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Nocturnal Bruxism: Still a Nosological Conundrum?

Corresponding Author:
Dr. Eva Gjovreku,
Dentistry specialist, Crystal University, Tirana, Albania - Albania

Submitting Author:
Dr. Gentian Vyshka,
Lecturer, Biomedical and Experimental Department, Faculty of Medicine, University of Tirana, Rr Dibres 371 - Albania

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Author(s): Gjovreku E, Vyshka G

Abstract

Sleep bruxism is a frequent and bothersome, albeit benign condition. Diagnosed initially from dentistry specialists, it has become gradually a neurological enigma, since several neurotransmitters and situations not related to dental malocclusion might cause teeth grinding and atypical facial pain. We have studied three cases with surface EMG, performed in the morning hours, thus registering electrical activity bilaterally and simultaneously in ipsilateral masseters and temporal muscles. A clear dissociation in electrical activity at rest was seen, with higher values of spontaneous discharges in the masseter muscles (up to 35%), confronting with a normal activity in the temporal muscles. Such a finding favours the dystonic hypothesis of bruxism, and will therefore justify usage of ad hoc drugs, such as botulinum toxin, among others.

Introduction

Bruxism is a stereotypical disorder of movement, characterized from teeth grinding or clenching during sleep. The disorder was initially described from Marie Pietkiewicz, but she originally used the term bruxomanie [1]. Such a term was later changed into bruxism, and entered in the clinical usage. The disorder seems fairly frequent, with prevalence values oscillating between 8 to 31%, according to different sources [2].

As long as bruxism might be present during sleep or during daytime, authors have coined the term sleep (nocturnal) bruxism, and prevalence studies generally refer only to the latter [3]. Teeth grinding is an epiphenomenon of internal tension in a very large number of adults, although very few refer it and request medical assistance. Due to the generally benign character of the disorder, dentistry specialists are the first to diagnose it, since bruxism might cause teeth damage, temporo-mandibular dysfunction and atypical facial pain [4-6].

Methods

According to the specialistic literature, sleep bruxism and teeth grinding are considered as concomitant to:

1. Peripheral factors such as dental malocclusion;
2. Psychosocial influences, namely stress and anxiety levels;
3. Central causes, related to neurotransmitter systems in the telencephalon or in the basal ganglia.

Of course, minor anatomical anomalies such as malocclusion will predispose for the disorder, but the fact is that sleep bruxism episodes might persist even after correction of dental causative factors. This is true to that extent, that considering bruxism principally a movement disorder, authors have tried and applied botulinum toxin to relieve clinical signs [7].

Genetic traits have been traced and formulated in sleep bruxism as well, but the models are not uniform [8, 9].

Authors have been raising the hypothesis of a dopaminergic dysfunction in the genesis of sleep bruxism, and the first case was reported from Magee in 1970, in a patient suffering from the disease of Parkinson, showing consistent teeth grinding under L-Dopa therapy [10]. Other sources have reached a completely different conclusion, suggesting even a therapeutic value of Dopa-therapy specifically in bruxism [11]. Other catecholamines such as adrenaline have been under scrutiny, with suggestions that beta-blockers might be helpful. Conversely, serotonin was imputed in aggravating the disorder, and selective serotonin reuptake inhibitors (SSRI), a new class of antidepressants drugs, show among other side effects the teeth grinding and bruxism episodes, among other [12].

Results

Surface electromyography was performed to three persons, whose spouses were complaining continuous nocturnal rumours related to teeth grinding; only two of the patients referred facial pain, and the third one being completely free from symptoms. All three cases were recently seen from dentistry specialists and malocclusion was excluded as a probable causative factor.

Surface EMG was performed in the morning, at a maximum of three hours after arousal, and findings

were repeated in four consecutive sessions in following days. Muscular activity was registered at the masseters and temporal muscles of the same side for two minutes, and thereafter on the other side, with the patient being at rest, in supine position, in a quiet environment.

A summary of electromyographic findings and one of the traces registered are shown respectively at the Table no. 1 and Figure no. 1 (see below).

Figure 1: Trace above, masseter activity at rest. Trace below, temporal muscle of the same side. Note the obvious difference in amplitude and the frequency' discharges (same amplification).

Spontaneous activity was registered in terms of fibrillations and complex electrical discharges, with patient at rest (no chewing or swallowing in course).

Discussion

Bruxism is considered etiologically different under two aspects: as a local disorder; and as a neurological problem [13]. Our data suggest that a clear dissociation in the electrical activity was seen between electromyographic traces in the masseter, when comparing with the ipsilateral temporal muscle. Such data are compatible to some extent with other findings; and registration over both muscular territories is an indispensable condition for obtaining reliable data [14, 15].

A limitation of the present study was the fact that registration was performed during day hours; a thorough polysomnographic study with night hour's registration would of course be more helpful towards a better understanding of the entire pathological process that characterizes bruxism, diurnal or nocturnal in its nature.

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Illustrations

Illustration 1

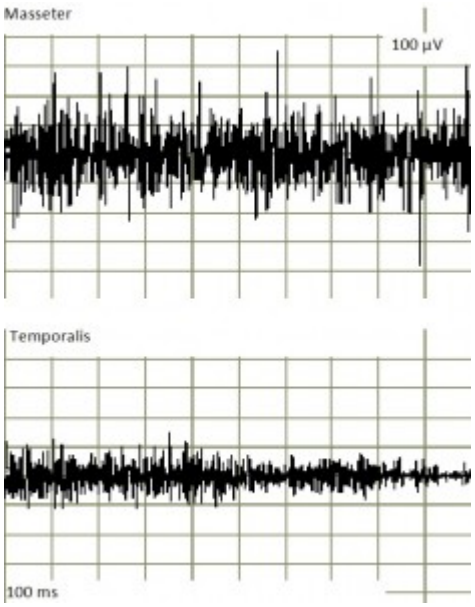
Table

Presence of spontaneous activity and respective percentage in the muscular territories controlled through surface electromyography

Case	Age (years)	Sex	Right masseter	Right temporal muscle	Left masseter	Left temporal muscle
1	37	Male	Spontaneous activity (>30%)	No spontaneous activity	Spontaneous activity (30%)	Spontaneous activity with norm (15%)
2	44	Male	No spontaneous activity	No spontaneous activity	Spontaneous activity (30%)	No spontaneous activity
3	38	Female	Spontaneous activity (40%)	Spontaneous activity within norm (10%)	Spontaneous activity (30%)	No spontaneous activity

Illustration 2

Surface electromyography of masseter and temporalis (ipsilateral)



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