in children.

With respect to gender, no differences seem to exist [Huynh et al., 2009] albeit few studies reported an increased prevalence in males [Lam et al., 2011; Renner et al., 2012]. Similarly, socio-cultural environment did not emerge to significantly influence bruxism prevalence. Indeed, no differences were showed neither if considering developed cities compared to developing ones [Renner et al., 2012], nor if cities were compared to rural areas [Liu et al., 2005].

Etiology and pathophysiology

Multiple risk factors have been hypothesised to increase the likelihood of developing bruxism in children. While AB is mainly due to psycho-social factors, SB’s pathophysiology appears more complex and centrally mediated [Manfredini et al., 2020]. The onset of the repetitive MMs activity during sleep occurs contextually with micro-arousal — i.e. physiologic brain switches from sleep to aroused states with a rise in autonomous activities — but probably it is not directly under the influence of the cortex [Lavigne et al., 2007]. However, many unsolved issues still exist due to the lack of studies, both in adults but especially in children. Among those observed, sleep problems, parafunctional habits and psycho-social factors emerged to be the most likely associated factors with paediatric SB.

Significant correlations between bruxism and emotional states in young individuals were supported already fifty years ago [Lindqvist et al., 1972]. This relationship has been further strengthened by the evidence of increased catecholamines’ urinary levels in children reporting bruxism [Vanderas et al., 1999]. The same authors also reported a positive dose-response trend as the risk of developing bruxism appeared to increase with the increase of epinephrine and dopamine levels. Personality traits seem also to be strongly associated with SB in children. Children with SB revealed higher level of responsibility and neuroticism [Castroflorio et al., 2015]. Similarly, children living in an unhealthy family environment, as in cases of divorced parents, appeared more susceptible to bruxism, supporting the negative role played by anxiety and stress [Castroflorio et al., 2015; Petit et al., 2007; Rossi et al., 2013].

Parafunctional habits such as biting objects were reported moderately associated with both paediatric SB and AB [Carra et al., 2011; Castroflorio et al., 2015]. It is also well known that such behaviours often represent reactive responses to psychosocial factors in bruxism [Manfredini et al., 2020]. Children with both SB and AB reported sleep disturbances more frequently than otherwise healthy individuals. Sleeping less than 8 hours per night, frequent awakenings, long sleep latency, restless sleep and having the light on or noises meanwhile sleeping are also reported to increase the risk of SB in children [Carra et al., 2011; Castroflorio et al., 2015]. Significant correlations between SB and Sleep Disordered Breathing (SDB) — habitual snoring and Obstructive Sleep Apnea — and nasal obstruction has emerged also in children and adolescents [Ng et al., 2002; Grechi et al., 2008; Di Francesco et al., 2004].

Additionally, heavy exposition to passive smoking emerged to be significantly associated with SB [Castroflorio et al., 2015]. By contrast, dental occlusion and facial skeleton morphology are no longer considered risk factors for bruxism [Manfredini et al., 2020]. Even among children the biological plausibility concerning the cause-and-effect hypothesis of such correlation was not demonstrated [Vanderas et al., 1995]. In such a scenario, early occlusal treatments are not scientifically supported to prevent or control bruxism in children. Few studies have investigated the role of hereditary predisposition in bruxism. Recently, polymorphisms in genes involved in dopamine metabolism have been found to be associated with bruxism in children, namely Dopamine Receptor D2 (DRD2), Ankyrin repeat and kinase domain containing 1 (ANKK1) and Catechol-O-methyltransferase (COMT) [Scarrot et al., 2022]. Similarly, polymorphisms in the gene Actinin Alpha 3 (ACTN3), also identified to be positively associated with bruxism in adults [Nicot et al., 2021], have been proposed to contribute to bruxism etiology even in children [Kücher et al., 2020]. Overall, the phenotypic variance attributable to genetics emerged to be high in children [Hublin et al., 1998]. Bruxism was also demonstrated to be a persistent trait, as up to 86% of adults with bruxism reported it also during the childhood [Hublin et al., 1998]. Additionally, children whose parents had a positive history for bruxism when they were children emerged 1.8 times more likely to develop bruxism [Cheifetz et al., 2005].

Signs, symptoms and comorbidities

Bruxism is characterised by several signs and symptoms that are not but always diagnosed all together. Tooth wear is often present in children with both AB and SB [Carra et al., 2011; Castroflorio et al., 2015]. Teeth may appear somewhat flat on incisal and occlusal surfaces — teeth wear facets — with an irregular pattern [Restrepo et al., 2006]. However, tooth wear is not pathognomonic for bruxism as it could be only the sign of grinding [Lobbezoo et al., 2018] and it might be due to other clinical conditions [Corica et al., 2014]. Clinicians need
additionally to be aware of tooth fracture, chippings or cracks, failure of restorations, widening of periodontal ligament, linea alba, teeth impressions on the tongue and traumatic lesions [Manfredini et al., 2020].

A close relationship exists between bruxism and Temporomandibular Disorders (TMDs) in both adults [Manfredini et al., 2020] and children [Toscano et al., 2009]. Indeed, functional musculoskeletal limitations commonly accompany bruxism: MMs hypertrophy, soreness and pain, difficulties in opening the mouth, joint sounds and/or pain [Fernandes et al., 2012; Manfredini et al., 2012]. Despite the limited number of studies, this correlation seems to be confirmed in children and supported by a biological plausibility as bruxism mechanisms are somewhat similar in both adults and children [Manfredini et al., 2020]. Children with SB and AB experienced higher frequency of joint click, muscles fatigue and difficulties in yawning than control subjects [Carra et al., 2011]. In a recent meta-analysis, children with bruxism emerged almost 3 times more likely to develop TMDs if compared to non-SB individuals (OR 2.97, 95% CI 1.72±5.15) [De Oliveira Reis et al., 2019].

Headaches are frequently found in children with SB compared to otherwise healthy subjects [Carra et al., 2012; Corvo et al., 2003]. Children suffering from tension-type headache were demonstrated to report SB by far more frequently than control subjects [Vendrame et al., 2008]. Similarly, migraine emerged to increase the risk of suffering from sleep disorders in children, including SB [Miller et al., 2003]. However, the cause-and-effect of such association is still debated and need to be further clarify.

SB has been described together with behavioural problems such as Attention Deficit Hyperactivity Disorder (ADHD), drowsiness and poor school results [Carra et al., 2011; Chiang et al., 2010]. ADHD patients frequently suffer from concomitant sleep disturbances, especially SD [Walters et al., 2008] and are prescribed with medications that increase the risk of SB [Malki et al., 2004]. In these occasions SB may be merely a secondary sign. However, a close connection has been described between ADHD and other sleep-related movement disorders, such as Restless Leg Syndrome [Walters et al., 2008], thus a direct link between SB and ADHD should not be neglected.

The impact of bruxism on children’ quality of life has not been profoundly evaluated and controversies have emerged. Only one study pointed out that SB negatively affect the quality of life in children [DeAlenca et al., 2017]. In particular, the self-perception and the social interaction appeared to be the most significantly involved domains. By contrast, other authors did not find any correlation [Antunes et al., 2015].

Diagnosis

Diagnosis of bruxism is generally complex and mainly clinical [Lobbézoo et al., 2017]. Recently, an international scientific consensus was reached to standardize the diagnostic approach for bruxism [Lobbézoo et al., 2018]. Reportedly, the combination of instrumental and non-instrumental assessments is necessary. Although more feasible with adults, the same or somewhat similar rules may be relevant for children too.

Up to now, the most reliable diagnostic tool in children is the report of teeth grinding by parents or caregivers [Manfredini et al., 2020]. However, most children sleep away from their parents and Parents who keep their bedroom door open were demonstrated to refer a 1.7 times greater incidence of SB in their children than those who keep it closed [Cheifetz et al., 2005]. Questionnaires or interviews should investigate frequency and intensity of any dental clenching or grinding, both during wakefulness and sleep. Taken medications and/or potential comorbidities, especially sleep disturbances and psychiatric disorders, need to be addressed too. Parents might be asked to keep a daily diary to monitor their children’s behaviour and habits. An oral interview must be accompanied by an accurate clinical examination. Clinicians must search for lesions on soft tissues - cheeks and tongue – and teeth - wear and fractures. MMs hypertrophy and temporomandibular complaints should not be neglected [Manfredini et al., 2020]. Furthermore, micrognathia, retrognathia, macrognathia and hypertrophy of the tonsils and adenoids might be assessed due to their pathogenetic role in SDB [Guileminault et al., 2018].

Electromyography (EMG) measurements have been proposed as a potential instrumental tool to evaluate bruxism as it represents a muscular behaviour. Both during wakefulness and sleep — in this case supplemented with video and audio recordings — EMG outcomes display a plausible behavioural pattern of MMs activity about frequency and magnitude. Despite that, many questions still exist concerning cut-offs and the actual interpretation of the data, already in adults [Lobbézoo et al., 2018]. The scarcity of literature on children further complicates the assessment. Additionally, it must be kept in mind that children tend to be uncooperative and reticent to enter sleep laboratories.

Management

Currently, no evidence exists to support any kind of therapeutic options for bruxism in children [Restrepo et al., 2009]. In children bruxism may be considered a behaviour that just need to be followed over time. Moreover, given that SB is suggested to progressively decline after childhood in the majority of children, management should be based on the identification of the underlying condition. If any, sleep disorders and/or psychological disturbances must be pointed out and carefully managed. When complaints are reported and/or signs of damage to orofacial structures appear, then conservative approaches are recommended.

Psychological and muscular relaxations techniques emerged effective in reducing signs of bruxism in children under 6 years of age [Restrepo et al., 2001]. Unfortunately, self-awareness education and biofeedback to control bruxism may still be a challenge in children. Similarly, although widely used for treating bruxism in adults, Oral Appliances (OAs) have not been proven in children with bruxism [Restrepo et al., 2009].

Probably, the cause lies in the concerns regarding the restriction induced over maxillary growth. Despite that, in children with severe TMD sign and symptoms, the use of an OA with a central expansion screw to allow normal development has been recently suggested [Viscuso et al., 2020].

As pointed out by Castroflorio et al. [2015], sleep habits may have a relevant role in paediatric bruxism pathogenesis, thus sleep hygiene measures must not be neglected. Furthermore, in case of SDB diagnosis or suspicion, appropriate treatment should be advised. Indeed, tonsillectomy and adenotonsillectomy emerged effective in reducing the frequency of SB in paediatric patients [Di Francesco et al., 2004; Eftekharian et al., 2008]. Limited number of evidence exist concerning medications used to control bruxism. Hydroyzine appeared effective in reducing bruxism in children, probably by increasing sleep depth, relaxing muscles and decreasing anxiety. Nevertheless, studies supporting efficacy and safety of hydroyzine for long-term therapies in children are lacking [Gale et al., 2016; Bruni et al., 2018].
In the light of such scenario, a combined multidisciplinary approach should be advocated to manage bruxism and its comorbidities in children. Moreover, the cooperation of parents is unavoidable.

Conclusions
Bruxism represents a common finding during childhood whereas several aspects need to be further investigated. At this point, however, the construct about the nature of bruxism has clearly shift from occlusal beliefs to a biopsychosocial model even in children. Sleep, personality, anxiety and stress, and headaches are the factors towards whom research questions must be addressed in order to better define diagnostic tools and management strategies.

References