# The Evaluation of Polysomnography in Patients with Nocturnal Bruxism Referring to Mashhad Faculty of Dentistry

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## ABSTRACT

**BACKGROUND AND OBJECTIVE:** Nocturnal bruxism is considered as a major cause of temporomandibular disorders. Although nocturnal bruxism is normally diagnosed clinically, polysomnography is also a gold standard diagnostic test for this disorder. This study aimed to evaluate the physiological events in patients with nocturnal bruxism through polysomnography analysis.

**METHODS:** This retrospective study was conducted on 20 patients who were diagnosed with nocturnal bruxism based on the sleep disorders questionnaires of the International Committee of Sleep Disorders (ICSD) and physical examinations. The sleep patterns and bruxism of these patients were also evaluated via polysomnography.

**FINDINGS:** In this study, 15-25% of the patients diagnosed with nocturnal bruxism reported various problems related to temporomandibular joint (TMJ) involvement. In addition, 85% of the patients complained of facial pain and tenderness, and the most frequent symptoms during the examinations were TMJ clicking, joint noises and limited mouth opening.

**CONCLUSION:** According to the results of this study, the quality and quantity of sleep in bruxism patients are at a lower level compared to healthy individuals.

**KEY WORDS:** Nocturnal bruxism, Polysomnography, Diagnosis.

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# Introduction

**B**ruxism is defined as a para-functional oral habit consisting of spasmodic or rhythmic grinding or clenching of the teeth (1, 2). According to self-reports of several patients, the prevalence of bruxism decreases with age, dropping from 14-20% in children to 8% in adults less than 60 years of age and 3% in adults over 60 years old (2). The prevalence of bruxism is not significantly different between men and women (3, 4); however, some researchers consider the incidence to be relatively higher among male patients (5). Bruxism is classified into two different categories: 1) daytime and nocturnal bruxism, based on the time of occurrence and 2) primary (with no specific medical or psychological causes) and secondary bruxism (associated with the use of drugs or neurological disorders), based on the type of occurrence.

Nocturnal bruxism is mostly known to occur during sleep (3, 6). In general, natural sleep consists of two main stages: 1) the non-REM stage, also known as nonrapid eye movement sleep, including the state of light sleep (phases I and II), and heavy sleep (phases III and IV), and 2) the REM stage, also known as rapid eye movement sleep or active sleep. These stages are repeated every 90-110 minutes (7, 8), and evidence suggests that nocturnal bruxism episodes most frequently occur during the stages of light sleep (phases I and II) (2).

The most common symptoms of nocturnal bruxism are temporomandibular joint (TMJ) pain, teeth sensitivity and mobility, poor quality of sleep, fatigue, headaches (especially in the temporal region), neck pain and masticatory dysfunction, and teeth grinding noises, which is often reported by the patient's spouse or parents (2, 3).

According to several specialists, the most common complications caused by bruxism, which are normally observed during dental examinations, include abnormal tooth wear, crenated tongue, gingival recession, maxillary and mandible bone protrusion, masseter muscle hypertrophy, decreased saliva, breakage of teeth restorations and limited mouth opening (2, 3, 6).

Several studies have attempted to determine the causes of bruxism and the associated complications. A number of these studies have proposed occlusal changes as the main contributing factor, while some others have propounded multiple factor hypotheses attributing this disorder to various agents such as genetics, mental and psychological disorders, anger and stress levels, environmental factors, catecholaminergic balance between the central nervous system and autonomic nervous system, and the consumption of substances such as alcohol, tobacco and caffeine (1, 3, 9). Bruxism is normally diagnosed based on the observation of dental wear by specialists (9); however, recent studies, which are based on electromyography (EMG) records, are indicative of a statistically insignificant correlation between dental erosion and bruxism (10).

Other diagnostic approaches of bruxism involve the use of tools such as pressure-sensitive intraoral devices, EMG and polysomnography (3, 10). Although EMG is considered an economical diagnostic measure, it is likely to affect the patient's normal sleep pattern due to multiple electrodes which are connected to the skin. Furthermore, due to the lack of muscle activity detection caused by coughing, sneezing and yawning during sleep, the patients may manifest falsely high positive results (3, 10).

Recently, the use of polysomnography in sleep laboratory for the diagnosis of bruxism, as well as the recording of the associated features, has been recognized as a gold standard diagnostic test (5, 10). Polysomnography is able to record EMG (masseter and temporalis muscle activities), Electroencephalogram (EEG, brain activity) and Electrocardiogram (EKG, cardiac activity) (2, 3).

According to several studies conducted via polysomnography, sleep bruxism is associated with certain physiological activities in the patient's brain and heart. The first observed physiological phenomenon is the increased activity of the sympathetic autonomic nervous system which occurs approximately one minute before the bruxism episode.

Along with this incidence, a decrease occurs in the parasympathetic nervous system, and all these factors will ultimately lead to a significant increase in the activity of the cerebral cortex. This phenomenon has been recorded approximately 4 seconds prior to the manifestation of bruxism.

Moreover, a slight increase in the patient's heart rate occurs about one second prior to the bruxism, which is normally followed by the bruxism episode involving the gnashing of the teeth and tooth contacts with or without sounds (11, 12).

Regarding the common symptoms of bruxism occurring in the patient's mouth and face, which are occasionally destructive, this study aimed to investigate the probable causes of bruxism as well as the other symptoms of this disorder affecting different body parts.

#### **Methods**

This cross-sectional study was conducted on 24 patients referring to the Specialized Clinic of TMJ and Occlusion affiliated to Mashhad Faculty of Dentistry, who complained of nocturnal bruxism. The participants consisted of 13 women and 11 men ageing between 18-50 years. In order to select the subjects, a questionnaire was designated based on the International Committee of Sleep Disorders (ICSD) for the evaluation of nocturnal bruxism. A polysomnography system (Stellate, Canada) was used to investigate the patients. This system is capable of assessing parameters such as REM and non-REM sleep disorders, epileptic seizure disorders, sleeprelated rhythmic movements and breathing disorders, and mental illnesses such as panic. All the physiological events were recorded by infrared cameras during the patients' sleep monitoring. In addition, the sound of teeth grinding was recorded by a microphone mounted above the patients' heads or an audio receiver connected to the patients' cheeks in the form of an electrode.

The EEG signals were received by 20 electrodes placed in different areas of the subjects' heads, and we only needed 8 electrodes in order to evaluate bruxism. Eventually, patients who complained of nocturnal bruxism (ageing between 18-50 years) and had one or more of the following factors were enrolled in this study: 1) abnormal tooth wear; 2) history of bruxism (at least 3 nights a week during the past 6 months); 3) masticatory muscle pain and 4) discomfort while waking.

The patients were required to be committed to the completion of the project, and the exclusion criteria of the study were as follows: 1) the use of medications which could lead to changes in sleep pattern and 2) presenting with no definite systemic disease or mental disorder. All the patients were clinically examined regarding their masticatory system functions including muscles, TMJ and teeth by a prosthetic specialist. Following that, the questionnaires on the masticatory system health and oral symptoms associated with bruxism were completed by the examiner, and the patients were referred to the sleep clinic of Ibn Sina Hospital for polysomnogram testing using a polysomnography device (Stellate, Canada). Finally, the existence of bruxism, as well as the quality and quantity of the patients' sleep structure, were evaluated.

Moreover, the following associated factors were examined in all the patients: the number of bruxism episodes within one hour and one night, total time of bruxism episodes within one night, bruxism time index (BTI), number of bruxism episodes during phases I, II, and III of non-REM sleep, percentage of bruxism episodes occurring in different sleeping positions (supine, on the right or left side), and the maximum masseter-muscle contraction. The obtained data from the questionnaires, physical examinations and polysomnogram were analyzed by SPSS V.11.5 and tables were also drawn.

#### Results

Out of 24 participants in the study, 4 patients were excluded due to lack of confirmation for bruxism by the polysomnography device (N=2), and lack of cooperation in the study (N=2). Eventually, a total of 20 individuals, including 11 women and 9 men, were enrolled in the study.

In these patients, the mean total sleep time (TST) was 384.5±42 minutes, the mean time from the onset of sleep to the non-REM stage (NRSL) was 22.85±11 minutes, and the mean time from the onset of sleep until the beginning of the REM stage (RSL) was 105.3±19 minutes. During the non-REM stage of the bruxism patients in this study, phase I, II and III constituted about an average of 11.05±1.8%, 53±1.9%, and 16.9±2.5% of the patients' sleep, respectively. Furthermore, the mean sleep quality of the patients, which was obtained through dividing the total TST by the total sleep duration, was estimated as 85.35±4.7% (Table 1). In addition, the mean number of bruxism episodes in the studied patients was calculated as  $22.9\pm10.4$ , while the mean number of bruxism episodes in one hour was recorded as 3.65±1.8. Additionally, the mean of the total duration of nocturnal episodes (TDur./n) was estimated as  $220\pm93$  seconds (Table 2).

According to the results of this study, bruxism was present in a mean of  $1.05\pm0.5\%$  of the patients' sleep time (BTI in Table 2). Moreover, the mean numbers of nocturnal bruxism episodes during different phases of non-REM sleep were as follows: 1)  $11.05\pm5.2$  episodes during phase I (EPi/NI); 2)  $10.6\pm4.8$  episodes during phase II (EPi/NII) and 3)  $1.25\pm1.4$  episodes during phase III (EPi/NII).

With regard to the sleeping positions during bruxism episodes, the patients were in the supine position in approximately half the cases  $(51\pm11\%)$ , while the rest of the subjects were sleeping on their left or right side during the incidence of the episodes. However, no significant difference was observed between the occurrence of bruxism and these positions (Table 2). The maximum intensity of the massetermuscle contraction in patients before sleeping was recorded as  $505\pm102 \mu v$ , while the mean intensity of the muscle contraction changed to  $186\pm39 \mu v$  during the nocturnal episodes of bruxism (Table 2).

 Table 1. Mean changes in sleep quality and quantity

 from polysomnogram of patients with nocturnal bruxism

Variable	Mean±SD
Actual sleep time in total sleep time (min)	384.5±42.07
Period from sleep onset to non-REM (minute)	22.85±11.60
Period from sleep onset to REM (minute)	105.3±19.71
Phase I of NR to the total sleep time (percent)	$11.05 \pm 1.85$
Phase II of NR to the total sleep time (percent)	53±1.91
Phase III of NR to the total sleep time (percent)	16.9±2.55
Sleep quality	85.35±4.70

Table 2. Mean quality and quantity of sleep frompolysomnogram of patients with nocturnal bruxismand sleeping position of patients

Variable	<b>Mean±SD</b>
Bruxism episodes in one night (number)	22.9±10.48
Bruxism episodes in one hour (number)	3.65±1.88
Total time of bruxism episodes in one night (seconds)	220±93.99
Bruxism activity to total sleep time (percent)	$1.05\pm0.58$
Bruxism episodes during NRI phase (number)	11.05±5.20
Bruxism episodes during NRII phase (number)	10.6±4.89
Bruxism episodes during NRIII phase (number)	$1.25 \pm 1.42$
Bruxism episodes in supine position (percent)	51±11.29
Bruxism episodes on the right side (percent)	29.5±8.34
Bruxism episodes on the left side (percent)	19.5±9.46
Maximal masseter-muscle contraction (microvolt)	505±102
Masseter-muscle contraction intensity during	186±39.01
bruxism episodes (microvolt)	

#### **Discussion**

According to the results of this study, the bruxism of the investigated patients differed from the findings of polysomnogram tests conducted on normal individuals outside Iran in terms of the quality and quantity of sleep. Furthermore, the amount of deep sleep in the subjects of the present study was less than that of healthy individuals, while the amount of light sleep in these patients was estimated to be higher than that of normal individuals (2, 13, 14). The difference in the sleep pattern of the studied patients might be due to the changes in the individuals' routine place of sleep as well, which was one of the inevitable limitations of the

present study also mentioned by previous studies in this regard (13). With regard to the variables related to bruxism, BTI could be considered as a major index in the evaluation of this disorder. In the current study, the patients experienced 1% of bruxism activity during their night's sleep on average, while this amount was reported to be 1.4% in one study by Saletu et al. (15) and about 0.9% in a research conducted by Camparis et al. (16). In their research conducted over the years on bruxism at a sleep laboratory using a polysomnography device, Lavigne et al. indicated that in most cases, bruxism episodes would occur during the light sleep phases (i.e. non-REM, phases I and II), while patients with bruxism tended to have less deep sleep compared to healthy individuals, and this difference in the sleep pattern might be a contributing factor for bruxism (17), as confirmed by the present study.

According to a number of researchers, the severity of bruxism is equal to 60% of the maximum intensity of the intentional force used by the patients to clench and grind their teeth before falling asleep at the sleep clinic while undergoing polysomnography. This figure signifies a considerable amount of force, which is normally used for mastication or other functional activities. One of the interesting findings of the present study was that out of 10 patients, 2 cases applied this maximum force to clench their teeth before falling asleep. Evidently, bruxism episodes during sleep are highly likely to cause certain complications for these patients (1).

In this study, the maximum intensity of the masseter-muscle contractions during the episodes was found to account for 36.83% of the maximal contraction of the masseter muscle in the patients who were observed to experience it before sleep. In one study conducted by Camparis et al., bruxism episodes and the total time of bruxism were reported to be 7 times per hour and 350 seconds per night, respectively (16), which are higher than the obtained results of the present study. This difference might be due to the higher severity of bruxism in the subjects of that study, as well as other differences in the polysomnography, sleep clinic, race and age of the investigated subjects. In another study by Okason et al., bruxism was observed to be either more frequent in the supine sleeping position, or not affected by the patients' sleeping position significantly. Moreover, it was reported that patients with bruxism tended to change their sleeping position more often than the normal subjects (1). In our study, about half of the studied patients experienced episodes of bruxism while sleeping in the supine position. With respect to the limitations of the current study, it seems that bruxism patients participating in this project had a lower level of sleep quality and quantity compared to healthy individuals, and bruxism was found to be more frequent in the supine position compared to other sleeping positions.

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### References

1.Okason JP. Management of temporomandibular disorders and occlusion. 6<sup>th</sup> ed. Elsevier Health Sciences;2008.p.130-63.

2. Trindade Mde O, Rodriguez AG. Polysomnographic analysis of bruxism. Gen Dent. 2014;62(1):56-60.

3.de la Hoz-Aizpurua JL, Díaz-Alonso E, LaTouche-Arbizu R, Mesa-Jiménez J. Sleep bruxism. Conceptual review and update. Med Oral Patol Oral Cir Bucal. 2011;16(2):e231-8.

4. Glaros AG. Incidence of diurnal and nocturnal bruxism. J Prosthet Dent. 1981;45(5):545-9.

5.Huynh NT, Rompré PH, Montplaisir JY, Manzini C, Okura K, Lavigne GJ. Comparison of various treatments for sleep bruxism using determinants of number needed totreat and effect size. Int J Prosthodont. 2006;19(5):435-41.

6.Ilova S, Zolger D, Castrillon E, Car J, Huckvale K. Biofeedback for treatment of awake and sleep bruxism in adults: systematic review protocol. Syst Rev. 2014;3:42.

7.Huynh N, Kato T, Rompré PH, Okura K, Saber M, Lanfranchi PA, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res. 2006;15(3):339-46.

8.Rompre PH, Daigle-Landry D, Guitara F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res. 2007;86(9):837-42.

9.Rintakoski K. sleep bruxism–genetic factors and psychoactive Substances: Studies in Finnish twins. Department of Public Health, Hjelt Institute and Department of Oral Public Health, Institute of Dentistry, Faculty of Medicine University of Helsinki on April 4<sup>th</sup> 2014. Available at: https://helda.helsinki.fi/ bitstream/handle/10138/44685/ rintakoski\_ dissertation.pdf? sequence=1

10.Kim JH, McAuliffe P, O'Connel B, Diamond D, Tong Lau K. Development of bite guard for wireless monitoring of bruxism using pressure-sensitive polymer. International Conference on Body Sensor Networks, Singapore. 2010;109-116.

11.Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med. 2003;14(1):30-46.

12.Kato T, Rompre P, Montplaisir JY, Sessle BJ, Lavigne GJ. Sleep bruxism: An oromotor activity secondary to microarousal. J Dent Res. 2001;80(10):1940-4.

13.Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res. 1996;75(1):546-52.

14.Kim H, Han HJ. Polysomnographic study of sleep bruxism in adults: preliminary study. J Korean Sleep Res Soc. 2011;8(1):9-13.

15.Saletu A, Parapatics S, Anderer P, Matejka M, Saletu B. Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatry Clin Neurosci. 2010;260(2):163-74.

16.Camparis CM, Formigoni G, Teixeira MJ, Bittencourt LR, Tufik S, de Siqueira JT. Sleep bruxism and temporomandibular disorder: Clinical and polysomnographic evaluation. Arch Oral Biol. 2006;51(9):721-8.

17.Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: An overview for clinicians. J Oral Rehabil. 2008;35(7):476-94.