

SLEEP BRUXISM NEUROBIOLOGY

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ABSTRACT

Primary SB pathophysiology has been associated with mild dysfunction of the basal ganglia, psychological social factors, tooth interference, genetic influences and a combination of these factors that lead to increased motoneural excitability during sleep. In primary SB, arousals trigger autonomic nervous system and chewing muscle activity, which lead to teeth grinding. The main signs and symptoms of SB include grinding noises, dental wear, masseter and temporal muscle hypertrophy, local pain, temporomandibular joint disorders, and unrefreshing nocturnal sleep. Primary SB manifestations are highly variable over time. A primary SB clinical diagnosis is made by evaluation of a patient's history and a dental examination. Sleep studies are indicated to rule out an associated sleep pathology, such as sleep-disordered breathing, and to evaluate sleep-state teeth grinding and arousals. Therapeutic approaches to secondary SB consist of managing the underlying medical, psychiatric or substance-related conditions. Primary SB therapeutic treatment includes several approaches which are customized to the subject's clinical characteristics. Dental treatment with mouth guards primarily aims to prevent orofacial damage and pain. Pharmacologic therapy with muscle relaxers, benzodiazepines, antidepressants other than Selective Serotonin Reuptake Inhibitor (SSRIs), or anti-epileptic drugs can be employed. Botulin toxin type A IM applications into masticatory muscles might be considered as an alternative for unresponsive cases of primary and secondary SB.

Keywords: teeth grinding, sleep bruxism, physiopathology, treatment.

INTRODUCTION

Bruxism is described as a parafunctional motor activity, no matter whether it occurs during sleep or wakefulness (1). Bruxism occurring during wakefulness, or daytime bruxism (DB), and primary sleep bruxism (SB) are different clinical entities as they have different causes, occur during distinct states of consciousness (sleep and wakefulness) and have different etiologies, which call for different treatment strategies (2).

DEFINITION OF SLEEP BRUXISM

Sleep bruxism is conceptually defined as a movement disorder, as it is an involuntary and undesirable activity that occurs during sleep. In operational terms, sleep bruxism is characterized by activity of the masticatory muscles during sleep, resulting in dental contact, local secondary symptoms and systemic symptoms (2). According to the International Classification of Sleep Disorders (ICSD - 2005), SB is a movement disorder related to the sleep state, and is defined as stereotyped and periodic movements characterized by teeth grinding or jaw clenching caused by rhythmic contraction of masticatory muscles during sleep, thus causing both local and systemic symptoms (3). Sleep bruxism is classi-

fied into two types: primary or idiopathic, and secondary, which is associated with other medical diseases, drugs, mental disorders and sleep disorders (1,2).

EPIDEMIOLOGY

SB onset occurs soon after tooth eruption, and its prevalence throughout the life cycle is close to 100%. Nevertheless, SB punctual prevalence ranges from 14 to 20% in children up to eleven years of age; among these, 85% suffer from SB in adulthood (4,5).

The most comprehensive study on SB prevalence was performed by Ohayon et al. in 2001 (6). The authors conducted 1,057 phone interviews with people living in Germany, the United Kingdom and Italy, and found, in the people interviewed, the prevalence of SB episodes occurring at least twice a week was between 8 and 20%; the highest rates were registered among subjects aged 19-44 years, but no significant differences between genders were found. Two Brazilian studies reported by Braz and Palma et al. in 1987 and 1995 evaluated the parasomnia prevalence among 1000 people living in the city of Sao Paulo, Brazil. They registered complaints of teeth grinding among 6-8% of the study population (7,8).

SB prevalence declines with age, as it is only 3% among people aged above 60 years (9,10).

SB ETIOLOGY AND COMORBIDITIES

SB is classified as primary when no clear medical cause is present. Etiology of most SB cases is primary. SB is classified as secondary when it is a comorbidity or when it is caused by a clinical, neurological or psychiatric disorder, use or suppression of substances or medications, or when it is associated with another primary sleep disorder (1, 2) (Table 1).

Secondary SB may be associated with other movement disorders (Parkinson's Disease, Huntington's Disease, Shy-Drager Syndrome, cervical dystonia, oromandibular dystonia, epilepsy, late oral dyskinesia, Gilles de la Tourette syndrome, hemifacial spasms, base ganglion infarction) (2,11-14). Patients with cerebellar hemorrhage, olivo-ponto-cerebellar atrophy, dementia, fibromyalgia, miofacial pain, or tinnitus, may show secondary SB (2,15). Children and adults with development disorders, such as cerebral paralysis, autism with mental retardation, hyperactivity, and attention deficit in patients suffering from Rett syndrome, schizophrenia, post-traumatic stress disorder, and nervous bulimia show a higher prevalence of secondary SB (2,16).

SB may be caused by or be associated with abuse of substances, including alcohol, tobacco, caffeine (high doses), cocaine, amphetamines, ecstasy, or antipsychotic drugs that antagonize dopaminergic neurotransmission, serotonin reuptake (fluoxetine, sertraline, paroxetine, citalopram, escitalopran, venlafaxine), fenfluramine, or calcium channels (flunarizine) (17-20).

SB is associated with restless leg syndrome and nocturnal myoclonus (periodic limb movement), as these syndromes are present in approximately 10% of SB cases (10). Approximately

3-5% of obstructive sleep apnea-hypoapnea syndrome suffer show SB, with full remission when sleep obstructive apnea-hypoapnea is treated (6,21).

SB may show a subclinical and early manifestation of REM sleep behavior disorder, a parasomnia most frequently reported in elderly males (22-24). SB may be associated with comorbid non-organic insomnia coupled with anxiety and/or depression (2).

Table 1: Sleep bruxism classification

Sleep Bruxism Classification
1 - Primary or Idiopathic
1.a - Peripheral
- Occlusal factors
1.b - Central
- Central nervous system aminergic imbalance
2 - Secondary
2.a - Associated with medications or other substances:
- Selective serotonin reuptake inhibitor antidepressants
- Calcium channel blockers (flunarizine)
- Antidopaminergic drugs
- Antipsychotics (dopamine neurotransmission antagonists)
- Amphetamines, ecstasy
- Caffeine
- Cocaine
- Tobacco
2.b - Associated with Sleep Disorders (according to ICSD 2005*)
- Restless Legs Syndrome
- REM Sleep Behavior Disorder
- Obstructive sleep apnea-hypoapnea
2.c - Neurological disorders
- Huntington's Disease
- Hemifacial spasm
- Parkinson's Disease
- Dementias
- Multiple system atrophy
- Olivopontocerebellar atrophy
- Gilles de la Tourette syndrome
- Cerebellar vascular accident
- Myofascial pain
- Mental retardation
- Attention deficit-hyperactivity disorder
- Rett Syndrome
- Comatose state
- Post-anoxia encephalopathy
2.d - Psychiatric Disorders
- Schizophrenia
- Affective Disorders
- Nervous Bulimia – Nervous Anorexia
2.e - Other Diseases
- Xerostomy
- Gastroesophageal reflux
- Fibromyalgia
- Sjögren's Syndrome

CLINICAL MANIFESTATIONS

Primary SB is a highly heterogeneous chronic condition with a broad spectrum in terms of frequency, variety and severity of symptoms; in milder cases, several weeks may pass without any clinical manifestation, or bursts of extended periods of activity may be observed, coupled with psychological stress (5,9,25,26).

Table 2 summarizes the main clinical characteristics of bruxism

Table 2

Sleep Bruxism Clinical Characteristics
- teeth grinding
- noises heard by other people
- orofacial automatisms and excessive tongue and lips movements
- leg movements
- excessive sweating during sleep (23%)
- nightmares
- palpitations (62%)
- short awakenings/arousals
- sudden awakenings plus tachycardia
According to what is reported by patient, symptoms felt upon arousal in the morning
- pain or discomfort in jaw muscles (86%)
- temporal headache (48-65%)
- jaw rigidity and reduction of its motility, generating masticatory difficulties at breakfast (40%)
- reduction in intensity of teeth grinding triggered by local muscular pain
- exacerbation of symptoms above added by emotional tension
- teeth hypersensitivity to liquid cold or hot nutrients, and to air
- sensation of a dry mouth
- tongue, lips and cheek wounds
- toothaches
- sore throat (69%)
- cervicalgia (69%)
- thoracic and abdominal pain (69%)
- diffuse muscular pain
Symptoms related to sleep (72%-80%)
- nonrefreshing sleep
- excessive daytime sleepiness
- fatigue
Observed by dentists or doctors
- exacerbation of periodontal diseases
- dental wear/shining fillings
- abrasion and enamel wear with dentine exposure
- rupture of dental restorations
- broken teeth
- reduction in saliva flux
- masseter, temporal, medial and lateral pterigoid muscles hypertrophy
- pain upon muscle palpation, and painful temporomandibular articulation
- tongue slashes
- parotitis or sialolithiasis
- gingivitis
Other symptoms
- traces of personality prone to anxiety
- decrease in libido (31-50%)
- systemic arterial hypertension

Secondary SB clinical manifestations are more persistent.

The most important symptom of teeth grinding is the characteristic noise similar to that produced by rubbing “granite against granite”.

Approximately 40% of patients suffering from bruxism complain about orofacial pain, masticatory myalgia, and morning jaw rigidity. Toothaches and symptoms of temporomandibular articulation dysfunction often lead suffers to search for dental treatment (2,9,27-29).

Bader et al. report that 19% of SB sufferer have systemic arterial hypertension; from 31% to 50% have erectile dysfunction, tinnitus, muscular tension, nocturnal sudoresis, and palpitations (9,30).

In general, SB patients do not show any side effect due to their interrupted sleep for extended periods of time (26). Nevertheless, around 70-80% of individuals diagnosed with SB show symptoms of fragmented sleep with moderate daytime sleepiness, a perception of not getting refreshing sleep, nocturnal arousals, a larger number of body movements during sleep, excessive sweating or nightmares; these symptoms develop over a period of up to five years of the onset of bruxism (31,32,33). Daytime neurocognitive symptoms, such as attention and memory deficits, may occur both in adults and children suffering from SB (16,34).

Stress and anxiety are contributing factors to SB. In fact, SB sufferers have a more aggressive and hyperactive temper, being prone to anxiety, stress, and development of psychosomatic symptoms (35). SB sufferers are 1.3 times more prone to develop anxiety disorders compared to the population in general (28,36). Prevalence of teeth grinding is higher in adult patients who live under constant emotional tension and who are hyperactive, aggressive, or whose personality has traces of impulsiveness (37,38).

PHYSIOPATHOLOGY

Multiple factors with different weights participate in primary SB physiopathology. It is believed that an association exists between the development of SB and masticatory motor control alterations, autonomic nervous system activity during sleep, alterations in psychological factors related to central nervous system (CNS) aminergic neurotransmission factors related to saliva homeostasis and gastric control and dental occlusional disorders.

RHYTHMIC MASTICATORY MUSCLE ACTIVITY DURING SLEEP

Alterations in motor control and in sympathetic autonomic nervous system control during sleep play a central role in normal and abnormal masticatory organization during sleep, as well as in SB genesis (2,36,39).

The sleep period is characterized by a partial reduction (NREM sleep) and a total reduction (REM sleep) in motor activity and axial skeletal neuromuscular, appendicular and masticatory tone (40). Despite these findings, phasic orofacial muscular activity remains during NREM sleep. Such activity is termed rhythmic

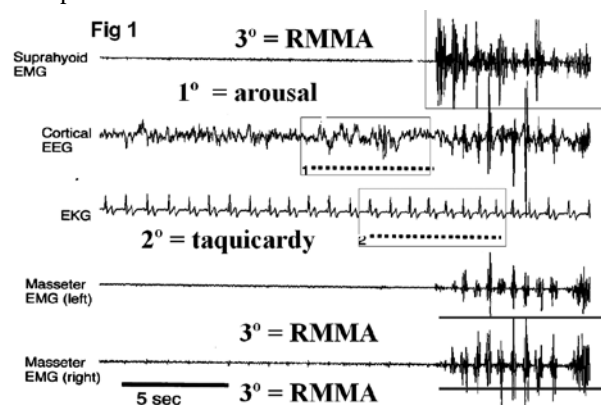
masticatory muscle activity during sleep (RMMA) (41). RRMA is a normal and automatic activity of the masticatory muscular system (41). It shows a characteristic coactivation pattern of opening and closing of the jaw muscular system followed by salivation and swallowing. RMMA bursts have a short duration and are registered exclusively during sleep, in both normal individuals and SB sufferers (41).

Studies conducted at Montreal University, Canada, with volunteer subjects demonstrated that 58% of the normal individuals showed RMMA without dental contact and/or teeth grinding (2,41). The registered RMMA episodes were not associated with arousals by the sleep EEG, nor were phasic raises in cardiac frequency registered (41). The present study has found that the frequency of RMMA episodes declines significantly from the fourth decade of life onwards, in parallel with a decline in SB prevalence. RMMA episodes are thus considered a normal orofacial motor manifestation (2,39,41). However, SB sufferers show quantitative and qualitative differences with respect to RMMA bursts.

RMMA frequency in primary sleep bruxism sufferers is from three to eight times greater than in normal volunteers (5,39,41). In SB sufferers, with each occurring RMMA episode, there is an increase in the duration of the episode and in the amplitude of neuromuscular discharge. There is also an increase in the number of bursts, and a reduction in intervals times between bursts. In SB sufferers, over 90% of RMMA bursts evolve to dental contact and teeth grinding (5,41). Dental contact during RMMA episodes generates a physical strength which may range from 300 to 8000 g (42). Such physical strength may cause ischemia of the dental structure, leading to acute pain and, subsequently, to a chronic inflammatory process with chronic dental pain.

Moreover, RMMA episodes occurring in association with dental contact show a typical temporal sequence. Approximately 4 seconds before the occurrence of an RMMA episode, an arousal takes place. This event is registered in the NREM sleep EEG. It is followed by a rise in cardiac frequency and, soon afterwards, dental contact (5,41,43) (Figure 1).

Figure 1. Temporal sequence typical of RMMA bursts. As per what has been registered in NREM sleep encephalogram, approximately 4 seconds before a RMMA episode happens an arousal occurs, followed by a phasic increase in cardiac frequency; soon afterwards, dental contact takes place.



Arousal is thus the first step that triggers RMMA episodes in SB; no arousals take place during RMMA episodes in normal volunteers who do not suffer from SB (43).

In 2003, Kato et al. demonstrated that young sufferers of bruxism showed seven times more RMMA episodes with teeth grinding, induced by artificial sound stimuli with intensity to provoke an arousal when compared to normal volunteers paired for age and sex (44). Both the arousal recorded by EEG during sleep with activation of the sympathetic autonomic nervous system and artificial sound awakening occur before the RMMA episode, which includes jaw contact and teeth grinding.

An arousal typically does not cause motor activation in normal volunteers, but does cause excessive motor activation in SB sufferers (44). Thus, teeth grinding during sleep might be considered a response to excessive activation of the masticatory motor system, with abnormal oromotor activity secondary to arousal (1,2,5,9,39). As a matter of fact, a study using transcranial magnetic stimulation demonstrates an excessive motor response with an increase in excitability of the trigeminal tract in young adults suffering from SB (42). For susceptible phenotypes, factors such as medical diseases, anxiety, consumption of recreational drugs, and stress trigger arousals which lead to tooth grinding bursts during RMMA episodes (2). The cause of spontaneous arousals during primary SB is not well established (43).

AUTONOMOUS NERVOUS SYSTEM ACTIVITY IN SB SUFFERERS

There are two types of functional alterations in the autonomic nervous system of SB sufferers; phasic alterations and tonic alterations.

Autonomic nervous system phasic alterations are represented by the transitory activation of the sympathetic autonomic nervous system associated with arousals, and with RMMA episodes followed by teeth grinding (43,45).

A study conducted in 2003 demonstrated that an alteration in the autonomic nervous system occurs during sleep in subjects with SB (46). The study included a spectral analysis of the cardiac frequency in SB sufferers during NREM sleep; this analysis showed an increase in the sympathetic tonus around 3 minutes before the occurrence of a sleep bruxism episode. An increase in sympathetic tonus leads to an arousal, which follows the sequence of RMMA. Such an imbalance in the autonomic nervous system may lead to an arousal, as well as to RMMA with dental contact.

Sympathetic nervous system tonic alterations have been demonstrated in a recent study that performed spectral analysis of the cardiac frequency among volunteer SB sufferers and healthy volunteers that were paired for sex and age. The spectral analysis shows a statistically significant increase in cardiac frequency in SB sufferers (47).

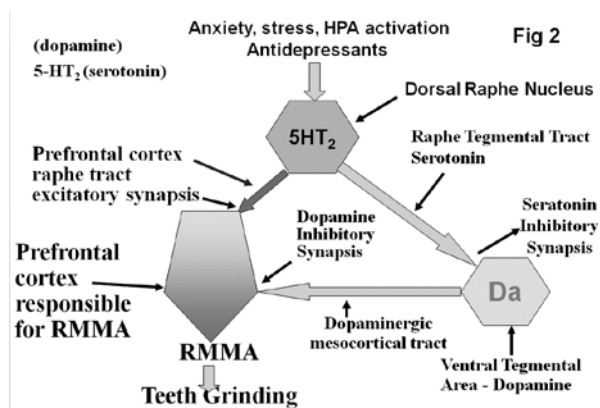
Thus, there are indications that an autonomic dysfunction with an increase in sympathetic nervous system tonus is present in primary SB sufferers (2,46).

NEUROTRANSMITTERS AND NEURAL CHANNELS IN BRUXISM

A functional anatomic structure in the CNS has not yet been identified as a neural center responsible for the involuntary oromandibular movements (1,2). Nevertheless, there is evidence of integrated participation of dopaminergic neurotransmission in the mesencephalic ventral tegmental area, noradrenergic channels in the locus ceruleus and serotonergic channels in the raphe dorsal nucleus and prefrontal cortex, in SB genesis and modulation.

Figure 2 depicts the serotonergic and dopaminergic channels involved in normal masticatory activity. The axons of the serotonergic neurons in the dorsal raphe nucleus are projected via the raphe tegmental tract to the ventral area of the dopaminergic mesencephalic tegment, and to the prefrontal cortex via the prefrontal raphe cortex. The prefrontal raphe cortex tract is excitatory, whereas the raphe tegmental tract inhibits the dopaminergic neurons of the ventral mesencephalic tegmental area. The dopaminergic neurons of the ventral mesencephalic tegmental area are projected towards the prefrontal cortex via the prefrontal mesencephalic cortex tract, thus inhibiting involuntary oromandibular activity (1).

Figure 2: Neural channels in SB



Mesencephalic raphe tegmental tract:
origin: dorsal raphe nucleus
projection: ventral mesencephalic tegmental area
neurotransmitter: serotonin
polarity: inhibitory

prefrontal cortex raphe tract:
origin: dorsal raphe nucleus
projection: prefrontal cortex
neurotransmitter: serotonin
polarity: excitatory

prefrontal cortex mesencephalic tract:
origin: ventral mesencephalic tegmental area
projection: prefrontal cortex
neurotransmitter: dopamine
polarity: inhibitory

An increase in serotonergic and noradrenergic activity caused by selective serotonin reuptake inhibitors increases synaptic transit in the raphe tegmental tract, which in turn inhibits the dopaminergic activity of the ventral mesencephalic tegmental area and increases synaptic transit in the prefrontal raphe cortex excitatory tract (17). Reduction in dopaminergic activity in the ventral mesencephalic tegmental area reduces synaptic transit in the prefrontal cortex mesencephalic tract, leading to disinhibition of prefrontal cortex activity. The combined effects of an increase in serotonergic excitatory activity of the prefrontal raphe cortex tract and a reduction in dopaminergic inhibitory activity of the prefrontal cortex mesencephalic tract would disinhibit the prefrontal cortex, thus allowing for the expression of oromandibular automatisms during NREM sleep (2,39).

HYPOTHESIS FOR THE SB DOPAMINERGIC EFFECT

Indications of an imbalance in dopaminergic neurotransmission in the context of SB are found in CNS imaging, pharmacological and epidemiological study results.

Parkinson's disease sufferers may show teeth grinding during treatment with levodopa, which is likely the result of drug effect of presynaptic inhibition. Administration of low dose levodopa has a therapeutic effect for the treatment of primary SB as it attenuates tooth grinding episodes (1,2,9).

It is known that chronic use of dopaminergic drugs results in dopamine receptor hypersensitivity and may lead to manifestations of late dyskinesia and teeth grinding (20). Hyperdopaminergic states induced by amphetamines, MDMA (methylenedioxyamphetamine or ecstasy) and cocaine may induce teeth grinding both during sleep and wakefulness (1).

Association of SB with other disorders related to alterations in dopaminergic transmission supports a role for the dopaminergic system in primary SB. Approximately 10-20% of SB sufferers show restless leg syndrome symptoms with periodic limb movements during sleep (PLMD) (10).

Selective serotonin reuptake inhibitor antidepressants (SSRIs) may cause extrapyramidal symptoms, such as fine tremors of the upper limbs, acatysia, dystonia, parkinsonism, tardive dyskinesia, and teeth grinding. An increase in serotonergic transmission induced by SSRIs produces a dopaminergic reduction in mesocortical and nigrostriatal circuits; this reduction would disinhibit the prefrontal cortex, causing oromandibular automatisms and teeth grinding during sleep (17). SSRIs are thus contraindicated for the treatment of anxiety or depression symptoms in SB sufferers (1,2).

Studies using CNS neuroimaging suggest a role for alterations in dopaminergic neurotransmission in SB sufferers (48-50).

PSYCHOPHYSIOLOGICAL ASPECTS

Studies utilizing physiological registers demonstrate that SB sufferers and normal controls show increases in the masticatory neuromuscular tonus, voluntary masticatory activity, and cardiac frequency when facing stressing stimuli (37,51,52).

Hypothetically, behavioral factors, such as anxiety, tension, negative emotions and frustrations would cause an increase in activity of the hypothalamic pituitary adrenal axis, with an increase in CRF, ACTH, cortisol and adrenaline secretion by supradrenal glands, both during sleep and wakefulness (36). Release of adrenaline by supradrenal glands would facilitate an increase in neuromuscular tonus, reduction in the saliva secretion rate, both during sleep and wakefulness, and tonic increase in basal activity of the sympathetic nervous system, with a subsequent increase in the frequency of RMMA episodes and teeth grinding during sleep (1).

Some individuals showing these behavioral factors continue to grind their teeth even after specific treatment for dental malocclusion; this reinforces the importance of the behavioral theory of SB. Despite the fact that SB is not a primary psychological disorder, the behavioral component may be an important causal factor. We suggest that this component should be adequately assessed and treated when present. Psychological responses to stressing factors in SB sufferers are quite distinct, in view of the personality profile characteristic of SB sufferers (34).

PERIPHERAL DENTAL FACTORS

In the 1960's, dental theory stated that "peripheral" elements, such as dental malocclusion and dental and jaw mechanical factors, were the cause of SB. This theory has since been challenged and has now been deemed inconclusive (2). Three important observations act to dispute a required role for dental malocclusion in the generation of SB:

1. Toothless individuals also present RMMA episodes, indicating that dental surface contact is not a causal factor that triggers oromotor activity during sleep (1,2).

2. The first event occurring in the SB sequence is activation of the autonomic nervous system followed by awakening, an increase in cardiac frequency and eventually, coactivation of masticatory muscles and dental surface contact; not the other way round. That is, dental contact is not required as the first event that leads to awakening (41,43,44,53).

3. The study conducted by Lazic et al. did not document a significant correlation between occlusal alterations in SB sufferers and the degree of masticatory electromyographic activity in individuals carrying moderate to severe SB. This finding indicates that peripheral factors are likely not instrumental in SB genesis and intensity (53,54).

We conclude that dental mechanical contact is a late occurrence in the sequence of SB events, and that peripheral factors derived from a presumed dental contact via periodontal mechanoreceptors are not required for coactivation of masticatory muscle activity (39,53).

XEROSTOMY

RMMA episodes with or without teeth grinding stimulate saliva secretion and lubrication of the upper digestive aerial tract (55).

Medical and psychiatric disorders or medications which cause

a reduction in saliva secretion may induce a compensatory increase in RMMA episodes, which may develop into teeth grinding. Factors that may lead to a reduction in saliva secretion include:

1. Medications: antidepressants, antihypertensives, anticholinergics, antihistamines, muscle relaxers, antiemetics, antiparkinsonians, chemotherapeutics;

2. Head and neck radiotherapy;

3. Systemic diseases: Sjogren's Syndrome, cystic fibrosis, sarcoidosis, tuberculosis, diabetes mellitus, thyroid diseases, hepatic insufficiency, chagasic megaesophagus, cerebral palsy, paralysis of the 7th cranial pair, AIDS;

4. Mental disorders: bulimia, nervous anorexia, depression, anxiety.

GASTROESOPHAGEAL REFLUX – GERD

The causal hypothesis that correlates gastroesophageal reflux with reduced salivation is based on the well-known fact that the presence of acid in the distal third of the esophagus stimulates saliva secretion, in order to neutralize the acid present in the esophagus 55. The presence of saliva in the oral cavity triggers swallowing, and RMMA which, in turn, stimulate salivation.

According to some authors, the presence of GERD is a risk factor for SB (56).

CLINICAL DIAGNOSIS

It is crucial to screen for all risk factors for secondary SB by checking for the use of antidepressive medications, recreational drugs, and associated medical or mental conditions in order to establish an etiological diagnosis of SB.

Seven major symptoms and signs significantly contribute to the bruxism clinical diagnosis (1):

1. Noises due to teeth grinding;

2. Dental wear (one or more teeth);

3. Complaints about pain and masticatory muscle weakness and occasional headaches or migraines (temporal muscles);

4. Tooth sensibility to cold and/or heat;

5. Masseter and temporal muscle hypertrophy;

6. Crepitation of the temporomandibular articulation or reduction in the amplitude of jaw opening;

7. Signs of wounds to the tongue.

According to the International Classification of Sleep Disorders published in 2005, diagnosis of primary bruxism calls for the following items 2:

The patient or their relatives are aware of tooth grinding noises during the patient's sleep period.

1. Presence of abnormal dental wear;

2. Presence of jaw discomfort, jaw fatigue or pain, or muscle pain upon arousal in the morning;

3. Signs of masseter muscle hypertrophy.

POLYSOMNOGRAPHIC EVALUATION

The physiologic variables that are monitored during polysomnography for the investigation of SB are: EEG, electrooculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), aerial flux (nasal and oral), breath effort (thoracic and abdominal), and blood gases (oxygen and hemoglobin saturation) (1).

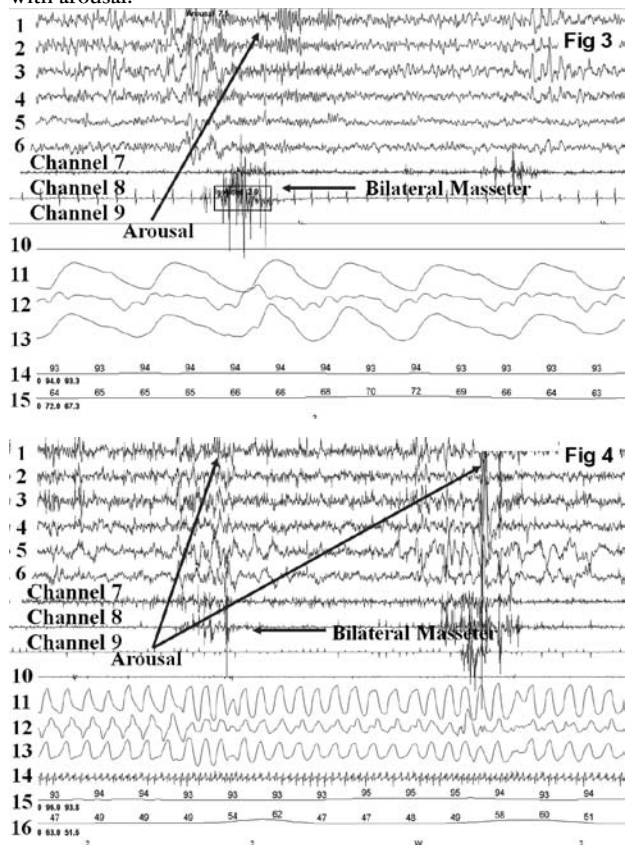
SB video-polysomnographic evaluation should include electromyography using masseter and temporal muscle electrodes, as well as a simultaneous audiovisual register of the anterior tibialis muscles. The audiovisual register is important for identifying bruxism episodes, as well as swallowing and snoring. It is also important for registering body movements and tooth grinding sounds (2).

Video-polysomnography is not necessary for the diagnosis of SB, but it is important for ruling out other sleep disorders, such as sleep apnea syndrome, PLMD, REM sleep behavioral disorders, and gastroesophageal reflux (1-4).

OROMANDIBULAR POLYSOMNOGRAPHIC MANIFESTATIONS DURING SLEEP

The large majority (80-85%) of bruxism bursts occur in NREM sleep stages 1 and 2, which precede the transition from stage 2 to REM sleep in the second half of the sleep period (2) (Figures 3 and 4).

Figures 3 and 4: Polysomnography with bruxism episode associated with arousal.



Episodes of neuromuscular activity with teeth grinding occurring during REM sleep are termed destructive bruxism.

Variability of oromotor activities occurring during sleep is greater in cases of mild to moderate SB, calling for the use of polysomnography for two consecutive nights. In severe SB cases, there is lower variability and, therefore, polysomnography performed over just one night is sufficient (26).

OTHER MANIFESTATIONS DURING SLEEP

Approximately 60-80% of tooth grinding episodes are associated with abnormal and periodic electromyographic activity of anterior tibial muscles that is characteristic of PLMD (1,2).

SLEEP ARCHITECTURE

In general, sleep macroarchitecture in SB sufferers below 40 years of age without manifestations of chronic pain is normal; that is, sleep latencies, quantity of different sleep stages and number of arousals are normal (2,16).

In populations of patients above 40 years of age, SB is generally accompanied by complaints of poor sleep quality. Alterations in sleep architecture with an increase in the number of changes in sleep stages, increase in the frequency of arousals, reduction in the amount of delta sleep and REM sleep, and increase in the amount of REM sleep normally occur in this group (32,33).

Arousals with sympathetic activation and subsequent tooth grinding bursts cause sleep fragmentation which, in turn, motivates subjective symptoms, such as poor sleep quality, excessive daytime sleepiness and fatigue, without substantially altering sleep architecture (40,57).

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