



BRUXISM AND TEMPOROMANDIBULAR DISORDERS IN CHILDREN: AN OVERVIEW.

Orthodontics

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ABSTRACT

Bruxism is nonfunctional clenching or grinding of the teeth. It is a destructive habit that may result in tooth wear. Although research on bruxism is extensive, its etiology remains debatable. The literature suggests that bruxism is correlated with both pathologic and psychological conditions. The purpose of this review is to enlighten the scientific community about the increasing cases of bruxism in children, etiology and treatment of the same. The article also emphasizes the role of dentists in identifying and correcting certain factors that can cause bruxism.

KEYWORDS

INTRODUCTION:

According to the American Academy of Sleep Medicine, bruxism has been defined as “repetitive jaw muscle activity characterized by the clenching or grinding of teeth and/or bracing or thrusting of the mandible”.¹ There have been different updates regarding the definition of bruxism based on etiology and whether it is sleep bruxism or daytime bruxism.

Sleep bruxism was defined as a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic), while awake bruxism is a masticatory muscle activity during wakefulness, characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible.² It is not a disease, but when severe may lead to an imbalance of the stomatognathic system. Many treatment modalities have been suggested, but there is no consensus about the most efficient.

The pathophysiology of bruxism is still not clear. It is considered multifactorial with potential influences of the central nervous system (CNS), including oral motor activities, regulation of sleep-wake cycle, autonomic and catecholaminergic as well as genetic and psychosocial influences. The role of dental occlusion remains confusing.³

“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.”⁴

Sleep bruxism is subject to constant discussion not only among dentists, but also in other health areas due to potential etiologic associations. Epidemiological studies with different methodologies and populations have been conducted, for this reason, the prevalence of sleep bruxism varies in different age groups. In young adults aged between 18 and 29 years old, it is of 13%, reducing to 3% in individuals over 60 years of age.⁵ Still, when sleep bruxism is related to children, major doubts remain. Due to variations in the prevalence of bruxism in children, a systematic and critical analysis of current literature is necessary to obtain more accurate data. Thus, the aim of this review is to discuss, based on scientific evidence, the patterns of bruxism in children, etiology, diagnosis and possible treatment planning which will greatly improve the quality of life of the suffering child and aid in its balanced growth and development physically and psychologically.

Diagnosis of bruxism The bruxism diagnosis is complex and should

be performed with validated tools. In this study, the measurement tool was considered validated when there was a distinction between sleep and awaking bruxism and used for the diagnosis of bruxism guidelines from the American Academy of Sleep Medicine (AASM). Only three studies followed these requirements. In regard to sleep/awake bruxism, Lobbezoo et al proposed the following diagnostic grading: “possible,” when is based only on a positive self-report; “probable,” when is based on a positive clinical inspection (findings in physical examination that justify the habit), with or without a positive self-report; or “definite,” when is based on a positive instrumental assessment (electromyography for awake bruxism and polysomnography for sleep bruxism), with or without a positive self-report and/or a positive clinical inspection. Restrepo, Gomez, and Manrique however reported that polysomnography is not a representative examination in children, and it is costly and time-consuming. In 1990, Marbach et al had already suggested that basing the diagnosis of bruxism on patient self-report is potentially tendentious, depending on what the dentist may have asked the patient. Regarding children, the report from parents or guardians can also limit the diagnosis. In diagnosing bruxism, the reviewed studies used either a clinical examination, an interview/questionnaire, or both. The clinical indicators of diagnosing this parafunction were the presence of dental wear/attrition and bruxofacets. The historical indicator of bruxism was grinding or clenching reported by the subject or parent during the interview or on the questionnaire.^{6,7,8}

Etiology:

Similar to adults, the etiology of Sleep bruxism in children is multifactorial, involving central elements and associations with some sleep disorders. The literature reports an association with habitual snoring, somniloquy (talking while sleeping), nightmares and can change a child's internal biological clock, thus affecting sleep duration and quality.

From a broader perspective the etiology can be malocclusions, psychological conditions and anxiety, disturbed sleep patterns and certain systemic conditions leading to generalized stress in the child.

The etiology of bruxism has been attributed to systemic factors such as intestinal parasites, subclinical nutritional deficiencies, allergies, and endocrine disorders; to local factors, especially malocclusion; and to psychological factors. It has been reported that bruxism is an initiating factor of craniomandibular dysfunction in adults. Also, studies conducted on children and adolescents reported significant correlations between this parafunction and signs and symptoms of craniomandibular dysfunction.

The hypothesis tested by the reviewed studies is that malocclusion,

especially occlusal interferences, can initiate and maintain forceful clenching or grinding of the teeth. Statistically significant correlations were found between different types of morphologic malocclusion such as Class II and III molar relationship, deep bite, overjet, and dental wear or grinding. Mediotrusion interferences, anterior-posterior and vertical distance between retruded and intercusp positions, and lateral shift of the mandible together with nonfunctional side interferences were the types of functional malocclusion correlated with dental wear or bruxofacets.

The main non-dental cause of pain in the orofacial region among children and adolescents is temporomandibular disorders (TMD), which is defined as a set of disorders involving the masticatory muscles, temporomandibular joint, and associated structures. The prevalence of TMD in children and adolescents is pretty high. The remarkable variation in both TMD and bruxism prevalence can be attributed to different research methodologies, clinical criteria for diagnosis, population samples and examination procedures. In addition, studies with children are even less enlightening about the extent to which bruxism may be related to TMD. Although the studies showed high risk of bias, the meta-analysis showed that children with bruxism have greater chance of developing TMD. Future studies with better methodological criteria and validated diagnostic tools are needed.

There is also evidence that, in younger children, bruxism may be a consequence of the immaturity of the masticatory neuromuscular system. No catecholamine measurement of any type was performed on the patients in this report. Nevertheless, Vandas and others have demonstrated that stress and anxiety may be directly related to bruxism, as patients suffering from bruxism show a higher catecholamine level, generally ascribed to emotional stress. Also, it is important to assess the impact of psychiatric disorders on childhood parasomnias.^{9,10,11,12}

Treatment of Bruxism: Pharmacological

Five medications were tested for treating sleep bruxism. Flurazepam (15 mg/day) was compared with a placebo in a clinical crossover trial, and individuals reported a decrease in the occurrence of bruxism via self-report. Hydroxyzine (25–50 mg/day and 5–25 mg/day) used for 4 weeks shows a reduction in the parent-reported bruxism score. However, a decrease in the Clinical Global Severity score was more significant for hydroxyzine than the placebo. Similarly, Trazodone (0.5 mg/kg/day) presented a significant reduction in the frequency of bruxism and morning pain after 2 and 4 weeks of intervention. One clinical case was reported using imipramine (25 mg/day) for 1 month and showed a reduction in parent-reported bruxism via the visual analog scale. On the other hand, diazepam (2.5 and 5 mg/day) was also tested and did not show a statistical difference from the placebo in an RCT, despite the fact that both the placebo and the diazepam were able to decrease self-reported bruxism. Various side effects of these drugs were reported, including drowsiness, nausea, vomiting, irritability, dry mouth, insomnia, confusion, aggression, headache, and diminished appetite. The studies on medications did not monitor the individuals for long periods (generally up to 4 weeks) to observe if signs of bruxism returned after the intervention ended.^{13,14}

Psychological intervention One study evaluated two psychological approaches as interventions for reducing bruxism. The techniques used were targeted muscle relaxation and reaction competence, which were applied to 33 children in a nonrandomized clinical trial. The authors report that both approaches were able to reduce parent-reported bruxism, although no comparisons were performed between the two techniques. These therapies did not present contraindications or side effects.^{15,16}

Physical therapy intervention Quintero et al. conducted a randomized clinical trial, evaluating in 26 children (3 to 6 years old) the effectiveness of physical therapy to reduce bruxism. Reduced occurrence of parent-reported tooth-grinding or tooth-clenching during sleep was observed by 77% in the physical therapy intervention group and 15.38% in the control group. The authors suggested that physical therapy intervention can be considered as effective in reducing self-reported bruxism in young children, without reported side effects.^{17,18}

Occlusal splints/appliances Four studies were found evaluating occlusal splints/appliances. The use of the occlusal splint correlated to a reduction in self-reported bruxism on a visual analog scale, in 30 children (7 to 10 years old). Self-reported bruxism by parents decreased by 76.7%. Moreover, a significant reduction in the signs of bruxism (headache and muscular discomfort) were observed with the use of the bite guard, although bite-guard wear and tear was not observed in any patient in this case series study. Similar results were observed in a clinical case of a 5-year-old girl with chronic headaches due to bruxism. The girl's bruxism reported by parents stopped during the use of the occlusal splint and morning headaches also decreased. On the other hand, Restrepo et al. evaluated even younger children (3 to 4 years old) and observed, after the use of the rigid occlusal splints, a 20% reduction in parent-reported bruxism which was not statistically different from the mean of the control group (15%). Also, dental wear and tear and pain, when opening the mouth, did not differ between the control and experimental groups. Although most of the studies only used the self-report to observe the reduction in bruxism, a randomized crossover study investigated the use of a mandibular advancement device in three different positions (neutral/advanced/free splints) for 1 week, evaluating the reduction in sleep bruxism measured by RMMA by way of a polysomnographic device. This study follows 16 adolescents (mean age of 14.9) and observed a significant reduction in the RMMA index, although no differences were observed in relation to the three tested positions. Moreover, a reduction in the intensity of headaches was observed in 57% of adolescents.^{19,20,21}

Orthodontic interventions Three studies employing orthodontic interventions were found and fulfilled the eligibility criteria, where one used fixed appliances and two used rapid maxillary expansion. As for the fixed appliance, 50 children and adolescents (7 to 16 years old) that used this appliance demonstrated a reduction in self-reported bruxism (9%) and a reduced prevalence of headaches (13%). Similarly, rapid maxillary expansion for 6/8 months (in 12 children—4 to 11 years old) was able to produce a significant reduction, observed in self-reported bruxism (58.4% of children). Bellerive et al. used non-instrumental (clinical examination) and instrumental methods (RMMA index via polysomnography and electromyography) to diagnose sleep bruxism and observed a significant reduction in the RMMA index and episodes (in 65% of children and adolescents between 8 and 14 years old) after palatal expansion.^{22,23,24}

CONCLUSION:

Various studies show there is an increasing incidence of bruxism in children. It is the clinicians responsibility to be aware about the etiology, diagnosis and treatment of bruxism to help make the child's quality of life better.

REFERENCES:

1. Pingitore G, Chrobak V, Petrie J. The social and psychologic factors of bruxism. *J Prosthet Dent* 1991; 65(3):443–6.
2. Attanasio R. Nocturnal bruxism and its clinical management. *Dent Clin North Am* 1991; 35(1):245–52.
3. Nilner M. Prevalence of functional disturbances and diseases of the stomatognathic system in 15–18 year olds. *Swed Dent J* 1981; 5(5-6):189–97.
4. Nilner M, Kopp S. Distribution by age and sex of functional diseases of the stomatognathic system in 7–18 year olds. *Swed Dent J* 1983; 7(5):191–8.
5. Laberge L, Tremblay RE, Vitaro F, Montplaisir J. Development of parasomnias from childhood to early adolescence. *Pediatrics* 2000; 106(1 Pt 1):67–74.
6. Bayardo RE, Mejia JJ, Orozco S, Montoya K. Etiology of oral habits. *ASDC J Dent Child* 1996; 63(5):350–3.
7. Vandas AP, Manetas KJ. Relationship between malocclusion and bruxism in children and adolescents: a review. *Pediatr Dent* 1995; 17(1):7–12.
8. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 2001; 28(12):1085–91.
9. Monaco A, Ciammella NM, Marci MC, Pirro R, Giannoni M. The anxiety in bruxer child. A case-control study. *Minerva Stomatol* 2002; 51(6):247–50.
10. Graf H. Bruxism. *Dent Clin North Am* 1969; 13(3):659–65.
11. Kopp S. Pain and functional disturbances of the masticatory system — a review of etiology and principles of treatment. *Swed Dent J* 1982; 6(2):49–60.
12. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. *J Prosthet Dent* 1984; 51(4):548–53.
13. Restrepo CC, Alvarez E, Jaramillo C, Velez C, Valencia I. Effects of psychological techniques on bruxism in children with primary teeth. *J Oral Rehabil* 2001; 28(4):354–62.
14. Okeson JP. Temporomandibular disorders in children. *Pediatr Dent* 1989; 11(4):325–9.
15. Hachmann A, Martins EA, Araujo FB, Nunes R. Efficacy of the nocturnal bite plate in the control of bruxism for 3 to 5 year old children. *J Clin Pediatr Dent* 1999; 24(1):9–15.
16. Pidcock FS, Wise JM, Christensen JR. Treatment of severe post-traumatic bruxism with botulinum toxin-A: case report. *J Oral Maxillofac Surg* 2002; 60(1):115–7.
17. Vandas AP, Papagiannoulis L. Multifactorial analysis of the aetiology of craniomandibular dysfunction in children. *Int J Paediatr Dent* 2002; 12(5):336–46.
18. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. *J*

- Behav Med 1980; 3(4):385-7.
19. Marbach JJ, Raphael KG, Janal MN, Hirschhorn-Roth R. Reliability of clinician judgements of bruxism. *J Oral Rehabil* 2003; 30(2):113-8.
 20. Lobbezoo F, Ahlberg J, Glaros Ag, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil* 2013; 40(1): 2- 4.
 21. 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.
 22. Bader G, Lavigne GJ. Sleep bruxism: an overview of an oromandibular sleep movement disorder. *Sleep Med Rev* 2000; 4(1): 27- 43.
 23. Manfredini D, Restrepo C, Diaz- Serrano K, Winocur E, Lobbezoo F. Prevalence of sleep bruxism in children: a systematic review of the literature. *J Oral Rehabil* 2013; 40(8): 631- 642.
 24. Antonio AG, Pierro VS, Maia LC (2006) Bruxism in children: a warning sign for psychological problems. *J Can Dent Assoc* 72: 155-159
 25. Restrepo CC, Vásquez LM, Alvarez M, Valencia I (2008) Personality traits and temporomandibular disorders in a group of children with bruxing behaviour. *J Oral Rehabil* 35:585-593
 26. Cheifetz AT, Osganian SK, Allred EN, Needleman HL (2005) Prevalence of bruxism and associated correlates in children as reported by parents. *J Dent Child (Chic)* 72:67-73
 27. Manfredini D, Serra-Negra J, Carboncini F, Lobbezoo F (2017) Current concepts of bruxism. *Int J Prosthodont* 30:437-438.
 28. Katsaros C (2001) Masticatory muscle function and transverse dentofacial growth. *Swed Dent J Suppl*:1-47