

Sleep Bruxism: A Review

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ABSTRACT

Sleep Bruxism is a common phenomenon in children with prevalence varying from 3.5 % to 40.6 % in children and 16.5 % in adults. It is not a movement disorder or sleep disorder, rather, a rhythmic or non-rhythmic masticatory muscle activity characterized by teeth grinding or clenching during sleep which starts soon after the eruption of deciduous incisors. This article aims to review sleep bruxism in detail.

Key Words: Sleep Bruxism, Grinding, Clenching, masticatory muscle activity

INTRODUCTION

Sleep bruxism was firstly described by Maria Pietkiewicz in 1907[1]. There are various definitions till date, but, according to recent updated definition given by “International Classification of Sleep Disorder (ICSD,2018)” - *Sleep bruxism is a rhythmic or non -rhythmic masticatory muscle activity during sleep and is not a movement disorder or a sleep disorder in otherwise healthy individuals*” [2].

Its prevalence varies from 3.5% to 40.6 % in children and 16.5 % in adults. It starts at 1 year of age soon after the eruption of deciduous incisors and gradually decreases with the age [3].

Sleep bruxism episode mostly occur in stage 1 and stage 2 (light sleep) of Non- Rapid Eye Movement (NREM) sleep and occasionally (<10%) during Rapid Eye Movement (REM) sleep. Sleep bruxism during REM can be subclinical manifestation of sleep behaviour disorder (Parasomnia where vivid dreams are acted out during sleep), as, during this stage muscles are relaxed up to the stage of paralysis and only brain is active [4].

Although Sleep bruxism is not a pathology or disorder but as it is mainly regulated by central nervous system, so, it could be a possible symptom of underlying deleterious health conditions or habits. Its etiology is also complex and multifactorial [5].

In recent years, Sleep bruxism has gained more attention from dental health professionals as it can lead to various dental problems such as masticatory muscle atrophy, temporomandibular disorder, tooth wear, fracture of restoration or teeth, hypersensitivity of teeth and even failure of implants / prosthesis [6].

ETIOLOGY

It can be divided into

- 1) Peripheral (morphological)
- 2) Central (pathological and psychological) [7]

Peripheral factors	Central factors
Occlusal discrepancy	1. Stress 2. Genetic factor 3. Alcohol, tobacco use, caffeine consumption 4. Drugs 5. Neurological disorder 6. Vitamins and minerals deficiency

Peripheral risk factors play only a minor role in etiology of sleep bruxism than central factors which activates CNS during sleep and contributes to sleep bruxism.

- 1. **Exogenous risk factors:** It includes smoking, heavy alcohol intake, caffeine, medications or drugs [8].
 - Alcohol: Consuming excess alcohol disrupts sleeping patterns and cause changes in behaviour and motor activity by altering the levels of glutamate, dopamine, serotonin and extrapyramidal region which in turn triggers muscle to hyperactivate and teeth to grind [9,10].
 - Tobacco and caffeine use: Tobacco and caffeine is stimulant which affects dopaminergic system and increases muscle activity and thus, leads to bruxism [10,11].
 - Drugs: Certain drugs increase the sleep bruxism episodes such as phenethylamines (amphetamine, Dextroamphetamine, Levoamphetamine, Lisdexamfetamine, Methylphenidate) and selective serotonin reuptake inhibitors (Fluoxetine, Paroxetine, Sertraline, Citalopram) [12].
- 2. **Stress and Psychosocial factor:** stress and anxiety disorders such as obsessive-compulsive disorder, social anxiety etc increases masticatory muscle activity, periodic pain and thus, commonly associated with sleep bruxism [13].

- 3. **Sleep disorders:** Sleep-Bruxism - Rhythmic masticatory muscle activity episodes tend to occur during recurrent microarousals (3-10s) period (natural activities during sleep that consist of repetitive rise in heart rate, muscle tone and brain activity 8-15 times /hour of sleep). Sleep disorders include Obstructive sleep apnoea, insomnia etc. These sleep disorders along with microarousals prevent sound consistent sleep and thus tend to increase risk of sleep bruxism [14,8].
- 4. **Upper Airway Resistance Syndrome:** In this syndrome, pressure in the airways is increased which leads to choking. This obstructive respiration during sleep can cause mandible to fall back and triggers reflex action to open airway by increasing masseter-tone. It further activates sympathetic system to open the airway pushing the jaw forward, which then initiates grinding in attempt to increase volume in the airway [15].
- 5. **Comorbidities:** Includes sleep apnoea, gastro-oesophageal reflux disorder, cerebral palsy, stroke, brain damage, dementia or Alzheimer’s disease, traumatic brain injuries. Sleep bruxism is secondary disorder for all these conditions. Research studies has shown that sleep bruxism plays protective role during sleep by maintaining patency of

airways or stimulating saliva flow to moisturize oropharynx [16].

6. **Parkinson disease:** Levodopa used for treatment of this disorder is converted to dopamine by brain which aids in body movement. Its long-term use can cause sleep bruxism [17].
7. **Fibromyalgia:** It is characterized by musculoskeletal pain along with fatigue, sleep disturbances, memory and mood changes which can increase the risk of sleep bruxism [18].
8. **Down syndrome:** It is genetic disorder also known as trisomy 21. Sleep bruxism in this case is common side effects of characteristic facial/oral features present such as short teeth and roots, missing teeth, class III malocclusion, crowding [19].
9. **Autism:** Antipsychotic medication used in autism can lead to Sleep bruxism [20].
10. **Genetic basis:** rs6313HTR2A SNP is involved in pathogenesis of sleep bruxism [21].
11. **Vitamins and minerals deficiency:** Vitamin -D maintains calcium homeostasis. Low vitamin -D and Ca level is associated with hypocalcaemia that affects neuromuscular function which can initiate sleep bruxism, whereas, mineral such as magnesium deficiency causes neuromuscular irritability, weakness, generalized anxiety, insomnia and headache which all can lead to sleep bruxism [22].
12. **Iron:** Iron deficiency is associated with restless leg syndrome and Bruxism is manifestation of the same as both share common features and malfunction of same pathway (mesocortical dopaminergic pathway) [23,24].
13. **Omega – 3 fatty acids:** Indirectly linked to sleep bruxism as it induces stress and increase corticosterone levels which can cause grinding of teeth [25].
14. **Added sugar consumption and excessive screen -timing:** Excessive screen timing and added sugar consumption basically alters sleep, affects cortisol homeostasis and can

cause depression and attention deficit hyperactive disorder related symptoms. Studies has shown association of all these factors with sleep bruxism[26] .

CLINICAL SIGNS AND SYMPTOMS

Bruxism has both positive and negative effects with various signs and symptoms such as attrition, loss of vertical dimension, wear facets (highly polished, smooth, flattened wear pattern on enamel), abfraction, chipping, cracks /fracture of teeth, periodontal ligament recession, bone loss, buccal exostosis or tori, audible occlusal sound, temporomandibular disorder, sensitivity of teeth, pain and hypertrophy of masseter or temporalis muscle, headache, ear pain [27-35].

DIAGNOSIS

Diagnostic criteria for sleep bruxism according to international classification of sleep disorder (third edition) [36] :-

Presence of (1) Regular or frequent audible sounds of grinding during sleep.

- (2) One or more of following clinical signs
- transient morning jaw muscle pain or fatigue
 - headache (temporal)
 - jaw locking on awakening

Further, Diagnosis can be divided into:

- 1.) Chairside diagnosis
- 2.) Definitive diagnosis

1) **CHAIRSIDE DIAGNOSIS:** Bruxism can be diagnosed chairside by *subjective reports, questionnaires, clinical examination* and *trial splints* such as *Bruxchecker* (visualises functional tooth contact area), *Brux-core plate* (observes wear facets of intra oral appliances), *Intra-splint force detector* (measures bite force loaded on intra-oral appliance) [37]

2) **DEFINITIVE DIAGNOSIS:** It can be done by electrophysiological tools such as

- Polysomnography (PSG): It is considered as gold standard for the diagnosis of bruxism. It measures brain activity (electroencephalogram); eye

movement (electrooculogram); jaw/leg movements (electromyogram); heart rate /rhythm (electrocardiogram); oxygen saturation; thoracoabdominal movements; oronasal airflow [38,39].

- Ambulatory tests: It can be done at home which unlike Polysomnography only assess overall masticatory muscle activity rather than sleep staging and audio-video recordings. Portable devices used for such tests are *Bite-strip*, *Grind-care* (assess masseter muscle activity by electromyographic recordings); *Brux-off* (assess masseter muscle contraction and heart rate) [40,41].

MANAGEMENT

There is no treatment that can completely cure sleep bruxism. Management of SB depends on three basic principles² –

- 1.) Bruxism may be a behaviour that does not need any treatment.
- 2.) Treatment is indicated when there is presence of negative clinical consequences.
- 3.) It is mostly sign of any underlying disease.

Management should be based on clinical consequences, until, specific cause is determined. Treatment of sleep bruxism includes various approaches such as Psychological e.g. biofeedback, hypnotherapy, cognitive-behavioural therapy, stress and relaxation management; Pharmacological and Dental treatments [42] which is as follows:

PSYCHOLOGICAL APPROACH

It includes: -

- a) Biofeedback Therapy
- b) Cognitive -Behavioural Therapy
- c) Hypnotherapy
- d) Stress and Relaxation Management

A) BIOFEEDBACK THERAPY

It is a technique that measures masticatory muscle activity in case of sleep bruxism using external electrodes which is linked to electromyograph that provides individual information about body activities (biofeedback) with the help of speakers or

supra-cutaneous electrical impulses or by releasing bad taste from occlusion splint on grinding in order to promote change in behaviour.

A pilot study by *Gu et al.* tested dental splint with extrabuccal biofeedback and reported significant reduction in frequency and duration of bruxism.

Hara et al. tested an occlusal splint with intrabuccal biofeedback and reported reduction in sleep bruxism frequency [2,43].

B) COGNITIVE -BEHAVIOURAL THERAPY

It comprises of muscle relaxation, recreation, enjoyment and sleep hygiene measures. Sleep hygiene measures include avoiding caffeinated drinks before sleeping, maintaining proper ventilation in bedroom, relaxing close to bedtime [44].

C) STRESS AND RELAXATION MANAGEMENT

Preventing stress can reduce bruxism activity significantly. Meditation, Yoga, listening to relaxing music can be some of the ways to reduce stress and thus sleep bruxism.

D) HYPNOTHERAPY

It is an effective approach to deal with bruxism. It works by inducing a relaxed state of mind and bringing awareness of the condition as well as addressing the underlying causes, which can sometimes include complex and deep seated emotional and psychological issues. When we are in this state, our unconscious is open to suggestion and re-patterning. So, thoughts leading to teeth grinding can be interrupted. As teeth grinding can often be a reaction to stress and anxiety, using hypnosis for stress may reduce bruxism [45]

E) PHYSIOTHERAPY

The proper mandibular resting position is as follows: lips together, teeth having contact only when swallowing. Muscle relaxation exercises are recommended to stretch them and balance overactivity of masticatory muscle. It is important treatment option in

patients with pain and fatigue of the jaw muscles as it is effective in relieving pain and restoring muscle and joint mobilization, and it also reinforces counselling or cognitive behavioural strategies.

Muscle relaxation exercise includes the following steps:

- I. Set the mandible in a relaxed position.
- II. Protrude the mandible to reach teeth-to-teeth contact.
- III. Opening mouth.
- IV. Closing the mouth until teeth-to-teeth contact. Returning the jaw to starting position.

Above exercise is recommended three times a day, with 20 repetitions for a month, freely but without force, roughly at the speed of their heartbeat [46].

PHARMACOLOGICAL APPROACH

- DRUGS

Drug therapy includes the use of benzodiazepines, anticonvulsants, beta-blockers, serotonergic and dopaminergic agents, antidepressants, muscle relaxants.

Among above all drugs Clonazepam (a benzodiazepine) significantly decreases sleep bruxism index by $42 \pm 15\%$. According to Saletu A, et al. 1 mg of clonazepam improves bruxism and sleep efficiency; but can cause apnea and hypoapnea as a side effect, although not significantly.

Propranolol, an adrenergic receptor blocker, also cause reduction in teeth grinding, but at the same time reduces quality of sleep.

Clonidine significantly decreases rhythmic masticatory muscle activity as compared to clonazepam.

Some of the homeopathic medicines such as *Melissa Officinalis* (MO) or *limoncillo*, and *Phytolacca Decandra* (PD) can cause significant reduction in sleep bruxism [47,48]

- NUTRITIONAL SUPPLEMENTS

Magnesium plays a vital role in nerve and muscle function which led the researchers to the suspicion that bruxism can occur due to insufficient consumption, or inefficient

utilization of this metal. Magnesium-deficient diet leads to frequent teeth grinding in both sleeping and awake pigs. Suggested treatment in humans involves magnesium supplements.

Research study by *Lehvila*, showed significant decrease (and sometimes even disappearance) in the frequency and duration of grinding episodes occurred in patients who took, once a day, a tablet of vitamins and minerals (consisting 25 mg {in children} or 100 mg {in adults} of magnesium), for at least five weeks.

The best possible treatment option involves taking following drugs daily:

Magnesium (approximately 100 mg), Calcium (150 mg), and Pantothenic acid (50 mg), combined with at least the following: Vitamins A (1,000 IU), C (300 mg), E (60 mg), and Iodine (0.1 mg—100 mcg).

If bruxism subsides, patients should be advised to continue taking these pills. If no improvement is observed after 8 weeks or so, this approach may be given up.

Magnesium should not be taken in case of renal impairment and acute dehydration and also if it causes diarrhoea, other adverse reactions, or if it interferes with other medicines. One should not prescribe more than 100 mg a day, as taking too much, or prolonged treatment, can cause fatigue and respiratory problems and even cause hypermagnesemia, which can further lead to nausea, vomiting, lethargy, and blockage of the bladder [49].

- BOTULINUM TOXIN THERAPY

Botulinum toxin (BTX-A) is a peripheral cholinergic synapse-blocking agent that produces motor weakness to the point of paralysis. According to some research studies Botulinum toxin was found to reduce the number of bruxism events and intensity. The Botulinum toxin is commercially available as a freeze-dried powder of 100 U which was reconstituted with 2 mL of sterile normal saline to a concentration of 5 U/0.1 mL. A dose of 25 U of Botulinum toxin is injected with 1-mL syringe with a 29-gauge,

0.5-inch needle into each masseter muscle. It is injected into two sites of each masseter muscle. First site is the inferior, most prominent part of the masseter muscle when the subject is asked to clench, and the other site is 1–2 cm vertically away from the first site. Both the sites are separated by 1-2 cm in the center of middle third of masseter muscle. Patients should be examined for three nights with PSG for checking reduction in sleep bruxism activity. The first night is before injection, the second is 4 weeks after injection, and the last 12 weeks after injection.

Contraindicated in:

- (1) previously received Botulinum toxin injection into both the masseter and the temporalis muscles for the past one year;
- (2) patients on medications affecting muscle relaxation (e.g., antiepileptic drugs, benzodiazepines, aminoglycoside, curare-like agents);
- (3) skin infection at the site of injection;
- (4) allergy to Botulinum toxin injection;
- (5) neuromuscular disease;
- (6) pregnant females [50].

DENTAL TREATMENT
- ORAL APPLIANCES
OCCUSAL SPLINTS

It is also known as bite splints, bite plates or night guards. It is removable appliance that can be classified according to material types, occlusal contacts, and condylar position. E. g. - Soft polyvinyl splints, Hard splints, Full coverage (Stabilization splints/Michigan splint), Palatal splints, Partial coverage splints, Anterior Repositioning (protrusive) splints, Posterior (distraction or pivot) splints. Based on designs, occlusal splints can unload, stabilize, and improve functions of the TMJ as well as reduce abnormal muscle activity, muscle pain, and improve functions of the masticatory motor system.

Soft polyvinyl splints - soft vacuum-formed splints are easy to construct and fit, but less durable and difficult to adjust. It increases masseter and temporalis muscle activity and

can lead to increased muscle pain/ discomfort in some patients.

Hard acrylic splints – It is more durable and more effective in reducing bruxism. It includes stabilization and anterior splints.

Repositioning (protrusive) and posterior (distraction or pivot) splints - are not used for managing sleep bruxism.

Stabilization splint – Stabilization splint is full coverage flat plane appliances designed with balanced contacts with all opposing teeth in centric relation. It reduces muscle activity and prevents the unwanted consequences of bruxism, such as grinding noises, tooth wear and associated pain.

Palatal splint is the splint with only palatal coverage and no occlusal coverage. This device does not interfere with the occlusion in any mandibular movements and it was adjusted for maximum tooth intercuspation, and any tooth contact upon mandibular movement is eliminated.

Partial coverage Anterior splints - example, the nociceptive trigeminal inhibition or NTI splint. It only makes contact with anterior teeth in the opposing dentition. It is based on the ability of anterior bite stops to reduce both temporalis and masseter activity during clenching and grinding.

Both anterior and stabilization splints reduce sleep bruxism on the first night and at 1 week but had no effect on sleep microarousals. Anterior splints are more effective than stabilization splints. It must be used with caution as it allows for unwanted tooth and condylar movements or over-eruption of uncovered teeth, if worn continuously for longer period.

For aggressive consistent grinders, a harder occlusal splint should be recommended. A hard/ soft type is 1 mm soft polyurethane for the inner layer and a 2 mm hard, more durable co-polyester outer layer. For less

aggressive and episodic grinders, a softer occlusal splint may be recommended.

Custom made occlusal splint are usually the best option for the patient. It is more comfortable and patient compliance is improved. The process for constructing this appliance is a two-appointment procedure.

At the first appointment, impressions of the maxillary and mandibular arches are taken. The impressions can be sent to a dental laboratory or the appliance can be fabricated in the office. Usually, occlusal splint is fitted to the maxillary. Sometimes a mandibular occlusal splint will be made if the patient has trouble with gagging or comfort. The second appointment is for delivery and to confirm the occlusal splint fits, feels right to the patient and to instruct them on the care of the occlusal splint[42,44].

- **RESTORATION OF SEVERE DENTAL WEAR ASSOCIATED WITH BRUXISM**

With Composite resin, crowns, direct and indirect restorations and ceramic veneers.

- **TREATMENT OF THE ABFRACTIONS**

As a result of greater occlusal stresses produced by bruxism, it increases the prevalence of non - carious cervical lesions. The materials usually used to restore abfractions are glass ionomers, resin-modified glass ionomers, and nanoceramic composite resins that have recently been introduced [51].

RECOMMENDED TREATMENT IN CHILDREN

Bruxism in children is so common that it is often considered normal behaviour. In addition, studies have shown that incidence of bruxism decreases from around nine to ten years of age. Management of sleep bruxism should be considered only when there is severe tooth damage and child reports pain, interrupted sleep, or the noise which is sufficient to disturb parents. It must be considered that they are in a stage of

development and growth of the maxillary structures, so each intervention carried out should be done with caution.

Chisini L, et al. suggest psychological treatment, physical therapy and the use of occlusal plates, but it is necessary to have an exhaustive and periodic controls if this type of treatment is carried out, since, for example, the use of splints can possibly restrict adequate maxillary growth, so, frequent appointments should be scheduled to monitor child bone growth and eruption of permanent teeth while placing splints in children.

A soft-based material is chosen to protect the primary teeth, thickness has to be sufficient (2mm) to prevent perforation and increase resistance to impact. Space should be created for erupting teeth in the splint to allow normal eruption. Despite the fact that bone growth of the alveolar process can be disturbed by the splint and consequently lead to orthodontic defects, the occlusal splint can decrease muscle activity and provide greater comfort to patients.

Gupta et al.,2010 reported significant reduction in grinding sounds following the use of occlusal splint in children. A systematic review done by *Macedo et al.,2007* concluded that occlusal splint offers benefits with regard to tooth wear.

Nonetheless, as the splints to be used in children could affect growth and development, it was recommended to have frequent follow-ups and use them for a period of 2-3 months.

Holmgren et al have shown in his study that splints do not stop bruxism but do redistribute the load borne by teeth and masticatory system.

Magnesium administration in children can also lead to significant reduction in sleep bruxism. Various studies recommend roughly 1.5 mg of magnesium per kg body weight combined with other vitamins and minerals once a day for at least five weeks.

Local therapies can also be considered in bruxing children which includes avoiding chewing gum, sleeping without a pillow, lying on back, wet heat, and no television

before bedtime. Children with upper airway obstructions or severe psychological problems should be referred to specialists.

Additionally, also pharmacological therapies such as flurazepam 15 mg/day, hydroxyzine 25-50 mg/day, and 5-25 mg/day for 4 weeks and diazepam 2.5 and 5 mg/day [49,51,53-57].

Acupuncture has been successfully used to treat bruxism in children with downs syndrome, resulting in a reduction in the activity of the masseter and anterior temporal muscles, as well as anxiety. Stimulation of specific acupuncture points, with the use of needles (dry needling), infrared radiation, electric current, or laser, altering the dynamics of circulation and relaxing muscles, so relieves muscle spasm, inflammation, and pain. Acupoint stimulation with laser is recommended in pediatric patients as it is painless and requires a shorter exposure time.

Furthermore, photobiomodulation can also be used in children with Down syndrome. study by Salgueiro et al. showed that acupuncture point treatment relieved the symptoms of bruxism in children and decreases cortisol level. Physiologically, photobiomodulation stimulates cells and promotes blood circulation, vasodilatation, analgesia, and anti-inflammatory, such as reduces edema and accelerates the healing process of injured tissues[58].

Rapid palatal expansion leads to reduction in the Rhythmic Masticatory Muscle Activity which can decrease bruxism activity in children [59].

Some studies have connected bruxism in children to dehydration. Inculcating a good habit of regularly drinking water can help in reducing bruxism in children [60].

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