Suppression of Sleep Bruxism: Effect of Electrical Stimulation of the Masseter Muscle Triggered by Heart Rate Elevation

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Purpose: To examine whether electrical stimulation of the masseter muscle triggered by heart rate elevation preceding sleep bruxism (SB) can actively suppress SB. ***Materials and Methods:*** Ten volunteers who were aware of their SB habits participated in the study. Baseline electromyogram (EMG) activity of the unilateral masseter muscle and electrocardiogram (ECG) signal were recorded on the first night. The individual mean sensation and pain thresholds to electrical stimulation of the unilateral masseter muscle were determined in awake subjects before the experiment. On the second night, electrical stimulations at either of the two threshold intensities were automatically generated and delivered to the masseter muscle on the opposite side from where electrodes were placed immediately after the heart rate exceeded 110%. On the third night, electrical stimulations at the other threshold intensity were delivered. ***Results:*** The numbers of SB events per night and per hour, the number of EMG bursts per SB event, and the duration of SB events decreased significantly on the nights when stimulation was applied compared with the baseline data. There were no significant differences between cases where the sensation threshold was used as the stimulation intensity and those in which the pain threshold was used as the stimulation intensity. ***Conclusion:*** The results suggest that electrical stimulation of the masseter muscle triggered by heart rate elevation can significantly suppress SB. *Int J Prosthodont 2014;27:80–86. doi: 10.11607/ijp.3330*

Sleep bruxism (SB) is abnormal involuntary masticatory muscle activity and is classified as a form of parasomnia in the international classification of sleep disorders.[1] Various causal factors such as malocclusion,[2] emotional stress,[3–5] and hereditary factors[6–8] have been suggested. Previously, peripheral factors such as malocclusion and occlusal interference were regarded as the most important etiologic factors of SB. However, SB has recently come to be considered to be primarily induced centrally.[9] Furthermore, since SB occurs unconsciously during sleep, the force during SB sometimes exceeds maximum occlusal force.[10] Such strong force leads to tooth wear, fracture of the tooth crown and root, jaw muscle discomfort, stiffness, fatigue, and temporomandibular disorders.[11,12] In addition, SB disturbs the bed partner's sleep due to the noise associated with tooth grinding. SB was often found as a comorbidity of obstructive sleep apnea—a serious sleep disorder.

SB has been treated through various methods, such as occlusal adjustment,[13] splint therapy,[14–16] psychologic treatment,[17] and medical treatment.[18] Currently, the occlusal splint (soft or hard type) is mainly used during sleep, not to suppress SB but to reduce as much as possible the harmful effects on the stomatognathic system.[19]

The possibilities of suppressing SB frequency and duration through audio,[20–23] vibrational,[24] and gustatory[25] stimulations have been reported. Furthermore, the suppression of SB through electrical stimulation of the lips or temporal muscle has also been reported,[26,27] suggesting that stimulation of the trigeminal nerve area may actively suppress SB. However, since these studies used masticatory muscle activity as a stimulation trigger, SB is suppressed after occlusal forces have already been loaded on the stomatognathic system, which is disadvantageous.

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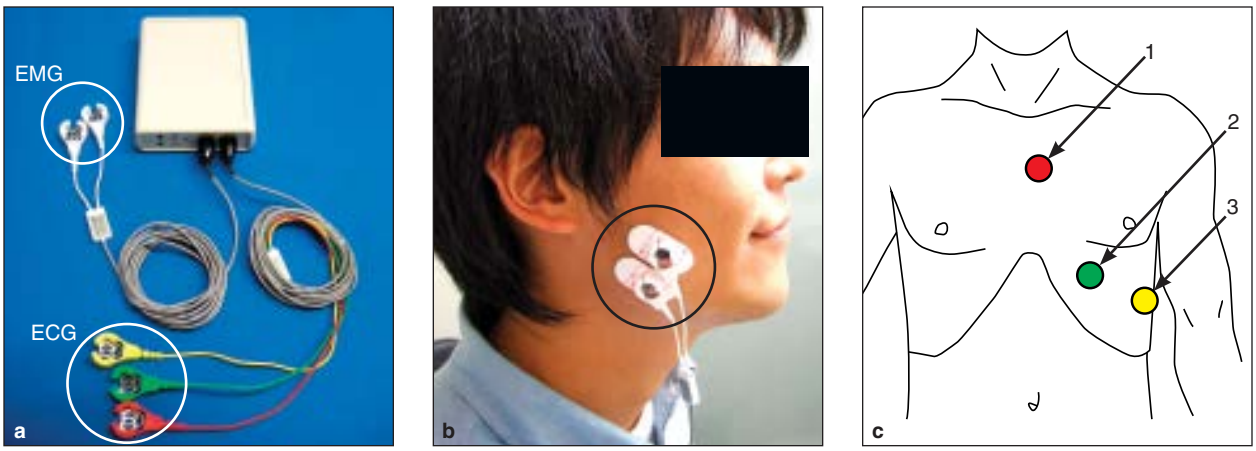

Fig 1 **(a)** Telemetry system for EMG and ECG. **(b)** Bipolar electrodes were placed on the unilateral masseter muscle for EMG recordings, and electrical stimulation was applied to the masseter muscle on the contralateral side. **(c)** Positions of the three electrodes used for ECG recordings.

On the other hand, the central nervous system has been determined to play an important role in the etiology of SB because SB is a part of the arousal phenomenon during sleep and its expression is modulated by neurotransmitters.[28] The onset of SB has also been found to be preceded by high-frequency electrocardiogram (ECG) activity and sympathicotonia characterized by heart rate and blood pressure elevation.[9,29,30] Therefore, the authors decided to use heart rate elevation as a trigger for suppression of SB before its onset.

The goal of this study was to examine whether electrical stimulation of the masseter muscle triggered by heart rate elevation can actively and effectively suppress SB.

Materials and Methods

Subjects

Ten young subjects (six men, four women; mean age: 26.7 ± 3.5 years) were recruited from students and faculty members in the Department of Fixed Prosthodontics, Osaka University, who were aware of their SB habits. Prior to the experiment, preliminary EMG recordings were performed, and subjects who met at least one of the Lavigne et al[31] criteria for SB (the number of SB events per night/hour, the number of EMG bursts per SB event) were selected. Subjects exhibiting or having a history of other sleep disorders and cardiovascular disturbance, subjects missing two or more molars, with the exception of the third molar, and subjects with pain in the orofacial region were excluded.

All participants provided written informed consent. This study was approved by the Ethics Committee of the Osaka University Graduate School of Dentistry (no. H22-E14).

Experimental Methods

The electromyogram (EMG) from unilateral masseter muscle and the ECG were recorded at home for three consecutive nights using a portable EMG and ECG telemetry system (EMG-ECG Telemeter 00, Harada Electronic Industry) (Fig 1). Participants used the recording system at home following instructions that described in detail how to set up the system and record data prior to measurement. Alcohol and caffeine consumption were forbidden on the day of recording. Disposable electrodes (Vitrode F, Nihon Kohden) were used for the EMG and ECG recordings. Data were sampled at 1,000 Hz, transmitted to the receiver, and stored in a laptop computer (Let's Note CF-T2, Panasonic).

EMG signals were rectified and integrated. EMG activities both larger than the mean resting state by three SDs and lasting longer than 0.25 seconds were defined as an EMG burst. Twenty-nine multiple EMG bursts lasting more than 3 seconds were defined as one SB event. Thirty-one EMG activities with disturbed ECG signals were assumed to be body movements and were excluded from the analysis. The number of SB events per night, the number of SB events per hour, the number of EMG bursts per SB event, and the duration of SB events were calculated.

The heart rate was calculated from the R wave to R wave (RR) interval of the ECG. The heart rate was calculated from the RR interval of the ECG using the following equation: heart rate (beats/minute) = 60/RR interval(s). Based on a previous study, a gradual increase in heart rate was observed starting from 10 heart beats before the SB event, and the heart rate of B1 (one beat before the onset of an SB event), A1, A2, and A3 (one, two, and three beat(s) after the onset of an SB event) became significantly higher than that of

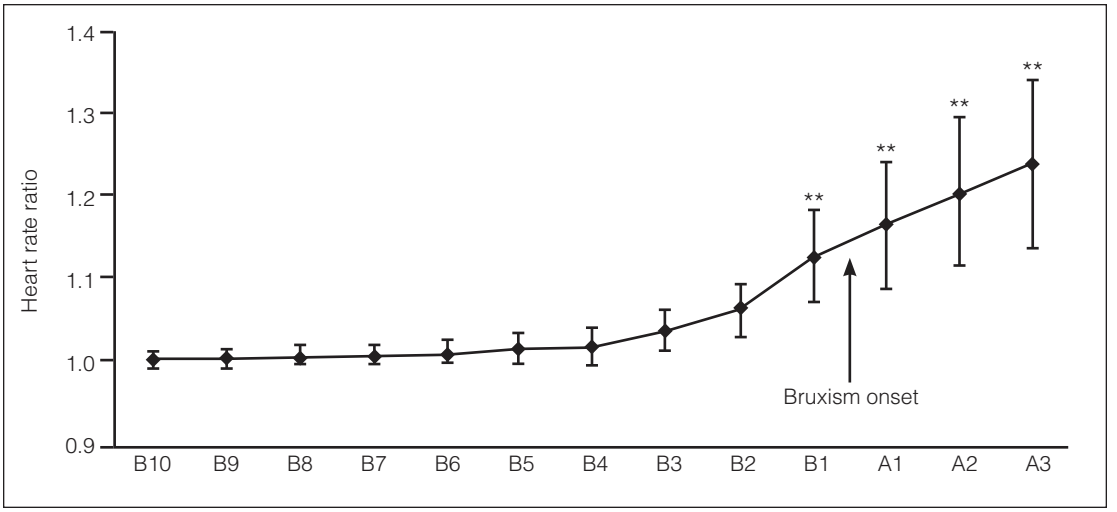

Fig 2 Ten heart beats before (B10 to B1) and three heart beats after (A1 to A3) the onset of an SB event were analyzed. A gradual increase in heart rate was observed starting from 10 heart beats before the SB event. The increase became significant at B1 compared with B10 (Dunnett test, $P < .01$) and reached 110% or more at B1 to A3.

B10 (10 beats before the onset of an SB event) (Fig 2). Based on this result, the threshold value for predicting an SB event was set at an increasing heart rate of 110% when the heart rate of B10 was set at 100%. Electrical stimulations (1 ms pulse, 10 Hz, 500 ms) from the stimulation device (NS101, Unique Medical) were automatically provided on the masseter muscle on the opposite side from where EMG recording electrodes were placed immediately after the heart rate exceeded 110% (Fig 2). The mean sensation and pain thresholds were used for the intensity of stimulation. The sensation and pain thresholds were determined in the waking hours as follows: each subject was asked to raise his/her hand when he/she felt an electrical stimulation or pain while the intensity was increasing. This procedure was repeated three times after incrementing the stimulation intensity by 0.1 mA each time, and the mean intensities were calculated and determined to be the thresholds. Therefore, the stimulation intensities differed among individuals (Table 1). These two intensities were selected because they are conducted in the central nervous system and can possibly modulate the occurrence of SB.

No stimulation was provided on the first night, although all of the experimental equipment was prepared in the usual manner. Stimulation at the sensation or pain threshold was randomly applied to each subject on the second and third nights. If stimulation at the sensation threshold was applied on the second night, then stimulation at the pain threshold was applied on the third night and vice versa. The intensity of stimulation was not changed over the same night because the SB frequency varied with sleep cycle and sleep depth in one night.[30,32] To compare the SB suppression effects of the two stimulation intensities with that of no stimulation (baseline), the numbers of SB events per night and per hour, the number of EMG bursts per SB event, and the duration of SB events were calculated.

Each subject provided responses to a questionnaire on sleep immediately after waking the next morning after EMG recordings were carried out. The questionnaire consisted of four simple questions to determine *(1)* the number of times the subject was awakened during sleep, *(2)* the number of times the subject noticed stimulation during sleep, *(3)* the visual analog scale (VAS) of fatigue in the maxillofacial region, and *(4)* the VAS of sleep quality. The VAS was from 0 to 100, with 0 = no fatigue and 100 = imaginable maximum fatigue and 0 = imaginable maximum discomfort of sleep quality and 100 = no discomfort of sleep, respectively.

Statistical Analysis

Statistical analyses were carried out using the Statistical Package for the Social Sciences (SPSS, IBM) version 17.0 for Windows. Analysis of variance for repeated measurements followed by Tukey tests was used to determine the significance of between-group differences for the number of SB events per night and per hour, the number of EMG bursts per SB event, the durations of SB events, the number of awakenings during sleep, the VAS of maxillofacial

Table 1 Stimulation Intensity and Responses to Four Questions of a Self-Administered Questionnaire

	Baseline			Sensation			Pain			*P*
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	
Stimulation intensity (mA)	–	–	–	0.54	0.22	0.20–0.90	1.21	0.83	0.40–3.40	0.0051*
No. of noticed stimulations/ total stimulations	–	–	–		2/7,060			9/10,903		0.431†
No. of awakenings	0.90	0.99	0–2	0.70	0.82	0–2	1.00	1.56	0–5	0.355‡
Maxillofacial fatigue (VAS)	5.30	10.83	0–34	5.10	9.85	0–30	3.90	9.36	0–29	0.073‡
Sleep quality (VAS)	43.70	11.21	21–56	45.90	10.18	21–58	44.20	12.34	21–62	0.446‡

VAS = visual analog scale.
*Wilcoxon signed rank sum test, †chi-square test, ‡Tukey test. There were no significant differences in the total number of awakenings, degrees of maxillofacial fatigue, or sleep quality.

fatigue, and the VAS of sleep quality. A chi-square test and a Wilcoxon signed rank sum test were used to analyze the number of electrical stimulations that the subjects noticed and the stimulation intensity, respectively. A *P* value of less than .05 was considered to be statistically significant.

Results

The mean intensity ± 1 SD for the sensation threshold was 0.54 ± 0.22 mA, and the mean intensity for the pain threshold was 1.21 ± 0.83 mA (Table 1). The mean sleep time was 5.31 ± 0.96 hours, and the mean analysis time was 4.81 ± 0.96 hours (the time periods 20 minutes after falling asleep and 10 minutes before waking up were eliminated from the analysis).

The total number of electrical stimulations applied over 20 nights (10 subjects for two nights) was 17,963 (sensation threshold: 7,060, pain threshold: 10,903). Therefore, each subject received approximately 900 occurences of stimulation in one night. Of these stimulations, only 11 were noticed by subjects during sleep (sensation threshold: 2, pain threshold: 9). Similarly, the total number of awakenings was 9 during the first night when no stimulation was applied, 7 when the sensation threshold was applied, and 10 when the pain threshold was applied. There were no significant differences between these numbers. There were also no significant differences in the degree of maxillofacial fatigue or sleep quality (Table 1).

A total of 324 SB events was observed in all subjects on the first night with no stimulation, and most of these events (299, 92.3%) were accompanied by heart rate elevation over 110% immediately before the onset of the SB event. Similarly, the total number of incidences of heart rate elevation over 110% was 8,324 on the first night, although only 3.9% of these incidences was related to SB.

All of the four EMG measurement parameters (the mean number of SB events per night/hour, the mean number of EMG bursts per SB event, and the mean number of duration of SB events) decreased significantly in the second and third nights when the sensation and pain thresholds were applied in comparison with the first night (*P* = .001) (Fig 3). Sex difference was not observed in the reduction of SB, although the sample size was not sufficient.

Discussion

The cutoff criteria for SB introduced by Lavigne et al[31] were *(1)* more than four bruxism episodes per hour, *(2)* more than six bruxism bursts per episode and/or 25 bruxism bursts per hour of sleep, and *(3)* at least two episodes with grinding sounds. Among the 10 participants in this study, 10 had more than four SB events per hour and 7 had more than six EMG bursts per SB event.

Most SB studies have used 10%[33,34] or 20%[18,29] of the EMG amplitude of the maximum voluntary contractions (MVC) as an EMG burst threshold for detecting SB. Taking the variability of MVC within a subject into consideration, an EMG burst threshold larger than the mean resting state plus 3 SDs was applied in the present study. This method will be useful when the patient cannot exert the maximum voluntary clenching force because of muscle pain or other pathologic conditions. When this EMG burst threshold by use of SD was compared to MVC after the experiment, it corresponded to approximately 10% to 30% MVC.

Sleep microarousal characterized by an increase in EEG and EMG frequencies has been frequently described as a physiologic adaptation to endogenous or environmental influence.[35] The excessive presence of microarousals experimentally induced by auditory or

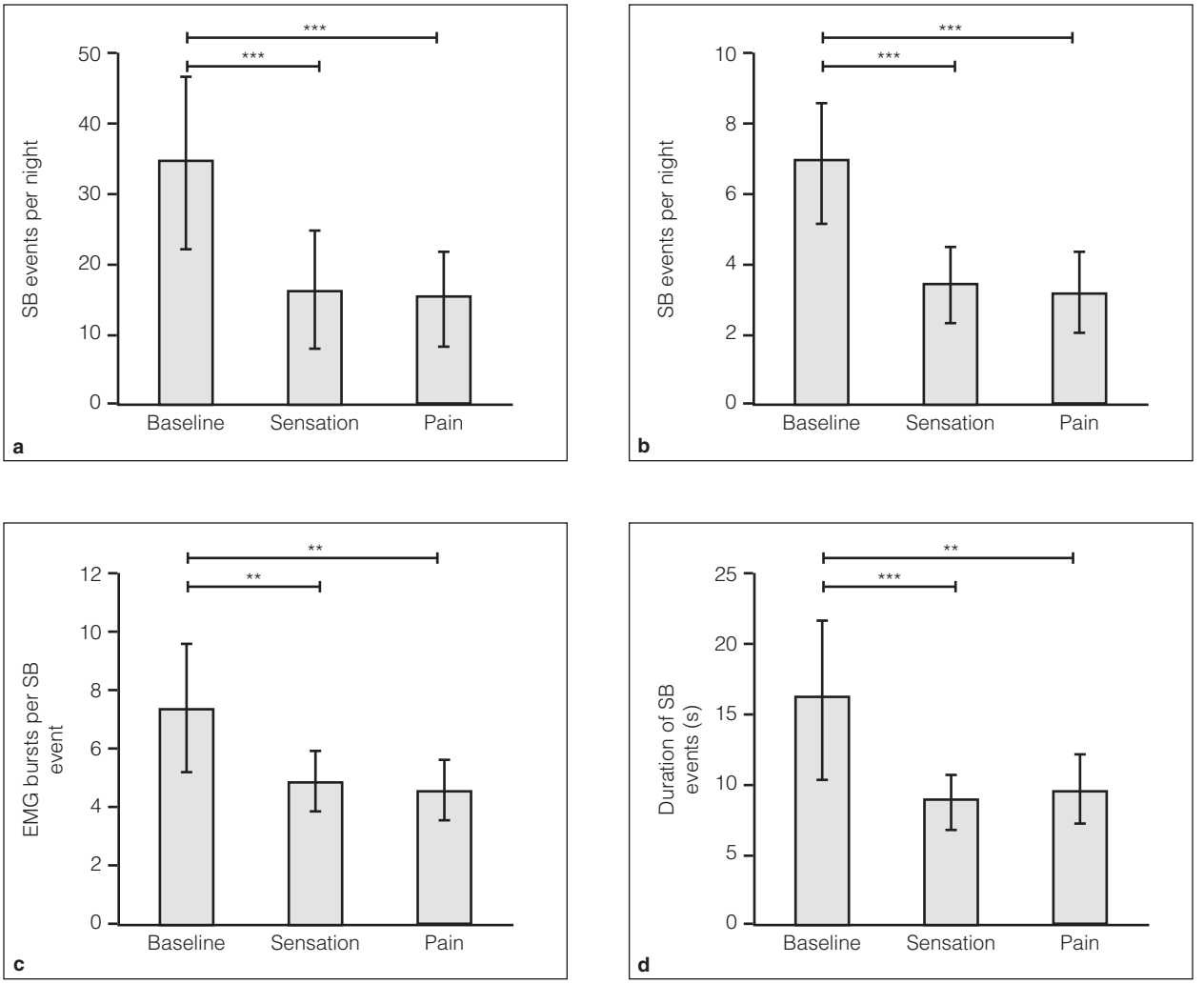

Fig 3 **(a)** Number of SB events per night, **(b)** number of SB events per hour, **(c)** number of EMG bursts per SB event, and **(d)** duration of SB events for 3 recording nights (mean values ± SD, n = 10, $^{**}P < .01$, $^{***}P < .001$).

vibrotactile stimulation causes sleep fragmentation, which can result in the loss of sleep continuity, leading to reports of poor sleep quality, fatigue, somnolence, and a possible decrease in mental and physical function during daytime.[36,37] Hida et al[38] indicated that submental stimulation for several successive nights in patients with obstructive sleep apnea reduced the frequency and duration of apnoeic episodes, with an associated improvement in sleep quality and daytime sleepiness. In the present study, a number of electrical stimulations were performed with no reference to SB, but the degree to which this affected the sleep continuity and the sleep cycle is unclear, although there are no differences in the number of awakenings, the maxillofacial fatigue estimated by VAS, or the sleep quality estimated by VAS based on the responses to the questionnaire. As such, in the future, more subjects should be examined for a longer period using polysomnography to objectively investigate the influence of electrical stimulation on sleep.

The sensitivity of SB detection was considerably high, but specificity was low due to arousal phenomenon without SB, body movement, snoring, or sleep-talking. Therefore, a method in which the electrical stimulation is not given when the heart rates increase by those nonfunctional movements should be found.

The mechanism of SB suppression by electrical stimulation is assumed to be due to the reflex inhibitory response of jaw-closing muscles. However, this mechanism remains unclear. In this study, the number of SB events per night and per hour were suppressed to approximately 45% of baseline values. Similarly, the

number of EMG bursts per SB event and the duration of SB events were suppressed to approximately 60% of baseline values. Nishigawa et al[26] reported that the number of SB events per hour was reduced to 63% of baseline values by electrical stimulation of the lips, and Jadidi et al[27] reported the number of EMG bursts per hour was reduced to just under 50% of baseline values by electrical stimulation of the temporal muscle. Although the experimental methodology and EMG burst definition of these studies are not the same, the SB suppression effect in the present study is equivalent to or superior to those previous reports.

Through electrical stimulation, the number of subjects who met the criteria of Lavigne et al[32] regarding the number of SB events per night decreased from six when no stimulation was applied, to one when the sensation threshold was applied, and to zero when the pain threshold was applied. Similarly, the number of subjects who met the criteria regarding the number of SB events per hour decreased from 10 when no stimulation was applied, to 3 when the sensation threshold was applied, and to 3 when the pain threshold was applied. The number of subjects who met the criteria regarding the number of EMG bursts per SB event was reduced from seven when no stimulation was applied, to two when the sensation threshold was applied, and to one when the pain threshold was applied. The suppression effect on SB events demonstrated in the present study might be insufficient because the parameters of the three criteria for estimating the degree of SB proposed by Lavigne et al[31] were not necessarily reduced within the normal range in all subjects. However, the authors believe that the degree of SB suppression can be increased by attempting to find a more effective threshold setup for triggering onset and a more ideal electrical waveform.

It is not clear why there were no significant differences between the suppression effects of the sensation threshold intensity and the pain threshold intensity. However, this enables the use of a milder electrical stimulation to suppress SB.

When the heart rate elevation remains around 110%, the electrical stimulation occurred continuously in a short time. This is the main reason why numerous electrical stimulations were applied over one night (832.4 times on the first night and approximately 900 times on the second and third nights). Assuming that one electrical stimulation is applied for one SB episode or a series of continuous electrical stimulation that occurred within three seconds is placed into one stimulation, the number of electrical stimulation is 65.3 (13.06 per hour; 0.22 per minute) on the first night. This number is almost equivalent to a previous report.[29]

Although small sample size without a control group and the use of portable devices, not polysomnography, are limitations of this study, this is the first report indicating that SB may be effectively decreased by electrical stimulations of the masseter muscle triggered by heart rate elevation. To implement this automated SB suppression system practically, miniaturization, weight reduction, and simplified recording methods are necessary.

Conclusion

The results of this study suggest that electrical stimulation of the masseter muscle triggered by heart rate elevation significantly suppresses sleep bruxism.

Acknowledgments

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Literature Abstract

Prevalence of dentine hypersensitivity and study of associated factors: A European population-based cross-sectional study

Dentin hypersensitivity (DH) is a common oral pain condition. The aim of this observational, cross-sectional, epidemiologic study was to assess the prevalence of DH and relative importance of its risk factors present in 18- to 35-year-old Europeans. A total of 3,187 patients from general dental practices in France, Spain, Italy, United Kingdom, Finland, Latvia, and Estonia were recruited. Sixty-four examiners were calibrated in their respective countries, obtaining a Kappa agreement of 0.75+. The evaluation was done clinically by cold air tooth stimulation, followed by obtaining the patient's pain rating and investigator's pain rating. Other variables such as erosive tooth wear and gingival recession were recorded, while nature of DH, erosive dietary intake, and toothbrushing habits were accounted for in a questionnaire given to each patient. A total of 1,339 (41.9%) patients reported pain on tooth stimulation and 1,810 (56.8%) scored 1 or higher on Schiff scale for at least one tooth. A highly significant association was noted between questionnaire-reported sensitivity and clinically elicited sensitivity. All three measures used in the assessment of DH were found to be complementary of each other. Clinically elicited DH was also found to be correlated with erosive toothwear and gingival recession. Risk factors such as acid reflux, vomiting, sleep medications, energy drinks, smoking, and acid dietary intake may contribute to possible erosive challenges leading to loss of hard tissue and, eventually, to DH. In conclusion, even though the prevalence of DH is high, many patients appear to reflect good coping mechanisms as reflected in a lower clinical reporting of pain.

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