

Demystifying Bruxism: A Review

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Abstract: *Bruxism is a parafunctional habit characterized by teeth grinding and clenching. Parafunctional habits system are any non functional neuro- muscular habits of the stomatognathic system that is caused due to constant repetition of an act that is pleasant for an individual. Bruxers have been majorly divided as: nocturnal and diurnal. The multifactorial etiologic model of bruxism includes genetic, neurophysiologic, psychoemotional and pharmacological factors; as a result definitive understanding of bruxism has always been elusive. Stress is one of the major factors for diurnal bruxism. Owing to its multifactorial nature no single treatment strategy has been effective in controlling the muscle contraction among these patients. The consequences that it has on the natural dentition and dental implants and its relationship to temporomandibular joint disorders have gained interest over the past few years. Bruxism presents a varying trend among children and adults. As a result proper diagnosis and treatment planning holds utmost importance in the rehabilitation of the patients with bruxism.*

Keywords: biofeedback, electromyogram, parafunctional, polysomnography

1. Introduction

Jaw movements involve wide range of orofacial muscle activities which are grouped into function and parafunction. Functional movements involves chewing, swallowing and speech.[1] Whereas parafunctional movements involve those neuromuscular activities of the stomatognathic system resulting from repetition of activities generally pleasant for individuals. Parafunctional habits include biting foreign objects, pressing tongue against teeth, clenching and grinding correlated with muscle and TMJ tenderness.[2]The American Academy Of Orofacial Pain describes bruxism as “diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing and grinding of teeth.”[3]On the other hand The American Sleep Disorder Association defines it as “Tooth grinding or clenching during sleep plus one of the following: tooth wear, sounds, or jaw muscle discomfort in the absence of medical disorder.”[4]

Bruxism is a harmful, involuntary oral motor condition that causes detrimental intra oral effects like tooth wear, damaged restorations, root fractures, tooth hypermobility, implant failures, masticatory muscle and joint pain or discomfort. Besides this, extraoral signs and symptoms manifest as: hypertrophied facial muscles, reduced mouth opening upon awaking and frequent headaches. The etiologic factors are not very clear and efforts are being made to unravel the mechanism behind bruxism.[5]

Bruxers may be majorly divided into two types: nocturnal grinders and diurnal clencher.[6] Nocturnal bruxers are considered to be true bruxists as they grind the teeth. Diurnal bruxers have mainly clenching action. The most putative factor for diurnal bruxism is stress. In 1983,

clenching and grinding were distinguished; clenching as centric bruxism and grinding as eccentric bruxism.[7]

Clenching of the teeth is forceful closure of the opposing dentition in a static relationship of the mandible to the maxilla in either maximum intercuspation or an eccentric position.[8]

Grinding of the dentition is forceful closure of the opposing dentition in a dynamic maxillomandibular relationship as the mandibular arch moves through various excursive positions.[8]

According to various studies it has been found that there exists a significant relationship between diurnal bruxism and myofascial pain.[6] The frequency of diurnal bruxism is more in females compared to males.

Nocturnal bruxism has been studied more extensively and needs proper diagnosis. There are numerous predisposing factors for nocturnal bruxism: occlusal factors, sleep arousals, personality traits, psychological factors etc. Owing to the multifactorial nature of the problem and various ways to diagnose it; it becomes clear that one single treatment modality cannot be used to cure the problem.[9]

Bruxism has also been classified into primary(idiopathic) and secondary(iatrogenic).[10] Primary bruxism includes day time and sleep bruxism in the absence of any medical condition. Secondary bruxism is associated with underlying disorders such as neurologic, psychiatric, sleep disorders, use of medication and/or any possible combination of these.[11–14]

The prevalence of bruxism shows a varying trend and has different distribution among the children and the adults and

shows a decreasing trend with progressing age. The prevalence of sleep bruxism in children ranges from 5.9 to 49.6%, depending on the method of diagnosis used and characteristics of the sample.[15,16] Large scale epidemiological surveys have revealed that self reported teeth grinding has prevalence of about 8% in general adult population with no sex difference.[17–19] The frequency of sleep bruxism among adults is 12.8%±3.8%. [17] Contrary to this little information is available on awake bruxism.[20]

2. Etiology

The etiology of bruxism is complex, controversial and multifactorial. Peripheral, psychological and central or pathophysiological factors have been associated with sleep bruxism.

2.1 Central/ pathophysiological factors:

a) Sleep

The central nervous system controls most of the voluntary and involuntary activities of our body. The involuntary activities are further controlled by the sympathetic and parasympathetic autonomic nervous systems. The parasympathetic responses mainly work during the rest phase and the sympathetic responses during the active phase.[9]

Rhythmic masticatory muscle activities are frequently observed during sleep in 60% of individuals; however when it occurs 3 times more frequently with an intensity approximately 30% more than normal, it is supposed to be associated with sleep bruxism.[21]

Since bruxism is associated with sleep, the sleep patterns have been studied extensively to find out the stages associated with it. Sleep is divided into two phases: rapid eye movement (REM) and the non rapid eye movement (NREM) phases. [22] Bruxism occurs in the second stage of NREM sleep which is associated with sleep arousals. This phase is also associated with increased body movements, increased heart rates and respiratory changes and increase in muscle activity. According to studies 86% of bruxism is associated with the arousal phase of sleep and in 80% of times in bruxism episode leg movements are seen. [23]

b) Psychological factors

Psychological stress has been contributory to sleep bruxism; anxiousness, aggressiveness and hyperactivity are more frequently seen in individuals with sleep bruxism. A few studies showed that when sleep bruxism patients experienced emotional or physical stress there was an increase in masseter EMG activity. [24] A study by Bayar et al., showed significant difference in depression, anxiety, hostility, phobic anxiety and paranoid ideation between non bruxers and sleep awake bruxers. [25]

It has been observed that there is possible presence of TMD in bruxers. There are associations between psychopathological symptoms and TMD and bruxism is one of the major risk factor for TMD.[26]

On studying the behavioral pattern, bruxists are thought to be perfectionists with increased tendency for aggression and anxiety.[25] A study by Katayoun E. revealed that adolescents with bruxism were 4 to 68 times more prone to psychological disorders.[27]

Maria Giraku et al., used Bruxcore- Bruxism- Monitoring Device to assess the role of stress as a factor for sleep bruxism. A significant correlation was found between 'daily problems', 'trouble at work', 'fatigue', 'physical problem' and 'coping strategy escape'. [5]

c) Peripheral factors

The influence of occlusal interferences playing a role in bruxism has been found to be controversial. Ramfjord was the first to study the clinical phenomenon of bruxism with electromyographic (EMG) technique. The study held occlusal characteristics responsible for the initiation of the disorder especially the discrepancies between retruded contact position and intercuspal position and the presence of balancing contacts as major etiologic factor for bruxism. But Ramfjord EMG study lacked controls and used indirect measures for assessing bruxism, hence, the upcoming of better controlled studies have put aside the role of occlusal factor as an etiology of bruxism.[23,28] A study was conducted by Sujimoto where the influence of occlusal factors on the magnitude of sleep bruxism was studied. The subjects were divided into three groups- high sleep bruxers, moderate sleep bruxers and low sleep bruxers. Bruxism was also divided into three types: grinding, clenching and tapping. It was found that while grinding and clenching were predominant, tapping was not prevalent during sleep. In high bruxers grinding was the predominant activity. They also found that molar tooth contacts caused high sleep bruxism activity.[29]

3. Diagnosis

3.1 Clinical manifestation and symptoms:

The diagnosis of bruxism involves a thorough clinical judgement and knowledge of recent diagnostic modalities in the field.

The criteria for screening patients with moderate to severe sleep bruxism are[30]-

- 1) Tooth grinding sounds- 3 to 5 nights per week for over 6 months.
- 2) Tooth wear- on incisal edges on anterior teeth and occlusal surfaces of posterior teeth
- 3) Masticatory muscle pain or fatigue in the morning
- 4) Masseter muscle hypertrophy

According to Lavinge And Manzini the criteria for sleep bruxism are:[31]

- 1) History of tooth grinding noise
- 2) Presence of wear facets on the teeth
- 3) Presence of headache in the temporal region
- 4) Fatigue of mandibular musculature during night time or day
- 5) Lockjaw or difficulty in opening the jaw
- 6) Presence of dental hypersensitivity
- 7) Hypertrophy of the masseter muscle

3.2 Tooth wear

Tooth wear has been thought to one of the major signs of bruxism. However not all studies have been found to support this view. Evidence of shiny spots on the enamel or dentin exposure may be there in bruxers.[32] However, tooth wear can be found in 40% of healthy, non tooth grinding population. [33]

Pullinger et al designed a four point scale to assess the level of attrition. 0-no wear; 1- minimal wear on tip of cusp or occluding planes or on incisal edges, 2-flattening of cusps or grooves; and 3- total loss of contour or dentin exposure.[34]

Pergamalian et al studied the association between wear facets, bruxism and the severity of facial pain in patients with TMJ disorder and found the following results:1) positive and significant correlation exists between tooth wear and age of the patient 2) the amount of tooth wear is not different among various bruxers, and 3) the number of painful joints were different in different bruxers but painful muscle sites were not significantly different in various bruxers.[35]

Proper diagnosis and examination of tooth wear caused due to bruxism is of high clinical importance because if unattended it causes various problems like sensitivity due to dentinal tubule exposure, pulp exposure and tendertooth. The contact points open up due to continued tooth wear leading to loss of arch integrity and pathological tooth movements. This over a long period of time leads to a collapsed vertical dimension of the patient with impaired mastication, hence portraying an appearance of an older individual.[5]

3.3 Polysomnography

Polysomnography is used for the diagnosis of study of various sleep disorders. It measures the physiological movement through the entire course of night using electrodes and sensors in the laboratory. It involves the use of electroencephalography (EEG), electrooculography (EOG) and electromyography (EMG) (submental, suprahyoid, tibialis anterior, mentalis, masseter and temporal muscles). [36]

Diagnosis of sleep bruxism involves various investigations like total sleep time, sleep onset latency, distribution of sleep stages, periodic limb movement, obstructive sleep apnoea and hypoapnoea and frequency of arousals and microarousals. According to Roehr et al arousal is defined as unconscious transition from 3-15 seconds of cerebral impulse of EEG activity either done or accompanied by tachycardia and increase in EMG. [37]

Five tasks are done for signal recognition and calibration of EMG amplification: 1) voluntary clenching (maximal intercuspal occlusion) 2) jaw movement (lateral and protrusive) 3) rhythmic contractions 4) swallowing and 5) coughing. The data analysis for sleep bruxism were done on the following basis:

- a) Sleep: The following sleep parameters were assessed. Total sleep time, sleep efficiency (percentage of of

actual time while asleep), sleep latency (time before sleep stage 1), first REM sleep stage latency and waking time after sleep onset plus the number of awakenings, movement arousals, sleep stage changes and percentage of time spent in each sleep stage per 20 second epoch.

- b) Leg Muscle activity: periodic leg movement activity (PLM) during sleep is recorded. EMG potentials of the anterior tibialis muscles were analyzed and it was considered positive if the score was more than 20 movements per hour. [38]
- c) Jaw muscle activity: EMG events can be defined and scored in different types of episodes: a) phasic (rhythmic) b) tonic (sustained) c) mixed (both phasic and tonic).[24]
- d) Phasic episode consisted of 2 EMG outbursts of 0.25 to 2 seconds duration separated by 2 interburst intervals. Tonic episodes consisted of bursts lasting for more than 2 seconds.[39]

A polysomnographic study by Rossetti et al. showed that there is a significant association between rhythmic masticatory muscle activity during sleep bruxism and myofascial pain. It was also concluded that daytime clenching constitute a stronger risk factor for myofascial pain than rhythmic masticatory muscle pain during sleep bruxism.[40]

3.4 Bruxcore plates

Use of Bruxcore plates is an objective way of measuring bruxism. It is worn by patients in a similar way to occlusal splints and they adapt to it easily. These are standardized polyvinyl plates, with four coloured layers laminated with total thickness of 0.51mm, have microdots of 0.14mm diameter printed on one surface with edible ink. There are 47.2 dots per centimeter, or 2228 dots per square centimeter. [25] A study conducted by C.J. Pierce and E.N. Gale suggested that bruxcore measured different bruxing pattern compared to that of an EMG. It is likely that bruxcore plates register grinding not clenching, while EMG measures muscle activity above 20µV threshold (i.e. both clenching and grinding).[41–43]

3.5 Detection of bite forces

Andrez D.Lantada et al. developed a prototype that uses magnetic field communication scheme similar to low frequency radiofrequency identification (RFID) technology. The reader generates a low frequency magnetic field that is used as an information carrier and powers the sensor. The system used intramouth passive sensor and an external interrogator which remotely records and processes information regarding the patient's dental activities. It permits the quantitative assessment of bite force, without requiring intra-mouth batteries and can provide supplementary information to polysomnographic readings, providing adequate early diagnostic methods and initiating corrective actions before irreversible dental wear appears. [44]

3.6 Electromyogram (EMG)

With the help of electromyogram the number, duration and magnitude of bruxism events can be calculated. However it

is less reliable than polysomnography used in sleep laboratory because of presence of confounding oro-facial activities such as sight, coughing and talking which cannot be differentiated from sleep bruxism.[45] A study by T. Castroflorio aimed at comparing the “probable” sleep bruxism to diagnosis based on clinical assessment with “instrumental” sleep bruxism. Bruxoff software was used to score sleep bruxism episode based on the following feature: mean masseter EMG amplitude at least 10% of maximum voluntary clenching activity preceded by approximately 20% increase in heart rate beginning 1second before rhythmic masticatory muscle activity onset. He used portable device with EMG and ECG which showed excellent diagnostic accuracy.[46]A study by T.Kato evaluated the masseter EMG activity during sleep and sleep bruxism. The study showed that masseter burst frequency tends to be higher in sleep bruxism patients. In non bruxers it was 0.7 hertz while in bruxers it was 0.9 hertz. [30]Lavinge et al. reported that mean activity of masseter EMG burst in RMMA is 30-40% higher in bruxers than non-bruxers. [36]

3.7 Treatment of Bruxism

Treatment of bruxism is challenging and involves various approaches: occlusal, psychological and pharmacological.

Occlusal therapy-

- a) Occlusal appliances
- b) Selective Occlusal grinding/ Occlusal intervention

Biofeedback mechanism for

- a) Diurnal bruxism
 - b) Sleep bruxism
- Pharmacological approach
Non-Pharmacological approach

3.7.1 Occlusal appliances:

Use of occlusal appliances has been considered as first line of strategy in relieving the symptoms of bruxism. Various appliances used are occlusal bite guard, bruxism appliance, bite plane, night guard and occlusal devices. ‘Bruxism S splint’ has been used along with orthodontic treatment to treat bruxism.[45] There has been use soft as well as hard occlusal devices that have been used as splints; however the reliability of soft splints has been less. Hard occlusal maxillary splint have shown to reduce masseter activity. [32] “Michigan type” occlusal splint have been used for 4 weeks in patients with bruxism. It provides greater stability of joint components, establishes favourable occlusal status, reorganization of neuromuscular activity, decreases hyperactivity of muscles and reestablishment of balanced muscle function.[47–49] In a study by Gomes et al., it was observed that a combination of massage therapy and occlusal splint therapy led to greatest improvement in the quality of life related to both physical (physical function, general health, vitality) and emotional (role emotion and mental health) and maximum reduction in pain in patients with bruxism.[50]

3.7.2 Occlusal intervention

There are various school of thoughts that having controvertial opinions on the role of occlusal interferences in

bruxism. According to Greene et al., occlusal rehabilitation mutilates the dentition much beyond what bruxism has created. [51] Karadachi et al. suggested that elimination of interferences in occlusion has no influence on bruxism activities. [52] Greene and Marbach, Bader and Lavinge and Lavinge and Manzini suggested that not every bruxer has such interferences and not every person with such interference is a bruxer.[53–55] A study by Safri A. was done on 59 subjects in which 28 were bruxers and 31 non bruxers. According to this study there was statistically significant difference between bruxing and protrusive interferences. However, no significant relationship in centric and other eccentric movements were found. According to Dawson, presence of occlusal interferences can cause parafunctional movements which was not there before the interferences and disappeared completely with the elimination of all the interferences. [56] According to Ramjford discrepancies between retruded contact position and intercuspal position and also the presence of mediotrusive (balancing side) contacts during articulation were thought to be etiology of bruxism. [28]Ramjford conducted a 45-60 minutes EMG recordings to prove that removal of occlusal interference reduces bruxism.[28] However, his critics stated that such short examination period did not hold significant value.[23]

Treatment considerations: The rehabilitation of patients having bruxism using fixed dental prosthesis should preferably involve usage of materials less prone to fracture such as metal occlusal surface instead of conventional porcelain, with uniform force distribution. In patients requiring extensive restorations protective occlusal splint or nightguard should be advised to wear at night.

4. Biofeedback

Biofeedback mechanism is primarily based on unlearning the habit by making the patient aware of it. It follows the concept of ‘aversive conditioning’. Biofeedback mechanisms are different during day and night time. Biofeedback modalities could be of various types- CES(contingent electrical stimulation), audiofeedback and visual feedback. Stimulations like vibration and gestation are also available.[57]

Daytime bruxer biofeedback mechanism:

Mittleman used EMG to provide auditory biofeedback.[58] Flat occlusal splints can also be used for providing biofeedback mechanism.

Night time bruxer biofeedback mechanism:

Nissani used taste stimulated mechanism to stop bruxism. Rupture of capsule containing aversive substances were used to make the patient aware.[59] Sound blasts were also used to awake the patient from sleep. However the major drawback of this had been that arousal from sleep frequently may lead to other problems and might cause sleepiness during day time.

Contingent electrical stimulation(CES) involves applying low level electrical stimulation to the muscles when they become active, i.e. during the bruxism episode.[60] Contingent electrical stimulation(CES) showed significant

reduction of EMG activity within 6 weeks of the treatment period. [61] However, this result differed in different studies. One of the reasons for this difference could be the environment surrounding the subjects were different during the recording night. Also the interference of sleep related movements account for the inconsistent treatment outcomes.

Pharmacological approach:

Drugs that decrease the release of acetylcholine at neuromuscular junction decrease bruxism. [45] Positive effect on bruxism is seen in patients by drugs stimulating the dopaminergic neurotransmission. [62] Lobbezoo et al., conducted a controlled clinical trial in which levodopa (L-DOPA) resulted in significant decrease in the bruxism episodes per hour of sleep in patients free of psychopathic disorders. [63] DA D1/D2 receptor agonist, Pergolide has been found to reduce polysomnographically confirmed sleep bruxism. [64]

Albin et al., proposed basal ganglia disorder model according to which the major reason for etiopathogenesis of bruxism was the depletion of DA innervations in basal ganglia area of the brain. Hence drug enhancing DA transmission, such as L-DOPA, and other DA D2 receptor agonist eg. Bromocriptine, have positive effect on bruxism. [65]

Laving et al., studied the neurobiological mechanism involved in sleep bruxism and questioned the role of striatal DA in sleep bruxism. [66]

Mascara et al., stated that oral motor control is deputed not only to striatal dopaminergic neuron but also to other brain areas including ventral tegmental areas and brain stem nuclei. [67]

Botulinum toxin

Botulinum toxin type A is effective in controlling the involuntary orofacial movement, secondary bruxism and awake bruxism. [68–72] According to a few studies BoTN-A reduced frequency of jaw motor events and decreased bruxism induced pain. [73,74] In a study by Shim et al., evaluation of BoTN-A injection was done on patients who did not respond to oral splint treatment. It showed reduction in intensity rather than the generation of contraction in jaw closing muscles. [75] Due to paucity of literature further clinical trials are advocated regarding positive effects of botulinum toxin.

Patients with neurological disorder:

Secondary bruxism is one of the common features of patients with neurological disorders. Most common among these are cranial and cervical dystonia which is associated with awake teeth grinding in combination with dystonic/dyskinetic movement, drug resistant temporal lobe epilepsy, seizures which is associated with long duration of teeth grinding and Huntington's disease. [60] These disorders are characterized by acute onset of severe teeth grinding that occurs mainly during wakefulness and that cannot be prevented voluntarily. [76,77] Levodopa and carbidopa have reported marked improvement in 1 week in patients with multiple system atrophy presenting with constant awake teeth bruxism. [78] Galatamine has also had effective results in patients with Alzheimer and associated awake

bruxism. [79] However, there are case reports related to such studies and no long term controlled trials for patients with neurological disorders.

5. Other Non-Pharmacological Approaches

a) Accupuncture

Accupuncture points are certain energy points that are on the channels of energy flow. They have different electrical resistance than the surrounding tissue and when they are stimulated through needles, infrared lasers or electrical currents, they send signals to the CNS, causing it to block the passage of pain, promoting balance between Yin (negative) and Yan (positive). [80] The introduction of needles into specific points on the energy meridians would generate stimulus in the nerve ending of muscles, which is sent to the CNS from where it is recognized and translated at 3 levels- A) hypothalamic levels- its activation leads to the release of endorphins (pain killers), cortisol (anti-inflammatory) and serotonin (antidepressant) in blood stream and CSF. B) level of midbrain- causes the activation of neurons in the grey matter releasing endorphins. C) level of spinal cord- causes activation of interneurons and release of dynorphins. [81,82] Dallanora et al., showed that after the application of acupuncture there is reduction in the activity of masseter and temporalis muscle upto 5 days. [80] Needling of certain points leads to release of endogenous substances such as endorphins that have analgesic, anti-inflammatory and relaxing effects and hence acupuncture is used in the treatment of chronic disorders. [81]

b) Yoga

A study was done to assess the effect of the daily practice of yoga therapies among school employees. It included asana, pranayam, dharana and dhyana. Results showed increase in calmness, comfort, cheerfulness and significant decrease in cognitive mind and body stress.

6. Conclusion

Bruxism is a harmful and involuntary oro-motor condition that involves clenching, bracing, gnashing and grinding of teeth. The etiology of bruxism is complex, controversial and multi-factorial. Peripheral, psychological and central or pathophysiologic factors have been associated with sleep bruxism. Its management involves a holistic approach with high level clinical expertise and thorough understanding of the factors governing the situation. Occlusal appliances are indispensable to treat bruxism and prevent its consequences. With the advent of better technology and understanding high quality randomized control clinical trials have become the need of the hour with more focus on non-pharmacological and psychological approaches being warranted.

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