Bruxism: Conceptual discussion and review

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ABSTRACT

Bruxism is commonly considered a detrimental motor activity, potentially causing overload of the stomatognathic structures. The etiology of bruxism is unclear, but the condition has been associated with stress, occlusal disorders, allergies and sleep positioning. Due to its nonspecific pathology, bruxism may be difficult to diagnose. Unfortunately, very little data exists on the subject of a cause-and-effect relationship of bruxism to the point that expert opinions and cautionary approaches are still considered the best available sources for suggesting good practice indicators. The present paper reviewed current concepts on bruxism, etiology, diagnosis and management, underlining its effects on dental structures in an attempt to provide clinically useful suggestions based on scientifically sound data.

KEY WORDS: Occlusion, oral dyskinesis, parasomnia, somatoform disorders, tooth-grinding

Mandibular movement's biomechanics is a function of the neurologic input from cortical and stomatognathic sources acting to initiate or restrict muscular contracture.

There exists fixed relationship between maxillary teeth and cranial base, just as mandibular teeth have a fixed relationship to the condyle, contact of their respective occlusal surfaces may directly influence condylar position or movement.

Dysfunction ideally refers to function that never exceeds the integrity or adaptive limits of the structural elements of the masticatory system. In clinical experience, it was seen that the tolerance of the components of the masticatory system can be exceeded by both acute trauma and chronic trauma.

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Acute trauma

E.g., Accident, blow to the face, long dental appointment.

It is an initiating event leading toward a chronic condition so accurate documentation, and careful monitoring may prove valuable.

Chronic trauma

Repeated experience of any type exceeding the tolerance of the affected masticatory system structure.

Parafunctional occlusal habits and postural stress, with or without occlusal discrepancies may produce musculoskeletal disharmony and orthopedic instability of the temporomandibular joint (TMJ).

Bruxism

Bruxism, derived from the Greek word *brygmós*, meaning "gnashing of teeth."

The American Academy of Orofacial Pain in 2008[1] defined bruxism as a diurnal or nocturnal parafunctional activity that

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includes unconscious clenching, grinding or bracing of the teeth.

"Nonfunctional contact of the mandibular and maxillary teeth resulting in clenching or tooth-grinding due to repetitive and unconscious contraction of the masseter and temporalis muscles."[2]

"Parafunctional grinding of teeth or an oral habit consisting of involuntary rhythmic or spasmodic nonfunctional gnashing, grinding or clenching of teeth other than chewing movements of the mandible, which may lead to occlusal trauma and periodontal break down." [3]

Bruxism is listed in the International Classification of Sleep Disorder (ICSD).

Bruxism is third most common form of sleep disorder right behind sleep, talking and snoring.

Nomenclature

- 1901: Karolyi M – "traumatic neuralgia"
- 1907: Marie Pietkiewicz – "bruxomania"[4]
- 1931: Frohman – "bruxism"[5]
- 1972: Drum –"emotional loaded parafunction"[6]
- 1971: Ramjford Ash – "centric and eccentric bruxism."[7]

Epidemiology

The ICSD-R states that 85–90% of the general population grind their teeth to a degree at some point during their life, although only 5% will develop a clinical condition.[8]

Studies have reported that awake bruxism (AB) affects females more commonly than males,[3] while in sleep bruxism (SB), males are as equally affected as females.[8,9]

A 2013 systematic review of the epidemiologic reports of bruxism concluded a prevalence of about 22.1–31% for AB, 9.7–15.9% for SB, and an overall prevalence of about 8–31.4% of bruxism generally.

The review overall concludes that bruxism affects males and females equally and affects elderly people less commonly.[10]

Classification

Bruxism may be classified according to several criteria[11]

- By when it occurs: [12]
  - Awake bruxism: This is presented when the individual is awake
  - Sleep bruxism: This is presented when the individual is asleep
  - Combined bruxism: This is present in both situations.
- By etiology:[12]
  - Primary, essential or idiopathic bruxism:
    - For which no apparent cause is known.
  - Secondary bruxism:
    - Secondary to diseases (coma, icterus, cerebral palsy)
    - Medicinal products (e.g. antipsychotic medication, cardioactive medication)
    - Drugs (e.g. amphetamines, cocaine, ecstasy).
- By motor activity type:
  - Tonic: Muscular contraction sustained for more than 2 s
  - Phasic: Brief, repeated contractions of the masticatory musculature with three or more consecutive bursts of electromyographic activity that last between 0.25 and 2 s apiece
  - Combined: Alternating appearance of tonic and phasic episodes.

Approximately, 90% of the episodes of SB are phasic or combined, unlike in AB, where episodes are predominantly tonic.[11]

- By period of occurrence:
  - Past bruxism
  - Current or present bruxism.

They are frequently difficult to tell apart.

- By severity[8]
  - Mild as occurring less than nightly, with no damage to teeth or psychosocial impairment
  - Moderate as occurring nightly, with mild impairment of psychosocial functioning; and
  - Severe as occurring nightly, and with damage to the teeth, tempormandibular disorders and other physical injuries, and severe psychosocial impairment.

- While awake
- Worsens throughout the day, may not be present on waking
- Rarely associated
- Usually clenching, occasionally clenching and grinding
- Stronger evidence for a relationship, but not conclusive
- 22.1-31%
- Mostly females
- Unclear.

Etiology

The cause of bruxism is generally accepted to have multiple causes though largely unknown.[3,13,14] Bruxism is debated whether this represents a subconscious habit or is entirely involuntary though it is a parafunctional activity.

Psychosocial factors

Studies have reported significant psychosocial risk factors for bruxism, mainly a stressful lifestyle, and this evidence is increasing, but still not conclusive.[3,15,16] Emotional stress is considered to be the main triggering factor.[17]

Genetic factors

Research suggests that there may be a degree of inherited susceptibility to develop SB.[18] 21–50% of people with SB have

a direct family member who had SB during their childhood, suggests that there are genetic factors involved.[9]

Medications

- Dopamine agonists
- Dopamine antagonists
- Tricyclic antidepressants,
- Selective serotonin reuptake inhibitors
- Alcohol, cocaine, and
- Amphetamines.

In some reported cases, where bruxism is thought to have been initiated by selective serotonin reuptake inhibitors, decreasing the dose resolved the side effect.[19] Other sources state that reports of selective serotonin reuptake inhibitors causing bruxism are rare,[20] and it only happens with long-term use.[15]

Occlusal interferences

An occlusal interference may refer to a problem, which interferes with the normal path of the bite, and is often used to describe as a localized problem with the position or shape of a single tooth or group of teeth.

A premature contact is a term that refers to one part of the bite meeting sooner than other parts, means that the rest of the teeth occlude later or are held open. e.g. a new dental restoration on the tooth (e.g. a crown), which has a slightly different shape or position to the original tooth may contact too soon in the bite.

A deflective interference refers to interference with the bite that changes the normal path of the bite. A common example of a deflective is an over-erupted upper third molar, often because the lower third molar has been removed. When the jaws are brought into occlusion here, the lower posterior teeth contact the prominent third molar before the other teeth, and the lower jaw has to be brought forward to get the rest of the teeth to meet.

The difference between a deflective interference and the premature contact is that the latter implies a dynamic abnormality in the bite.

Other possible associations

- Parkinson's diseases[21]
- Torus mandibularis[22]
- Oromandibular dystonia[2]
- Rett syndrome[10]
- Down syndrome[13]
- Trauma[15]
- Atypical facial pain.[8]

Physiopathology

Sleep bruxism is related with rhythmic masticatory muscle activity,[11,23] a specific type of masticatory muscle activity that is characterized by rhythmic, pseudo-masticatory jaw movements occurring once or twice per hour of sleep, at a frequency of approximately 1 Hz [Figure 1].

Sequelae

Bruxism is usually detected because of the effects of the process (most commonly tooth attrition and pain), rather than the process itself.

The large forces that can be generated during bruxism can have detrimental effects on the components of masticatory system, mainly teeth, the periodontium and the articulation of the mandible with the skull (the TMJ).

The masticatory muscles that act to move the jaw can also be affected.

It has been estimated that during clenching or grinding, the individual might impose a load of over 20 g on a tooth over periods of 2.5 s per clenching. This is far in excess of normal functional stresses and causes.

Flow with in the visco-elastic periodontal ligament and distortion of alveolar bone, from which the tissue which delay recovering.

- Affect the proprioceptive nerve endings → affect the protective reflex mechanism leading to abnormal muscle activity → habit is perpetuated
- In the absence of gingival inflammation or periodontal destruction, the supporting tissues may adopt to the load of primary occlusal trauma where as
- In early to moderate periodontitis → adaptive response is same
- In severe periodontitis → rate of disease progress is accelerated.

Utilized, over and above the normal function.[18]

Clinical findings

Although there is no connection between bruxism and gingival inflammation or periodontitis, bruxism definitely has the potential to cause tooth wear, fracture and periodontal and muscle pain and it is a major cause of tooth mobility.

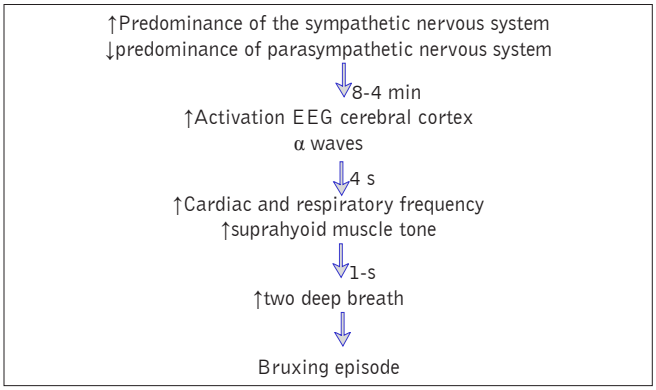

Figure 1: Time sequence of physiological phenomena of micro-arousal with rhythmic masticatory muscle activity preceding an episode of bruxism

Diagnosis

Early diagnosis of bruxism is advantageous, because of possible damage that may be incurred and the detrimental effect on quality of life.[3] A diagnosis of bruxism is usually made clinically,[24] and is mainly based on the person's history (e.g. reports of grinding noises) and the presence of typical signs and symptoms.

Symptoms

- Grinding of teeth, accompanied by a characteristic sound that may even awaken the Bruxer's bed partner
- Pain in the TMJ
- Pain in the masticatory and cervical muscles
- Headache (especially in the temporal zone when the patient wakes up in the morning)
- Hypersensitive teeth
- Excessive tooth mobility
- Poor sleep quality: Tiredness.

Signs

- Abnormal tooth wear
- Tongue indentations
- Linea alba along the biting plane
- Gum recession
- Presence of torus maxillaris and/or mandibularis
- Increase in muscle activity (this is recorded by the polysomnograph)
- Hypertrophy of masseter muscles
- Reduction of salivary flow
- Breakage of fillings and/or teeth
- Limitation of mouth-opening ability.

Bruxism – diagnostic features comprise the following
- Occlusal sounds during sleep
- Functional tooth wear (i.e.,) attrition facets
- Periodontal changes
- Tooth mobility pattern
- Widened Periodontal ligament (PdL) space
- Trabeculation of the alveolar bone
- Exostosis formation
- Masticatory muscle tiredness
- Recurrent migraine
- Fractured filling (or) split teeth
- Soreness of the oral mucosa beneath the denture
- Tender on percussion
- Mucosal ridging of tongue and cheeks
- Increase in size of temporal and masseter, lt. pterygoid
- Deviation of the lower jaw on opening, limited opening
- Increased mobility of teeth.

International classification of sleep disorder-R diagnostic criteria

The ICSD-R listed diagnostic criteria for SB.[8] The minimal criteria include both of the following:

- Symptom of tooth-grinding or tooth-clenching during sleep, and
- One or more of the following:
  - Abnormal tooth wear
  - Grinding sounds
  - Discomfort of the jaw muscles.

With the following criteria supporting the diagnosis:
- Polysomnography shows both
  - Activity of jaw muscles during sleep
  - No associated epileptic activity
- No other medical or mental disorders (e.g. sleep-related epilepsy, which may cause abnormal movement during sleep).
- The presence of other sleep disorders (e.g. obstructive sleep apnoea syndrome).

Treatment

- First Step: Eliminate the causative factors such as
  - Quitting smoking
  - Quitting alcohol
  - Quitting Coffee uptake
- Second Step: Discussion with patients
- Third step: If occlusal disharmony positive-coronoplasty.

According to Welden E. Bell.[25]

Daytime Bruxism, can be controlled by habit training to voluntarily leave the teeth separated or ajar.

The use of remainders such as a small piece of chewing gum patted along the occlusal surface of the molar teeth may be helpful.

Nocturnal Bruxism may be reduced by sleeping flat on the back without a pillow, by autosuggestion that "I will not clench my teeth while asleep" or by positive posthypnotic suggestion.

He also formulated tension control program that include:
- Medicinal therapy in the form of tranquilizers (i.e. a dose of 25 mg hydroxyzine 1 h before bedtime)
- Psychological care → counseling, autosuggestion, hypnotherapy (or) psychotherapy
  - Relaxation techniques, stress management, behavioral modification, habit reversal and hypnosis (self-hypnosis or with a hypnotherapist[13]
  - Cognitive behavioral therapy has been recommended by some for treatment of bruxism[26]
- Bio feedback technique
- Genuine understanding and empathy.

The treatment of Bruxism is also done by classifying the patients into reversible and irreversible.

Irreversible

- Occlusal adjustment

Only at initial stages (through selective grinding, occlusal rehabilitation, orthodontics)

- Oral rehabilitation.

Reversible

- Contributing control factors – control of psychic factors:
  - Psychotherapy
  - Relaxation
  - Yoga.
- Medicines:[12]

Benzodiazepines, anticonvulsants, beta blockers, dopamine agents, antidepressants, muscle relaxants.

Clonazepam,[27] levodopa,[27] amitriptyline,[27] bromocriptine,[27] pergolide, clonidine, propranolol, and l-tryptophan.

Botulinum toxin

Botulinum toxin (Botox) has been suggested as a treatment for bruxism,[20] however there is only one randomized control trial, which has reported that Botox reduces the myofascial pain symptoms.[2] This scientific study was based on thirty people with bruxism who received Botox injections into the muscles of mastication and a control group of people with bruxism who received placebo injections.[2]

In 2013, a further randomized control trial investigating Botox in bruxism started.[2] There is also a little information available about the safety and long-term follow-up of this treatment for bruxism.[12]

Mechanism of action

Botulinum toxin causes muscle paralysis by inhibition of acetylcholine release at neuromuscular junctions.[19] Botox injections are used in bruxism on the theory that a dilute solution of the toxin will partially paralyze the muscles and lessen their ability to forcefully clench and while aiming to retain enough muscular function to enable normal activities such as talking and eating, grind the jaw.

Bite guards

Occlusal shield

Covers the occlusal surface, incisal edges and facial and lingual tooth convexities. Splints may also reduce muscle strain by allowing the upper and lower jaw to move easily with respect to each other.

Treatment goals include: Constraining the bruxing pattern to avoid damage to the TMJs; stabilizing the occlusion by minimizing gradual changes to the positions of the teeth, avoiding tooth damage and revealing the extent and patterns of bruxism through examination of the markings on the splint's surface.

A dental guard is typically worn during every night's sleep on a long-term basis. However, a meta-analysis of occlusal splints (dental guards) used for this purpose concluded that "there is not enough evidence to state that the occlusal splint is effective for treating SB.[28]

Anterior bite plane

When attrition is severe (or) where there has been a collapse of the posterior segment.

A repositioning splint is designed to change the occlusion or bite of the patient. It is debated relating the efficacy of such devices. Some writers suggested that irreversible complications can result from the long-term use of mouthguards and repositioning splints. Random controlled trials with such devices generally show no benefit over other therapies.[29-31]

Another partial splint is the "nociceptive trigeminal inhibitor tension suppression system" dental guard. Only the upper front teeth are snapped by this splint. Preventing the tissue damages primarily by reducing the bite force from attempts to close the jaw normally into a forward twisting of the lower front teeth has been theorized. The intent is for the brain to interpret the nerve sensations as undesirable, subconsciously and automatically reducing clenching force.

Conclusion

Bruxism is a parafunctional oromotor habit with a high prevalence in the general population. The signs and symptoms of bruxism are detectable, but unfortunately, the hypothesized etiologies and mechanisms of their actions have not been substantiated satisfactorily. At present, there is no effective treatment to eliminate bruxism permanently. Therefore, the therapeutic approach is steered towards attempting to prevent damage and to treat the pathological effects of bruxism on the structures of the masticatory system.

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