ORIGINAL ARTICLES

Comparison of the inhibitory effects of oral appliances on sleep bruxism: A pilot study

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Sleep bruxism (SB) is believed to be a masseter muscle activity caused by the central nervous system, thus distinguishing it from diurnal bruxism, which is thought to be an acquired behavior. Various local factors have been associated with the onset of SB, but a direct relationship with occlusion has been ruled out, whereas psychological stress is typically believed to be closely involved. The present study monitored both bruxist and non-bruxist subjects to observe their SB profiles and then compare and investigate the SB-inhibiting effects of several removable oral appliances. The subjects were teachers, undergraduate students, and postgraduate students from the Asahi University School of Dentistry. Informed consent was obtained from all subjects after carefully explaining the study aims and details. This study was conducted with the approval of the Asahi University School of Dentistry is Ethical Review Board (approval no. 22095). A comparison of the incidence of bruxism events when not wearing an oral appliance and when wearing the 3 types of appliances (thin plate, thick plate, SP) showed that the incidence decreased when wearing any of the 3 appliances relative to when no appliance was worn. Furthermore, a comparison of the event incidences of the 3 appliances demonstrated that the thin plate had the highest event incidence.

Key words : Sleep bruxism, oral appliances, EMG

Introduction

Sleep bruxism (SB) is believed to be a masseter muscle activity caused by the central nervous system¹⁾, thus distinguishing it from diurnal bruxism, which is thought to be an acquired behavior. According to Kato et al.²⁾, surveys conducted in the USA, Europe, and Asia show that the incidence of SB based on subjective awareness of tooth-grinding sounds declines with age, from 10–20% in children to 5–8% in adults and 2–3% in the elderly. However, a study by Attanasio claims that 85–90% of the population has SB³⁾, but only a minority is aware of it, and most cases do not involve grinding sounds.

Depending on its severity, SB with these onset characteristics can cause various disorders in the organs of the stomatognathic system⁴⁾ and has also been identified as an important contributing factor to temporomandibular joint disorder (TMJD)⁵⁾. An epidemiological study by Agerberg et al.⁶⁾ found that people who experienced SB often had functional pain in the temporomandibular joint (TMJ) and muscles, while a study of patients complaining of TMJ and craniofacial pain by Fricton et al.⁷⁾ reported a strong link between clenching and grinding.

Various local factors have been associated with the onset of SB, but a direct relationship with occlusion has been ruled out⁸⁾, whereas psychological stress is typically believed to be closely involved. In any case, the prevailing view is that SB is not independently caused by these factors, but rather by the complex interrelation of a variety of determinants⁹⁾. A physiological role in the form of stress release has also been ascribed to SB¹⁰⁾, and there is currently no means to eradicate this. As such, our facility provides a range of symptomatic therapies to prevent and

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mitigate damage to the stomatognathic system when SB is suspected, and it has garnered considerable recognition for the effects of fitting stabilization splints. Recently, however, a study by Minagi et al.¹¹⁾ described the effects of a plate-type oral appliance designed to fill the entire palatal concavity without involving occlusion. Nevertheless, the SB-inhibiting effect of these oral appliances has yet to be properly clarified¹¹⁻¹⁵.

The present study monitored both bruxist and nonbruxist subjects to observe their SB profiles and then compare and investigate the SB-inhibiting effects of several removable oral appliances.

Methods

1. Subjects

The subjects were teachers, undergraduate students, and postgraduate students from the Asahi University School of Dentistry who had no missing teeth or mental disorders and who did not take medication on a regular basis. The bruxist group consisted of 8 men who were aware of their SB or who met at least one of the following inclusion criteria; (1) had been made aware of tooth-grinding sounds by another person; (2) had abnormal dental attrition or shiny spots on restorations; (3) experienced TMJ pain or discomfort upon waking; (4) felt fatigue or pain in the masseter muscles; or (5) had masseteric hypertrophy^{16,17)}. The non-bruxist group, on the other hand, consisted of 3 men who did not meet any of the above criteria (1)-(5).

Informed consent was obtained from all subjects after carefully explaining the study aims and details. This study was conducted with the approval of the Asahi University School of Dentistry's Ethical Review Board (approval no. 22095).

2. Data monitoring equipment & collection

Vital signs during sleep were monitored and recorded using the method described in Fig. 1 below. Since bruxism occurs due to occlusal muscle activity, muscle activity in the central portion of the masseter muscle was measured. Body movements were also measured to distinguish between brain waves used

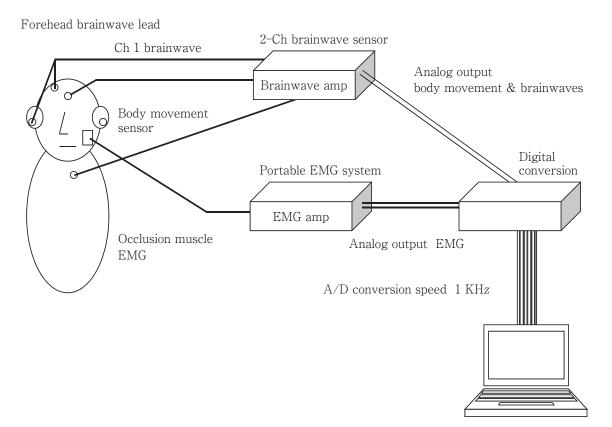


Fig. 1 Vital sign measurement and data collection

to determine sleep onset (hypnagogia) and masseter muscle activity indicating SB onset. Although there was a risk of misinterpreting masseter activity during swallowing as a bruxism event (herein 'event'), a preliminary experiment confirmed that masseter activity during swallowing did not exceed 20% of the maximum voluntary contraction (MVC) level in any of the subjects. Masseter activity was determined with electromyography (EMG) by placing an electrode on the central part of the left masseter muscle so as to be parallel to the muscle fibers¹⁸⁾. Brain wave activity was determined with electroencephalography (EEG) by attaching the indifferent electrode to the forehead and the reference electrodes to both earlobes (A1), as well as the frontal pole (Fp1), and using a 2-channel brainwave sensor (DL-160). Body movements were detected as acceleration signals converted to voltage using a piezoelectric element attached to the manubrium. Vital signs were converted from analog to digital (A/D) signals using a data collection system (UAS-108S, Unique Medical Co., Ltd., Tokyo, Japan) at a sampling frequency of 1 kHz and then recorded and analyzed using a personal computer.

Data collection was performed in the homes of each subject. Subjects were instructed to measure muscle activity on the measurement days by placing the designated electrodes on the same part of the body¹⁹⁾. The subjects received an explanation on how to place the electrodes and operate the measurement device and were required to practice until they were proficient. They were also instructed to perform the measurements on days when their physical and mental state was close to normal, rather than during times of anxiety, stress, or frustration. Furthermore, to prevent sleep interference by the attached electrodes and leads connected to the device for recording vital signs during events²⁰⁾, the subjects were required to familiarize themselves with the device by practicing how to use it 3 days prior to measurement for a period of at least 2 days.

3. Oral appliances

The maxillary oral appliances worn by the subjects during sleep are shown in Fig. 2.

 Thin palatal plate (thin plate): Made of a quickcure resin covering the entire palate at a thickness of 1.5 mm, the thin plate was anteriorly and laterally aligned with the palatal cervical margin of the subjects' teeth, and its posterior part was made consistent with the line connecting the distal region of the rear-most molar crowns.

- (2) Thick palatal plate (thick plate): Based on the appliance described by Minagi et al.¹¹⁾, the thick plate was made of a quick-cure resin covering the entire area from the deepest part of the palate to the cervical region and using the same positions as that of the thin plate.
- (3) Splint (SP): This appliance was a stabilization splint covering the edge-to-edge bite with a 1.5-2.0-mm resin that brought the mandibular buccal cusp into occlusal contact with a 0.5-0.7-mmdiameter contact area so as to be guided by the front teeth and canines and to separate the molar region during anterior and lateral movement of the mandible. In addition, the three types of oral appearances were randomly attached to the subjects.

4. Analysis parameters

1) Identifying bruxism events

The root mean square (RMS) of the EMG's source waveform was calculated using a time constant of 60 msec. Each subject was instructed to perform a maximum voluntary bite for 2 seconds 5 times prior to measurement, and the mean of the maximum EMG amplitude was regarded as 100% MVC²¹⁾. A bruxism event was defined as muscle activity that exceeded 20% of this MVC muscle activity level and continued for at least 0.25 seconds. Artifacts deemed to be obvious body movements and noise based on the EMG source waveform were excluded from the data.

2) Event measurement variables

Events identified based on the above criteria were measured in terms of the following variables:

- (1) Event incidence per hour of sleep (events/hour);
- (2) EMG level during each event onset;
- (3) Duration of each event (sec).

5. Experimental procedure & tasks

Experiment 1 investigated the event profiles of 3 bruxism and 3 non-bruxism subjects. The selected subjects were required to collect the data at their homes for 3 nights, and the masseter EMG results (event incidence, EMG level at event onset, and

Thin plate Resin-based palatal plate with 1.5-mm palatal thickness anteriorly and laterally aligned with the teeth and posteriorly consistent with the line connecting the distal
region of the rearmost teeth
 Thick plate Resin-based palatal plate covering the entire area from the deepest part of the palate to the cervical region using the same positions as the thin plate.
SP Resin-based maxillary stabilization splint
Fig. 2 Oral appliances

duration), as well as the correlation between EMG level and duration, were compared between the 2 groups.

Experiment 2 compared the SB-inhibiting effects of the 3 oral appliances (thick plate, thin plate, and SP) based on event incidences when the appliances were worn and when they were removed. This experiment selected 2 of the bruxist group subjects from experiment 1 who had the highest bruxism event incidence. The event incidence data from each device were collected for each subject on 2 different days at least 2 weeks apart.

Experiment 3 gathered data on the thick plate and SP appliances (which had a significant SB-inhibiting effect in experiment 2) from 5 bruxists on 2 different days and compared them according to event incidence, EMG level at event onset, and correlation between EMG level and duration.

6. Analytical procedure

Experiment 1 analyzed the event incidences of the

bruxist and non-bruxist groups using Welch's t-test. Intergroup comparison of EMG levels at event onset and duration was done using the Mann-Whitney U-test, and interday comparison of individual subjects was performed using the Kruskal-Wallis test followed by multiple comparison testing using the Steel-Dwass method. The correlation between the EMG level and duration was analyzed using Spearman's rank correlation test.

In experiment 3, event incidence was analyzed using 'between-subjects' and 'among-3-applianceconditions' (i.e. no appliance, thick plate, SP) 2-way repeated measures ANOVA. This was followed by Bonferroni multiple comparison testing. The EMG levels at event onset and duration were analyzed using the Kruskal-Wallis test followed by multiple comparison testing using the Steel-Dwass method. Furthermore, the correlation between the EMG level and duration was analyzed using Spearman's rank correlation test.

Results

1. Experiment 1

1) Event incidence

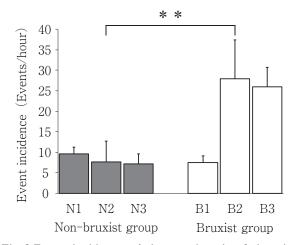
The average event incidence per hour of sleep (3 days) was 7.2–9.7 times in the non-bruxist group and 7.6–28.0 times in the bruxist group (Fig. 3).

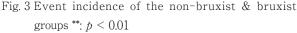
Analysis of event incidence using Welch's t-test revealed that the bruxist group had significantly more events (20.5 \pm 11) than the non-bruxist group (8.2 \pm 3.1) (p < 0.05).

2) Muscle activity during event onset

(1) EMG level

The EMG muscle activity level of each subject during event incidence is shown in histograms prepared based on a class-interval of 10% MVC (Fig. 4).





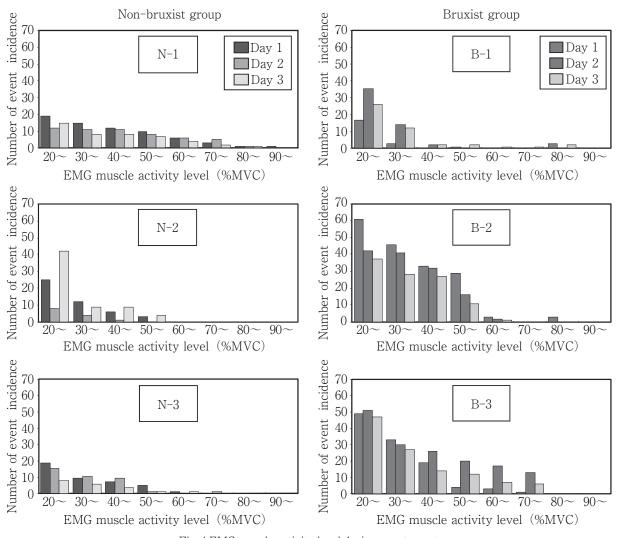


Fig. 4 EMG muscle activity level during event onset

Events in both the non-bruxist group and bruxist group occurred most often at 20–30%MVC, with the number of events tending to decline as the EMG level increased. While 1 subject in the non-bruxist group 0 (N-1) showed a high level of muscle activity on day 1 at 90–100% MVC, even subjects in the bruxist group, such as B–1 on day 1, had a relatively low level of muscle activity at \leq 50% MVC. The results of the Mann-Whitney U test did not indicate any significant intergroup difference in EMG levels at event onset. Kruskal-Wallis testing of between-day variation of individual subjects revealed a significant difference in 2 bruxist subjects (B–1 and B–2) (p < 0.05).

(2) Duration

The duration of muscle activity of each subject during event incidence is shown in histograms prepared based on a class-interval of 0.5 seconds (Fig. 5).

The most common duration of muscle activity in both the non-bruxist group and bruxist group was 0.25-0.75 seconds, with muscle activity subsequently declining as duration increased. The Mann-Whitney test results showed no significant intergroup difference. Kruskal-Wallis testing of between-day variation of individual subjects showed a significant difference in 2 bruxists (B-1 and B-2), as for the EMG levels (p < 0.05). 3) Correlation between duration of muscle activity during event onset and EMG levels. The correlation between duration of muscle activity during event onset and EMG level is shown for each subject in Fig. 6.

The results of Spearman's rank correlation test demonstrated that the correlation coefficient r was distributed between 0.32 and 0.45 in the non-bruxist

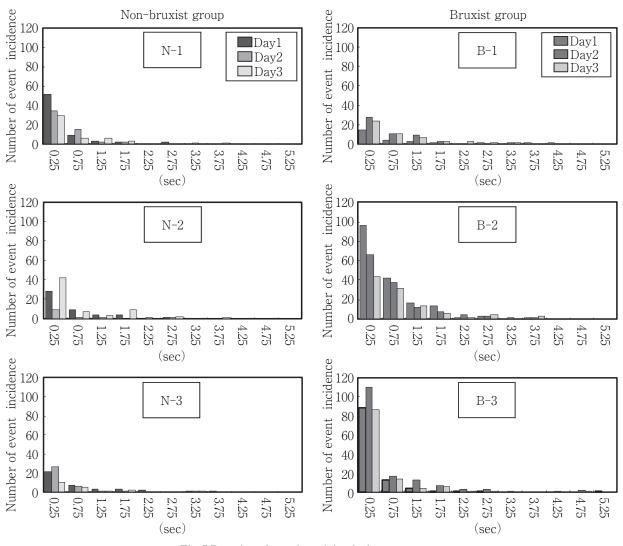


Fig. 5 Duration of muscle activity during event onset

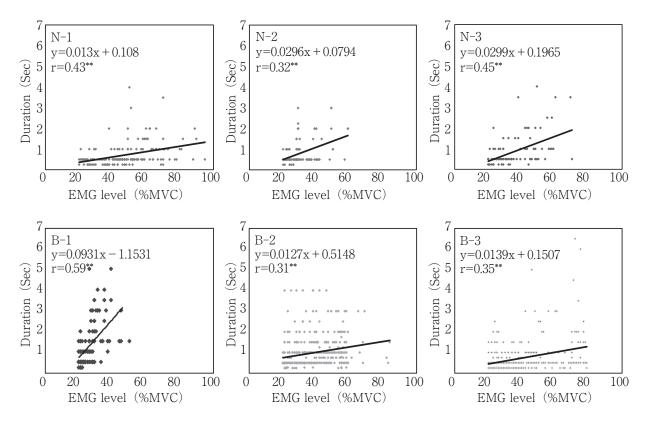


Fig. 6 Correlation between muscle activity duration and EMG level **: p < 0.01

group and between 0.31 and 0.59 in the bruxist group, all of which were significant.

2. Experiment 2

1) Event incidence

The event incidences (events/hour) when not wearing any oral appliance and when wearing the 3 types of appliances are shown for each subject in Fig. 7.

The average event incidence was greatest when no appliance was worn (30 events/hour), followed by the thin plate (22.1 events/hour), SP (18.9 events/hour), and thick plate (10.1 events/hour).

3. Experiment 3

The findings of experiment 2 demonstrated that wearing the thick plate or SP significantly reduced event incidence compared to when no appliance was worn and had a considerable SB-inhibiting effect; this was further examined.

1) Event incidence

The average event incidence (events/hour) of the 3 measured instances is shown for each subject in Fig. 8. The event incidence was 18.9–46 events/hour when

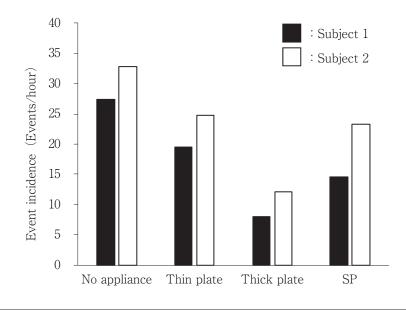
not wearing any appliance, 4.8–13.9 events/hour for the thick plate, and 11.4–29.5 events/hour for the SP. The results of 2–way ANOVA using the subjects and appliance conditions as factors revealed significant differences for both factors (p < 0.01). Meanwhile, multiple comparison testing between appliances showed that thick plate and SP event incidences were significantly lower than that of no plate, and that the event incidence was significantly lower with the thick plate than with the SP (p < 0.05).

2) EMG level

The EMG muscle activity level of each subject during event incidence is shown for each appliance condition (no appliance, thick plate, SP) in histograms prepared based on a class-interval of 10% MVC (Fig. 9). Event incidence was greatest for all 3 appliance conditions at 20-30% MVC, with the number of events tending to decline as the EMG level increased. The Kruskal-Wallis test results failed to reveal any significant differences among the 3 appliance conditions.

3) Duration

Duration of muscle activity during bruxism



		No appliance	Thin plate	Thick plate	SP
Subject 1	Dayl	31.78	18.55	10.60	14.22
	Day2	22.89	20.44	5.45	14.89
Subject 2	Day1	38.89	26.22	10.00	23.56
	Day2	26.60	23.33	14.22	23.11

Fig. 7 Event incidence of each appliance condition, and raw data

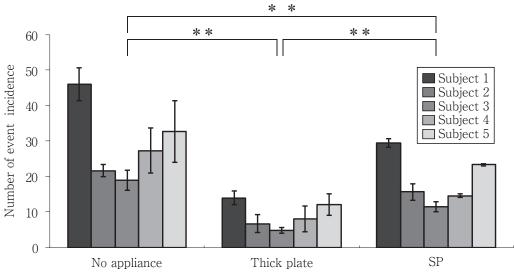


Fig. 8 Event incidence with 3 oral appliances **: p < 0.01

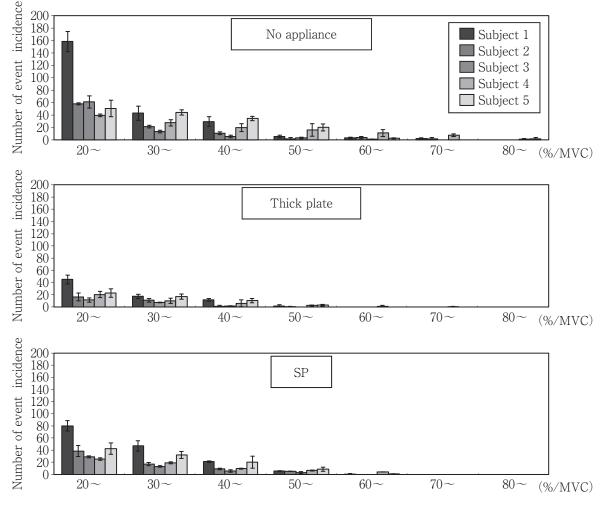


Fig. 9 EMG muscle activity level during event onset

event onset is shown for each subject in histograms prepared based on a class-interval of 0.5 seconds (Fig. 10). Bruxism events occurred most frequently for all 3 appliance conditions at a duration of 0.25-0.75 seconds, with the number of events subsequently decreasing as the duration increased.

The Kruskal–Wallis test results revealed significant differences in event duration among the 3 appliance conditions (p < 0.01), while the Steel–Dwass multiple comparison test results indicated that event duration was significantly shorter when wearing the thick plate or SP compared to when not wearing any appliance (p < 0.01).

4) Correlation between duration of muscle activity during event onset and EMG levels. The correlation between duration of muscle activity during event onset and EMG level is shown for each appliance in Fig. 11. The correlation coefficients of each appliance condition were similar at r = 0.539 for no appliance, r = 0.530 for the SP, and r = 0.507 for the thick plate.

Discussion

The results of experiment 1 showed that event incidence was significantly higher in the bruxist group than in the non-bruxist group, but the fact that many bruxists in the present study experienced fewer events than those reported by Mikami et al.²²⁾ can be attributed to the different criteria used by the respective studies to determine bruxism, and the current lack of clear-cut criteria for diagnosing the disorder. There was also a considerable variation in SB incidence even in the bruxist group, with the low-incidence bruxists experiencing virtually the same number of events as their non-bruxist group

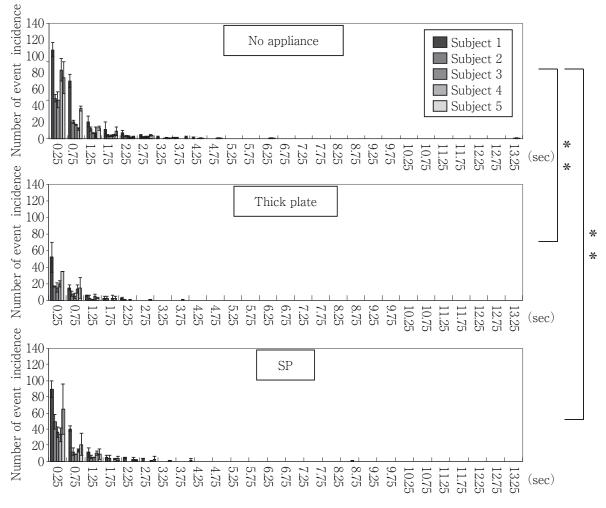


Fig. 10 Duration of muscle activity during event onset **: p < 0.01

counterparts. This finding supports the assertion of Rugh et al.²³⁾ that bruxists do not necessarily exhibit constant SB behavior every night but are instead prone to intermittent bouts of severe bruxism; this suggests that the measurements were likely conducted at a time when the subjects' SB incidence happened to be low.

The absence of intergroup (i.e. bruxist group versus non-bruxist group) differences in bruxism EMG levels and duration in the present study differed from the findings of a study by Ichiki et al.²⁴⁾ wherein the duration and amount of muscle activity varied between the bruxist group and the non-bruxist group without any observed difference in the number of bruxism events.

As already stated, this can also be ascribed to differences between the studies in terms of their respective event definitions, assessment criteria, and diagnostic standards²⁵⁾. Meanwhile, a look at the individual subjects reveals that some had very high EMG muscle activity levels during event onset (80-90% MVC) despite belonging to the non-bruxist group, while some had relatively low levels of muscle activity despite belonging to the bruxist group. This suggests that the non-bruxists may have engaged in frequent bruxism characterized by clenching of the jaw in the vicinity of the intercuspal position despite not being aware of it or exhibiting any other signs of the disorder²⁶⁾. On the other hand, the fact that bruxist subjects had masseter muscle fatigue upon waking despite low bruxism frequency and EMG levels suggests that bruxers with low muscle contraction frequency are more likely to complain of masseter muscle pain and fatigue than those with high muscle

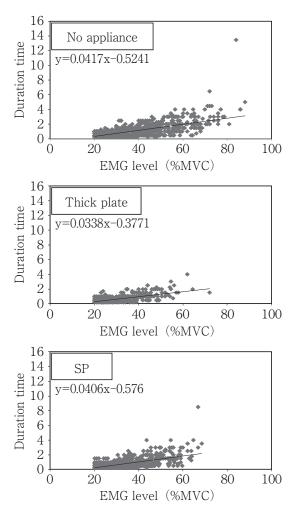


Fig. 11 Correlation between duration and EMG level collection **: p < 0.01

contraction frequency, as reported by Rompre et al.²⁷⁾.

Next, the study findings showed that the EMG level during event onset and duration of muscle activity differed between measurement days for some bruxist group subjects but not for others. A study by Clark et al.²⁸⁾ also discovered day-to-day fluctuations in the EMG levels of bruxism patients monitored for 10 consecutive days, while a study by Reding et al.²⁹⁾ found day-to-day fluctuations in bruxism muscle activity and described the need for daily monitoring. Furthermore, a study by Baba et al.³⁰⁾ that measured forceful tooth-to-splint contacts of bruxers for 5 consecutive nights found that the average duration of bruxism events per hour varied from day to day by 200% or more.

On the other hand, if we extrapolate Rugh et al.'s³¹⁾ finding that bruxism is strongly influenced by daily

mental stress, the lack of day-to-day fluctuations in muscle activity among some patients may have been due to them having performed the measurements on days when their physical and mental state was close to normal and free of anxiety, stress, or frustration.

Moreover, the correlation between EMG level and duration of muscle activity during event onset was significantly positive for all subjects. Okura et al.³²⁾ also reported a correlation between bruxism event duration and peak %MVC, and that absolute muscle activity increased in conjunction with prolonged bruxism events. Ware et al.³³⁾ asserted that defensive reactions to harmful stimuli generally decline during REM-stage sleep, so that although muscle contractions should not occur, bruxism also happens during REM sleep and is likely to exert a destructive effect on the body. Taking all of these points into account, we can surmise that an increase in prolonged REM-stage events or high EMG level events has a major impact on stomatognathic tissues, given the correlation between the two.

In experiment 2, although only 2 subjects were monitored, the oral appliance with the greatest SBinhibiting effect was the thick plate, followed by the SP, while the thin plate was the least effective. Similarly, a study by Minagi et al.¹¹⁾ on the therapeutic effects of palatal appliances of varying thicknesses on patients with myofascial pain reported that a thick palatal appliance corresponding to the thick plate of the present study was the most effective.

As in experiment 1, the incidence of SB events for the 5 bruxist subjects in experiment 3 when not wearing any oral appliance (18.9-46.0 events/hour) was often lower than the bruxist group incidences reported by Lavigne et al.²⁵⁾ and Mikami et al.²²⁾ Although this is conceivably due to the abovementioned reasons, there is a need to clarify the criteria for evaluating bruxism, as well as the definition of bruxers.

In any case, bruxers clearly have a higher incidence of bruxism events than their non-bruxer counterparts, and the resulting impact on their stomatognathic system cannot be overlooked.

Clinical treatment of bruxism typically involves splint therapy of the occlusal surfaces. However, while a stabilization splint that inhibits SB¹⁵⁾ reportedly reduces nocturnal muscle activity, it is not necessarily effective in all patients¹²⁾. A separate study by Harada et al. found that stabilization splints are effective in treating bruxism, but the effects decreased over time³⁴⁾. A systematic review by Fricton on the types of symptoms that intraoral splints are effective in treating confirmed that stabilization splints were particularly effective in remedying strong temporomandibular disorder (TMD) pain³⁵⁾. In addition, a study by Baba et al.³⁶⁾ reported that stabilization splint therapy cannot eliminate bruxism but can at least protect the teeth from attrition and reduce TMJ pressure, which suggests that it is currently the first-line treatment for SB in TMD patients.

Accordingly, the effects of stabilization splints in treating TMD have generally been acknowledged in recent years.

On the other hand, a study by Greene et al.¹⁴⁾ found that wearing a palatal splint with no occlusal contact elicited a complete response in 40% of myofascial pain syndrome patients, while a study by Minagi et al.¹¹⁾ reported that a palatal splint that filled the entire palate was very effective in inhibiting nocturnal occlusal muscle activity. However, there are also studies that reject these purported effects¹³⁾, and although some previous studies deny the benefits of both stabilization and palatal splints, the results of the present study imply that both the thick plate and SP are effective in inhibiting bruxism.

Ichiki et al.³⁷⁾ investigated the effects of a stabilization splint and a control splint that does not cover the occlusal surfaces on bruxism and found that wearing either splint was effective in reducing bruxism in all patients, regardless of their shape.

The present study did not find any significant differences between the thick plate and SP in terms of EMG levels and duration of muscle activity, but event incidence was clearly lower when wearing the thick plate than when wearing the SP, suggesting that the thick plate had a greater SB-inhibiting effect than the SP appliance.

Incidentally, the fitted SP inhibits bruxism by stretching the occluded muscle spindles during biting compared to when no appliance is worn. Even if the contraction force of occluded muscles is equal, the amount of muscle information transmitted to the brain presumably increases, making it possible to acquire the same amount of kinesthetic information that would be expected from a weaker bite³⁸⁾. Moreover, it is conceivable that the thick plate reduced bruxism by inhibiting the mandibular movements accompanying the disorder as a result of diminished tongue activity owing to stimulation of the back of the tongue by the plate's base portion, which is in turn caused by narrowing of the tongue space³⁹⁾.

Conclusions

The following 5 conclusions can be drawn as a result of examining the effects of 3 types of oral appliances in inhibiting sleep bruxism.

The incidence of bruxism events (events/hour) was higher in the bruxist group than in the non-bruxist group, but there was no significant difference between the 2 groups in terms of EMG level and duration of muscle activity during event onset.

A comparison of the incidence of bruxism events when not wearing an oral appliance and when wearing the 3 types of appliances (thin plate, thick plate, SP) showed that the incidence decreased when wearing any of the 3 appliances relative to when no appliance was worn. Furthermore, a comparison of the event incidences of the 3 appliances demonstrated that the thin plate had the highest event incidence.

Comparing the incidence of bruxism events when not wearing an oral appliance and when wearing an oral appliance (thick plate, SP) revealed a decline in the event incidence with both appliances relative to when no appliance was worn. Furthermore, comparing the event incidences of the 2 appliances indicated that the thick plate had a lower event incidence than the SP. There was no difference between the appliance conditions in terms of EMG levels during event onset, and the duration of muscle activity was shorter when wearing the thick plate or SP than when not wearing any appliance.

Significant correlations between EMG level and duration of muscle activity during bruxism event onset approaching 0.5 were observed for all oral appliance conditions (i.e. no appliance, thick plate, SP).

Refarece

 Lobbezoo F and Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. 2001; 28: 1085–1091.

- 2) Kato T and Lavigne GJ. Sleep bruxism: A sleep-related movement disorder. *Sleep Med Clin*. 2010; 5: 9–35.
- Attanasio R. An overview of bruxism and its management. *Dent Clin North Ame*. 1997; 41: 229–241.
- Minoru Ai. Jaw dysfunction-approach from occlusion. Tokyo: Ishiyaku Publications; 1983: 197–198.
- Yoshinori Kobayashi. The role of Bruxism in the development of so-called temporomandibular disorders. Dental journal. 1989; 29: 37–52.
- Agerberg G and Carlsson GE. Functional disorders of the masticatory system. II. Symptoms in relation to impaired mobility of the mandible as judged from investigation by questionnaire. *Act Odont Scand.* 1973; 31: 335–347.
- Fricton JR, Chung SC, Kroening RJ and Hathaway KM. TMJ and craniofacial pain: Diagnosis and management. St Louis · Tokyo: *Ishiyaku EuroAmerica*, *Inc*; 1988: 27–28.
- Ash MM. Paradigmatic shifts in occlusion and temporomandibular disorders. *J Oral Rehabil*. 2001; 28: 1–13.
- 9) Kazumi Baba. How to understand bruxism, Definition/ diagnosis/morbidity/measurement method/ pathology, From past studies and Dr. Clark's studies. Quintessence. 1997; 16: 55-67.
- 10) Sadao Sato, Katsuji Tamaki, Koji Sakakibara. The clinical cause of bruxism and its clinical response Chapter 2 Physiological significance of bruxism. Tokyo: Quintessence Publishing; 2009: 14–21.
- 11) Minagi S, Shimamura M, Sato T, Natsuaki N and Ohta M. Effect of a thick palatal appliance on muscular symptoms in craniomandibular disorders: a preliminary study. *J Cranio-mandib Pract*. 2001; 19(1): 42-47.
- 12) Clark GT, Beemsterbore PL and Rugh JD. Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. *J Oral Rehabil.* 1981; 8: 279– 286.
- 13) Dao TT, Lavigne GJ, Charbonneau A, Feine JS and Lund JP. The efficacy of oral splints in the treatment of myofascial pain of the jaw muscles: a controlled clinical trial. *Pain*. 1994; 56: 85–94.
- 14) Greene CS and Laskin DM. Splint therapy for the myofascial pain dysfunction (MPD) syndrome: a comparative study. J Am Dent Assoc. 1972; 84: 624–628.
- 15) Huynh N, Manzini C, Rompre PH and Lavigne GJ. Weighing the potential effectiveness of various treatments for sleep bruxism. *J Can Dent Assoc.* 2007; 73: 727-730.
- Thorpy MJ. The International classification of sleep disorders. ASDA. 1990: 168–185.
- 17) Lavigne GJ and Montplaisir JY. Bruxism epidemiology,

diagnosis, pathophysiology, and pharmacology. New York: Raven Press, Ltd; 1995: 387-404.

- 18) Shigeyoshi Fujii, Yasushi Hiraga, Taiji Nakamura, Yoshinori Komatsu, Yoshinori Kobayashi, Shozo Yanagita. Study on the effect of Bite plane: 1st report: Overnight observation findings of masseter electromyogram, electrocardiogram and respiratory curve of Bruxism patients by application of telemeter system. Odontology. 1980; 68: 141-142.
- 19) Hideo Kodama. Effect of tongue invasion on jaw muscle activity. Odontology. 1978; 65: 1008–1042.
- 20) Wruble MK, Lumley MA and Mcglynn FD. Sleeprelated bruxism and sleep variables: A critical review. *J Craniomandib Disord: Facial & Oral Pain.* 1989; 3: 152–158.
- 21) Ikeda T, Nishigawa K, Kondo K, Takeuchi H and Clark GT. Criteria for the detection of sleep-associated bruxism in humans. *J Orofac Pain*. 1996; 10: 270–282.
- 22) Said Mikami, Taihiko Yamaguchi, Kazuki Okada, Akihito Gotouda, Shinpei Matsuda. Application of the ultraminiature cordless electromyogram measurement system to assessment of masseteric activity during nocturnal sleep. J. Jpn, Soc, Stomatognath. Funct. 2009; 15: 121-130.
- 23) Rugh JD and Harlan J. Nocturnal bruxism and temporomandibular disorders. Advances in neurology. New York: Raven press; 1988: 329–341.
- 24) Ichiki Rika, Tsukiyama Yoshihiro, Koyano Kiyoshi. Development of a portable electromyographic recording device and an application for the evaluation of the day to day variation of nocturnal masseter mascle activity. J Jpn. Soc. Stomatognath. Funct., 1999; 6: 67-77.
- 25) Lavigne GJ, Rompre PH and Montplaisir JY. Sleep bruxism. validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res.* 1996; 75: 546–552.
- 26) Kei Yugam. Continuous Evaluation of Muscle Activity with Occlusal Tooth Contacts Part II. Nocturnal Tooth Contacts and Muscle Activity. J. stomatol. Soc. 1996; 63: 31-41.
- 27) Rompre H, Daigle-Landry D, Guitard F, Montplaisir JY and Lavigne GJ. Identification of a sleep bruxisim subgroup with higher risk of pain. *J Dent Res.* 2007; 86: 837–842.
- 28) Clark GT, Rugh JD and Handelman SL. Nocturnal masseter muscle activity and urinary catecholamine levels in bruxers. *J Dent Res.* 1980; 59: 1571–1576.
- 29) Reding GR, Zepelin H, Robinson JE Jr, Zimmerman SO and Smith VH. Nocturnal teeth-grinding: all-night psychophysiolo-gic studies. *J Dent Res.* 1968; 47: 786– 797.

- 30) Baba K, Takeuchi H, Ikeda T and Clark GT. Application of intrasplint force detection system for bruxism in sleeping subjects. *J Oral Rehabil.* 1998; 25: 227.
- Rugh JD and Solberg WK. Electromyographic studies of bruxist behavior. *Calif Dent J*. 1975; 43: 56–59.
- 32) Kazuo Okura. The Multi-telemetering Measurement and Analysis of Sleep Associated Bruxism. Jpn Prosthodont Soc. 1997; 41: 292-301.
- Ware JC and Rugh JD. Destructive bruxism. sleep stage relationship. Sleep. 1988; 11: 172–181.
- 34) Harada T, Ichiki R, Tsukiyama Y and Koyano K. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. *J Oral Rehabil.* 2006; 33: 482–488.
- 35) Fricton J. Current evidence providing clarity in management of temporomandibular disorders: summary of a systematic review of randomized

clinical trials for intra-oral appliances and occlusal therapies. *J Evid Base Dent Pract.* 2006; 6: 48–52.

- 36) Kazuyoshi Babaa, Yasuhiro Onoa, and Akira Nishiyama. Diagnosis and Management for Temporomandibular Disorders under Consideration of the Etiology. —Association between Parafunctional Activity and Development of TMD—. Jpn Prosthodont Soc. 2009; 1: 7-12.
- 37) Ichiki R, Koyano K and Tsukiyama Y. Effect of occlusal splint designs on nocturnal masticatory muscle activity. *J Dent Res.* 1999; 330 (Special Issue): 78.
- 38) Toshifumi Morimoto. Physiological considerations on bite plane therapy for the PMD syndrome. Proceeding of Japanese Society of Stomatognathic Function. 1991; 9: 191–192.
- Kobayashi Yoshinori. Sleep Bruxism and Sleep Apnea. J Jpn. Soc. Stomatognath. Funct. 2009; 15: 95–120.