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ORIGINAL ARTICLE

Urinary levels of catecholamines among individuals with and without sleep bruxism

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Abstract
Introduction Sleep bruxism (SB) is characterized by repetitive and coordinated mandible movements and non-functional teeth contacts during sleep time. Although the etiology of SB is controversial, the literature converges on its multifactorial origin. Occlusal factors, smoking, alcoholism, drug usage, stress, and anxiety have been described as SB trigger factors. Recent studies on this topic discussed the role of neurotransmitters on the development of SB.

Objective Thus, the purpose of this study was to detect and quantify the urinary levels of catecholamines, specifically of adrenaline, noradrenaline and dopamine, in subjects with SB and in control individuals.
Materials and methods Urine from individuals with SB (n = 20) and without SB (n = 20) was subjected to liquid chromatography. The catecholamine data were compared by Mann–Whitney's test ($p \leq 0.05$).
Results Our analysis showed higher levels of catecholamines in subjects with SB (adrenaline = 111.4 μg/24 h; noradrenaline = 261,5 μg/24 h; dopamine = 479.5 μg/24 h) than in control subjects (adrenaline = 35,0 μg/24 h; noradrenaline = 148,7 μg/24 h; dopamine = 201,7 μg/24 h). Statistical differences were found for the three catecholamines tested.
Conclusion It was concluded that individuals with SB have higher levels of urinary catecholamines.

Keywords Sleep bruxism · Etiology · Catecholamines · Adrenaline · Noradrenaline · Dopamine · Urine

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Introduction

Sleep bruxism (SB) has been defined as a parafunction, which is characterized by coordinated, repetitive mandible movements and non-functional teeth contacts (clenching and tooth-grinding) during sleep time. The prevalence of SB is about 8% of the population [1]. Although various theories have been formulated about its etiology, SB etiology remains unknown [2, 3].

In the past, the commonly held belief was that SB was caused by occlusal alterations. Recent studies, however, have demonstrated that premature teeth contacts and other occlusal alterations are not the main cause of SB [1, 3]. Stress, emotional alterations, diseases of the central nervous

system, sleep disorders, drugs, and chronic pain are some of the factors now believed to be involved in SB etiology [4–6]. However, the intrinsic neurological mechanisms of SB etiology are not known.

Furthermore, the role of the central nervous system (CNS) in the etiology of SB deserves further attention, as some CNS-related drugs stimulate or inhibit SB [1, 4, 7–13]. Recent reports have also indicated a relationship between SB and rhythmic muscular activity [1].

Catecholamines are neurotropic substances involved in the rhythmic activity of masticatory muscles, mainly during sleep [14, 15]. Evidence points to the participation of dopamine, adrenaline, noradrenaline, and serotonin on SB etiology and modulation [2, 6, 14, 16–18]. Dopamine levels should play an important role in the genesis of repetitive, coordinate mandible movements. As this kind of muscular activity is closely related to SB, some works have suggested that dopamine and CNS neurotransmission are involved in SB [17, 19–22] and other forms of bruxism etiology [16].

Considering the possible role of catecholamines in SB etiology, the present paper aims to compare the urinary levels of dopamine, noradrenaline, and adrenaline in individuals with and without SB.

Material and methods

Subject selection

Two hundred male adults, completely dentate, aged between 30 and 35 years, were evaluated (medical history and dental examination). From this initial sample, 40 healthy individuals were selected and divided into two groups of equal number (n=20). One group was composed of individuals presenting sleep bruxism and the other was the control group.

The criteria used to identify SB were: (1) wear of anterior teeth on incisal border; (2) wear of occlusal posterior teeth (in both conditions, the worn borders of teeth fit the antagonist arch in excursive mandible movement, with anatomical change of the teeth. When wear of the occlusal posterior teeth was present, individuals were considered bruxers only if open bite was also present); (3) bed partner reports of frequent sleeping sounds of teeth grinding; and (4) white line at buccal mucosa, teeth-impressed tongue, and spontaneous soreness of masticatory muscles (taken together, these signs and symptoms define SB on this study). These diagnostic criteria for SB were based on the American Academy of Sleep Medicine diagnostic and coding manual [23].

Individuals not presenting all the aforementioned signs or symptoms were classified as non-bruxers. None of the selected volunteers for SB group presented symptoms of wake-time clenching.

For both groups, individuals that take medications, which may interfere with catecholamines (amphetamines and amphetamine-like compounds, appetite suppressants, bromocriptine, buspirone, caffeine, carbidopa-levodopa, clonidine, dexamethasone, diuretics [in doses sufficient to deplete sodium] ethanol, isoproterenol, labetalol, methyldopa, MAO inhibitors, nicotine, nose drops, propafenone, reserpine, theophylline, tricyclic antidepressants, and vasodilators). Those with removable dentures as well as alcohol and/or illegal drugs users were also excluded. Furthermore, volunteers who presented variables that influence cathecolamine levels, such as cardiovascular conditions, stress level, chronic pain, and sleep disturbance/sleep disorders, were also excluded.

This research project was approved by the institution's research ethics committee (Protocol CEP/UNITAU 331/05) as it adhered to all the ethical aspects.

Sampling procedures

All the volunteers enrolled in the study received written and oral instructions about the procedures to obtain urine samples, based on earlier studies [17, 21].

The entire urine volume excreted in a 24-h period was collected in an appropriate bottle containing 20 ml of 50% chloridric acid. The first urine excreted that day was discarded, while all subsequent urine was collected and the hour of excretion recorded up to the first urine of the following day. The bottle was kept in the refrigerator during the collection day and transported to the laboratory for analysis in a styrofoam container.

The volunteers also observed the following instructions: (1) Do not use vaginal creams 24 h before sampling; (2) Do not collect samples during menstruation; (3) Do not smoke, drink alcoholic beverages, soft drinks, coffee or tea 12 h before and during sampling; (4) Do not eat chocolate or bananas 12 h before and during sampling; (5) Do not engage in vigorous exercise in the 7 days before sampling; and (6) Avoid stressful situations in the 7 days before sampling.

The samples were subjected to a liquid chromatography analysis to identify and quantify adrenaline, noradrenaline, and dopamine. The data obtained were analyzed statistically first by the Anderson–Darling test and then by the Mann–Whitney test (p<0.05) to compare the groups for each catecholamine.

Results

Figure 1 presents the average values of catecholamines for both groups. A statistically significant (p=0.00) positive correlation was found between catecholamine levels and the presence of SB.

Discussion

Considering the multiple factors involved in SB etiology [1], mainly those relating to the central nervous system [4–13], the present study evaluated urinary catecholamine levels in individuals with and without SB. We found that these levels were significantly higher in the group presenting SB than in the control group.

Reference urinary levels are lower than 60 μg/24 h for adrenaline, and 200 μg/24 h for noradrenaline and between 65 and 400 μg/24 h for dopamine [24]. It is worth noting that in our study, all the individuals suffering from SB presented higher levels of adrenaline and noradrenaline, and only three subjects had dopamine levels within the normal parameters. In contrast, all the control subjects showed normal urinary levels of all the catecholamines tested here.

We used a clinically based method for bruxism diagnostic, used by some [21, 25], but not all authors [1]. The so-called gold standard for diagnosing bruxism for research proposals is the polysomnography [1], but it is not available for all the researchers in this field, and in the dental practice the most used method is still being the clinically based one. To minimize the risk of incorrect diagnostic, we grouped a number of clinical signs and symptoms to define bruxism, as described above.

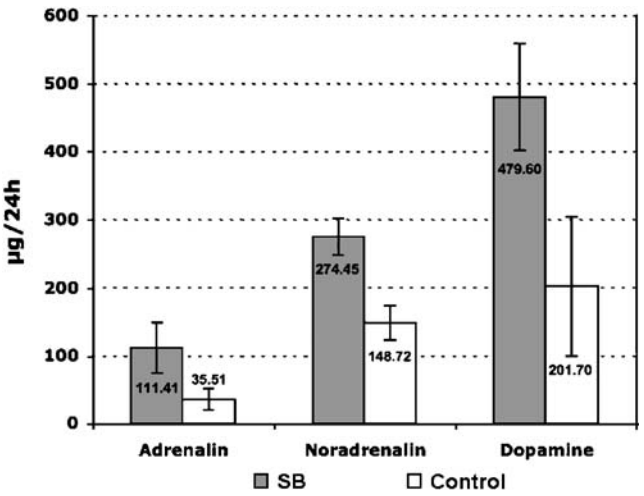

Fig. 1 Distribution of average urinary levels of catecholamines by group

Our results are consistent with those encountered by Clark et al. [17], but these authors did not test for dopamine urinary levels. However, Vanderas et al. [21] also tested for dopamine, but in children, with results similar to those shown here. As the study proposed by them [17, 21], our study is correlational and a causal relationship among catecholamines and bruxism is not demonstrated.

L-Dopa was the first neurotransmitter associated with SB. Magee [18] used L-Dopa to treat a patient presenting Parkinson's disease and found that this patient also presented SB after the L-Dopa treatment. However, Lobbezoo et al. [14] verified that a low dosage of L-Dopa diminished SB episodes. Other studies [9, 10] have linked antidopaminergic drugs with SB. Lavigne et al. [2] showed that bromocriptine, a dopamine agonist, had no effect on SB and suggested that this result was due to bromocriptine's link to the D2 receptor for dopamine. However, this result does not exclude dopamine's role in SB, as other dopamine receptors (D1, D3, D4) should be involved in its etiology.

Ecstasy (methylenedioxymethamphetamine) should also produce a dopaminergic effect. Among the side effects of this drug, SB has been reported by 70% of ecstasy users. This drug causes the release of serotonin, dopamine, and noradrenaline [13].

Multiple studies [7, 8] have related selective inhibitors of serotonin reuptake, such as fluoxetine, sertraline, fluvoxamine, and paroxetine, with SB. However, this relationship is not fully understood. Selective inhibitors of serotonin reuptake increase serotonin in synapses, inducing dopaminergic cells to produce less dopamine. With less dopamine in the prefrontal cortex, there is less control of undesirable movements, enabling the onset of SB [11]. In contrast, Stein et al. [12] reported two clinical cases of SB that diminished during the use of selective inhibitors of serotonin reuptake.

The controversial role of neurotransmitters in SB [1] and other forms of bruxism [16] is probably due to the complex system formed by numerous neuron-chemicals and its receptors, regulating and downregulating a net of neurologically mediated effects.

Despite the controversy in the aforementioned literature regarding the influence of catecholamines in the etiology of SB, our findings suggest that catecholamines probably do play a role in SB etiology, as stated earlier [17, 21], and should therefore encourage new clinical trials involving larger samples and additional outcome analyses, such as electromyography of masticatory muscles, polysomnography, single proton emission computerized tomography (SPECT) and functional magnetic resonance imaging (fMRI), which

should help to investigate a possible causal relationship between cathecolamines and SB.

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