Bruxism and Children - A Review

Ateet Kakti

ABSTRACT

Bruxism is very common in children. One in five children up to the age of 11 years are reported to have sleep bruxism, although the real figure is probably higher as parents often are unaware. Children who brux usually begin at 4–8 years of age, the numbers increase between 10 and 14 years of age and then start declining after the age of 14 years. Many factors associated with children who brux are anxiety, personality, attention-deficit/hyperactivity disorder, and other parafunctional habits. The most important characteristic of bruxism is that there is non-functional contact of mandibular and maxillary teeth resulting in clenching or grating of teeth. Nonetheless, the paper contains references to a number of management strategies that may be useful in protecting the dentition, periodontium, and the jaw musculature and temporomandibular joint from this potentially destructive behavior.

Keywords: Anxiety, Bruxism, Children, Clenching, Grating, Grinding, Occlusal, Stress.

How to cite this article: Kakti A. Bruxism and Children - A Review. Int J Prev Clin Dent Res 2018;5(2):S58-62.

Source of support: Nil

Conflicts of interest: None

INTRODUCTION

The term bruxism is said to come from the Greek word "brychein," which means "to grind or gnash the opposing rows of upper and lower molar teeth." Bruxism is involuntary, excessive grinding, clenching, or rubbing of teeth during non-functional movements of the masticatory system.^[1,2] Reported prevalence in children ranges from 7% to 15.1%,^[3-5] with girls apparently more frequently affected.^[6] Bruxism can occur during the day or night.^[7] In general, patients clench their teeth throughout the day and gnash and clench them during sleep.^[8] However, nocturnal bruxism is more frequent; it varies with the individual and has been related to emotional or physical stress.^[9] Bruxism usually causes tooth wear as evidenced by wear facets that can range from

Lecturer

Department of Preventive Dentistry and Pediatric Dentistry, Riyadh Elm University, Riyadh, Kingdom of Saudi Arabia

Corresponding Author: Ateet Kakti, Lecturer, Department of Preventive Dentistry and Pediatric Dentistry, Riyadh Elm University, Riyadh, Kingdom of Saudi Arabia. e-mail: ateetprof@gmail.com

mild to severe and can be localized or found throughout the dentition.^[9] Other traumas to the dentition and supporting tissues include thermal hypersensitivity, tooth hypermobility, injury to the periodontal ligament and periodontium, hypercementosis, fractured cusps, and pulpitis and pulpal necrosis.^[2] Children specifically who brux usually begin at 4-8 years of age, the numbers increase between 10 and 14 years of age and then start declining after the age of 14 years. Many factors associated with children who brux are anxiety, personality, attention-deficit/hyperactivity disorder, and other parafunctional habits.^[4] Episodes of bruxism last around 4 s and occur approximately 6 times per hour. They tend to occur in clusters throughout the night with the majority in Stage 2 and rapid eye movement (REM) sleep. One study found that 60% of bruxism episodes were accompanied by arousal.^[1-4] In children, bruxism may be related to the growth and development of the jaws and teeth. Children may brux because their maxillary and mandibular teeth do not occlude properly and comfortably as they are erupting. Children may also grind their teeth due to tension, anger, or as a response to pain from an earache or teething. The most important characteristic of bruxism is that there is non-functional contact of mandibular and maxillary teeth resulting in clenching or grating of teeth.^[5,6]

ETIOLOGY OF BRUXISM

The etiology is triggered by local factors, systemic, psychological, occupational, and hereditary. Irregular and crowded teeth and insufficient contact between the upper and lower teeth are the justified factors which explain the causes of bruxism. Unconscious habits such as nail biting, chewing pencils, and pen can be effective in jaw clenching. In some cases, having large tonsils that cause upper airway obstructions, have been considered as the involved factor. Bruxism risk factors are including factors such as excessive anxiety, beverage containing caffeine (coffee, chocolate, and cola), cigarettes, narcotics, and psychotropic drugs. Some hyperactive children are also suffering from this disorder.^[7] Sometimes, children who have mental retardation, cerebral palsy, or using certain drugs are at risk of bruxism. More surprisingly, given birth to the second child and unconscious and too much attention to that child is known as one of the causes of jaw clenching. Siblings or

parents argument or fighting or problems in school and kindergarten are other factors which are considered.^[8-10]

DIAGNOSIS OF BRUXISM

The diagnosis of bruxism is descriptive in nature, contingent on the presence of symptoms such as acknowledged tooth grinding, pain in the temporomandibular joints or jaw musculature, temporal headache, tooth hypersensitivity or mobility, and poor sleep quality. These subjective symptoms are coupled with clinical signs such as abnormal tooth wear, tongue indentations, the presence of a linea alba along the biting plane of the buccal (cheek) mucosa, gum recession, masseter hypertrophy, and/or broken fillings or teeth. Attempts have been made to provide a more formal definition of nocturnal bruxism based on sleep architecture and the number of episodes occurring during different phases of sleep. However, for some sleep variables, their sensitivity and specificity are problematic. For example, rhythmic masticatory muscle activity (RMMA) has been reported to occur once or twice per hour in Phases 1 and 2 of nonREM sleep for about 60% of non-bruxing individuals and 80% of patients as identified as bruxers. However, RMMA is also associated with other sleep disorders such as parasomnias, acid reflux, and obstructive sleep apnea and snoring so may not reliably differentiate bruxers from people with other sleep problems. In another study, sleep architecture including the number of microarousals, K=complexes, Kalphas, electroencephalographic spindles, and the density of slowwave activity (SWA) was assessed in a small group of subjects and controls. Based on findings from this research, the authors concluded that, in bruxism patients, good sleep was characterized by a low incidence of Kcomplexes or Kalphas and by the absence of any difference in other sleep microstructure variables or SWA. The extent to which these variables predict bruxism has not been further assessed for diagnostic accuracy.^[11] In diagnosing bruxism, particularly as a sleep disorder, their needs to be a welldefined exclusion criteria that separate bruxism from other sleep disorders such as sleep apnea, epilepsy, and REM disorder behaviors. In an initial effort to establish diagnostic criteria, Lavigne and Colleagues have defined a set of polysomnographic diagnostic criteria, based on the analysis of 18 bruxers and 18 asymptomatic subjects. On the basis of this preliminary data, they have suggested several cutoff criteria for defining bruxism. According to Solberg, et al.,^[12] a person defined as a bruxer should have the following:

- 1. >4 bruxism episodes per hour.
- 2. >6 bruxism bursts per episode and/or 25 bruxism bursts per hour of sleep.
- 3. At least two episodes with grinding sounds.

The validity of this suggested criteria has, to date, not been challenged by additional research with larger numbers and variable levels of bruxism severity, but this first effort at establishing sleep criteria may prove useful research and selective clinical situations. Another possible criterion that might be considered in the diagnostic criteria includes microarousals defined by increased autonomic, cardiac, and respiratory activity which, according to one report, tends to repeat 8–14 times per hour of sleep in affected individuals.

CONSEQUENCES OF BRUXISM

Chronic bruxism, based on published reports, can cause mildtosevere tooth wear, tooth cusp fracture, attached gingival recession, tooth mobility, fractured restorations, masticatory muscle pain, and temporomandibular disorders (TMD). In addition, literature includes other possible effects such as jaw opening limitation, temporal headache, ear symptoms (e.g., plugging, tinnitus and subjective hearing loss), migraines, neck pain, insomnia, and depression.^[13] In a retrospective analysis of parent report surveys, Insana and Colleagues found a high prevalence of sleep bruxism among preschool children (36.8%). In addition, in a subset of preschoolers who received additional behavioral and neurocognitive assessments, bruxism was also associated with increased internalizing behaviors and increased health problems, which, in turn, were associated with decreased neurocognition. The researchers concluded that pediatric sleep bruxism may serve as a warning sign for possible adverse health conditions and the need for early intervention.^[14]

TREATMENT OF BRUXISM

Effective management of bruxism relies on the recognition of potential causative factors associated with the condition. For example, since daytime or diurnal bruxism may be confounded by factors such as stress, distress, and other psychosocial parameters, considering interventions such as habit modification, relaxation therapy, biofeedback, or counseling may be appropriate. In the patient with sleep bruxism (which does not appear to be impacted by psychological or psychosocial factors), appropriate intervention might include appliance therapy and medication. In patients with medication or drug-induced bruxism, medication withdrawal or a change of medication type to one less likely to cause bruxism should be considered. If street drugs are involved, intervention should include drug and perhaps psychological counseling. Bruxism occurring in patients with neurogenic abnormality (e.g., Parkinson disease, dystonia, and Huntington disease) might benefit from

Botox injection of the muscles of mastication. Dietary counseling and management may be necessary in some cases as well (excessive caffeine use and tobacco use). The health-care provider attempting to manage bruxism should understand that nocturnal or sleep bruxism is not going to be cured by intervention. Moreover, the behavior is likely to decrease with age. Daytime bruxism can sometimes be effectively eliminated through intervention, suggesting a cure, but recrudescence of the condition is common. The following sections outline the more commonly recommended management strategies for bruxism.^[15]

APPLIANCE THERAPY

Appliance therapy has been extensively studied from 1966 to the present day, and several extensive reviews have been published in the past 10 years. Occlusal splints are generally appreciated to prevent tooth wear and injury and perhaps reduce nighttime clenching or grinding behavior rather than altering a causative malocclusion. In addition, they are unlikely to significantly reducing nocturnal behavior. The type of appliance that has been studied and suggested as helpful in managing the consequences of nocturnal bruxism is the flatplane stabilization splint, also called an occlusal bite guard, bruxism appliance, bite plate, and night guard. This appliance can vary in appearance and properties. It may be laboratory processed or constructed in the dental office and be fabricated from hard or soft material. The typical appliance covers either all of the maxillary or mandibular teeth. No determination has been made whether significant differences exist in terms of outcome between soft, hard, mandibular, or maxillary splints, but some clinicians feel that soft splints can increase clenching behavior in some patients. However, even if no appreciable change occurs in nocturnal behavior consequent to splint therapy, the appliance serves to protect the dentition. Appliances have also been used to retrain daytime clenching, but other less costly strategies may be equally or more efficacious in managing behavior modification of this daytime activity.^[16]

Complications

Some splints are made to cover only the anterior teeth, and when these are worn during the day as well as at night, the posterior teeth can erupt. When this happens, the removal of the appliance then results in an anterior open bite that can be significant, necessitating the need for otherwise unnecessary orthodontic treatment. Soft splints have been linked to the development of malocclusion. Improper tooth cleaning during splint use can lead to dental caries, and, in some cases, appliance use may result in TMD. A less significant complication is the staining of teeth.^[17]

PHARMACOLOGY

Historically, benzodiazepine-type drugs and muscle relaxants have been prescribed by clinicians in an attempt to reduce nocturnal bruxism. Given the past decade of brain research related to neurotransmitters associated with bruxism, a major focus of recent research has centered on additional potentially useful serotonergic and dopaminergic drugs for the management of sleep bruxism and also the use of acetylcholineinhibiting formulations such as botulinum toxin.^[18,19] In a placebocontrolled, singleblind, non-randomized trial using polysomnography and psychometry, 10 subjects were given 1 mg clonazepam 30 min before lights out. The result was a significant improvement in the mean bruxism index (from 9.3 to 6.3/h of sleep). Furthermore, per the authors, significant improvement occurred in total sleep, total sleep time, sleep efficiency, sleep latency, and time awake during the total sleep period. Periodic leg movements also were noted to decrease significantly.^[20,21] As for the effect of serotonergic medications on nocturnal bruxism, the evidence for efficacy is poor. In a placebocontrolled randomized controlled trial (RCT), a serotonin precursor, Ltryptophan, was not found to be effective in reducing the behavior. The effect of antidepressant medications is mixed.^[22,23] As suggested in an article published in Clinical Neuropharmacology, antidepressant drugs may exert deviating effects on bruxism: Either they exacerbate the condition (selective serotonin reuptake inhibitors [SSRIs]) or they are inert in their effects (amitriptyline). Serotonin reuptake inhibitor medications (SSRIs) have been observed to increase bruxism. In these cases, a reduction in the dosage or withdrawal and substitution with another antidepressant may help to reduce behavior that is significant and pathologic. It should be appreciated that drug manipulation should not occur in the absence of physician consultation. Another approach to managing the patient with SSRIinduced bruxism is possible cotreatment with gabapentin.^[24] Dopaminergic medication also appears to demonstrate potential utility in reducing nocturnal bruxism.^[25] The D2 receptor agonist bromocriptine was studied in six subjects using a doubleblind, placebocontrolled polysomnographic and neuroimaging study with a single cross-over design.^[26] The bromocriptine subjects demonstrated a 20-30% greater reduction in bruxism episodes per hour of sleep, although the number of bursts per episode was not significant between groups. However, bromocriptine-induced significantly lower rootmean-square EMG levels in this limited study, suggesting that the drug may be useful in treating bruxism.

Complications

Dentists are most likely to prescribe benzodiazepine and muscle relaxant drugs for the management of severe bruxism. Prescribing clinicians should be familiar with the general effects, side effects, and potential drug interactions involving these classes of medications. These medications should only be prescribed short term for maximum benefit. In at least one study involving clonidine use for bruxism, the drug is reported to have caused severe morning hypotension in 20% of participants. In addition, longterm use of Ldopa has been associated with an increase in bruxing behavior.^[27]

BOTULINUM TOXIN A

The most recent advocated pharmacological approach to managing the pathological effects of nocturnal and diurnal bruxism is injection of the muscles of mastication with botulinum toxin A. This neurotoxin inhibits the release of acetylcholine from the neuromuscular junction, essentially rendering the muscle incapable of activity. This approach to therapy may find its greatest application in the treatment of severe bruxism associated with coma, brain injury, amphetamine abuse, Huntington disease, autism, and Parkinson disease but is also being used to treat more minor cases of bruxism as well. Complications include bleeding, spot tenderness, bruising, toxic reactions, and infection.^[28]

RELAXATION AND BEHAVIORAL STRATEGIES

The use of relaxation strategies such as meditation, hypnosis, guided imagery, selfmonitoring, habit retraining, cognitive behavioral intervention, and biofeedback in the management of nocturnal bruxism has not been validated by sound scientific studies (e.g., RCTs and studies using nocturnal polysomnography and psychometry). Nonetheless, since these interventions are non-invasive and have been shown to be effective in controlling other forms of habitual behavior, they may have utility in treating diurnal or daytime behavior. Hypnosis appears partially supported by a number of case reports suggesting that longterm effects might be possible in such management. Other relaxation techniques, including meditation, are theorized to reduce stress and improve selfesteem and self-control. Improvement in the quantity and quality of sleep is also understood generally to reduce the episodes of bruxism. As a result, some physical medicine approaches such as cardiovascular toning programs, TENS, acupuncture, manual massage, and a few alternative naturopathic approaches have also been suggested for the management of bruxism. None have been studied appropriately to allow reasonable comment on their efficacy.^[29,30]

OCCLUSAL TREATMENT

In cases in which it can be determined that a specific tooth interference may be initiating bruxing behavior, the offending biting contact should be adjusted to remove the irritant. This does not mean that all the teeth should be adjusted to accommodate a theoretical ideal occlusal position every time.^[31]

CONCLUSION

Although bruxism can be caused by stress or anxiety, most often occurs during sleep due to crooked teeth, tooth loss, and supernumerary teeth. The reason for the formation of this oral parafunctional activity is unknown, but one may argue that crowded teeth, the arrangement of teeth out of the occlusion, inappropriate contact between the upper and the lower teeth, diseases such as malnutrition, nutritional deficiencies, allergies, and endocrine disorders, and psychological factors such as stress and anxiety can lead to bruxism in children. However, after tooth eruption, this habit is lost in most cases. If tooth clenching is diagnosed early, it can be treated by knowing the primary causes of this problem. However, if the treatment is late and bruxism becomes a habit, then, it must be treated with the method of modification's habit.

REFERENCES

- 1. Emodi-Perlman A, Eli I, Friedman-Rubin P, Goldsmith C, Reiter S, Winocur E, *et al.* Bruxism, oral parafunctions, anamnestic and clinical findings of temporomandibular disorders in children. J Oral Rehabil 2012;39:126-35.
- Graber TM, Rakosi T, Petrovic AG. Dentofacial Orthopedics with Functional Appliances. St. Louis: Mosby; 1985. p. 496-519.
- Restrepo C, Gómez S, Manrique R. Treatment of bruxism in children: A systematic review. Quintessence Int 2009;40:849-55.
- 4. Sari S, Sonmez H. The relationship between occlusal factors and bruxism in permanent and mixed dentition in Turkish children. J Clin Pediatr Dent 2001;25:191-4.
- 5. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. J Behav Med 1980;3:385-97.
- 6. Vanderas AP, Menenakou M, Kouimtzis T, Papagiannoulis L. Urinary catecholamine levels and bruxism in children. J Oral Rehabil 1999;26:103-10.
- Landry ML, Rompré PH, Manzini C, Guitard F, de Grandmont P, Lavigne GJ, et al. Reduction of sleep bruxism using a mandibular advancement device: An experimental controlled study. Int J Prosthodont 2006;19:549-56.
- 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;24:9-15.

- McDonald RE, Hennon DK, Avery DR. Diagnosis and correction of minor irregularities in the developing dentition. In: Pediatric Dentistry for the Child and Adolescent. 7th ed. Chicago: Mosby; 2000. p. 487.
- Williamson EH, Lundquist DO. Anterior guidance: Its effect on electromyographic activity of the temporal and masseter muscles. J Prosthet Dent 1983;49:816-23.
- 11. Ramfjord SP. Dysfunctional temporomandibular joint and muscle pain. J Prosthet Dent 1961;11:353-74.
- Solberg WK, Clark GT, Rugh JD. Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. J Oral Rehabil 1975;2:215-23.
- Casamassimo P. Periodontal considerations. In: Pinkham J, Casamassimo OS, Fields HW, McTigue DJ, Nowak A, editors. Pediatric Dentistry Infancy Through Adolescence. 2nd ed. Philadelphia, PA: WB Saunders Co.; 1994. p. 372.
- 14. Kato T, Dal-Fabbro C, Lavigne GJ. Current knowledge on awake and sleep bruxism: Overview. Alpha Omegan 2003;96:24-32.
- 15. Mittelman J. Biofeedback: New answer to dental pain. It can be administered easily and inexpensively in any dental office. Dent Manage 1976;16:21-2, 26-7.
- 16. Riolo ML, TenHave TR, Brandt D. Clinical validity of the relationship between TMJ signs and symptoms in children and youth. ASDC J Dent Child 1988;55:110-3.
- Nilner M, Lassing SA. Prevalence of functional disturbances and diseases of the stomatognathic system in 7-14 year olds. Swed Dent J 1981;5:173-87.
- Kampe T, Edman G, Bader G, Tagdae T, Karlsson S. Personality traits in a group of subjects with long-standing bruxing behaviour. J Oral Rehabil 1997;24:588-93.
- Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001;28:1085-91.
- 20. Olkinuora M. Psychosocial aspects in a series of bruxists

compared with a group of non-bruxists. Proc Finn Dent Soc 1972;68:200-8.

- 21. Lindqvist B. Bruxism in twins. Acta Odontol Scand 1974;32:177-87.
- Egermark-Eriksson I, Carlsson GE, Magnusson T. A long-term epidemiologic study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res 1987;66:67-71.
- Gunn SM, Woolfolk MW, Faja BW. Malocclusion and TMJ symptoms in migrant children. J Craniomandib Disord 1988;2:196-200.
- Huynh N, Lavigne GJ, Lanfranchi PA, Montplaisir JY, de Champlain J. The effect of 2 sympatholytic medications propranolol and clonidine on sleep bruxism: Experimental randomized controlled studies. Sleep 2006;29:307-16.
- Nash MC, Ferrell RB, Lombardo MA, Williams RB. Treatment of bruxism in Huntington's disease with botulinum toxin. J Neuropsychiatry Clin Neurosci 2004;16:381-2.
- Foster PS. Use of the calmset 3 biofeedback/relaxation system in the assessment and treatment of chronic nocturnal bruxism. Appl Psychophysiol Biofeedback 2004;29:141-7.
- Treacy K. Awareness/relaxation training and transcutaneous electrical neural stimulation in the treatment of bruxism. J Oral Rehabil 1999;26:280-7.
- de la Hoz-Aizpurua JL, Díaz-Alonso E, LaTouche-Arbizu R, Mesa-Jiménez J. Sleep bruxism. Conceptual review and update. Med Oral Patol Oral Cir Bucal 2011;16:e231-8.
- Shetty S, Pitti V, Babu CL, Kumar GP, Deepthi BC. Bruxism: A literature review. J Indian Prosthodont Soc 2010;10:141-8.
- Behr M, Hahnel S, Faltermeier A, Bürgers R, Kolbeck C, Handel G, *et al.* The two main theories on dental bruxism. Ann Anat 2012;194:216-9.
- 31. Aloe F. Sleep bruxism treatment. Sleep Sci 2009;2:49-54.