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Bruxism – Occlusal Parafunction and Its Clinical Significance

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Abstract

Bruxism is a Para-functional or motor habit with a high prevalence in the general population. Non functional contact of maxillary and mandibular teeth resulting in clenching or grating of teeth is called bruxism. Bruxism is not a specific entity of just one disease. Due to its nonspecific pathology, bruxism may be difficult to diagnose and treat. The signs and symptoms of bruxism are detectable and at present, there is no effective treatment to eliminate bruxism permanently. The present paper reviewed with current concepts on bruxism, etiology, diagnosis and management.

Keyword: Bruxism, Parafunctional

Introduction

Periodontal disease is the result of a complex of local and systemic factors and no single factor can initiate the disease. Eliminating the local factors, helps with considerable success in many cases, in other instances, the disease process persist or reappears , indicating that other etiologic factors are operant. The role of psychosomatic factors in the etiology of periodontal disease has been given increasing attention in the recent years. It is essential to determine whether psychosomatic factors give rise to pathologic processes in the periodontal structure. Psychosomatic is a term which has been defined as relating to or involving the influence of emotional stress or conflict on a somatic area , organ or bodily system. Investigations showed that whenever stress was imposed

on the human body, the first reaction of the body tissue was are ofadaptation and defense. If the stress was not removed , pathologic alteration took place in the tissue. Bruxism has a deleterious effect on the Periodontium. Bruxism and clenching are contributing factors in Chronic Destructive Periodontal Disease. The relationship between occlusal wear of the teeth and Periodontal ligament conditions were discussed which may help the practioners to become awareof the factors and take them into consideration in his/her treatment planning .

Bruxism

The term bruxism refers to a nonfunctional contact between maxillary and mandibular teeth often resulting in clenching and grinding[1]. It is a pathological act of the stomatognathic system that involves tooth grinding and clenching during parafunctional jaw movements. The term bruxism comes from the old greek word brygmos-gnashing of teeth[2]. According to New Gould medical dictionary Bruxomania derived from old greek word brychein – the grinding of teeth. The non functional clenching or grinding of teeth is known as bruxism was originally reported by Marie and Piet Kiewicz in 1907.The American academy of oro-facial pain defined bruxism as “Diurnal or nocturnal activity which includes clenching, gnashing, gritting and grinding of teeth. Patient can be clinically diagnosed based on the presence of excessive tooth wear which could not have been caused by mastication.Types of bruxism includes- Idiopathic and Iatrogenic**.**Idiopathic form includes clenching and grating as well as nocturnal bruxism. It is not linked to neurogenic or psychiatric disorders. The prevalence of bruxism in infancy is 14-20% and in adult population is 8-10%.According to Nadler, everyone will brux at least once in life [3,4,5]

Theories: Two different theories of bruxism:

Theory 1: (peripheral causes)

Local morphological disorders in the periphery such as malocclusion are the cause of clenching and gnashing. This ecological model is based on the theory that malocclusion results in reduced masticatory muscle tone. In the absence of occlusal equilibrium , motor neuron activity of masticatory muscle is triggered by periodontal receptors.This theory was followed and supported by study done by Ramjford who was first to carry out electromyographical investigations in patients with bruxism.[6]Ramjford proposed that bruxism is caused by discrepancies between retruded habitual contact positions as well as by balancing contacts. Occlusal correction always result in the disappearance of bruxism symptoms. Proved by means of EMG records of 45-60 minute duration.Critics- such a short period is insignificant failed to include randomised, blinded, control groups.

Occlusion theory critics:

So far nobody has been able to show how perfect occlusion should be achieved .No controlled clinical study has been able to show that bruxism symptoms can be significantly aborted either by removal of occlusal interferences/ by equilibration methods.

Importance of periodontium supporting occlusal theory:Occlusion determines the localization of biomechanical transmission. The intramuscular functional patterns of masticatory muscles are regulated via the receptors of periodontal apparatus. These functional patterns are modified by different motor tasks as well as by dislocation of the mandible in relation to maxilla. The receptors of PDL apparatus relay information on the location of the mandible in relation to the maxilla in the state of equilibrium.Basalganglia(caudate nucleus, putamen, and globuspallidus) are components of functional loops arranged in parallel that includes the

thalamus and cortex.The information flow in these compartment controls the organization of motor preparation and execution of muscular movements. Specific cortical areas send excitatory projection to the striatum. The striatum represents the input stage of the basal ganglia output nuclei i.e, the internal segment of the globuspallidus , the pars reticularis of substantianigra, and the central part of the palladium, exert GABA – mediated inhibition to the target nuclei in thalamus.

Roccabado pointed out that during involuntary deglutition, mandibular and maxillary teeth briefly make contact so that the receptors of both tooth rows become activated for a short time. If load is evenly distributed on all teeth during the final clamping position, receptors send information that mandible is in the best physiological position for the body and thus also for the sense of equilibrium. During malocclusion, premature and one-sided contact is registered . Receptors may interpret this contact in such a way that the mandible needs to be retracted to the resting position by muscular activity .If assuming a final clamping position is not possible because of malocclusion, movement patterns in the mot or cortex are constantly triggered in attempt to achieve the resting position .

Theory 2 : (central causes)[7]

Central disturbances in the area of the basal ganglia , for example sleep- related dysfuctions, are assumed to cause bruxism. Nocturnalparafuctional activity occurs in different stages of sleep[8]. Bruxism constitutes a sleep-related dysfunction (parasomnia) associated with sleep walking , talking or enuresis [9] .The inhibitory outflow is differentially modulated by two opposing but parallel pathway.

- Arises directly from the inhibitory striatal efferent and tends to disinhibit the thalamus stage of circuit.
- The indirect pathway first leads to the external segment of the globuspallidus, then passes to the subthalamic nucleus and finally to the output via an excitatory projection from subthalamicnucleus

Both pathways may be activated selectively and concurrently in association with cortically initiated movements.Then the inputs from the indirect pathway, which are reinforced by the direct pathway may smooth / break the cortically initiated motor pattern.Thus both pathways contribute to the motor pattern of initiated muscles allowing controlled purposeful movements.

Role of dopamine

Striatal operations are furthermore influenced by neurotransmitters such as dopamine. The overall influence of dopamine on the striatum may reinforce any cortically initiated activation and facilitate conduction via the circuits direct pathway which has excitatory effect on thalamus.

The antagonist to dopamine is acetylcholine eg. Parkinsons disease.

Imbalance in the circuit processing of the basal ganglia is thought to be responsible for muscle hyperactivity during nocturnal dyskinesia such as bruxism

Explanation : neural plasticity is based on the ability of synapses to change the way they work. Activation of neural plasticity can change the relationship between inhibition and excitation. Activation of neural plasticity can also change synaptic efficacy and create or eliminate contacts between nerve fibres and nerve cells and dendrites.

Causes for plasticity

- Faulty expression – cause hyperactive diseases such as central neuropathic pain , tinnitus.
- Lack of expression- development of autism form of diseases.

Classification

Bruxism may be classified according to its occurrence , etiology and by motor activity type.[10]

As per its occurance

- Awake bruxism- this is presented when individual is awake(common in females)
- Sleep bruxism- this is presented when the individual is under sleep (common in both the genders) [11,12]
- Combined bruxism- this is present in both the situations.

By its etiology

- Primary bruxism
- Secondary bruxism- seen secondary to diseases , medical products -eg. Cardio selective and anti - psychotic medications.
- Idiopathic- No apparent cause
- Drugs – amphetamine and cocaine.

By motor activity type

- Tonic – muscular contraction sustained more than 2 seconds
- Phasic- brief, repeated contraction of the masticatory musculature.
- Combined- alternating appearance of tonic and phasic episodes.

Period of occurrence

- Past bruxism
- Present bruxism

By severity

- Mild
- Moderate
- Severe

According to Berlin et al , bruxism is an uncommon habit of grinding of teeth and is not concerned with other activities. Habit of grinding unconsciously in night are called as nocturnal bruxism and those with consciousness are diurnal bruxism. Olkinuora has divided bruxism has strain and non strain bruxism.

Sub classification

- Local
- Systemic
- Psychological
- Occupational

Occupation

Boxers, watchmakers, die makers and diamond cutters require a high level of precision and alertness that results in greater amount of physical and mental stress and strain.

Etiology

The etiology of bruxism is multifactorial in nature.[13,14]

- **Psychosocial factors**: emotional stress is considered to be the main triggering factor.
- **Genetic factors**: people with sleep bruxism have a direct family member who had sleep bruxism during their childhood. Prevalence rate is 21-50% . This suggests that genetic factors are involved.

Medications

- Bruxism initiated by selective serotonin reuptake inhibitors is rare and gets resolved when the dosage is decreased. Cases have been reported only with long term use.
- Drugs : Dopamine antagonists, dopamine agonists, tricyclic antidepressants, selective serotonin reuptake inhibitors, alcohol , cocaine and amphetamine.

Pathophysiology

Bruxism has been linked to sleep disturbance, altered brain chemistry, the use of certain medications, smoking, alcohol conception, trauma and disease. These factors are to be involved in the precipitation of bruxism .In 86% of bruxism cases were associated with an arousal response, which is a sudden change in the depth of sleep, during which the individual either arrives in a lighter sleep stage or actively wakes up. Teeth contact during rhythmic,

masticatory muscle activity during sleep generates a physical strength ranging from 300 to 8000 grams. Certain disturbances in the central neurotransmitter system may be involved in the etiology of bruxism.

In bruxers , the balance between the direct and indirect pathways of the basal ganglia, a group of subcortical nuclei that are involved in the coordination of movements are affected. The direct output pathways goes directly to one of the 5 basal ganglia to the thalamus, from where the afferent signals project to the central . The indirect pathway passes by several other nuclei before the thalamus is being reached. If there is imbalance between both the pathways, movement disorders occurs like in Parkinson's disease. In case of bruxism, there may be imbalance between both output pathways without signs of degeneration of the nigrostriatal feedback loop. Long term use of L- dopa, neuroleptics are known to cause bruxism [15].

SSRI- selective serotonin reuptake inhibitor, acts on the brain to raise the levels of the neurotransmitter serotonin without raising the levels of norepinephrine and these medications thought to be a benefit in treatment of depression, anxiety and panic disorders. These medications have been reported to encourage bruxism.

Psychological factors

There is a common thought that the psychological stress contributes to bruxism. Usually bruxism is emotionally out of balance and they tend to develop more psychosomatic disorders. Anxiety , tension , frustration , negative emotions may increase the activity of hypothalamic pituitary, adrenal axis with an increase in ACTH, cortisol and adrenaline secretions by supradrenal glands [16] , both during sleep and wakefulness. Release of adrenaline by supra adrenal glands would facilitate an increase in neuromuscular tonus and tonic increase basal activity of the SNS with subsequent increase in the frequency of Rhythmic Masticatory Muscle Activity episode and teeth grinding during sleep along with decrease in the salivary secretory rate.

Bruxomania : It is defined as grinding or powdering of the teeth as the manifestation of neurosis usually during sleep. The term was derived from greek word brychein meaning to grind or gnash the teeth. The term was changed to bruxism by Frohman in 1931 [17]. He described bruxism as a dysfunctional jaw movements. Neuralgia traumates and occlusal habit neurosis are different terms used for bruxism. The use of the word parafunctional was suggested by Drum in 1962. Auther has labeled clenching as a definite entity to that of bruxism. Bruxism is a forceful rubbing or grinding in eccentric excursion and grinding in centric as in clenching.

Diagnosis and management

Diagnosis of bruxism is based on patients history and clinical examination.Parafunctional habits are potential source of trauma from occlusion. Bruxism is a pathologic activity of the stomatognathic system involving teeth grinding and clenching. Splint therapy can be used to decrease the trauma from occlusion forces and improve the patients comfort. Bruxism in everyday practice often remains undetected and badly treated.Peripheral factors like occlusal discrepancies and deviation in orofacial anatomy plays a minor role. So the focus is more on central factors psychological and pathophysiological factors are given importance for bruxism.

Clinical findings

Diagnosis of bruxism is usually made clinically. Most of the time it is based on patients history.

Types

- Irreversible – occlusal adjustment, oral rehabilitation
- Reversible – psychic factors, medications.

It is based on the case records and systems. Changes it causes in the masticatory system and its surroundingstructures will help to diagnose . The signs and symptoms of the patientincludes movementsof the mandible. Pain in the TMJ, masticatory and cervical muscle, headache , hypersensitive teeth, excessive tooth mobility, PDL and dental examination.

Observation

Opening and closingof the mandible is analyzed with an average maximum opening of 50mm.

Palpation of muscles of mastication for any elicitation of tenderness or pain.

Evaluation of soft tissue for any discomfort or any radiating pain.Abnormal tooth indentations, gum recession, reduced salivary flow, fracture of filling or teeth, limitation of mouth opening, exostosis formation.

Auscultation : Joint sounds and any changes such as clicks, pops, crepitation are recorded.

Load test- by bimanual palpation. This test includes test positive for the palpation of head of the condyle, surface of glenoid fossa and the tissue interposed in between.

Complete occlusal analysis should be done.

This includes – analysis of type of occlusion ,interocclusal rest space, opening and closing patterns, occlusal curves, centric occlusion, the intercuspal position , protrusive movements, laterotrusive (R&L)movements.

Recognition of bruxism

- Prominent masseter
- Taut and tense craniofacial muscles
- Facets
- Depressed nature of posterior teeth
- Depressed nature of saddle area

Management

After history, inform the patient-

It should be done in 3 different approach

- Behavior approach
- Occlusal approach
- Pharmoceutical approach

Behavior Approach:[18]

Bruxism during wakefulness can be trained to control their jaw muscle activity by auditory or visual feedback. [19] Sleep bruxism – auditory , electrical , vibratory or taste stimuli can be used for feedback.

Psychoanalysis, hypnosis, medications and self-monitoring are other types of behaviour approach.

Occlusal approach:

True occlusal intervention: this includes occlusal equilibration , occlusal rehabilitation and orthodontic movements.

Occlusal appliance: it is the most widely known and used therapeutic method (splint), stabilization appliance [20], anterior bite plane device, soft and resilience appliance are different types of appliances used in the management of parafunctional habit.

Pharmacological aspects:[21]

Drugs that affect the muscle function by exerting paralytic effect through an inhibition of acetylcholine release in nerve muscle junction in botulinum toxin can be admitted as a safe and efficient treatment for severe bruxism [22]. The treatment modality should be continued to patients who are refractory toconventional treatments.

Sequale

Generally bruxism is detected because of the presence of defects on the teeth like attrition and pain rather than the process itself. The component of masticatory system mainly teeth, the periodontium , the articulation of the mandible with the skull(TMJ) are affected due to the heavy forces generated during bruxism.

Bruxism and periodontium

Bruxism definitely has the potential to cause tooth wear, fracture, periodontal and muscle pain and it may be a major cause of tooth mobility. Opinion differs regarding

possible relationship between bruxism and periodontal breakdown. Several investigators stated that bruxism has a deleterious effect on the periodontium. The relationship between occlusal wear of the teeth and PDL condition was studied by Ainamo and Lahdenpua[23]. Patients with severe attrition didn't exhibit gingivitis decreased probing depth or increased loss of CAL than individuals without attrition. No clinical or histological evidence was found to state that bruxism could cause a shift of gingivitis to destructive periodontitis[24].

The absence of acausal link betweenbruxism and periodontal damage has been discussed in systematic literature assessment on bruxism and periodontium .An increased periodontal perception is the only plausible bruxism consequence on the periodontium [25]. It is also possible that teeth with decreased periodontal support and certain degree of mobility may act as a protective mechanism against bruxism via the same pathways that reduce bruxism activities in individuals with high restoration. Bruxism may precipitate condition and jeopardise survival of migrated or periodontally migrated teeth.

Inter-relationship between bruxism and the periodontium are discussed by Lund and Goldberg [26]. Eschler in an electromyographic study of bruxism – noted with inflammation , the relationship between periodontal receptor and masticatory muscle was altered. According to him a lower response threshold in the muscle secondary to the periodontal inflammation is mainly due to an increase in resting potential of the masticatory musculature.

Conclusion

Bruxism is a parafunctionaloromotor habit. Bruxism is listed in the international classification of sleep disorder (ICSD) it is the 3rd most common form of sleep disorder right behind sleep talking and snoring.The signs and symptoms are detectable and at present there is no effective treatment to eliminate bruxism permanently. Because of this the therapeutic approach is steered towards attempting to prevent damage to the teeth and surrounding structures.

References

1. Frohman B.: Dent . Cosmos 73:117,1931.
2. Manfredini D., Ahlberg J., Mura R., Lobbezoo F. Bruxism is unlikely to cause damage to the periodontium: Findings from a systematic literature assessment. J .Periodontol. 2015;86(4):546-555.
3. Hanamura H., Houston F., Rylander H., Carlsson G.E., Haralson T., Nyman S. Periodontal status and bruxism. A comparative study of patients with periodontal disease and occlusal parafunctions. J. Periodontol. 1987;58(3): 173-176.
4. Lobbezoo F, Ahlberg J, Glaros A, Kato T, Koyano K, LavigneGJ, et al. Bruxism Defined and Graded: an International Consesus. J Oral Rehabil 2013;40:2-4.
5. Lobbezoo, F., Naeije, M., 2001. Bruxism is mainly regulated centrally, not peripherally. J. Oral Rehabil. 28, 1085-1091.
6. American Academy of Sleep Medicine. International Classification of Sleep disorders, Revised : Diagnostic and Coding Manual. Chicago, Illinois: American Academy of Sleep Medicine.
7. Macedo CR, Silva AB, Machado MA, Saconato H, Prado GF. Occlusal splints for treating sleep bruxism(toth grinding). Cochrane Database Syst Rev 2007;17:CD005514.
8. Macedo CR, Machado MA, Silva AB, Prado GF. Pharmacotherapy for sleep bruxism. Cochrane Database Syst Rev 2009; 36:86-92. [John Wiley and Sons, Ltd].
9. PovedaRoda R, Bagan JV, Diaz Fernandez JM, Hernandez Bazan S, Jimenez Soriano Y. Review of temperomandibular joint pathology. Part I:

Classification, epidemiology and risk factors. Med Oral Patol Oral Cir Bucal 2007; 12:E292-8.

10. Bell WF. Temporomandibular disorders: Classification, diagnosis, management (Impact Factor: 3.54). Plat ReconstrSurg 1987;81:134.
11. Goldberg, G.: The psychological , physiological and hypnotic approach to bruxism in the treatment of periodontal disease. J Am SocPsychosom Dent Med 20:75, 1973.
12. Ainamo, J.: Relationship between occlusal wear of the teeth and periodontal health. Scand J Dent Res 80:505, 1974.
13. Lavigne GJ, ManziniC, Kato T. Sleep bruxism. In :kryger M, Roth T, Dement WC, ed. Principlesand practice of sleep medicine. 4th edn. Philadelphia, PA: Elsevier Saunders, 2005:946-959.
14. Bader , G. and Lavigne, G.J.2000. Sleep bruxism: an overview of an oromandibular sleep movement disorder. Sleep Medicine Reviews. 4,27.
15. Lobbezoo , F. and Lavigne, G.J. 1997. Do bruxism and temporomandibular disorders have a cause- and effect relationship . Journal of Orofacial Pain, 11,15.
16. Lobbezoo, F.,Lavigne, G.J., Tanguay, R. and Monstplaisir, J.Y.1997. the effect of the catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Movement disorders, 12:73.
17. Kampe T., Edman, G.,Bader, G.,Tagdae, T. and KarlssonS.1997. personality traits in a group of subjects with a longstanding bruxism behavior. J Oral Rehab. 24:581-7.
18. F Lobbezoo, J. Van Der Zaag, M.K.A. Van Selmset al 2008. Principles for the management of bruxism. Journal of oral Rehabilitation. 35;509-523.
19. Okeson; 'Management of Temperomandibulardisordersand occlusion. 5th edn:15,p.509.
20. Tan EK, Jankovie J.2000. Treating severe bruxism with botulinum toxin. J Am Dent Assoc. 131:211-216.