ISSN Print: 2394-7489
ISSN Online: 2394-7497
IJADS 2017; 3(4): 225-229

www.oraljournal.com
Received: 03-08-2017
Accepted: 04-09-2017

Renu Gupta
Professor and Head Department of Prosthodontics, H.P.G.D.C. Shimla, Himachal Pradesh, India

RP Luthra
Professor and Principal Department of Prosthodontics, H.P.G.D.C. Shimla, Himachal Pradesh, India

Akrant Mehra
Junior Resident Department of Prosthodontics, H.P.G.D.C. Shimla, Himachal Pradesh, India

Correspondence
Renu Gupta
Professor and Head Department of Prosthodontics, H.P.G.D.C. Shimla, Himachal Pradesh, India

Nocturnal bruxism: A review

Renu Gupta, RP Luthra and Akrant Mehra

Abstract
Most individuals, both adults and children, engage in nocturnal bruxist activity at some point in their lives and to varying degrees. The tissues of the masticatory system will generally adapt to this behavior; however, in some individuals the capacity for adaption will be exceeded by the cumulative forces of this mandibular parafunctional behavior, resulting in pain and dysfunction of the masticatory system. Many theories have been proposed regarding the etiological factors, and with time multiple treatment options has been explored for its management. This article gives a brief review regarding the various aspects of nocturnal bruxism.

Keywords: Parafunctional, catecholamine, biofeedback, occlusal splints

1. Introduction
The term “la bruxomanie” was first introduced by Marie Pietkiewicz in 1907. It was later adapted as “bruxism” to describe gnashing and grinding of the teeth occurring without a functional purpose. This tooth movement is produced by rhythmic or sustained–tonic contractions of the masseter and other jaw muscles; it usually occurs without patient awareness [1]. Sleep bruxism (nocturnal bruxism) has been defined by the American Sleep Disorders Association (ASDA) in its International Classification as a "stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep" (Thorpy, 1990) [3]. Various reputed institutions gave their own statements defining bruxism in order to cover all the salient features of it.

Table 1: Definition of both awake and sleep bruxism

Source	Definition
American Academy of Oro facial Pain (2008)	A diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth. In the absence of subjective awareness, past bruxism can be inferred from presence of clear wear facets that are not interpreted to be the result of masticatory function. Contemporary bruxism can be observed through sleep laboratory recordings.
The International Classification of Sleep Disorders (2005)	Sleep-related bruxism is an oral activity characterized by grinding or clenching of the teeth during sleep, usually associated with sleep arousals.
The Academy of Prosthodontics (2005)	1. The parafunctional grinding of teeth. 2. 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 – called also tooth grinding, occlusal neurosis.
American Sleep Disorders Association (ASDA)	"stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep"

This review literature will discuss the various aspects of sleep bruxism in brief.

2. Physiology and pathology of bruxism
Clinical complaints and associated damage or pain may justify the classification of bruxism as a wake-time ‘parafunction’ or sleep-related movement disorder but the mechanisms underlying

the genesis of bruxism remain under investigation because of the lack of a standardized methodology. For the past few decades, the search for the aetiology and physiology of sleep bruxism (SB) has been restricted to mechanical factors (e.g. occlusion) to adoptive or maladaptive behaviour (e.g. stress) and in extreme cases to a medical dysfunction of dopamine (DA). New avenues for investigation have also been emerged.

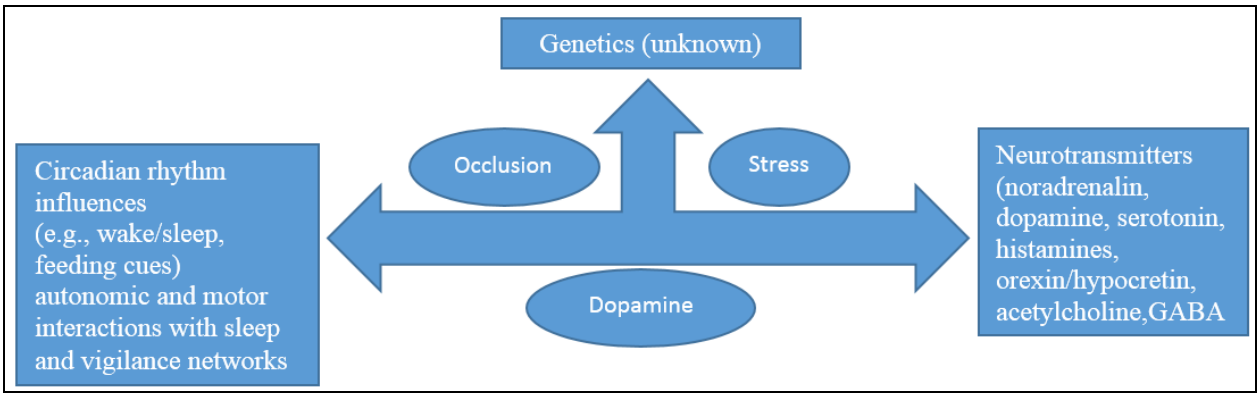

Fig 1: Evolution of the aetiology and pathophysiology of sleep bruxism (circles = older theories; arrows = new avenues). GABA, gamma-aminobutyric acid.

2.1 Sleep bruxism may be linked to multiple genetic factors or to a familial learned behaviour

Initial evidence of a genetic basis for a given motor behaviour or disorder is usually extracted from concordance in a population of twins or dominance in familial distribution. Studies based on questionnaires or tooth wear estimation in monozygotic and dizygotic twins show that there is a high genetic determinant in bruxism. A high concordance rate exists for the pattern of mastication in monozygotic twins (0.97) and dizygotic twins (0.61). On the other hand, a recent twin study did not find any genetic determinant for temporomandibular joint-related signs and symptoms. To initiate a genetic search based on family prevalence, all positive and negative bruxism cases need to be entered into a Mendelian tree. Specific analysis of gene candidates can be made from blood collection, i.e. DNA sampling under blind conditions for linkage analysis. Many limitations reduce the likelihood of finding genes directly associated with bruxism. SB is frequently concomitant with another sleep parasomnia, sleep talking; the shared genetic effect of both conditions is about 30%. This evidence suggests that awake or SB types will probably not be explained by single gene expression; it is more likely that gene heterogeneity drives the apparition of an oral motor behaviour or activity. Moreover, it is difficult to isolate the influence of the stress and anxiety that may cause a sequence of gene and protein activations in relation to observed changes in the autonomic and cerebral arousal systems.

2.2 Occlusal considerations in relation to sleep bruxism

Using tooth interference to explain bruxism became very popular following the publication of a study suggesting that occlusion may influence muscle activity, but it is important to note that this suggestion was based on electromyographic (EMG) data collected during the daytime. Recent literature contains little evidence to support the role of occlusion in the genesis of bruxism. The argument that selective occlusal adjustments in the dental curriculum and in therapy is based on existing knowledge and practice that operates irrespective of the concepts and methods of evidence based dentistry. A recent experimental study showed that occlusal interference was not associated with temporomandibular disorder or orofacial pain and did not significantly raise the frequency of EMG masseter muscle activity in young healthy female subjects. More importantly, clinicians tend to forget that tooth contact is not a dominant activity over a 24-hour cycle. Tooth contact has been suggested to occur for approximately 17.5 min over a 24-hour period. Its been estimated that SB related muscle activity lasted approximately 8 min over a complete sleep period that usually lasted between 7 and 9 h. The fact that some patients do report relief from so-called 'dental discomfort', pain or headache after an oral rehabilitation or an orthodontic treatment is not a sufficient proof to justify extensive treatment.

2.3 Stress and anxiety contributing factor to sleep bruxism

Many dentists share the opinion that bruxism, either clenching while awake or grinding during sleep, is associated with stress and anxiety. Two studies showed that patients with bruxism had elevated levels of catecholamines in their urine in comparison to nonbruxism subjects; such findings support a link between emotional stress and bruxism. Most bruxism patients report that they clench their teeth in periods of intense or frequent familial duties or increased work load. The literature demonstrates that self-report and clinical observation of tooth wear are one means of assessing bruxism in relation to the role of anxiety and stress. Tooth wear has been described as a weak indicator of current bruxism and does not discriminate clenching from grinding bruxism.

Tooth wear magnitude may be influenced by enamel density or by saliva quality and lubricating efficacy. In general practice, dentists need to recognizing psychological or psychiatric disorders, such as severe or pathological anxiety, mood and personality disorders. A survey reveal that subjects with an anxiety disorder (compatible to DSM-IV criteria) are slightly more at risk of reporting tooth grinding (TG). Furthermore, it is very difficult to isolate the role of stress and anxiety from concomitant changes in autonomic and motor excitability and a state of altered physiological vigilance.

2.4 Sleep bruxism is associated with a high level of oromotor activity in jaw muscles

Most current evidence supports the hypothesis that bruxism is centrally mediated under autonomic and brain arousal or vigilance influences. Among the various hypotheses proposed to explain SB, the most recent ones support the role of the

central nervous and autonomic nervous systems in the genesis of oromandibular activity during sleep. Some of the findings related to the neurochemistry and sleep related mechanisms that may contribute to the increased motor activity underlying the genesis of SB.

- **Catecholamine and neurochemistry**

On the basis of a case report in which one Parkinsonian patient was treated with L-dopa suggested the association of TG with dopamine (DA). L-dopa is a precursor of catecholamine such as DA and noradrenaline (NA). The putative role of DA in oromandibular movement disorder is indirectly supported by the presence of chewing-like and TG activity in schizophrenic patients treated with neuroleptics that act mainly on DA receptors. A study was performed in young and otherwise healthy SB subjects, using L-dopa, a DA receptor agonist bromocriptine and two adrenergic medications, a peripheral beta receptor blocker, propranolol and an alpha 2 agonist, clonidine. With L-dopa a significant reduction in SB and rhythmic masticatory muscle activity (RMMA) frequency in comparison to placebo was reported. Bromocriptine administration failed to show either a reduction in SB motor episodes or a change in DA striatal binding. No significant reduction in SB-RMMA with propranolol has been observed. However, the use of clonidine significantly reduced the SB-RMMA index by 60% in comparison to placebo. SSRI antidepressant medications are reported to trigger clenching in some susceptible individuals. A few reports suggest that gamma aminobutyric acid (GABA) may also have a role in SB. Substances with an affinity or structural analogy to GABA, such as clonazepam (also a muscle relaxant and anxiolitic), tiagabine and gabapentin have been reported to reduce SB-TG. The role of the cholinergic system (e.g. acetylcholinerelated medication) on the genesis of SB is unknown. The observation that TG is exacerbated by smoking provides an indirect clue to the potential interaction of nicotinic receptors with mechanisms responsible for SB. However, it remains to be discriminated if smoking increases the risk of bruxism as an oral habit or if it is an effect of nicotine on the cholinergic system, which is heavily involved in vigilance and related brain-arousal networks.

2.5 Sleep bruxism-related motor activation: role of arousal

The ascending and descending arousal systems play an important role in vigilance and sleep; neurochemicals (e.g. acetylcholine, monoamine, NA, serotonin, DA, orexin / hypocretin, histamine, etc.) in networks located in the lower brain up to the hypothalamus are secreted differently for either wake or sleep states. Awake bruxism is associated with stress and increased vigilance (a condition known to increase autonomic cardiac activity). Most SB episodes are under the transient influence of cardiac sympathetic activity (as a promoter of arousal).

2.6 Sleep structure and recurrent-cyclic arousals in relation to SB

A sleep cycle is composed of non-REM and REM periods of 90–110 min of sleep. A night of sleep is composed of 3 to 5 cycles. Non-REM sleep is further divided into light sleep with sleep stages 1 and 2, and deep sleep with sleep stages 3 and 4. Most SB episodes are observed in light non-REM sleep, whereas about 10% occur in REM sleep in association with sleep arousal. Arousals tend to recur 8–15 times an hour of sleep in young healthy subjects. Interestingly, it has been observed that SB tends to occur in relation to recurrent arousal within the so-called cyclic alternating pattern (CAP), which repeats every 20–60 s during non-REM sleep. This finding is further supported by the observation that most SB episodes occur in clusters in relation to CAP. Cyclic alternating pattern-related arousal are described as a natural process that act as a sensor for maintaining body homeostasis and as a protection sentinel during sleep. In brief, the genesis of most SB episodes follows: (i) a rise in sympathetic cardiac activity at -8 to 4 min; (ii) a rise in the frequency of EEG activity at -4 s; (iii) heart rate tachycardia starting at -1 heart beat; (iv) an increase in jaw-opener muscle activity probably responsible for mandible protrusion and airway opening; (v) an associated major increase in the amplitude of the respiratory ventilation; (vi) observable EMG incidents scored as SB-RMMA with or without TG.

3. Signs and symptoms [1, 4]

Table 2

Symptoms Frequent headaches	**Clinical signs** Increase in muscle size of temporalis and masseter
History or presence of fractured teeth or restorations	Temporalis, masseter or external pterygoid muscles tender to palpation
Repeated uncemented restorations	Mandibular deviation while opening
Jaw discomfort upon awakening	Limited occlusal opening
Muscle tenderness	Tooth mobility
Spouse awareness during sleep	Cervical abfraction of teeth
	Fracture of teeth or restorations
	Uncemented crowns or restorations
	Wearing of natural teeth

4. Diagnostic criteria

The International Classification of Sleep Disorders revised edition (ICSD-R) listed diagnostic criteria for sleep bruxism. The minimal criteria include both of the following:

A. symptom of tooth-grinding or tooth-clenching during sleep, and

B. One or more of the following:
1. Abnormal tooth wear
2. Grinding sounds
3. Discomfort of the jaw muscles

With the following criteria supporting the diagnosis:

C. polysomnography shows both:
4. Activity of jaw muscles during sleep
5. No associated epileptic activity

D. No other medical or mental disorders (e.g., sleep-related epilepsy, which may cause abnormal movement during sleep).

E. The presence of other sleep disorders (e.g., obstructive sleep apnea syndrome) [13].

5. Effects of bruxism on prosthetic restorations on natural teeth

Survival rates of conventional FDPs is 94% after 5 years and 89% after 10 years (Systematic reviews). The reason for most common technical failures includes loss of retention and fracture of material. The occurrence of such failures is greatest in patients with bruxing habits.

- With an opposing occlusion of tooth enamel, most clinicians and researchers agree that a metal occlusal surface, and preferably one of high noble content, is preferred in order to minimize wear of the natural dentition. Unpolished ceramics could be especially hazardous to opposing natural teeth.
- All things considered, metal or metal–ceramic restorations seem to be the safest choice in cases of high load conditions, although under extreme conditions, there is no material that will last for too long. Because of the risk of chipping of ceramic veneers in metal–ceramic restorations, many clinicians prefer gold–acrylic FDPs for heavy bruxers.
- Thus, for conventional fixed prosthodontics, single crowns should be constructed whenever possible and FDPs should be of minimal extension.
- An effective way to increase the retention of conventionally retained crowns on short, worn abutments is to include boxes and grooves, or parallel pins in the preparation.

5.1 Effects of bruxism on implant restorations

- Although bruxism was included among risk factors, and was associated with increased mechanical and/or technical complications, it had no impact on implant survival.

However, patients with bruxism have a higher incidence of complications on the superstructures of both of fixed and removable implant-supported restorations

5.2 Effects of bruxism on complete dentures

- Clinical experience indicates that bruxism is a frequent cause of complaint of soreness of the denture-bearing mucosa.
- The relationship between oral parafunctions and residual ridge resorption has not been investigated, but it is tempting, to include parafunctions as a possible factor related to the magnitude of ridge reduction [14].

6. Management

Commonly management can be categorized under four strategies, which are shown here below:

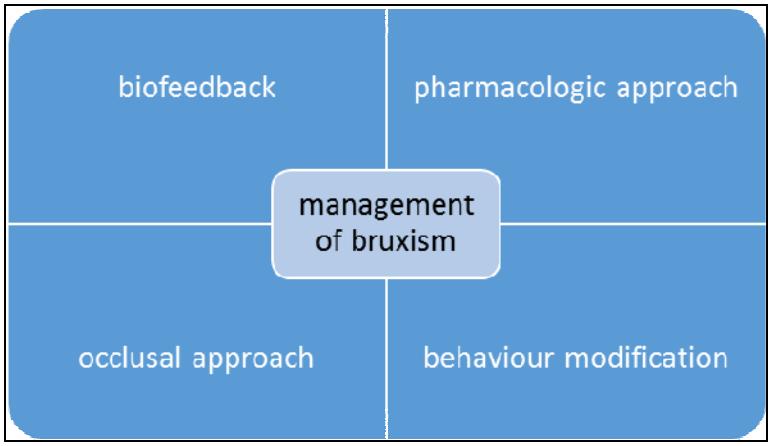

Fig 2: strategies for bruxism management

6.1 Biofeedback

- Biofeedback uses the concept 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 as well as for sleep bruxism.
- For sleep bruxism, auditory, electrical, vibratory and even taste stimuli can be used for feedback.
- In some of the case reports, a sound blast was applied as the aversive stimulus. The sound stimulus is supposed to actually wake up the patient, who is then supposed to switch off the sound and resume his ⁄ her sleep. The awakenings are a major disadvantage of such approaches, because sleep disruption may lead to serious side effects like excessive daytime sleepiness

6.2. Behavior management

a) Behavioral approaches that have been described in the literature for the management of bruxism includes psychoanalysis, autosuggestion, hypnosis, progressive relaxation, meditation, self-monitoring and sleep hygiene. Various techniques followed under this method includes:

a. **Autosuggestion:** This technique helps the bruxer become aware of the habit, even while asleep, by giving him⁄her the autosuggestion 'I'll wake up if I gnash my teeth' before falling asleep.

b. **Hypnotherapy:** Clarke and Reynolds on the basis of a stronger study design (viz. a case–control study) and by using nocturnal EMG recordings concluded that hypnotherapy provided profound relief from problems related to nocturnal bruxism

c) Relaxation: it including meditation, is supposed to produce a sense of self-esteem and control over one's body

d) Sleep hygiene: The objective of measures like 'avoid stimulants (e.g. caffeine, nicotine) for several hours before bedtime' and 'maintain a regular sleep schedule' is to promote better sleep. As bruxism mainly occurs in the lighter sleep stages and in relation to arousals, bruxism will probably

decrease.

6.3. Pharmacological approach

- Short-term administration of the muscle relaxant methocarbamol yielded 'good control and improvement of the bruxism habit. Another drug that affects muscle function, by exerting a paralytic effect through an inhibition of acetylcholine release at the neuromuscular junction, is botulinum toxin (botox). Reports claimed success of botulinum toxin in decreasing (clinically assessed) bruxism activity, especially in severe cases with co-morbidities like coma, brain injury, amphetamine abuse, Huntington's disease and autism. This drug can be administered as a safe and effective treatment for severe bruxers. However, this treatment modality should be confined to patients who are refractory to other (conventional) treatments.
- Catecholamine precursor L-dopa exerted a modest, attenuating effect on sleep bruxism. Likewise, sleep bruxism activity was reduced by the administration of low doses of the dopamine D1 ⁄D2 receptor agonist Pergolide in a severe bruxism case.
- It can be concluded that although some pharmacological approaches for bruxism seem promising, they all need further efficacy and safety assessments before clinical recommendations can be made.

6.4 Occlusal approach

- **A. occlusal interventions:** There is no support in the literature for the use of 'true' occlusal interventions like equilibration, rehabilitation and orthodontic alignment in the management of bruxism. In view of the current insights into the etiology of bruxism that the disorder is mainly regulated centrally and not peripherally future research on this category of management strategies for bruxism seems impractical.

- **B. Occlusal Appliances**
  Most prescriptions describe clinical and technical procedures for the manufacture of various types of splints. These splints have different names [e.g. occlusal bite guard (modified), bruxism appliance, bite plate, night guard (retainer), occlusal device] and slightly different appearances and properties, but in essence most of them are hard acrylic-resin stabilization appliances, mostly worn in the upper jaw.
- Although the concept of soft splints is appealing, 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, and because hard splints are suggested to be more effective in reducing bruxism activity than soft splints.
- The case reports that deal with occlusal splints in the management of bruxism usually describe success in extreme and⁄or special-category patients [6].

7. Conclusion

- Bruxism is a common parafunctional habit which usually has no serious effects, but may, in some patients, have pathological consequences.
- The etiology of bruxism is not well known, but it is agreed to be multifactorial. There is a common consensus that teeth contact is necessary condition for bruxism and bruxing without tooth contact is not bruxism.
- There is no specific treatment available at this time to stop bruxism, so that the focus has been to reduce the adverse effects of the habit. When prosthetic intervention is indicated in a patient with bruxism, efforts should be made to reduce the effects of heavy occlusal loading on all the components that contribute to prosthetic structural integrity [1]

8. References

1. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. Sleep Medicine Reviews. 2000; 4(1):27-43.
2. Holmgren K, Sheikholeslam A, Riise C. Effect of a full-arch maxillary occlusal splint on parafunctional activity during sleep in patients with nocturnal bruxism and signs and symptoms of craniomandibular disorders. THE journal of prosthetic dentistry, 1993, 69(3).
3. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep Bruxism: Validity of Clinical Research Diagnostic Criteria in a Controlled Polysomnographic Study. J Dent Res 1996; 75(1):546-552.
4. Lavigne GJ, khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for Clinicians. Journal of Oral Rehabilitation. 2008; 35:476-494.
5. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. Journal of Oral Rehabilitation. 2001; 28:1085-1091.
6. Lobbezoo F, Zaag JV, Selms MKA, Hamburger HL, Naeije M. Principles for the management of bruxism. Journal of Oral Rehabilitation 2008; 35:509-523.
7. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, *et al.* Bruxism defined and graded: an international consensus. Journal of Oral Rehabilitation. 2013; 40:2-4.
8. Okeson JP. The effects of hard and soft occlusal splints on nocturnal bruxism. JADA, f, 1987, 1(4).
9. Pierce CJ, Gale EN. A Comparison of Different Treatments for Nocturnal Bruxism. J Dent Res, 1988.
10. Ohayon MM, Li KK, Guilleminault G. Risk Factors for Sleep Bruxism in the General Population. CHEST / 119 / 1 / JANUARY, 2001.
11. Sheikholeslam A, Holmgren K, Ruse C. A clinical and electromyographic study of the long-term effects of an occlusal splint on the temporal and masseter muscles in patients with functional disorders and nocturnal bruxism. journal of or at rehabilitation. 1966; 6(13)-137-145.
12. Solberg WK, Clark GT, Rugh JD. Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. Journal of Oral Rehabilitalion. 1975; (2):215-223.
13. Vignatelli L, Bisulli F, Zaniboni A, Naldi I. Interobserver reliability of ICSD-R minimal diagnostic criteria for the parasomnias. J Neurol. 2005; 252:712-717.
14. Johansson A, Omar R, Carlsson GE. Bruxism and prosthetic treatment: A critical review. Journal of Prosthodontic Research. 2011; 55:127-136.