Sleep and Breathing (2019) 23:363–372
https://doi.org/10.1007/s11325-019-01782-x

DENTISTRY • ORIGINAL ARTICLE

Effects of a contingent vibratory stimulus delivered by an intra-oral device on sleep bruxism: a pilot study

Hirotaka Nakamura[1] · Masayuki Takaba[1] · Yuka Abe[1] · Shuichiro Yoshizawa[1] · Takeshi Suganuma[2] · Yuya Yoshida[1] · Yukari Nakazato[1] · Yasuhiro Ono[1] · Glenn T. Clark[3] · Kazuyoshi Baba[1]

Received: 28 June 2018 / Revised: 4 January 2019 / Accepted: 12 January 2019 / Published online: 26 January 2019

Abstract

Purpose Although sleep bruxism (SB) is one of the most important clinical problems in dental practice, there is no definitive method for controlling it. This pilot study evaluated the effects of contingent vibratory feedback stimuli using an occlusal splint for inhibition of sleep bruxism.

Methods Thirteen subjects with clinically diagnosed SB participated after providing an informed consent. Portable polysomnographic recordings were conducted in the subjects' home environment to make a definitive SB diagnosis and to evaluate the effects of the vibratory stimuli on SB. A force-based bruxism detection system, which used a pressure-sensitive piezoelectric film embedded in the occlusal splint, was utilized to trigger vibration feedback stimuli, which was scheduled to be applied intermittently for 30 min, at 30-min intervals.

Results The number of SB episodes (times/hour), the total SB duration (seconds/hour), the mean duration of SB episodes (seconds/episode), and the micro-arousal index (times/hour) were scored for each time period (with and without vibration). The effects of the vibration on these scores were tested (paired t test; $p < 0.05$). The number of SB episodes tended to decrease with the vibration stimuli, and the decrease in the total SB duration was statistically significant (14.3 ± 9.5 vs. 26.0 ± 20.0, $p = 0.03$). No substantial change was found in terms of the micro-arousal index.

Conclusions These study results suggested that the SB inhibitory system employing a vibratory stimulus might be able to suppress the total SB duration without disturbing sleep.

Keywords Biofeedback · Masseter EMG activity · Portable polysomnography · Sleep bruxism · Stabilization splint

Introduction

The International Classification of Sleep Disorders, 3rd edition (ICSD-3) defines sleep bruxism (SB) as "an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive (intense) sleep arousal activity" [1]. These involuntary masticatory muscle activities may cause and/or accelerate abnormal tooth attrition [2–4], dental restoration and tooth fractures [5], periodontal disease, and temporomandibular disorders [6].

Although SB is an important clinical problem in dental practice, there is no clear means of controlling SB, mainly because the etiology and neural mechanisms underlying SB remain unclear. Therefore, depending on the practitioner's concept of SB causes, various treatments have been proposed. These methods include occlusal splints (OS) [7], as well as pharmacological [8], behavioral (such as cognitive behavioral approaches [9]), and biofeedback approaches [10–12].

OS therapy has been one of the most common methods for managing SB and aims to minimize damage to the stomatognathic system and prosthetic restorations. OS does not inhibit masticatory muscle activity, which is associated with SB [13]; however, it may be able to protect the teeth or prosthetic restorations from occlusal force related to SB.

✉ Kazuyoshi Baba
kazuyoshi@dent.showa-u.ac.jp

[1] Department of Prosthodontics, School of Dentistry, Showa University, 2-1-1 Kitasenzoku, Ohta-ku, Tokyo 145-8515, Japan

[2] Department of Temporomandibular Disorders, Clinic School of Dentistry, Showa University, Tokyo, Japan

[3] Orofacial Pain and Oral Medicine Center and Distance Learning Office, Herman Ostrow School of Dentistry, University of Southern California Los Angeles, Los Angeles, CA, USA

Additionally, several drugs have been reported to inhibit SB, but their effects may vary among individuals [8] and they may have side effects [14]. Therefore, further research is required on this matter. Behavioral approaches, such as relaxation and sleep hygiene improvement, have been proposed; however, their effects on SB inhibition are reportedly limited [15, 16]. Biofeedback therapy, which uses contingent auditory, electrical, or vibratory stimulation, has been studied since the 1970s [10–12, 17, 18]. Contingent auditory feedback decreases nocturnal masseter muscle activity, as measured by portable EMG devices [17]; however, the sleep quality of patients and their sleep partner may be detrimentally affected by the feedback sounds. Electrical stimulus to the upper lip or the temporalis muscle reduces SB duration by 14–52% [10, 11]; however, this method causes a trace current in the body, limiting its use in some patients, such as those with pacemakers.

Vibratory stimuli are more acceptable than auditory or electrical stimulation to patients, and yields comparable inhibitory effects to other types of feedback [12]. A previous single-subject study found a 25% reduction in the number of SB episodes and a 44% reduction in SB duration, without substantial sleep disturbance, with this stimulus. However, the SB-inhibiting effect of this method requires further evaluation in sufficient numbers of SB patients, using polysomnographic (PSG) recordings, for accurate examination of the effects on both SB and sleep structure.

This study thus evaluated the effects of vibration stimulation on SB and sleep quality. The null hypothesis of this study was that vibration stimulation would have no effects on SB.

Materials and methods

Subjects

Thirteen healthy adults (five men and eight women; mean age [± SD]; 26.0 ± 3.0 years) were selected from the students and staff of the Showa University School of Dentistry, based upon clinical diagnostic criteria [19]. Inclusion criteria were as follows: (1) reports of tooth-grinding sounds by the subject's bed partner for at least three nights a week during the preceding 6 months, and (2) the presence of tooth attrition with dentin exposure on at least three occlusal surfaces, masseter muscle hypertrophy upon voluntary clenching, or symptoms of morning orofacial jaw muscle fatigue. Exclusion criteria were as follows: (1) two or more missing molars (excluding third molars) or use of a removable prosthesis; (2) use of medication with possible effects on sleep or motor behavior; (3) alcohol or drug abuse; (4) ongoing physical or dental therapy, including orthodontic treatment; (5) major neurological or psychiatric disorders; and (6) sleep disorder (e.g., sleep apnea syndrome, narcolepsy). All subjects underwent polysomnographic (PSG) recordings using a portable PSG device (Sleep Profiler, Advanced Brain Monitoring Inc., Goleta, CA, USA) and then definitive diagnosis was made using research diagnostic criteria for SB in a polysomnographic study: (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 (Fig. 1) [20].

This study was approved by the Ethics Committee of the Showa University School of Dentistry (No. 2014-034). Informed consent was obtained from the study subjects prior to their enrollment in the study.

Sample size

On the basis of our preliminary study, the vibratory stimulus was expected to lead to a decrease in 50% of the total SB duration per hour (24.9 ± 8.3 s). A sample size of 11 subjects were required to detect this change, assuming a risk of 5% ($\alpha = 0.05$) and a power of 80% ($\beta = 0.20$). After considering the possibility of dropouts, 13 subjects were recruited for this study.

SB inhibition system

The SB inhibition system was composed of an intra-splint force detector (ISFD) [21, 22] and the vibration and control units (iom Co., Ltd., Tokyo, Japan) (Fig. 2). The detail of the ISFD has been described in our previous study [21]. In brief, the ISFD used a 100-μm-thick deformation-sensitive piezoelectric film (Tokyo Sensor Co., Ltd., Tokyo, Japan), which was embedded 1 mm below the occlusal surface of a modified maxillary stabilization appliance, made of a heat-curing resin (Acron; GC Co., Ltd., Tokyo, Japan). With deformation, such as occurs with any substantial occlusal force, the piezoelectric film generates an electrical signal, which varies in accordance with the force applied to the film. The ISFD has an amplifier and a threshold-detection circuit, which generates an output signal when the preset threshold is surpassed. The output signal then drives the battery-powered vibration unit, and the schedule of the vibratory application was set on the control unit. The vibratory stimulus was set to occur when the electrical signal from the ISFD surpassed 15% of the maximum voluntary contraction level and was scheduled to be applied intermittently for 30 min, with 30-min intervals (Fig. 3).

Portable PSG recordings

PSG recordings were obtained with a portable device (Sleep Profiler, Advanced Brain Monitoring Inc.) (Fig. 4) [23] in the subjects' home environment, in order to evaluate sleep structure as well as SB. This device was a battery-powered recorder designed to acquire three frontopolar EEG signals between AF7 and AF8, AF7 and Fpz, and AF8 and Fpz; heart rate;

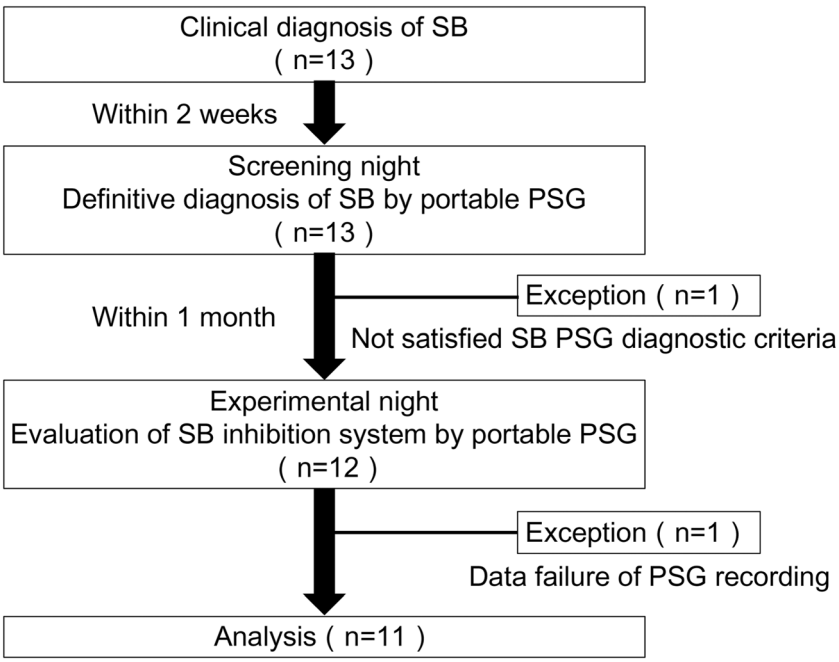

Fig. 1 Study protocol

snoring sound; pulse; head position; and EMG from the right masseter.

Sleep and SB scoring

Sleep data were scored automatically for each 30-s epoch by the analytical software attached to the PSG device (Sleep Profiler PSW012, Advanced Brain Monitoring Inc.) and the total sleep time, sleep efficiency, sleep latency, micro-arousal index, awakening index, and the percentage of time spent in each sleep stage were calculated. In addition, sleep questionnaires, which inquired about the number of awakenings during the night and quality of sleep by means of a 100-mm visual analogue scale (VAS, 0 indicating the best possible sleep quality, 100 indicating the worst possible sleep quality), were administered upon waking in the morning after both screening and experimental nights.

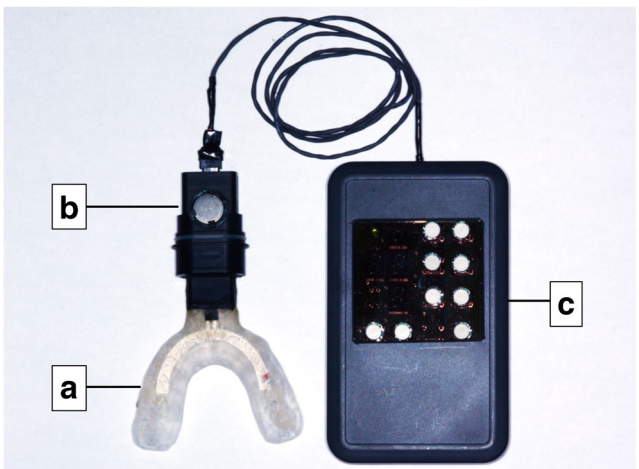

Fig. 2 Sleep bruxism (SB) inhibition system
Occlusal force signals detected by an intra-splint force detector (ISFD) (a) are sent to the vibration unit (b), which generates and applies vibration stimuli to the ISFD. The configuration of the vibratory application (intensity, duration, and timing) is set on the control unit (c).

The masseter EMG activity was utilized to evaluate SB episodes. First, all EMG amplitudes that exceeded approximately 10% of the maximum voluntary contraction were identified and then those that occurred during the awake state and that were associated with body movements, as determined by the software as well as by visual inspections by the scorer (YN), were excluded from the analysis. The remaining episodes were categorized into phasic episodes (three EMG bursts or more, each lasting 0.25–2.0 s), tonic episodes (EMG burst > 2.0 s), or mixed episodes (both types of bursts) [24] by the same scorer. The number of SB episodes per hour of sleep, the total SB duration (sum of the duration of all SB episodes) per hour of sleep, and the mean duration of SB episode (total SB duration/total number of SB episodes) were calculated for each subject for each night.

Statistical analysis

The number of SB episodes per hour (times/hour), the total SB duration per hour (seconds/hour), the mean duration of SB episode (seconds/episode), micro-arousal index, and the percentage of time spent in each sleep stage, with and without the vibration stimuli, on the experimental night were calculated. The sleep variables and the VAS score on the screening and experimental night were also calculated. The effect of the vibration stimuli on these values was tested using paired t tests ($p < 0.05$).

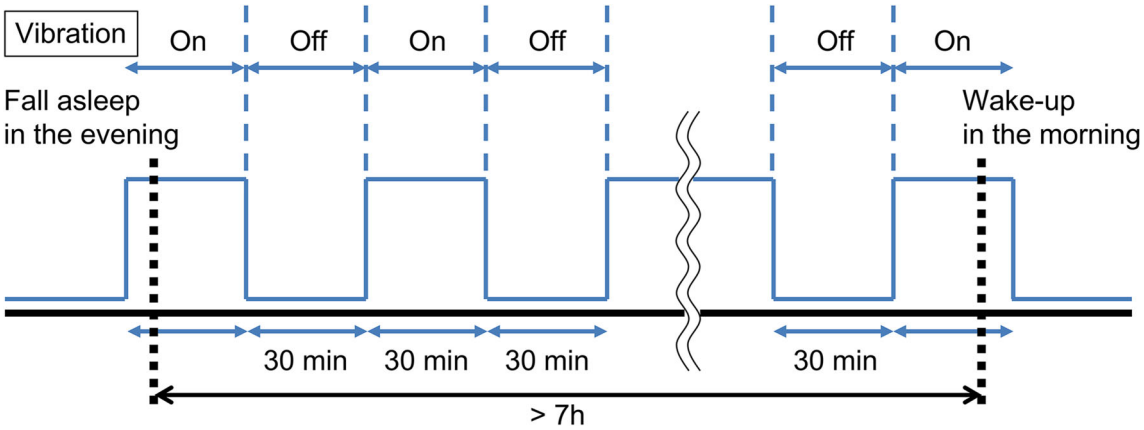

Fig. 3 Schedule of vibratory stimuli in the experimental night
Application schedule of vibratory stimuli was set so that vibrations were applied for 30 min, every other half hour, by the control unit.

Results

Two of the 13 subjects, who were initially recruited using clinical diagnostic criteria, were excluded from the study. One subject did not meet the PSG diagnostic criteria and the other had PSG recording failure (Fig. 1).

Sleep variables

All of the sleep variables gathered on the experimental night were within the normal ranges (Table 1). No subject reported being awakened during the experimental night. There was no significant difference in the VAS score between the screening and the experiment night (17.0 ± 14.5 vs 17.4 ± 18.2, p = 0.91). Furthermore, no significant difference was found in the sleep variables between the screening night and experimental night (Table 2). No substantial difference in sleep variables was found between periods with and without stimuli (Table 2).

SB variables

The mean total sleep time on the experimental night was 6.0 ± 0.8 h, where the vibratory stimuli were set to be applied for

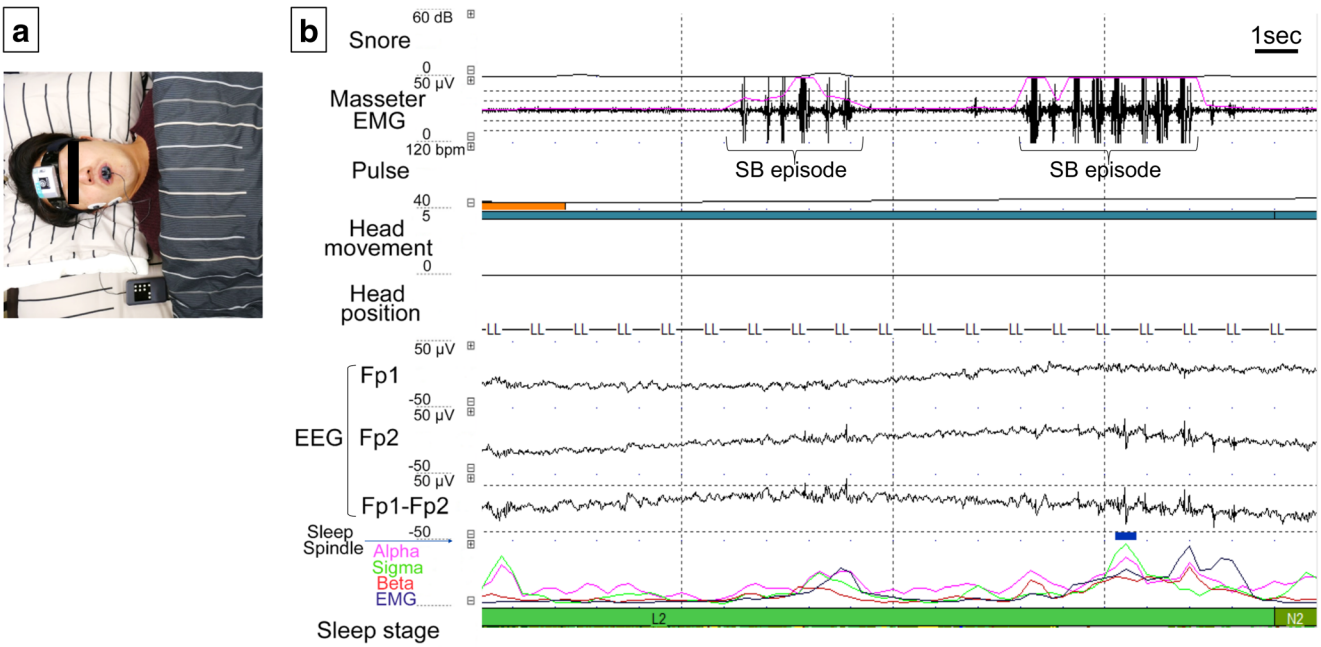

Fig. 4 Portable device and analytical software. Polysomnography recordings were conducted by using a portable device (Sleep Profiler, Advanced Brain Monitoring Inc.) (a). Sleep bruxism (SB) episodes were identified by visual inspection of the masseter EMG signal by a single scorer, with the help of attached software (b)
Pulse, upper: increase or decrease of the pulse rate compared with the average of last six pulsations (orange color), lower: sleep-awake status based on autonomic change (blue-green color: sleep, gray color: awake). Head movement, 0 (no movement) to 5 scale. Head position, S, supine; U, upstand; LL, lie left; LR, lie right; PL, prone left; PR, prone right. Sleep stage, L2, transition between N1 and N2.

Table 1 Comparison of sleep variables between the screening and the experimental night

	Screening night	Experimental night (with and without vibration)	*p* value
Total sleep time (min)	415 ± 100.4	339.8 ± 62.5	0.94
Sleep efficiency (%)	94.7 ± 4.9	92.3 ± 9.1	0.16
Sleep latency (min)	5.2 ± 5.1	4.7 ± 4.2	0.79
Micro-arousal index (times/h)	16.5 ± 10.9	17.5 ± 10.6	0.72
Awakening index (times/h)	1.9 ± 0.8	2.3 ± 1.1	0.12
Stage N1 (%)	6.2 ± 4.0	8.5 ± 7.2	0.26
Stage N2 (%)	54.7 ± 12.6	54.4 ± 14.9	0.93
Stage N3 (%)	19.9 ± 11.9	18.5 ± 8.6	0.64
Stage REM (%)	19.1 ± 7.0	18.3 ± 10.0	0.80

Data are presented as mean ± standard deviation

3.0 ± 0.4 h and not applied for 3.0 ± 0.4 h. The number of SB episodes per hour recorded during the period with vibration stimuli tended to be lower than those without these stimuli (3.0 ± 2.0 vs. 4.1 ± 2.8, $p = 0.05$); however, this trend did not reach statistical significance (Fig. 5). The same trend was found for the total SB duration per hour, and this result was statistically significant (14.3 ± 9.5 vs. 26.0 ± 20.0, $p = 0.03$) (Fig. 6). The mean duration of SB episodes during the period with vibratory stimuli decreased significantly in comparison with those without (4.7 ± 1.7 vs. 6.3 ± 2.4, $p = 0.03$) (Fig. 7).

Discussion

While the number of SB episodes was not significantly reduced by the vibratory stimulus, the total SB duration and the mean duration of SB episode were significantly reduced by 46% and 25%, respectively. Thus, the null hypothesis of this study was rejected and the study results suggested that the vibratory stimulus might be effective in reducing the total SB duration, but not the number of SB episodes. Biofeedback treatments that utilize event-triggered counter-stimulation are generally effective in controlling the duration, but not the occurrence of episodes [11], at least in one-night evaluations.

The literature has reported substantial fluctuation in SB levels across nights [25]. We here used intermittent vibratory feedback, where vibratory stimuli were applied every other half hour, instead of throughout the night, which allowed the comparison of stimulation periods with non-stimulation periods within the same night, and minimized the confounding effect of night-by-night fluctuation of SB.

The gold standard for SB monitoring is video-PSG recording. PSG recordings allow accurate evaluation of SB and sleep quality (or the presence of sleep disturbances), which is necessary for feedback stimulation studies, because such stimuli might detrimentally affect sleep quality. However, such studies generally require patients to sleep at a sleep laboratory, with the associated costs and technical complexity [20, 26]. Therefore, we utilized a portable PSG recording device (Sleep Profiler, Advanced Brain Monitoring Inc.), which allowed SB recordings and sleep monitoring in the subjects' home environment [23]. The results suggested that the vibration did not affect sleep structure or the frequency of micro-arousals substantially. This was also confirmed by the self-administered sleep questionnaires. However, while the automatic scoring of three-channel forehead EEG recordings will be helpful to understand sleep structure, these scorings provide less information

Table 2 Comparison of micro-arousal index and percentage of time spent in each sleep stage between periods with and without vibration stimuli during the experimental night

	With vibration	Without vibration	*p* value
Micro-arousal index (times/h)	15.8 ± 10.1	15.9 ± 11.7	0.97
Stage N1 (%)	7.3 ± 7.1	7.8 ± 6.6	0.86
Stage N2 (%)	48.7 ± 24.1	48.4 ± 17.3	0.97
Stage N3 (%)	13.7 ± 9.4	16.1 ± 7.6	0.38
Stage REM (%)	16.3 ± 13.8	12.6 ± 8.0	0.42
Total recording time (min)	186.0 ± 18.0	179.5 ± 24.8	0.20
Total number of threshold-exceeding episodes	17.6 ± 32.4	20.2 ± 18.3	0.41

Data are presented as mean ± standard deviation

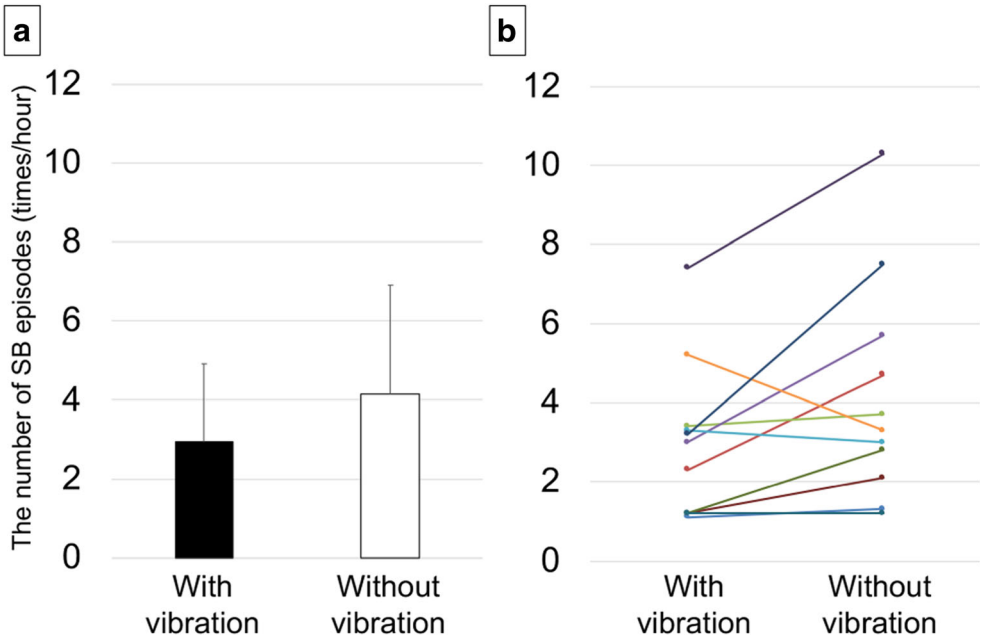

Fig. 5 Number of sleep bruxism episodes. Comparison of the number of sleep bruxism (SB) episodes per hour (times/hour) between the periods with and without vibration stimuli during the experimental night (a), individual value plots (b)

than manual scorings of full PSG recordings and may not be completely compatible with them; this should be regarded as one of the study limitations [23].

In our previous study, we utilized a stabilization splint with biofeedback vibratory stimuli to inhibit SB [12]. That study differed from the present study in that a rubberized strip (code switch), which included a pressure electro-conductive rubber, was placed on the splint and activated the electrical circuit upon detection of a vertical force of more than 300 g and reported significantly fewer SB episodes (25% reduction) and a shorter duration per episode (44% reduction). These findings were comparable with the current study results in that vibration stimuli had a higher impact on the duration of SB episodes than on the number of the SB episodes. A clear improvement of the current study over the previous study was that the number of study subjects was decided by sample size estimation, and they were selected based upon research diagnostic criteria for SB [20].

Other types of feedback stimuli have been investigated in the literature, generally showing weak-to-moderately positive

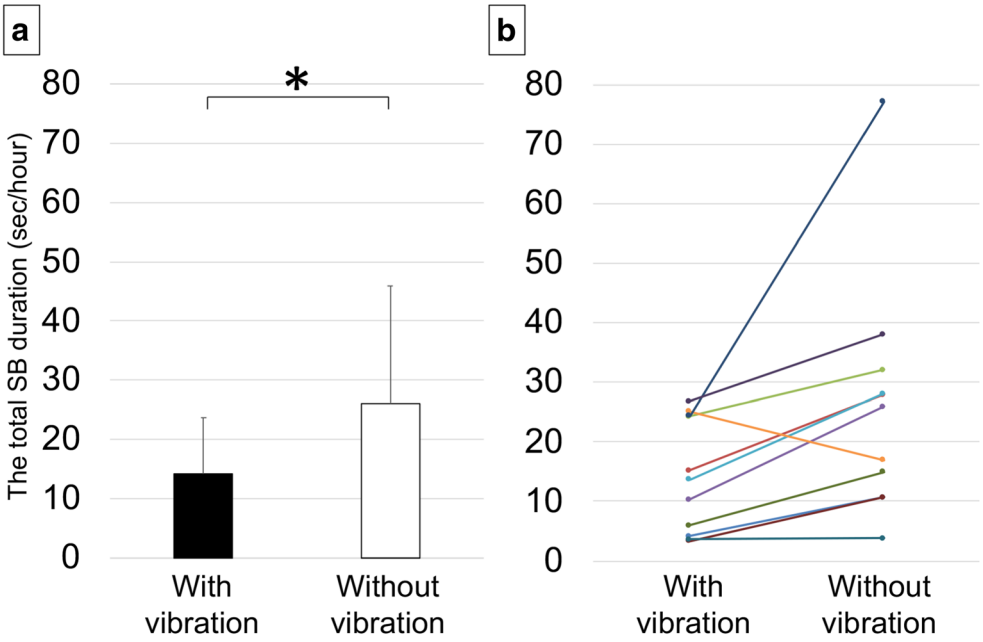

Fig. 6 Total sleep bruxism duration. Comparison of the total sleep bruxism (SB) duration per hour (seconds/hour) between the periods with and without vibration stimuli during the experimental night (a), individual value plots (b). *$p < 0.05$

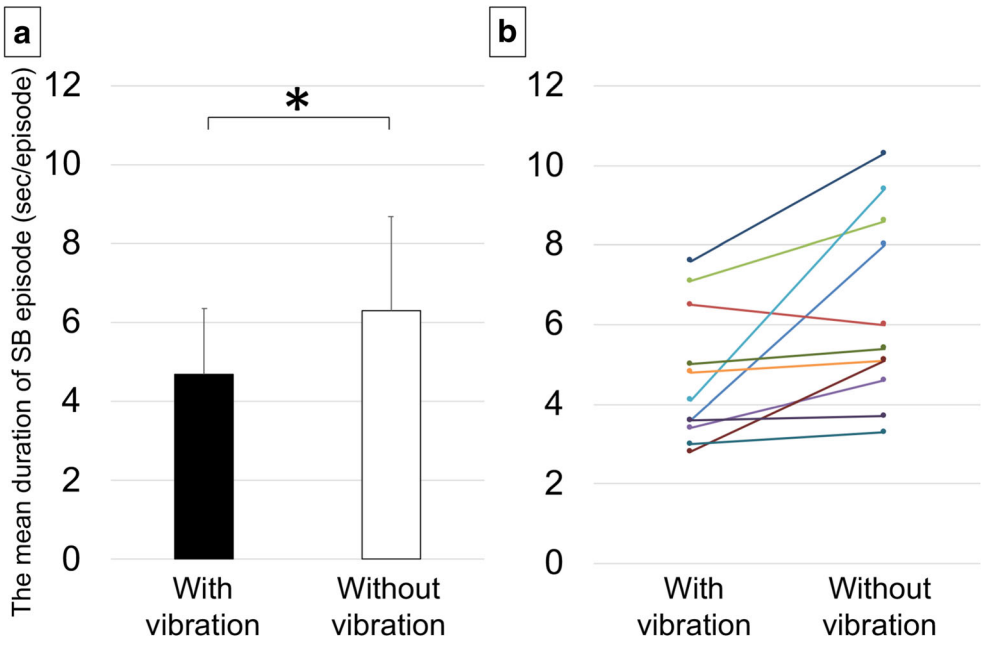

Fig. 7 The mean duration of sleep bruxism episode. Comparison of the mean duration of sleep bruxism (SB) episode (seconds/episode) between the periods with and without vibration stimuli during the experimental night (a), individual value plots (b). *$p < 0.05$

results (Table 3). Kardachi and Clarke [18] used contingent auditory feedback on nine subjects with SB and reported that this feedback did not reduce the number of SB events, but significantly reduced the duration and total SB activity. Rugh and Johnson [28] reported similar results using a similar EMG-activated contingent auditory feedback system in five SB patients. Finally, Pierce and Gale [29] evaluated masseter muscle activity during sleep in 100 SB patients before, during, and after five different experimental treatments. Both the EMG-measured frequency of SB episodes (50%) and the duration of SB activity decreased (50%) significantly with contingent auditory feedback therapy. However, contingent auditory feedback sometimes caused awakening or a sleep stage elevation and could also wake the sleep partner [17].

Additionally, low-level electrical shock of the trigeminal nerve has been used to control SB. Nishigawa et al. [11], who investigated electrical lip stimulation, reported significantly shorter individual SB episode duration, but not fewer SB episodes, similar to our results . Another study investigated electrical stimulation on the anterior temporalis muscle [10] and reported that the number of EMG episodes, which counted the number of EMG bursts and is therefore comparable with the SB episode duration, during sleep was significantly reduced (52 ± 12%) in myofascial temporomandibular disorder patients [10]. One of the important limitations of these studies was that they did not conduct PSG-based sleep monitoring, which is the gold standard method for evaluating SB, making the study results less reliable, as mentioned above. One laboratory-based PSG study investigated electrical stimulation on the anterior temporalis muscle and reported that the simulation did not cause major arousal responses in any sleep parameter, including SB-associated EMG episodes, in normal subjects [27].

Overall, independent of the type of stimulus used, such stimuli are consistently reported to be effective for controlling SB-associated EMG activity, but not for controlling SB completely, which agrees with our study results. Since it has been well documented that the majority of SB episodes occurs accompanied by micro-arousals [30], effects of feedback stimulation on the occurrence of SB episodes are expected to be limited, at least for the short term. In other words, biofeedback stimuli are triggered only after the start of SB episodes and therefore do not prevent their occurrence, but stop continuance of SB-related muscle activities (Table 3).

Since our system utilized the OS, the effects of the OS on SB should be considered. It has been well documented that the OS can potentially control SB for a short period of time [13]. Therefore, in order to identify the effects of vibration feedback, the confounding effects of the OS on the association between vibration feedback and SB needed to be minimized and controlled. For this purpose, intermittent vibratory stimuli were applied every other half hour and their effects were compared between these half-hour periods. In addition, it has been reported that the inhibitory effects of the OS are transient and that they do not last long [13]. In such cases, the use of the OS in combination with any type of biofeedback treatments that has potential to reduce SB would be justified, as the OS is useful for the protection of oro-dental structures from occlusal force related to SB. In this regard, our system facilitates better patient compliance than other types of biofeedback treatments, because patients are able to use the system as easily as they use the OS. In conclusion, within the limitations of this study, these study results suggest that intra-oral vibratory feedback has a weak suppressive effect on SB without disturbing sleep.

Table 3 Characteristics of biofeedback treatments

Articles [references]	Type of feedback simulation	Trigger for feedback	SB recordings	SB evaluation	Number of subjects	Application period	Results (% significant reduction)
Current study	Vibration	Occlusal splint, above 15% MVC threshold force	Portable PSG with masseter EMG channel	Number of SB episodes, total SB duration, mean duration of SB episode	11	1 night	Total SB duration, 46%; mean duration of SB episode, 25%
Watanabe et al. 2001 [12]	Vibration	Occlusal splint, above 300 g threshold force	Mechanical distortion detectable strip	Number of switch activation, duration of switch activation	1	4 months	Number, 25%; duration, 44%
Nishigawa et al. 2003 [11]	Electricity (lip)	Occlusal splint, above 220 g threshold force	Force detectable switch	Number of SB events, amplitude (% MVC), duration of SB event, and total time of all SB events	7	5 nights	Duration of SB event, 14%
Jadidi et al. 2011 [27]	Electricity (temporalis)	EMG activity above 10% or 20% MVC	Portable EMG of temporalis muscle	Number of EMG events per hour sleep based on a signal recognition algorithm	14	3 nights	No significant difference
Jadidi et al. 2013 [10]	Electricity (temporalis)	EMG activity above 10% or 20% MVC	Portable EMG of temporalis muscle	Number of EMG events per hour sleep based on a signal recognition algorithm	11	6 weeks	Number of EMG events/h, 52%
Kardachi and Clarke 1977 [18]	Sound	EMG activity above 100 microvolts	Masseter and temporalis EMG	Number of units (function of bruxing time and intensity)	7	more than 4 weeks	Number of units, 80%
Rugh and Johnson 1981 [28]	Sound	Masseter muscle EMG activity with at least 1-s duration above 20 microvolts	Portable masseter EMG	Number and duration of SB events and duration	5	8 nights	Duration, 39%
Pierce and Gale 1988 [29]	Sound	Frequency of bruxing episodes and duration of bruxing activity	Masseter EMG	Number of EMG bruxing episodes per hour, durations of EMG bruxing episodes per hour	100	2 weeks	Number of episodes and duration, about 50%

SB, sleep bruxism; *MVC*, maximum voluntary contraction; *PSG*, polysomnography; *EMG*, electromyography

Authors' contributions K Baba and H Nakamura designed the study. H Nakamura, M Takaba, Y Abe, and S Yoshizawa wrote the initial draft of the manuscript. Y Nakazato and Y Yoshida contributed to analysis and interpretation of data, and assisted in the preparation of the manuscript. All other authors have contributed to data collection and interpretation, and critically reviewed the manuscript. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Funding information This study was supported by Japan Society for the Promotion of Science KAKENHI Grant No. 16K20514, Grant-in-Aid for Young Scientists (B).

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee (Ethics Committee of Showa University School of Dentistry, No.2014-034) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Statement of informed consent Informed consent was obtained from all individual participants included in the study.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

1. Sateia MJ, Buysse DJ, Krystal AD, Neubauer DN, Heald JL (2017) Clinical practice guideline for the pharmacologic treatment of chronic insomnia in adults: an American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med 13:307–349
2. Abe S, Yamaguchi T, Rompre PH, De Grandmont P, Chen YJ, Lavigne GJ (2009) Tooth wear in young subjects: a discriminator between sleep bruxers and controls? Int J Prosthodont 22:342–350
3. Yoshida Y, Suganuma T, Takaba M, Ono Y, Abe Y, Yoshizawa S, Sakai T, Yoshizawa A, Nakamura H, Kawana F, Baba K (2017) Association between patterns of jaw motor activity during sleep and clinical signs and symptoms of sleep bruxism. J Sleep Res 26:415–421
4. Yoshizawa S, Suganuma T, Takaba M, Ono Y, Sakai T, Yoshizawa A, Kawana F, Kato T, Baba K (2014) Phasic jaw motor episodes in healthy subjects with or without clinical signs and symptoms of sleep bruxism: a pilot study. Sleep Breath 18:187–193
5. Lin WS, Ercoli C, Lowenguth R, Yerke LM, Morton D (2012) Oral rehabilitation of a patient with bruxism and cluster implant failures in the edentulous maxilla: a clinical report. J Prosthet Dent 108:1–8
6. Baba K, Haketa T, Sasaki Y, Ohyama T, Clark GT (2005) Association between masseter muscle activity levels recorded during sleep and signs and symptoms of temporomandibular disorders in healthy young adults. J Orofac Pain 19:226–231
7. Jokubauskas L, Baltrusaityte A, Pileicikiene G (2018) Oral appliances for managing sleep bruxism in adults: a systematic review from 2007 to 2017. J Oral Rehabil 45:81–95
8. Sakai T, Kato T, Yoshizawa S, Suganuma T, Takaba M, Ono Y, Yoshizawa A, Yoshida Y, Kurihara T, Ishii M, Kawana F, Kiuchi Y, Baba K (2017) Effect of clonazepam and clonidine on primary sleep bruxism: a double-blind, crossover, placebo-controlled trial. J Sleep Res 26:73–83
9. Manfredini D, Ahlberg J, Winocur E, Lobbezoo F (2015) Management of sleep bruxism in adults: a qualitative systematic literature review. J Oral Rehabil 42:862–874
10. Jadidi F, Castrillon EE, Nielsen P, Baad-Hansen L, Svensson P (2013) Effect of contingent electrical stimulation on jaw muscle activity during sleep: a pilot study with a randomized controlled trial design. Acta Odontol Scand 71:1050–1062
11. Nishigawa K, Kondo K, Takeuchi H, Clark GT (2003) Contingent electrical lip stimulation for sleep bruxism: a pilot study. J Prosthet Dent 89:412–417
12. Watanabe T, Baba K, Yamagata K, Ohyama T, Clark GT (2001) A vibratory stimulation-based inhibition system for nocturnal bruxism: a clinical report. J Prosthet Dent 85:233–235
13. Harada T, Ichiki R, Tsukiyama Y, Koyano K (2006) The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. J Oral Rehabil 33:482–488
14. Huynh N, Lavigne GJ, Lanfranchi PA, Montplaisir JY, de Champlain J (2006) The effect of 2 sympatholytic medications-propranolol and clonidine-on sleep bruxism: experimental randomized controlled studies. Sleep 29:307–316
15. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M (2008) Principles for the management of bruxism. J Oral Rehabil 35:509–523
16. Valiente Lopez M, van Selms MK, van der Zaag J, Hamburger HL, Lobbezoo F (2015) Do sleep hygiene measures and progressive muscle relaxation influence sleep bruxism? Report of a randomised controlled trial. J Oral Rehabil 42:259–265
17. Cassisi JE, McGlynn FD, Belles DR (1987) EMG-activated feedback alarms for the treatment of nocturnal bruxism: current status and future directions. Biofeedback Self Regul 12:13–30
18. Kardachi BJ, Clarke NG (1977) The use of biofeedback to control bruxism. J Periodontol 48:639–642
19. Dube C, Rompre PH, Manzini C, Guitard F, de Grandmont P, Lavigne GJ (2004) Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. J Dent Res 83:398–403
20. Rompre PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ (2007) Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 86:837–842
21. Baba K, Clark GT, Watanabe T, Ohyama T (2003) Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain 17:58–64
22. Takeuchi H, Ikeda T, Clark GT (2001) A piezoelectric film-based intrasplint detection method for bruxism. J Prosthet Dent 86:195–202
23. Levendowski DJ, Ferini-Strambi L, Gamaldo C, Cetel M, Rosenberg R, Westbrook PR (2017) The accuracy, night-to-night variability, and stability of frontopolar sleep electroencephalography biomarkers. J Clin Sleep Med 13:791–803
24. Lavigne GJ, Rompre PH, Montplaisir JY (1996) Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 75:546–552
25. Lavigne GJ, Guitard F, Rompre PH, Montplaisir JY (2001) Variability in sleep bruxism activity over time. J Sleep Res 10:237–244
26. Dutra KM, Pereira FJ Jr, Rompre PH, Huynh N, Fleming N, Lavigne GJ (2009) Oro-facial activities in sleep bruxism patients and in normal subjects: a controlled polygraphic and audio-video study. J Oral Rehabil 36:86–92
27. Jadidi F, Norregaard O, Baad-Hansen L, Arendt-Nielsen L, Svensson P (2011) Assessment of sleep parameters during contingent electrical stimulation in subjects with jaw muscle activity during sleep: a polysomnographic study. Eur J Oral Sci 119:211–218

28. Rugh JD, Johnson RW (1981) Temporal analysis of nocturnal bruxism during EMG feedback. J Periodontol 52:263–265
29. Pierce CJ, Gale EN (1988) A comparison of different treatments for nocturnal bruxism. J Dent Res 67:597–601
30. Kato T, Rompre P, Montplaisir JY, Sessle BJ, Lavigne GJ (2001) Sleep bruxism: an oromortor activity secondary to micro-arousal. J Dent Res 80:40–44