

Bruxism - A Periodontal Overview

Anil Melath¹, Arjun MR^{2*}, Mahesh Raj V³, Subair K⁴, Mohanapriya⁵ and Mohammed Nihal⁵

¹Professor and Head Department of Periodontics, Mahe Institute of Dental Sciences, India

²Reader Department of Periodontics, Mahe Institute of Dental Sciences, India

³Senior Lecturer Department of Periodontics, Mahe Institute of Dental Sciences, India

⁴Professor, Department of Periodontics, Mahe Institute of Dental Sciences, India

⁵Third Year Bds Student, Mahe Institute of Dental Sciences, India

*Corresponding author

Arjun MR, Reader Department of Periodontics Mahe Institute of Dental Sciences, India. E-mail: arjunjai2002@gmail.com

Received: May 11, 2022; **Accepted:** May 30, 2022; **Published:** June 03, 2022

Introduction

Tooth grinding is an activity particularly important to the dentist because of breakage of dental restoration, tooth damage, induction of temporal headache and temporomandibular disorder [1]. The term para function was introduced by Drum to suggest distinction between occlusion stress exerted during mastication and swallowing and occlusal stress which are brought into action outside of the normal function. Para functional activities are non-functional oromandibular or lingual activities that includes Jaw clenching, bruxism, tooth grinding, tooth tapping, cheek biting, lip biting, object biting etc. [2,3]. That can occur alone or in combination and are different from functional activities like chewing, speaking and swallowing.

Bruxism can occur during wakefulness or during or during sleep. Bruxism during daytime is called "Awake bruxism" or "Diurnal bruxism". Bruxism during sleep either during that time or during night is termed as "sleep bruxism" prevalence rate of awake bruxism and sleep bruxism is about to 20% and 8.16 % in adult population [4]. The disorder is appearing more frequently in the younger population. Onset of sleep bruxism is about one year of age soon after the eruption of deciduous teeth [5].

Definition

American Academy of orofacial pain

Bruxism- Defined as diurnal or nocturnal para-functional activity including clenching, bracing, gnashing and grinding of the teeth [6].

According to GPT- 8

Bruxism- defined as the para-functional of grinding of the teeth or oral habit consisting of involuntary rhythmic or spasmodic non-functional gnashing, grinding or clenching of tooth in other than chewing movement of the mandible which may lead to occlusal trauma [7].

Nomenclature

1901: Karolyi M - "traumatic neuralgia"

1907: Marie pietkiewicz - "Bruxiomania" [8]

1931: Frohman - "bruxism" [9]

1971: Ramjford Ash - "centric and eccentric bruxism" [10]

1972: Drum- "Emotional loaded parafunction" [11]

Epidemiology

The ICSD-R states that 90% of the general population grind their teeth to a degree at some point during their life, although only 5% will develop a clinical condition [7].

Studies have reported that awake bruxism affects females more commonly than males while in sleep bruxism, males are equally affected as females [12].

Classification

Bruxism may be classified according to several criteria by when it occurs [13].

- Awake bruxism -This is presented when individual is awake.
- Sleep bruxism - This is presented when the individual is asleep.
- Combined bruxism - present in both conditions.

By Etiology [14]

- Primary or idiopathic bruxism - no apparent cause known.
- Secondary Bruxism-Disease, medicinal products drugs.

By Motor Activity Type

- Tonic - Muscular contraction sustained for more than 2s.
- Phasic - Brief, repeated contraction of the masticatory musculature.
- Combined - Alternate tonic and phasic.

Approximately 90% of the episodes of SB are phasic or combined, unlike in AB, where it is tonic [15].

By of Period of Occurrence

- Past bruxism
- Current or present bruxism

By Severity [10]

- Mild - occurring less than nightly
- Moderate- occurring nightly
- Severe- occurring nightly and with damage to the tooth.

By Duration

- Acute
- Subacute
- Chronic

Etiology

Bruxism is considered to have multifactorial aetiology SB and grinding have been associated with peripheral factors such as tooth interference in dental occlusion. Psychosocial influence such as stress or anxiety and central or pathophysiological causes involving brain neurotransmitter or basal ganglia [16].

Patho Physiological Factors

As the bruxism often occurs during sleep, the Physiology of sleep has been especially the 'arousal response' in search of possible cause of disorder. 86% off Bruxism episodes were associated with arrows arousal responds along with involuntary leg movements [14]. This shows that bruxism is a part of arousal response indeed.

Recently it is derived that disturbance in central neurotransmitter system may be involved in the etiology of the bruxism [17]. Nicotine stimulates central dopaminergic activities which might explain the findings that cigarette smokers report bruxism two times more than non-smoker [18].

Psychosocial Factors

Bruxism differs from healthy individuals in the presence of depression, increased level of hostility and stress sensitivity. Bruxing children are more anxious than non-bruxers [19].

A multi factorial large scale population study to sleep bruxism revealed highly stressful life as a significant risk factor.

Genetic factors

Research suggests that there may be a degree of inherited susceptibility to develop sleep bruxism [19]. 21 -50% of people with sleep bruxism have a direct family member who had sleep bruxism during their childhood suggest that there are genetic factors involved.

Other possible associations

- Parkinson's disease
- Tones mandibularis
- Oromandibular dystonia
- Rett syndrome
- Down syndrome
- Trauma
- Atypical facial pain

Clinical Manifestation

Bruxism has the potential to cause Tooth wear, fracture, periodontal and muscle pain and it is a major cause of tooth mobility.

The clinical evaluation is complex because both bruxers and normal individuals may show some nocturnal para functional activity [20].

Dental enamel is the first structure affected from the para functional load. An abnormal wear of the tooth is the most common evidence of this condition. It may be restricted to single tooth or the entire mouth. Bruxism harmful habit causes relevant changes in the stomatognathic system structure. It causes friction, inflammation, pulpal necrosis and tooth mobility. Bruxism can also cause posture problem. In addition, it can affect masticatory muscle and posture muscle of the cervical spine which may cause muscular pain future chronic permanent changes.

Radiographic Features

Radiographic examination can show the Cors of lamina dura, changes in the periodontal space which can either disappear or be increased, root resorption, root fractures and pulp stone [20].

Bruxism was associated with cortical shape, the mental index (MI), Ante gonial index (AI), gonial index (GI), And ante gonial notch depth (AND), semilunar defects, cortical residues and porosity in the industrial cortex of the mandible where more prevalent in bruxers. The effects of Bruxism on fractal dimension which was found to be significantly reduced in the condylar region of patient with bruxism [21].

Cortical thickening in the mental and gonial regions of the mandible was observed in bruxers. Tiny bond peaks were detected in the cortex of the mandibular gonial Region in bruxers at a significantly higher rate than in non-bruxers. The masseter muscle insertion is at the gonial angle, the excessive bite force in bruxers could explain the higher 61 and Linny bone peaks seen. Cortical thickening in the mental region may occur in response to endosteal bone damage caused by premolar teeth in bruxers.

Antegonial notch depth was used to evaluate morphological changes in bruxers and is given by this distance along a perpendicular line from the deepest point of the mandibular inferior border notch concavity to the tangent through the inferior border of the mandible. AND was increased in bruxers. AND was greater in male bruxers. Male bruxers patient has gonial index and antegonial notes depth values then female bruxers. The radio morphometric variables, AND and rate of periodontal disease were compared between the bruxers and non-bruxers.

Effects of Bruxism

Periodontium

- Tooth mobility
- gingival recession
- pathological migration

Excessive pressure and tension produced alteration in periodontal ligament and inflammation passed into the altered area and resulted in necrosis of periodontium. This necrotic tissue would act test barrier preventing inflammation from extending into underlying periodontal disease.

Alveolar bone

Buttressing bone formation takes place as an attempt to compensate for lost bone.

Central

Buttressing bone formation occurs within the jaw, the buttressing endosteal cell deposit new bone which restores bony trabecular and reduces side of marrow spaces.

Peripheral

Mainly occurs in facial and lingual surface of alveolar plate.

Lipping

Depending on severity, peripheral buttressing may produce a shelf like thickening of alveolar margin.

Muscles

- functional disturbance of jaw dynamics
- Pain during Joe moment
- Hypertonicity or muscle hyperactivity
- Trismus- A spasm of jaw muscle that makes it difficult to open the mouth lock jaw
- Myofascial pain
- Mayo spasm- spasmodic contraction of muscle
- Swelling

Paediatric Patients

Anxiety and stress are the primary contributing factors to bruxism in children. Snoring and obstructive sleep apnea are closely related to children, but the mechanism behind this relationship remains unknown. This sleep disorder is often associated with mouth breathing and adenoids or tonsil hypertrophy or infection.

Children with behaviour and attention difficulties might exhibit more disruption of sleep pattern with predisposition to bruxism.

Implants

Bruxism can lead to implant failure by loss of integration and screw fracture.

Early integration loss

These are spontaneous avulsions of implants that are not yet Osseo integrated.

Late integration loss

Fibrous repair occurs more than bone formation which leads to fracture of implants that are partially Osseo integrated.

Delayed integration loss

Occurs in already integrated and prosthetically restored implants.

Consequences of Bruxism

Sleep bruxism is characterised by the clenching and grinding of the teeth. Clenching of teeth is a forceful closure of the opposite dentition in a static relationship of the mandible to the maxilla in either maximum intercuspation or an eccentric position. The lateral movement of the mandibular during grinding often exceeds the edge-to-edge relation of the canine [22]. Bite forces during sleep bruxism events can exceed the amplitude of maximum voluntary bite force when awake [23]. Protective neuromuscular reflexes that are operational during waking hours appear to be suppressed during sleep. This can lead to significant loading of teeth, the periodontium, TMJ as well as muscles of mastication.

Grinding involves the forceful closure of opposing dentition in a dynamic maxillomandibular relationship as the mandibular arch moves through areas excessive position. Bruxism is associated with increased mechanical and technical complications of tooth and implant supported prosthesis. When prosthodontic interventions are indicated in sleep bruxism patients effort must be made to reduce occlusal loading on all prosthetic components especially during sleep.

Diagnosis of Bruxism

Diagnostic criteria for sleep bruxism based on the international classification of sleep disorders,

- Presence of regular or frequent tooth grinding sounds during

sleep.

Other symptoms and signs include,

- teeth that are worn down ,flattened or chipped
- pulpitis
- Tooth ache
- partial crown fracture
- tooth migration

Muscular symptoms include [20]

- fatigue
- Increased tension in masticatory muscles especially the lateral pterygoid muscle, mandibular elevator muscle, masseter and temporal.

The most common muscular symptom is fatigue which is inability to resist during a sustained effort without having apparent signs and symptoms of pain and discomfort.

- Increased tooth pain or sensitivity
- Jaw neck or face pain or soreness
- Tightening of jaw muscles
- Earache because of violent jaw muscle contraction
- Chewed tissue on the inside of your cheek
- Insomnia
- Anxiety, stress and tension

Definitive diagnosis of sleep bruxism can be achieved using electrophysiological tools. Laboratory based PSG allows for the detection of SB as well as other sleep disorders including sleep apnea, periodic limb movement and parasomnias.

Objective PSG recordings include electromyogram (jaw movements), oronasal air flow and oxygen saturation.

Ambulatory test can be used to access overall masticatory muscle activity rather than SB [24].

Clinical Examination

The diagnosis of bruxism is based particularly on history, tooth mobility, tooth wear and other clinical findings.

Tooth Wear

Tooth wear is considered to be analogue to bruxism. A number of systems for the classification and measurement of incisal and occlusal tooth wear have been introduced. Tooth wear index which was developed with regarding to incisal and occlusal wear [25].

Intraoral Appliance

Bruxism activity can be evaluated using the intra oral appliance and is classified into,

- Observation of wear facet of Intra oral appliance
- Measurement of bite force loaded on the intra oral appliances [26].

Brux Core Plate

The Brux core bruxism- monitoring device (BBMD) is an intraoral appliance that was introduced as a device for measuring sleep bruxism activity [27] and the brux core plate evaluates bruxism activity by counting the number of abraded micro dots on its surface and by scoring the volumetric magnitude of abrasion.

Detection of Bite Force

The recording device was developed for sleep bruxism, an intra splint for detector (ISFD) which uses an intraoral appliance to measure the force being produced by tooth contact on the appliances [28]. The force is detected using a thin deformation sensitive piezo electric film. It was confirmed that the duration

of bruxism events during stimulated bruxism was correlated with that of masseter EMG.

Masticatory Muscle Electromyographic Recording

Among the various methods for the assessment of bruxism, the EMG recording has seen commonly used to measure actual sleep bruxism activity directly. The principal advantage is that the occurrence of bruxism can be assessed without intraoral devices. Which may change natural bruxism activity.

Portable EMG Recording Device

Sleep bruxism episodes were measured over an extended periods in patients' home with the use of battery-operated EMG recording devices [29].

This device can measure masticatory muscle activity more minutely also the number, duration and magnitude of bruxism.

Miniature Self-contained EMG Detector Analyser

It was developed as a screening test for moderate to high level bruxers. EMG signals detect tooth grinding and clenching and also biofeedback stimulation for reducing sleep bruxism activities [30].

Polysomnography

Polysomnographic recording for sleep bruxism generally includes Electro encephalogram, EMG, electrocardiogram along with simultaneous audio, video recordings. sleep bruxism activity is assessed based on EMG activity in the masticatory muscles. physiological changes related to sleep bruxism can also be monitored.

Management of Sleep Bruxism

Bruxism management relies on the recognition of the potential causative factors associated with the development of condition. Diurnal bruxism can be managed by considering intervention such as habit modification, relaxation therapy.

Methods to Manage Bruxism

Occlusal Adjustments

Premature contacts or occlusal interference have been associated with the development of bruxism. Some dental clinicians still believe that occlusal adjustment is requirement in the management of bruxism. But there seem to be no basis of evidence for performing such irreversible occlusal adjustments [31].

Occlusal Splints

Occlusal splints are worn at night to guide the occlusal movement so that the periodontal damage is minimal. The appliance covers overall the maxillary or mandibular teeth but it mostly worn in the upper maxilla. Occlusal splints are generally appreciated to prevent tooth wear, injuries and reduce night time clenching. The appliance that helps manage the consequences of nocturnal bruxism is that flat planned stabilization splints also called an occlusal bite guard, bruxism appliance, biteplate or night guard.

Hard acrylic resin stabilisation splints are suggested to be more effective in reducing bruxism activity than soft splints [31]. These appliances are also utilised to retain that daytime clenching.

Psychotherapy

Bruxism may often be related to stress. Psychotherapeutic approaches should be made to foster calmness. Patient counselling can lead to decrease intention and also create awareness of the habit. This will increase voluntary control and thus reducing para functional movements.

Physical Therapy

Physical therapy may be recommended if Bruxism is associated with muscle pain and stiffness.

Relaxation Training

In this method the patient is trained to relax the muscle group or voluntarily.

Restorative Treatment

If there is a severe attrition associated with bruxism, then endodontic therapy is recommended. Composite restoration or full coverage crowns restore vertical dimension and function.

Medication

Pharmacological management includes the use of Antianxiety agents, tranquilizers, sedatives and muscle relaxants. Medication such as diazepam can be prescribed for a few days to alter the sleep disturbance and anxiety level. Low doses of tricyclic antidepressants may be used to inhibit the amount of rapid eye movement (REM) sleep [32].

Electrical Method

Electro galvanic stimulation for muscle relaxation is currently used for the treatment of bruxism.

Biofeedback

This technique utilises positive feedback to enable the patient to learn tension reduction. It is based on the idea that bruxers can unlearn their behaviour. It is accomplished by allowing the patient to view an electromyography (EMG) monitor while the mandible is postured with minimal activity. For nocturnal bruxism auditory, vibratory or taste stimuli may be used [32].

Botulin Toxin

Botulin toxin, neurotoxin for muscle relaxation is currently used for bruxism. It is synthesised by Clostridium botulinum. It works by impeding acetylcholine production and blocking calcium channel in nerve endings temporally inhibiting muscle contraction.

Botulin toxin injection in the masseter and temporal muscle have been demonstrated to improve the quality of life of patient with Bruxism. The neurotoxin decreases the frequency of bruxism episodes, the severity of pain and intensity of masticatory force [33].

Conclusion

Bruxism is a parafunctional oromotor habit with a high prevalence in the general population. the signs & symptom of bruxism care is detectable but unfortunately the hypothesized etiologies & mechanism of their actions has not been substantiated satisfactory. Present there is no effective treatment to eliminate bruxism permanently. There for the therapeutic approaches started towards attempting to prevent damage and to treat the pathological effects of bruxism on the structures of the masticatory system.

References

1. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K (2008) Bruxism physiology and pathology: an overview of clinicians. J Oral Rehab 35: 476-494.
2. Burton C (1983) Bruxism. Thesis, University of Sydney.
3. Laati A, Macaluso GM (2002) Sleep bruxism as a motor disorder. Mov Disord 17: 67-69.
4. Glaros AG (1981) Incidence of diurnal and nocturnal bruxism. J Prosthet Dent 45: 545-549.
5. Sari S, Sonmez H (2001) The relationship between occlusal

- factors and bruxism in permanent and mixed dentition in Turkish children. *J Clin Pediatr Dent* 25: 191-194.
6. Reny de Leeuw (2008) American Academy of Orofacial Pain: Guidelines for Assessment 22: 190-200.
 7. AASM (2005) International classification of sleep disorders, 2nd edn. American Academy of Sleep Medicine, Westchester.
 8. Bader G, Lavigne G (2000) Sleep bruxism; an overview of an oromandibular sleep movement disorder. Review article. *Sleep Med Rev* 4: 27-43.
 9. Faulkner KD (1990) Bruxism: A review of the literature. Part I. *Aust Dent J* 35: 266-276.
 10. Drum W (1972) Die Autodestruktionstheorie. In: Drum W, editor. *Zahnmedizin für Ärzte*. Berlin: Verlag Die Quintessenz.
 11. Ramjford SP, Ash MM. (1971) 2nd ed. Philadelphia: WB Saunders Co.
 12. Shetty S, Pitti V, Satish Babu CL, Surendra Kumar GP, Deepthi BC (2010) Bruxism: A literature review. *J Indian Prosthodont Soc* 10: 141-148.
 13. Macedo CR, Machado MA, Silva AB, Prado GF (2009) John Wiley and Sons, Ltd. Pharmacotherapy for sleep bruxism. *Cochrane Database Syst Rev* 36: 86-92.
 14. Bader G, Lavigne G (2000) Sleep bruxism; an overview of an oromandibular sleep movement disorder. *Sleep Med Rev* 4: 27-43.
 15. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K (2008) Bruxism physiology and pathology: An overview for clinicians. *J Oral Rehabil* 35: 476-494.
 16. Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, et al. (1998) Sleep bruxism is an disorder related to periodic arousals of sleep. *J Dent Res* 77: 565-573
 17. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisier JY (1997) The effect of the catecholamine precursor l-dopa on sleep bruxism: a controlled clinical trial. *Mov Disord*. 12: 73.
 18. Ashroftt GW, Eccleston D, Waddell JL (1965) Recognition of amphetamine addicts. *Br Med J* 1: 57.
 19. Monaco A, Ciammella NM, Marci MC, Pirro R, Giannoni M (2002) The anxiety in bruxer child. A case-control study. *Minerva Stomatol* 51: 247-250.
 20. Lobbezoo F, Van Der Zaag J, Van selms MK, Hamburger HL, Naeije M (2008) principles for the management for bruxism. *J Oral rehabil* 35: 509-523
 21. Gulec M, Tassoker M, Ozcan S, Kaan Orhan (2020) Evaluation of the mandibular trabecular bone in patients with bruxism using fractal analysis. *Oral radiol* 20: 422-425.
 22. Yap AU (1998) Effects of stabilization appliances on nocturnal parafunctional activities in patients with and without signs of temporomandibular disorders. *J Oral Rehabil* 25: 64-68.
 23. Nishigawa K, Bando E, Nakano M (2001) Quantitative study of bite force during sleep associated bruxism. *J Oral Rehabil* 28: 485-491.
 24. Kato T (2004) Sleep bruxism and its relation to obstructive sleep apnea-hyponea syndrome. *Sleep Biol Rhythms* 2: 1-15.
 25. Ekfeldt A, Hugoson A, Bergendal T, Helkimo A (1990) An individual tooth wear index and an analysis of factors correlated to incisal and occlusal wear in an adult Swedish population. *Acta Odontol Scand* 48: 343-349.
 26. Baba K, Clark GT, Watanabe T, Ohyama T (2003) Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. *J Orofac Pain* 17: 58-64.
 27. Forgione A (1974) Simple but effective method quantifying bruxing behavior. *J Dent Res* 53.
 28. Takeuchi H, Ikeda T, Clark GT (2001) A piezoelectric film-based intrasplint detection method for bruxism. *J Prosthet Dent* 86: 195-202.
 29. Rugh JD, Solberg WK (1975) Electromyographic studies of bruxist behavior before and during treatment. *J Calif Dent Assoc* 3: 56-59.
 30. Minakuchi H, Clark GT (2004) The sensitivity and specificity of miniature bruxism detection device. *J Dent Res* 83.
 31. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M (2008) Principles for the management of bruxism. *Journal of oral rehabilitation* 35: 509-523.
 32. Beddis H, Pemberton M, Davies S (2018) Sleep bruxism: an overview for clinicians. *British dental journal* 225: 497-501.
 33. Fernández-Núñez T, Amghar-Maach S, Gay-Escoda C (2019) Efficacy of botulinum toxin in the treatment of bruxism: Systematic review. *Medicina oral, patologia oral y cirugia buccal* 24: 416-424.

Copyright: ©2022 Arjun MR, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.