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# Review Article

## All about bruxism-The teeth grinding

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***ABSTRACT*:**
Bruxism has turned out to be a mounting concern in current years due to its downbeat impact on the life value and also for being measured an imperative risk factor for temporomandibular dysfunctions. The grinding force compared to regular mastication is three to ten times more powerful enough to crack a walnut. Although bruxism is not a huge handicap, it can influence the quality of human life, especially through dental problems, such as tooth wear leading to inefficiency of mastication, pain in the facial region and tooth fractures. Hence early identification and preventive measures go a long way to protect the occlusion.
**Key words:** Bruxism, Occlusion, Dysfunction

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### INTRODUCTION

Stress related disturbances, including depression and anxiety, are a real problem in a highly developed society[1]. It is possible that different forms of stress have different effects on the child and the effects of suppressing emotions and motor activities burden the functionality of an organism resulting in several neuromuscular disorders. More often it results in the development of a various muscular parafunctional oral habits such as thumb sucking, finger biting or finger sucking, tongue thrusting, lip biting, mouth breathing and one such habit is bruxism. Thus in the light of the above knowledge a humble effort has been made to briefly explain the bruxism and its impact on the growing children.

Bruxism, (from Greek brychein, gnashing of teeth) is an oral parafunctional activity which may be diurnal or nocturnal grinding of the teeth, and is typically accompanied by the clenching of the jaw. The first description of this phenomenon in the scientific literature was by Marie and Pietkiewicz in 1907, when they introduced the French term “La bruxomanie” (a French term, translates to bruxomania). It was later adopted to “Bruxism” in 1931 by Bertrand Frohman who first coined the term bruxism, which comes from the Greek expression “brychienodontas”. (Figure1).

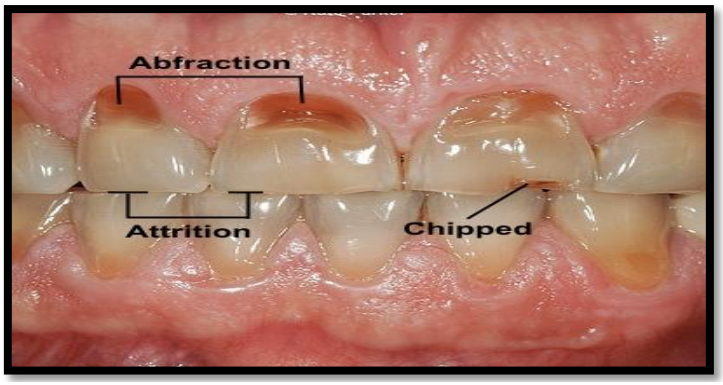


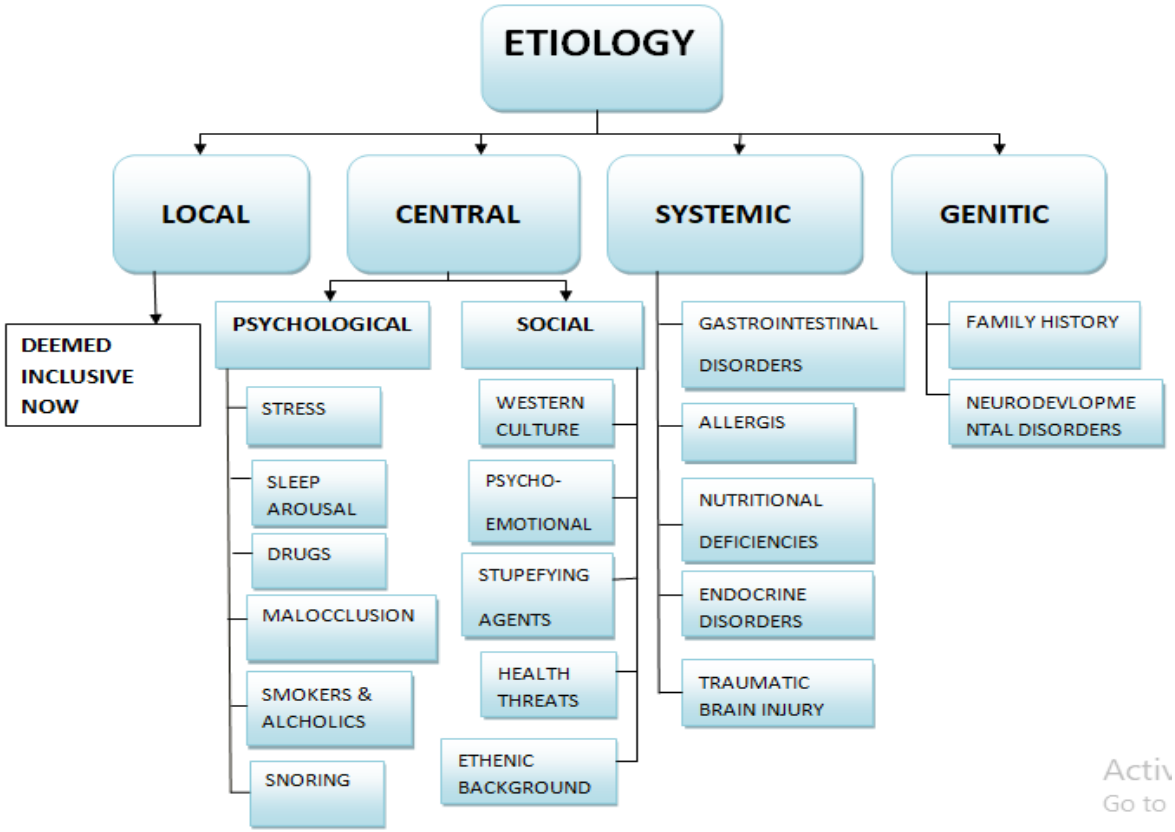

**Figure 1: Teeth grinding resulting in attrition, abfraction and chipping of the dentition.**

## INCIDENCE AND PREVALENCE

The true incidence and prevalence rates of bruxism is not known as over 80% of all bruxers may be unaware of the habit or be ashamed to admit it, hence they may dismiss any evidence of self-destructive behavior.[2] Clenching or grinding of teeth is common among three out of every ten children. In the general adult population the prevalence of bruxism ranges from 7% to 58%, with prevalence of awake bruxism ranging from 22.1% to 31% while that of sleep bruxism was more consistent at 13%.[3]. Thus, awake bruxism and sleep bruxism are considered as two separate entities of bruxism and hence prevalence of both the behaviors can effect the incidence and prevalence of bruxing episodes in different age groups, gender, socio-economic status and occupation of an individual.

## ETIOLOGY

Considering the latest literature the multifactorial etiology of bruxism is as under (Flowchart 1):




**Flowchart 1: Classification of etiological factors**.

Below is the brief description of each etiological factor predisposing to bruxism.

## I. LOCAL FACTORS

In the 1960's, dental theory stated that "peripheral" elements, such as dental malocclusion and mechanical factors, were the cause of sleep brusism. This theory has since been challenged and is now been deemed inconclusive. Thus peripheral factors derived from a presumed dental contact via periodontal mechanoreceptors are not required for coactivation of masticatory muscle activity.[4]

## II. CENTRAL /PSYCOSOCIAL FACTORS

Most current evidence (MieszkoWieckiewicz et al 2014) supports the hypothesis that bruxism is mediated by the central and autonomic nervous systems and especially by the dopaminergic system. The various psychosocial etiological factors related to both awake and sleep bruxism are as under:

### 1. Stress

Bruxers differs from healthy individuals in the presence of depression, increased levels of hostility and stress sensitivity. Various pathological emotional experiences more and more often result in the development of a muscular parafunction/bruxism.[16] Chronic stress belongs to the most destructive factors threatening a human organism and the warning reactions triggered by it manifest themselves as functional deficiencies of the nervous-muscle system and are the main etiologic factors of psycho-dependent bruxism.

### 2. Sleep arousal

Arousal response is a sudden change in the depth of the sleep during which the individual either arrives in th elighter sleep stage or actually wakes up. Sleep bruxism is an oromotor manifestation secondary to the microstructural sleep event 'micro-arousal' (i.e. an abrupt change in the frequency of cortical EEG that is occasionally associated with motor activity) (Macaluso et al 1998).

### 3. Drugs

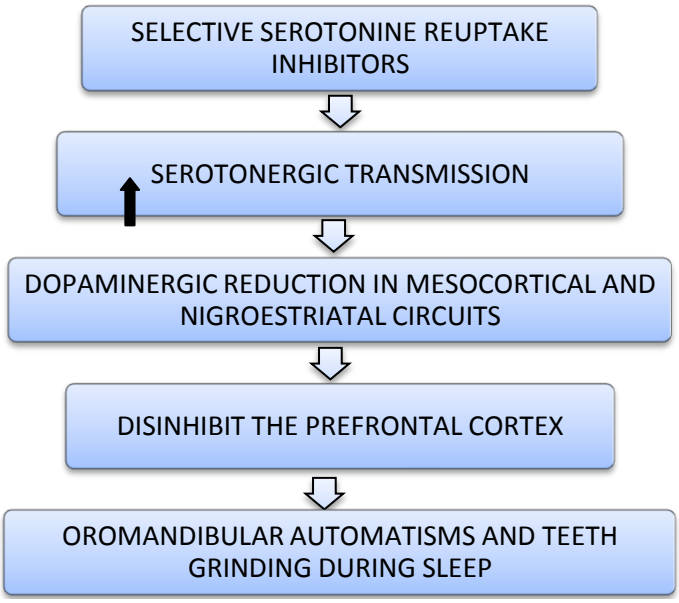
Anti-depressants and anti-psychotics drugs have been shown to increase bruxism. It is perceived that changes in the neurotransmission are responsible for teeth grinding.[5]

a) Dopamine precursors

Parkinson's disease sufferers may show teeth grinding during treatment due to the chronic (long-term) use of L-dopa which is likely the result of drug effect of presynaptic inhibition resulting in dopamine receptor hypersensitivity and may lead to manifestations of late dyskinesia and teeth grinding.[5]

a) Selective serotonine reuptake inhibitors antidepressants (SSRIS)

SSRIs have an indirect influence on the central dopaminergic system, which is the system that is thought to be involved in the genesis of bruxism (Flowchart 2).



**Flowchart 2: Antidepressants causing bruxism.**

(c) Amphetamine

The amphetamine-like medications that are used in the management of attention deficit hyperactivity disorder (ADHD), like methylphenidate, have bruxism as a possible side effect as reported by Malki et al.

**a) Smokers and alcholics**

Smokers of cigarettes report bruxism almost two times more than non-smokers (Lavigne et al,1997). Nicotine that stimulates the central dopaminergic activities explains the fnding that smokers can lead to bruxism episodes.[6]

**b) Habitual snoring**

In relation to sleep quality and architecture, bruxism and habitual snoring are found to be closely related.[7] There is an increased risk of reported sleep bruxism in the presence of loud snoring and obstructive sleep apnoea syndrome (OSAS).

**c) Malocclusions**

Significant factors in the development of occlusal parafunctions are malocclusions and abnormal bites. This is the imperfect alignment of the teeth that can cause irritation when the jaw is closed (ApostoleP.Vanderas).

**7. Social aspects**

Prevalence of bruxism depends on the development of civilization and the modern lifestyle. In this way the psychological aspect of occluso-muscle disorders becomes more significant[27]. In recent years, the number of patients suffering from bruxism has increased significantly due to various social aspects listed below.

a) Influence of western culture
b) Psychoemotional disorders
c) Stupefying agents
d) Change in health threats
e) Ethinic background

## III. SYSTEMIC FACTORS

The various systemic conditions predisposing to bruxism are as under:

**1. Gastrointestinal disturbances**

Among the systemic factors, gastrointestinal disturbances are one of the factors which can lead to bruxism. Bunting and Hill stated that bruxism may be caused by irritation elsewhere in the body, as for example, by hyperacid urine, or intestinal parasites were often named as causative.

**2. Allergies**

Bruxism may be caused by allergic processes, by asthma and by respiratory airway infection. Thus, bruxism may be a reflex of the central nervous system due to an increase in negative pressure in the middle and/or inner ear caused by allergic edema of the mucosa of the auditory tubes.[8]

**3. Nutritional deficiencies**

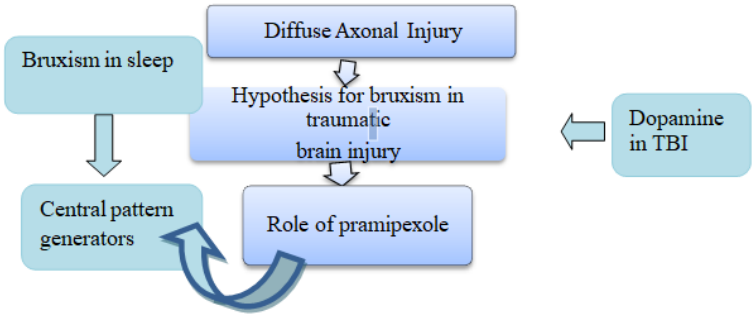
Miller (1943), Brauer and his coworkers (1947) mentioned subclinical nutritional deficiencies as predisposing factors to bruxism in children, for example, avitaminosis.

**4. Endocrine disorders**

'Miller' mentions vesical irritability and includes endocrine disorders such as an overactive thyroid producing nervous tension and, ultimately, bruxism.

**5. Traumatic brain injury**

Bruxism after brain injury is not often mentioned in literature. Certain speculations based on laboratory findings suggest the possibility of airway maintenance and respiration as underlying physiological mechanisms and the possibility of these events being mediated by a certain state of dopamine (DA) depletion (Flowchart 3).



**Flowchart 3: Hypothesis to explain the cause of bruxism in traumatic brain injury.**

**IV. Genetic**

Recent genetic studies (Aleksandra Calic and BorutPeterlin, 2015) indicate that the etiology of sleep bruxism involves the interaction of environmental and genetic factors. An indirect genetic effect, such as altered genetic sensitivity to environmental factors, most likely underlies the development of bruxism[9].

**SIGNS AND SYMPTOMS**

During bruxism the damage when lasts long with increased frequency and intensity, the patient will be having some or all of the following signs, symptoms and hence consequences of bruxism:

**1. Audible occlusal sounds**

Bruxism many times is diagnosed from a bed partner or someone observing the patient during a napping time who is disturbed due to the noise produced by grinding. The audibility is determined by the tooth-to-tooth friction, the area in which the grinding occurs and the force of the grinding. The sounds can be described as crunchy, chewing on gravel, rocks or marbles.[10]

**2. Fatigue and failuare of restoration**

When bruxism is involved, there is an abnormal increase in magnitude, frequency and duration elevating the pounds of pressure to increase to the maximum biting force of 200 to 300 pounds of pressure. While bruxing, the load can be as high as 1000 N, changing normal physiologic wear to severe wear, leading to fatigue failure and fractures of restorations or any previous treatment done in the mouth of a bruxing patient.[11]

**3. Wear patterns**

Wear facets are damage to the tooth, recognized as highly polished, smooth, flattened wear patterns on the enamel. The distal corners of the maxillary central and lateral incisors are very common areas where damage from bruxism is noticed. Signs of attrition on molars start with the cusps flattening out and can even wear through to the dentin[5] (Figure 2).

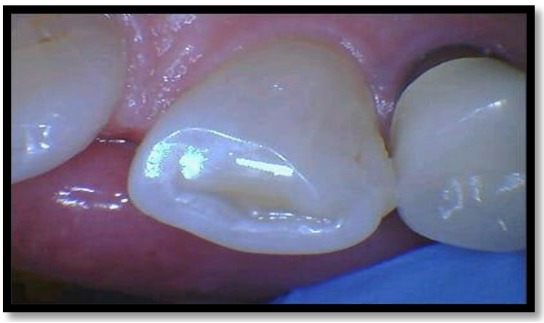


**Figure 2: Polished wear facets on lingual and occlusal surfaces of tooth.**

**4. Attrition**

Dental attrition is caused by tooth-to-tooth contact, resulting in loss of tooth tissue, usually starting at the incisal or occlusal surfaces. Bruxism is one the most common causes of attrition. Due to attrition, there is cupping appearance of anterior teeth and flattening of posterior teeth resulting in wearing down of the insisal and occlusal surfaces of the enamel, causing enough reduction to alter the vertical dimension and severe tooth sensitivity due to loss of enamel.[5] (Figure 3)

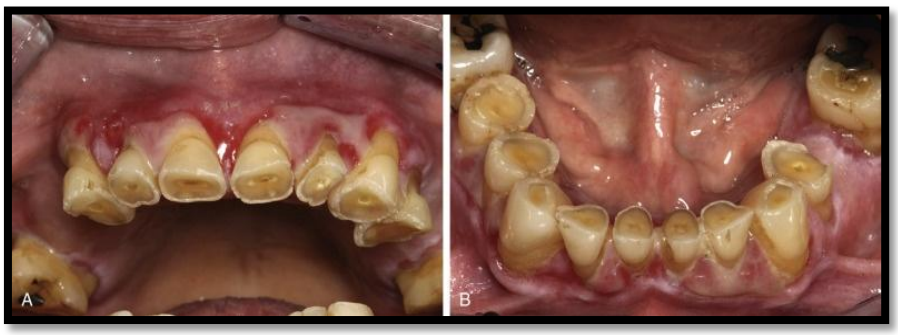



**Figure 3: Attrition and Cupping of incisors due to severe bruxism.**

**5. Abfraction**

It is a mechanism that explains the loss of enamel and dentin caused by flexure and ultimate tissue fatigue of susceptible teeth at locations away from the point of loading. This happens when the tooth has flexed greatly during the grinding process and the root surface begins to break down and form a scooped or notched out appearance.[11](Figure 4)


**Figure 4: Scooped out appearance called abfraction due to stress of constant grinding.**

**6. Periodontal recession and bone loss**

Bruxism can be a serious, aggravating factor in periodontal disease because it can interfere with the normal recovery time of the periodontium. This increases the risks for tissue breakdown and reduces the rate of regeneration (Figure 5). Localized bone destruction or an isolated periodontal pocket may occur with bruxing[11].

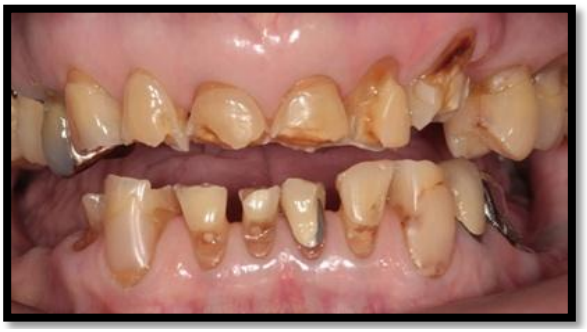


**Figure 5: Bruxism aggravating the periodontal disease and resulting in severe recession.**

**7. Mobility of the teeth**

With bruxism, the teeth are forcefully rocked back and forth in the socket, which can cause temporary mobility, progressing to risks of permanent mobility. Tooth mobility with sleep bruxism is greater in the morning and is significant when found in teeth with little or no evidence of periodontal disease. Such teeth may exhibit a dull percussion sound and patient may report soreness when biting on the tooth or teeth.[12]

**8. Buccal exostosis and tori**

Bony out-growths that form where there is an excessive amount of stress and tension placed on the teeth's underlying structures are called tori or exostosis. Due to bruxism excess strain and tension placed on the teeth and jaws, the body's defense is to produce extra boney material to support the teeth (Figure 6). This bone grows and becomes visible under the soft tissue.[5]

**Figure 6**: **Buccal exostosis, palatine and mandubular tori in a patient with wake bruxism**.

**9. Sensitivity of the teeth**

Sensitivity can be localized, generalized, constant or sporadic. One of the first symptoms of bruxism is hot and cold sensitivity to the teeth. This is caused by the flexing that occurs when teeth are ground from side to side. The thinning of enamel can cause underlying dentinal sensitivity.[5]

**10. Damage to the restoration**

Any reconstructive work that may have been done on the teeth could get destroyed by constant grinding. Cavities that are already present can get worse due to constant grinding.

**11. Acute or chronic pain**

Fortunately or unfortunately, bruxism aches and pains are a functional, healthy response. Any injury to the body causes other muscles, ligaments or tendons to overcompensate for the injury. Over time, those compensations also become weak and unproductive. Common aches or pains can progress from mild to severe depending on the aggressiveness and repetitiveness of the bruxing.[10]Any of these pains can be acute or chronic.

**a) Fatigue of Muscles**

When grinding the posterior teeth, the masseter and temporalis muscles are more involved resulting in hypertrophy of masseter as in (Figure 7 A& B) which shows midline shift and hypertrophy of left masseter muscle[10].

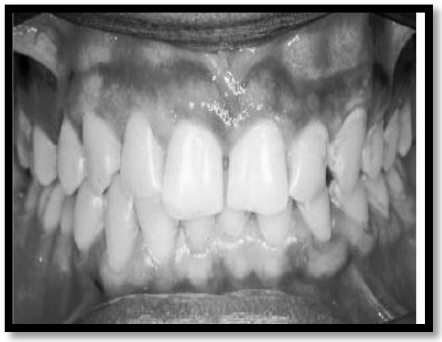


**Figure 7 A**: **Midline shift during occlusion in a patient having bruxism.**

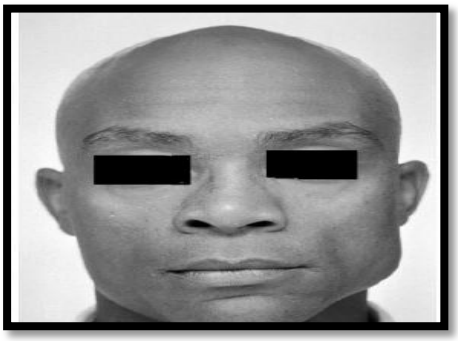


**Figure 7 B**: **Masseter muscle hypertrophy in bruxism patient.**

**b) Temporomandibular Disorder (TMD)**

Often clicking, popping, or grating noises are heard in bruxing patients TMJ. Occasionally, this can be a painful event that results in a reflex contraction of the chewing muscles which locks the TMJ in an open dislocated position[10]. This is referred to as an open lock.

**c) Headaches**

Headaches can be in the morning or night, constant or fluctuating and can range from a slight dull pain to an intense migraine.

**d) Pain in cheeks**

Cheeks can feel tired, worn or sore when chewing. During nocturnal bruxism, suction is created in the mouth, due to which there cheek bite and tonue. The symptoms of this can be recognized from scalloping of tongue and lineaabla on buccal mucosa of cheek (Figure 8 A& B).

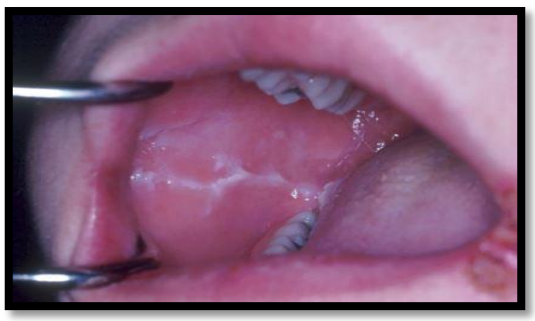


**Figure 8 A: Linea alba due to cheek bite in**

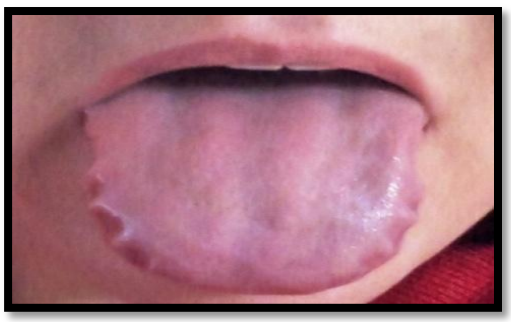


**Figure 8 B: Scalloping of tongue due**

**bruxism.** **to suction created in bruxism patients**.

**DIAGNOSIS**

The key to successful treatment is always early and correct diagnosis. Diagnosis of clenching is difficult, leading clinicians to often fail to detect the presence of clenching (false negatives), or to mistakenly tell people that they clench when in fact they aren't (false positives). The various diagnostic approaches to detect bruxism is as follows.

**I. NON-INSTRUMENTAL APPROACHES**

Non-instrumental approaches for assessing bruxism include:

1. **Self-report**
2. **Clinical inspection**

1. **Self-report**

Self-reported assessment of sleep or awake bruxism continues to be the primary tool in bruxism research and clinical practice. Questionnaires are adjuncts in both research and clinical situations[13]. Table 1 consists of the questions asked that can confirm bruxism via questionnaires:

| QUESTIONNAIRE |
|---|
| 1. Has anyone heard you grinding your teeth at night? |
| 2. Is your jaw ever fatigue or sore on awakening in the morning? |
| 3. Are your teeth or gums ever sore on awakening in the morning? |
| 4. Do you ever experience temporal headache on awakening in the morning? |
| 5. Are you ever aware of grinding your teeth during the day? |

**Table 1**: **Questionnaire for detecting bruxism.**

The positive response of the above questions can help confirm the condition in adjunct to other confirmatory tests.

2. **Clinical inspection**

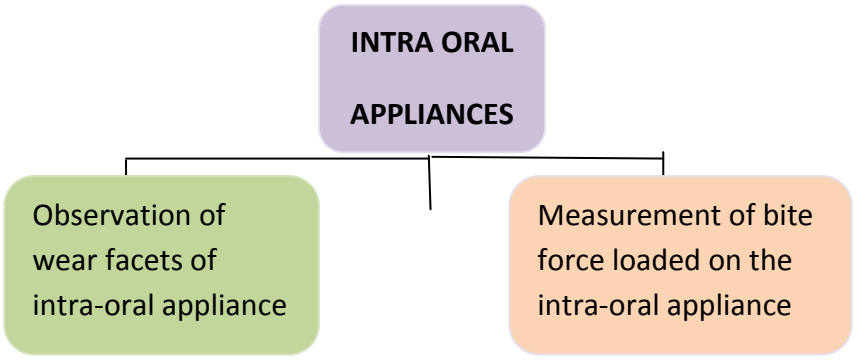
The clinical diagnosis of bruxism can be traced out by evaluating the tooth mobility, tooth wear and other clinical findings listed in (Table 2).

| CLINICAL AND ANAMNESTICAL INDICATORS FOR BRUXISM |
|---|
| **1.** Presence of tooth wear seen within normal range of jaw movements or at eccentric position |
| **2.** Presence of masseter muscle hypertrophy on voluntary contraction |
| **3.** Complain of masticatory muscles discomfort, fatigue or stiffness in the morning (occasionally, headache in temporal muscle region) |
| **4.** Tooth or teeth hypersensitive to cold air or liquid |
| **5.** Tongue or cheek indentations |

**Table 2: Clinical indicators to diagnose bruxism.**

**II. INSTRUMENTAL APPROACHES**

1. **Intra oral appliance:** Bruxism activity can be measured using the intra-oral appliance and is classified into two groups (Flowchart 4)



**Flowchart 4: Various intraoral appliances to diagnose bruxism.**

a) Bruxcore plate

The Bruxcore Bruxism-Monitoring Device (BBMD) is an intra-oral appliance that measures sleep bruxism activity objectively (Figure 9). The Bruxcore plate evaluates bruxism activity by counting the number of abraded microdots on its surface and by scoring the volumetric magnitude of abrasion[14].

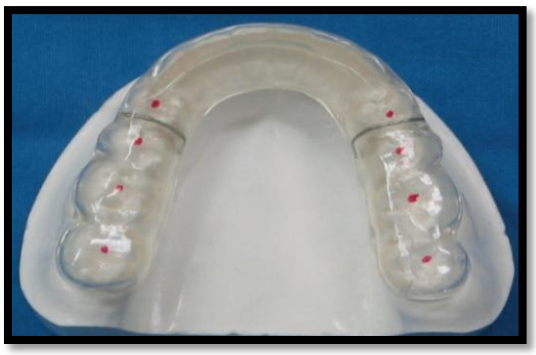


**Figure 9: Occlusal view of an intra-oral appliance for monitoring bruxism**.

b) Intra-splint force detector

An intra-splint force detector (ISFD) is a recording device designed for sleep bruxism (Figure 10), which uses an intra-oral appliance to measure the force being produced by tooth contact onto the appliance.

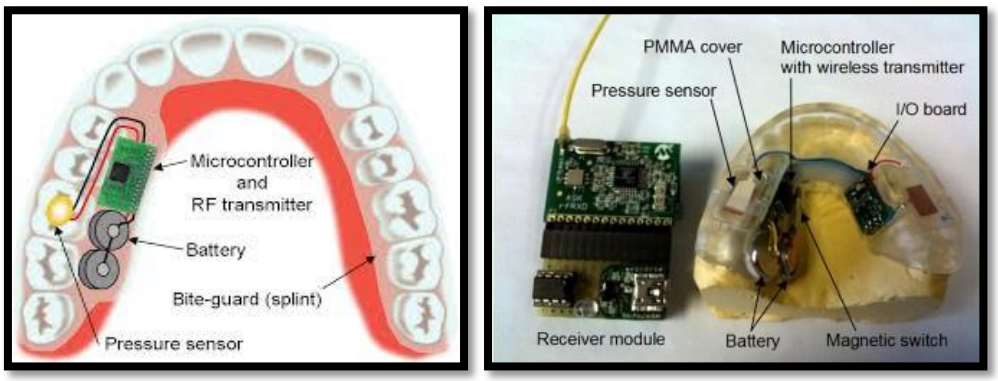



**Figure 10: Intra-splint force detector (ISFD) used for recording sleep bruxism.**

2. **Extra-oral appliances:** Bruxism activity can be measured using the intra-oral appliance as follows:

a) Masticatory muscle electromyographic recording

One among the plethora of assessment tools, Electromyographic (EMG) recordings may provide key evidence of awake bruxism as well as sleep bruxism. The main advantage is that the bruxism can be measured without intra-oral devices, which may change natural bruxism activity.[13]

b) Portable EMG recording devices**:**

Since the 1970s, sleep bruxism episodes were measured over a period of time in patients' homes with the use of battery-operated EMG recording devices. A surface EMG electrode with a built-in buffer-amplifier and a cordless type of EMG measurement system was developed as well, to improve the accuracy of recordings (Figure 11).

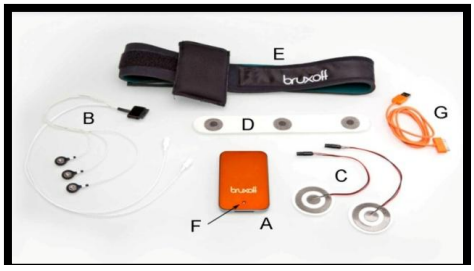


**Figure 11**: **Portable recording device with 3 channels allowing to record and score sleep bruxism**. (A. Bruxoff®. B. Connection cables. C. EMG electrodes. D. Cardiac electrode. E. Thoracic belt. F. On/off button. G. USB cable.)

c) Miniature self-contained EMG detector analyser**:** A miniature self-contained EMG detector–analyser (Bite-Strip) was developed as a screening test for moderate to high level bruxers (Figure 12). Its salient feature is that the number of bruxism events can be objectively estimated by simply attaching it to the skin over the masseter muscle[14].

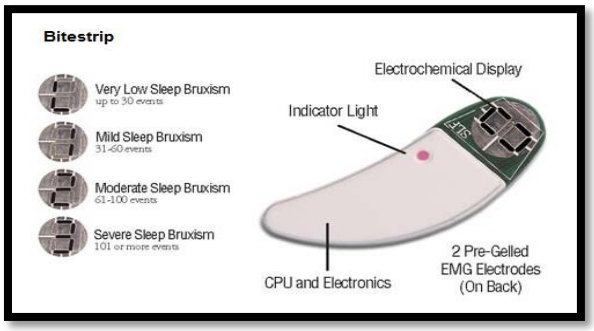



**Figure 12**: **Miniature self-contained EMG detector–analyser (Bite-Strip) to detect bruxism.**

## III. POLYSOMNOGRAPHY

Polysomnographic (sleep laboratory) recordings for sleep bruxism includes electroencephalogram, EMG, electrocardiogram and thermally sensitive resistor (monitoring air flow) signals along with simultaneous audio–video recordings.[15]

**Cut-off points:** More recently, the diagnostic consequences of the time-variant nature of sleep bruxism were quantified, yielding the suggestion to use cut-off points when diagnosing sleep bruxism[16]. The following polysomnographic diagnostic cut-off criteria are suggested:

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

When the polysonmnographic bruxism-related variables are combined under logistic regression, the clinical diagnosis is correctly predicted. The positive and negative predictive values of these cut-offs are evaluated according to Sacket et al. (1985).

## IV. GRADING

Despite an abundance of techniques, reliable and valid diagnostic tools for bruxism are scarce. Therefore, following the approach recently suggested for the grading of neuropathic pain , Lobbezoo et al in 2013 proposed a diagnostic grading system of ‘possible’, ‘probable’ and ‘definite’ sleep or awake bruxism for clinical and research purposes and to determine the likelihood that a certain assessment of bruxism actually yields a valid outcome[9]. Therefore, it is suggested that the grading system proposed in 2013 is transformed as follows:

1. **Possible sleep/awake bruxism**
   It was suggested that ‘possible’ sleep or awake bruxism should be based on self report, by means of questionnaires and/or the anamnestic part of a clinical examination.

2. **Probable sleep/awake bruxism**
   ‘Probable’ sleep or awake bruxism should be based on self-report plus the inspection part of a clinical examination.

3. **Definite sleep/awake bruxism**
   ‘Definite’ sleep bruxism should be based on self-report, a clinical examination, and a polysomnographic recording, preferably along with audio/video recordings. For polysomnography, cut-off points for a sleep bruxism diagnosis have been proposed.

## MANAGEMENT

Bruxism management relies on the recognition of the potential causative factors associated with the development of bruxism. The patient should be offered a comprehensive management plan while the condition is being treated.

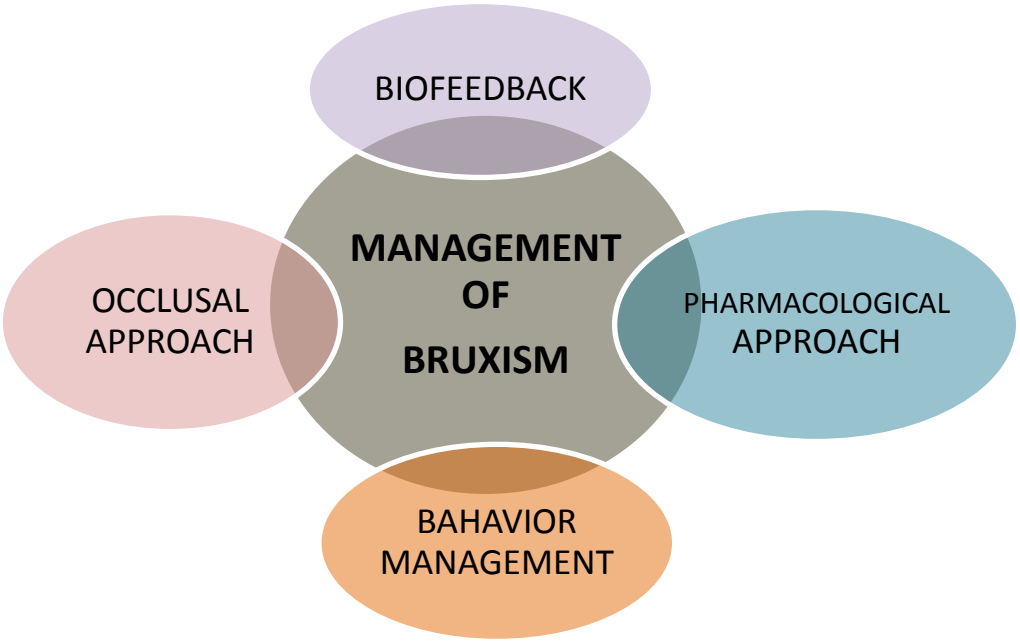



**Figure 13: Different management strategies of bruxism.**

Following is the stepwise treatment and recommendations for managing bruxism.

### I. WAIT AND WATCH

In few cases, bruxism may vanish spontaneously. In particular, young children often require different therapeutic approaches than adults. Juvenile bruxism is probably "a self-limiting condition which does not progress to adult bruxism and which appears to be unrelated to TMJ symptoms" (Kieser&Groeneveld, 1998). Either way, when the damage to a child or adult is severe, or when the habit persists, treatment is mandatory[30]

### II. SELF-CARE STEPS

One of the mainstays of bruxism treatment is that the patient is aware of the disease, and based on literature reports, following self-care measures should be adopted[29] (shino bay Aguilera et al; JCAD 2017):

1. Application of ice or wet heat to sore jaw muscles.
2. Avoid eating hard foods like nuts, candies, and steak.
3. Avoid chewing gum.
4. Averts jaw play and other bad oral habits. This refers to nonfunctional jaw movements without tooth physical touching.
5. Drink plenty of water every day.
6. Get plenty of sleep.
7. Learn physical therapy stretching exercises to help restore the action of the muscles and joints on each side of the head to get back to normal.
8. Massage the muscles of the neck, shoulders, and face.
9. Looks for small, painful nodules called trigger points that can cause pain throughout the head and face.
10. Relax the face and jaw muscles throughout the day.
11. The goal is to make facial relaxation a habit.
12. Try to reduce daily stress and learn relaxation techniques.
13. Wear a properly fitted mouth guard at night while sleeping.
14. Recommend taking ltheanine 100 to 200mg orally daily to help cope with stress

### III. REMOVAL OF ETIOLOGICAL FACTORS

#### 1. Stress

In some cases, emotional stress is alleged to trigger, or exacerbate, bruxism. Management should be done by first removing the causative factor, that is in assistance with psychiatric, the psychologically effecting chronic stress should be dealed with.

### 2. Counteracting trauma

In some cases, bruxism may commence shortly after dental procedures such as fillings, crowns, or bridges; after an injury to the mouth; or after a prolonged operation in or through the mouth. In this case, the corrective procedure should be undertaken as soon as possible, to prevent entrenchment of the bruxing habit.

### 3. Bruxism secondary to drugs

Antidepressant and antipsychotic medications may trigger bruxism in non-bruxers[16] (Brown & Hong et al, 1999). If bruxism developed shortly after the beginning of antidepressant "therapy," the prescribing clinician should be notified and consulted about the desirability of reducing the dose of the antidepressant, switching to another antidepressant, or prescribing a drug which will counteract the bruxism-inducing effect of the antidepressant.

### 4. Medical conditions

If an underlying medical condition, such as gastroesophageal reflux disease (GERD), is identified as the cause, treating this condition may improve bruxism.[17]

### 5. Bruxism associated with neurological disorders

The management of bruxism associated with neurological disorders focuses on improvement of chewing, speaking, swallowing, and feeding, which are severely compromised, and to relieve orofacial pain symptoms.

## IV. OCCLUSAL THERAPY

**Occlusal interventions**

The occlusal management strategies for bruxism contains the frequently used occlusal appliances. Hard splints are generally preferred over soft splints for practical reasons to prevent inadvertent tooth movements and also because hard splints are suggested to be more effective in reducing bruxism activity than soft splints. Occlusal splints have the following functions:-

1. To relax the muscles
2. To allow the condyle to seat in centric relation
3. To provide diagnostic information
4. To protect teeth and associated structures from bruxism
5. To reduce cellular hypoxia levels.

## V. BIOFEEDBACK APPROACHES

Biofeedback therapy may be an effective, novel, and convenient approach for the treatment of bruxism. This technique has been applied for bruxism during wakefulness as well as for sleep bruxism. While awake, patients can be trained to control their jaw muscle activities through auditory or visual feedback from a surface EMG. For sleep bruxism, auditory, electrical, vibratory and even taste stimuli can be used for feedback.[22]

**Principle:** Biofeedback uses the paradigm that bruxers can ‘unlearn’ their behaviour when a stimulus makes them aware of their adverse jaw muscle activities (‘aversive conditioning’) (Figure 14).

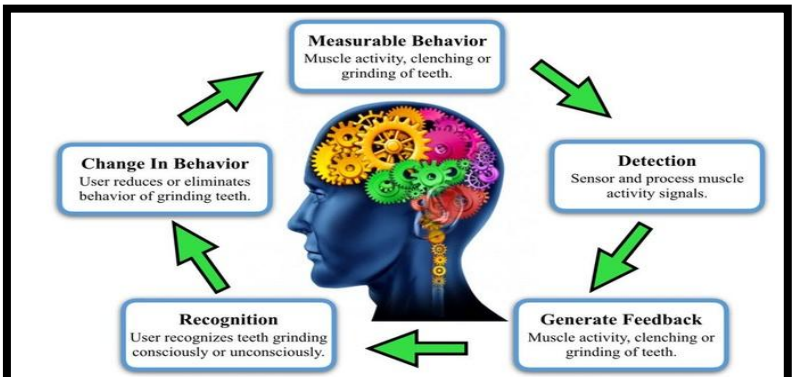



**Figure 14: Mechanism of biofeedback approach of managing bruxism**.

### 1. Sound Alarm Feedback

Mittelman[18] described an EMG technique that provides the daytime clencher with auditory feedback from his/her muscle activity letting him know the degree of muscle activity or relaxation that is taking place.

2. **Vibration Alarm**
   For the purpose of nerve system and muscle relaxation, a watch-style device around the patient's wrist vibrates to alert the patient of teeth grinding or clenching if the value of biting force and duration exceed the threshold. It was observed after 6 weeks therapy, that the average incidence of bruxism declined dramatically from 10 to 3 times during any given night, and the average duration of bruxism events was reduced from 21 seconds to 10.[19]

3. **Taste feedback**
   Nissani used a taste stimulus to awaken the patient. This stimulus was caused by the bruxism-related rupture of capsules filled with an aversive substance such as hot peppers (capsaicinoids), horseradish, quinine, mustard, ginger, garlic, onion, salt, or denatonium benzoate (with consent) in the dental appliance, to which long-term success was claimed.[11]

4. **Contingent electrical stimulation**
   For the use of biofeedback in the management of sleep bruxism, Cherasia and Parks published a technique which used contingent arousal from sleep with actual awakenings which reduces the masticatory muscle activity associated to sleep bruxism[20] (Figure 15).

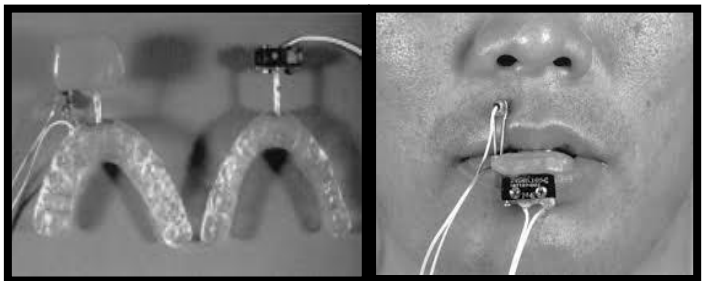


**Figure 15: Contingent electrical lip stimulation.**

Thus the major disadvantage of such approaches are awakenings because sleep disruption may lead to serious side effects like excessive daytime sleepiness. Therefore, such techniques need further attention, so that it can be applied for the safe treatment of patients with bruxism.

## VI. PHARMACOLOGICAL APPROACH

Drugs that have paralytic effect on the muscles through an inhibition of acetylcholine release at the neuromuscular junction decreases bruxism activity[21.]A range of drugs that have been suggested for pharmacological treatment are as follows in (Table 3).

| **PHARMACOLOGICAL AGENTS** | **DOSAGE (**per dose) |
|---|---|
| **Muscle relaxants, sedatives, anxiolytics** | |
| Diazepan | 2-10mg (orally 2 to 4 a day) |
| Clonazepan | 1.5 mg (orally per day divided into 3 doses) |
| Metocarbarnol, baclofen | 5 mg (orally 3 times a day for 3 days) |
| Zolpidem | 5-10mg (once a day prior bedtime) |
| **Dopaminergic agents** | |
| L-dopa | 3-5mg per day |
| Bromocriptine | 1.25 to 2.5 mg (orally once a day Increase in increments of 2.5 mg every 2 to 7 days) |
| Ropirinole | 0.5-10mg per day |
| Pergolide | 0.05 mg (orally once a day for the first 2 days) |
| **Beta-adrinergic agonists** | |
| Clonidine | 0.1 to 0.3 mg (orally once or twice a day) |
| **Antidepressants** | |
| Amitriptilyne | 25mg twice daily |
| Venlafaxine | 37.5-150mg (once in 4 days) |
| **Others** | |
| Botulinum toxin A | 25-100MU |

| | |
|---|---|
| Buspirone | 7.5 mg (orally 2 times a day) |

**Table 3: Various drugs and their dosage for treating bruxism.**

Thus it can be concluded that although some pharmacological approaches for bruxism seem promising, they all need further efficacy and safety assessments before clinical recommendations could be made.

## VII. NUTRITIONAL SUPPLEMENTS

### 1. Magnesium supplements

Magnesium's vital role in nerve and muscle function led at least two researchers to the suspicion that bruxism may be traceable to insufficient consumption, or inefficient utilization, of this metal. According to Ploceniak (1990), for instance, prolonged magnesium administration nearly always provides a cure for bruxism.[22]

### 2. Calcium and pantothenic acid (vitamin B5)

Cheraskin & Ringsdorf (1970) studied the effects of nutritional supplements on teeth grinders or clenchers. It was concluded that after surveying a year later, the active agents were calcium and pantothenic acid (vitamin B5) in which bruxism had vanished.[23]

## CONCLUSION

Bruxism is an umbrella term grouping together different motor phenomena of jaw muscles, such as grinding, and/or clenching of the teeth and bracing, and/or thrusting of the mandible. Allthough bruxism is not a huge handicap; it can influence the quality of life, thus whenever bruxism treatment is indicated, the disorder should be assessed by a multidisciplinary team that includes dentists, psychologists and medical specialists giving child the opportunity to learn to deal with their conflicts and tensions.

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