INTRODUCTION

For years, dentists have been fascinated and sometimes even obsessed with the phenomenon called bruxism. It has been underdiagnosed, overdiagnosed, and even misdiagnosed. Bruxism is known as parafunctional grinding of teeth or an oral habit consisting of involuntary rhythmic or spasmodic non-functional grinding or clenching of teeth in other than chewing movements of the mandible which may lead to occlusal trauma. At present a multi-factor etiology is accepted that includes genetic, neuro-physiological, psycho-emotional and pharmacological factors. The researchers should focus on bruxism behavior rather than bruxism as a disorder.

Tooth grinding is important to dentists because of breakage of dental restorations, tooth damage, induction of temporal headache and temporomandibular disorders.1 Bruxism is derived from the Greek word βρυγμός (brygmós), which means “gnashing of teeth”. People suffering with bruxism are also called “bruxists” or “bruxers” and the act is called “to brux”. The term ‘la bruxomanie’ was first introduced by Marie Pietkiewicz in 1907.2 It was later adopted as ‘bruxism’ to describe gnashing and grinding of the teeth occurring without a functional purpose. Glossary of Prosthodontic Terms (GPT-8)3 defines bruxism as parafunctional grinding of teeth or an oral habit consisting of involuntary rhythmic or spasmodic non-functional gnashing, grinding or clenching of teeth in other than chewing movements of the mandible which may lead to occlusal trauma.4 Bruxism may occur during the day or night time.5 Mostly, patients clench their teeth throughout the day as well as clench them during sleep. However, nocturnal bruxism is more common and it varies with the individual and is related to emotional or physical stress. Bruxism causes tooth wear and it is evidenced by wearing facets which varies from mild to severe and can be localized or may be seen throughout the dentition.6 It causes trauma to dentition and supporting tissues which includes thermal hypersensitivity, tooth hypermobility, injury to periodontal ligament, hypercementosis, fractured cusps and pulpal necrosis.7

Bruxism is defined as non-functional movements of the mandible with or without audible sound occurring during the day or night time.6 Sleep bruxism (SB) is a parafunctional oromotor habit and according to the second International Classification of Sleep Disorders (AASM 2005): it may be defined as an oromotor activity which is characterized by the clenching or grinding of the teeth during sleep, and is habitually associated with micro-arousals and is generally accompanied by sound.8 Bruxism may be considered as a normal habit of an individual, but in few circumstances, like an increase in the frequency of episodes of bruxism and the strength of masseter muscle contractions may turn into a phenomenon that may cause pathological consequences.9 Bruxism may change the results and the duration of treatments performed by clinicians. It may lead to fracture of natural teeth, prostheses, or dental restorations. Bruxism may also be a contraindication to implants; as it influence in implant placement and its prognosis.9

ETIOLOGY

Etiology of bruxism can be divided into three categories; they are psycho-social factors, peripheral factors and patho-physiological factors. The etiology of sleep bruxism is uncertain; the factors responsible are occlusal discrepancies and the anatomy of bony orofacial structures. Other factors include smoking, trauma, alcohol, drugs, systemic disease, stress or peer pressure. Heredity appears to play an important role in the occurrence of sleep bruxism. Recent studies suggest that sleep bruxism episodes of individual are part of sleep arousal response. It is a sudden change in the depth of sleep of an individual. Besides this, sleep bruxism appears to be a disturbance in the dopaminergic system.10 Previously, morphological factors like occlusal discrepancies and anatomy of the bony structures of orofacial region have been considered as pivotal factors for bruxism. However, nowadays these factors are thought to play only a minor role, as recent studies focuses more on patho-physiological factors.11
CLASSIFICATION

Bruxism may be classified according to several criteria.¹

According to occurrence:

a. Awake bruxism
b. Sleep bruxism
c. Combined bruxism

According to etiology:

a. Primary, essential or idiopathic bruxism: No apparent cause is known.
b. Secondary bruxism: Secondary to disease (e.g. coma, ictus, cerebral palsy), medicinal products (e.g. antipsychotic medication, cardioactive medication), drugs (e.g. amphetamines, cocaine, ecstasy).

According to motor activity type:

a. Tonic: Muscular contraction sustained for more 2 sec.
b. Phasic: Brief repeated contractions of masticatory musculature with three or more consecutive bursts of EMG activity that last 0.25 - 2 sec.
c. Combined: Alternating appearance of tonic and phasic episodes. Approximately 90% of the episodes of SB are phasic or combined, unlike in awake bruxism, where episodes are predominantly tonic.

According to presence:

a. Past bruxism
b. Present bruxism

THEORIES ON BRUXISM

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. There are few controversies regarding theories on bruxism.

Disorders, such as malocclusion may be the cause of clenching and gnashing. It is based on the theory that occlusal maladjustment leads to reduction in masticatory muscle tone. In the absence of occlusal equilibrium, motor neuron activity of masticatory muscles is triggered by periodontal receptors.

Second theory states that, a central disturbance in the area of basal ganglia plays an important role in causing bruxism. An imbalance caused due to the processing of basal ganglia is the main reason behind muscle hyperactivity during nocturnal dyskinesia such as bruxism. Few authors suggest that bruxism constitutes sleep-related parafunctional activity such as parasomnia. A recent study which explains the potential imbalance of the basal ganglia is neuroplasticity. Neural plasticity is based on the ability of synapses to change or modify the way they work. Due to activation of neural plasticity, changes in the relationship between inhibitory and excitatory neurons occur.¹²

SIGNS & SYMPTOMS OF BRUXISM¹

Signs:

- Grinding of teeth, which has a characteristic sound
- Pain in the TMJ
- Pain in the masticatory and cervical muscles
- Headache (especially in temporal zone when patient wakes up in the morning)
- Hypersensitive teeth
- Excessive tooth mobility
- Insomnia or poor sleep quality
- Tiredness

Symptoms:

- Abnormal tooth wear
- Hypertrophy of the masticatory muscles like masseter
- Reduction in salivary flow rate
- Gingival recession

ASSESSMENT & DIAGNOSIS OF BRUXISM

Sleep bruxism (SB) is an oro-mandibular behavior which is defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding and/or clenching.¹³ Sleep bruxism has been classified as sleep related movement disorder according to Classification of Sleep Disorders.³ Bruxism activity can be estimated using intra-oral appliance.¹⁴

Clinical methods to assess bruxism:

1. Questionnaires for evaluation or detection of bruxism
2. Clinical Findings
3. Tooth Wear

Questionnaire for detection of bruxism⁸

Scales: Yes / No / Don’t know

1. Do you have the habit of grinding your teeth while sleeping?
2. Has anyone heard you grinding your teeth while you are asleep?
3. After awakening, whether you find that you are clenching your teeth?
4. After awakening, whether you find pain in jaw or jaw fatigue?
5. After awakening, whether you usually have the feeling that your teeth are loose?
6. After awakening, whether you usually have sore teeth and/or sore gums?
7. After awakening, whether you usually have a headache in the temples?
8. After awakening, whether you usually have a jaw lock?
9. Have you ever realized that you were clenching your teeth during daytime?
10. Have you ever realized that you were grinding your teeth during daytime?
Tooth Wear Scores

Tooth wear can be considered to be an analogous to bruxism. The incisal or occlusal wear for a single tooth can be evaluated by the following scores:

0. No or negligible wear of the enamel
1. Obvious wear of enamel or wear through the enamel to the dentine in single spots
2. Wear of the dentine up to one-third of the crown height
3. Wear of the dentine up to more than one-third of the crown height; excessive wear of tooth restorative material, crown or bridgework

The individual tooth-wear index (IA) is calculated from the given criteria (scores of incisal or occlusal wear for each tooth of that individual). Tooth wear is a cumulative record of both functional and parafunctional activities and various other factors such as age, gender, diet and bruxism which are associated with tooth wear. Erosion by acidic drink is considered to be an important contributing factor for tooth wear.15

Detection of bite force

Takeuchi et al16 developed a recording device for sleep bruxism, an ‘intra-splint force detector (ISFD)’, which uses an intra-oral appliance to measure the force being produced by tooth contact onto the appliance.

A miniature self-contained EMG detector–analyzer (BiteStrip) was developed as a screening test for bruxism.17 It comprise of EMG electrodes, an amplifier, central processing unit (CPU) with software, a display which shows the outcome in the monitor, a light emitting diode and a lithium battery which records the number of masseter muscle activities above a threshold. The characteristic feature of this device is that a number of bruxism events can be estimated by simply attaching this device to the skin over the masseter muscle.16

Polysomnography

The physiological changes related to sleep bruxism like microarousal and tachycardia can also be monitored. Hence, a polysomnographic study allows multi-dimensional analyses of sleep-related physiological behaviors and studies which show sleep laboratory EMG-based assessments are reported to be very reliable. One of the major limitations is that a change in the environment for sleep may influence the actual behavior of bruxism.17

PATHOLOGY OF BRUXISM

- Inflammation of the periodontal ligament, leading to loosening of the teeth.
- Glossodynia: burning sensation on the tongue;19 possibly related to a coexistent “tongue thrusting” parafunctional activity.
- Indentations of the teeth in the tongue (“crenated tongue” or “scalloped tongue”).20

MANAGEMENT OF BRUXISM

Occlusal Intervention: Butler evaluated an occlusal adjustment procedure for the treatment of bruxism but without proper theoretical basis.21 Similarly, Frumker formulated a set of principles for a successful occlusal treatment on the basis of the idea that, better the occlusal anatomy and function, the easier the bruxers relieve tension in the masticatory and associated musculature.22

Figure 1: Occlusal splint

Occlusal Appliances: The second category of occlusal management includes occlusal appliances. These splints have different names (e.g. occlusal bite guard, bruxism appliance, bite plate, night guard) and have different appearances and properties (Fig 1). Mostly they are hard acrylic-resin stabilization appliances, which are mostly worn in the upper jaw. Hard splints are generally preferred over soft splints for practical reasons (e.g. soft splints are more difficult to adjust than hard ones) to prevent inadvertent tooth movements. Another reason is that hard splints are suggested to be more effective in reducing bruxism activity as compared to soft splints.23

Biofeedback: The principle behind biofeedback is based on the fact that bruxers can ‘unlearn’ their behaviour when a stimulus makes them aware of their adverse jaw muscle activities (aversive conditioning). This technique has been applied for bruxism during wakefulness and for sleep bruxism that is it can be used both in day and night time.
Mittelman described the use of biofeedback in the management of bruxism. He described an EMG technique which provides the daytime clencher with auditory feedback from his/her muscle activity letting the individual to know the degree of muscle activity or relaxation that is taking place.

**Pharmacological Approach:** Drugs which have paralytic effect on the muscles through an inhibition of acetylcholine release at the neuromuscular junction (botulinum toxin) causes decrease in bruxism activity especially in severe cases like coma, brain injury, autism and Huntington’s disease.

**CONCLUSION**

Sleep bruxism is a parafunctional oromotor habit with a high prevalence in the general population. Recently, a multifactor model showing different etiological events had been accepted which includes genetic, neuro-physiological, psycho-emotional and pharmacological factors. Researchers will have to evaluate other aspects as well to determine the risk and consequences such as tooth damage and pain by bruxism.

**REFERENCES**

19. Macedo, CR; Machado MAC, Silva AB, Prado GF. Pharmacotherapy for sleep bruxism; Cochrane Database of Systematic Reviews. John Wiley & Sons, Ltd.