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Clinical efficacy of stabilization splints for sleep bruxism

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Sleep bruxism has harmful effects such as dental attrition, denture disconnection, and root and crown fractures. With the aim of enabling alleviation of damage to the oromandibular system by sleep bruxism, the efficacy of stabilization splints—a type of oral appliance— in suppressing sleep bruxism was investigated.

For this study, three men who were subjectively aware of sleep bruxism were selected for the bruxism group, and three other men who were not aware were selected for the non-bruxism group. In each group, the frequency of bruxism events during sleep (events/hour) was determined, and the electromyographic level was measured for each bruxism event.

Bruxism events were observed even in the non-bruxism group, but the frequency of bruxism events was significantly lower than in the bruxism group. In addition, the bruxism event frequency was significantly lower in the stabilization-splint group than the non-stabilization-splint group.

These findings suggest that a stabilization splint is effective for preventing dental attrition, denture disconnection and suppressing sleep bruxism.

Key words: Sleep bruxism, oral appliances, EMG

Introduction

Sleep bruxism is thought to involve activation of the masticatory muscle due to excitation of the brain and central nervous system during sleep[1]. In the context of clinical practice relating to dentures, sleep bruxism have complications such as dental attrition, denture disconnection, root and crown fractures, and chipping of all-ceramic crowns. The incidence of sleep bruxism has been reported to be 85% to 90%[2]. Therefore, it is essential to ascertain the state of sleep bruxism with each patient when under prosthetic treatment is to be performed.

However, it is difficult for making the diagnosis on outpatient using electromyography or other measurement devices. In general, sleep bruxism comes to light during an interview with the patient or as reported by the person the patient sleeps with, and screening for sleep bruxism is often performed by collecting information about the presence or absence of intraoral bony protrusions and attrition[3]. In addition, taking mandibular movement as the index, sleep bruxism is broadly classified as being of the grinding type, in which the patient recognizes the bruxism; the clenching type is when the teeth are clenched tightly together[4], but atypical movement that does not belong to either type sometimes occurs, so care is needed with this classification.

In terms of factors associated with sleep bruxism, various local factors have been put forward, but direct relationships with occlusion can be ruled out[5], and psychological stress is thought to play an important part. As such stress has numerous different causes, it is difficult to identify and eliminate each cause.

In the present study, with the aim of enabling alleviation of damage to the oromandibular system by sleep bruxism, the modes of occurrence of sleep bruxism were investigated in the bruxism and non-bruxism groups, after which the suppressive efficacy on sleep bruxism of a stabilization splint—a type of oral appliance—were investigated.

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Materials and methods

1 Selection of subjects

Six male subjects were selected from among students and lecturers at the authors' university. They had no missing teeth, and their age range was 22 to 28 years. Three were placed in the bruxism group, in that they were aware of nocturnal sleep bruxism, whereas the other three were placed in the non-bruxism group, in that they were not aware of nocturnal sleep bruxism. Each of the subjects understood the principles and content of the study sufficiently and gave informed consent to participate. The study was approved by the Ethics Committee at Asahi University's School of Dentistry (approval no.: 22095).

2 Bruxism measurement method

The masseter muscle was used for electromyographic measurement. Muscle activity was generated by the central part of the left masseter muscle so as to run parallel to the masseter muscle fibers. Brain waves were used as the criteria for each subject after falling asleep and were generated using an indifferent electrode in the forehead region; further, a two-channel electroencephalography sensor (DL-160) from electrodes was affixed to both earlobes (A1) —as the reference electrodes—and to the forehead (Fp1). To determine the onset of bruxism, physical movements were assessed carefully to distinguish them from masseter muscle activity. Physical movements were detected as an acceleration signal after voltage transformation by a piezoelectric element affixed to the sternal manubrium. For each biological signal, the analog output was converted to a digital form by the data acquisition system (UAS-108S; Unique Medical Co., Ltd.) at a sampling frequency of 1 KHz, and the data was recorded and analyzed on a Personal Computer (Panasonic Corporation). These measurements were performed at each subject's own home. The site of electrode affixing was specified when measuring muscle activity, and the same site was used on each measurement date. Data were collected over the course of 2 days.

3 Stabilization splint

A stabilization splint, an oral appliance fitted to the maxilla while the patient is asleep, was used. The incisal edges were covered, with an occlusion surface area in the 1.5 to 2.0 mm range, and occlusal contact was achieved with the mandibular buccal cusp over a contact area with a diameter of 0.5 to 0.7 mm. When the mandible moved forwards or sideways, the splint was guided by the canines, so as to keep the molars separation.

4 Identification of bruxism events

In electromyography, root-mean-square processing of the source waves was performed with a time constant of 60 ms. Each subject was instructed to clench his teeth as hard as possible five times, for 2 s each, and the mean maximum amplitude was taken to be the 100% maximum voluntary contraction (MVC)[6]. Muscle activity at 20% MVC or higher that continued for 0.25 s or more was interpreted to be a bruxism event. The bruxism event parameters were frequency of events during sleep (events/hour), and electromyographic level of each event.

The source waves on the electromyographs were assessed, and items that were definitely judged to be physical movements or noise were excluded from the analysis.

5 Analysis methods

The frequency of bruxism events during sleep (events/hour) was compared between the bruxism group and non-bruxism group using Welch's t-test. In both the bruxism and non-bruxism groups, MVC muscle activity was measured at 10% MVC intervals, starting at 20% MVC. The electromyographic level of muscle activity at event onset was compared between the bruxism and non-bruxism groups at each 10% MVC interval using the Bonferroni multiple-comparison test.

The bruxism group was then taken to be the non-stabilization-splint group, and the subjects in the bruxism group with whom stabilization splints were fitted were taken to be the stabilization-splint group, and the bruxism event frequencies during sleep (events/hour) were compared between these using Welch's t-test. Maximum voluntary contraction muscle activity electromyographic levels at event onset were measured at 10% MVC intervals in the non-stabilization-splint group and the stabilization-

splint group. The Bonferroni multiple-comparison test was used to compare the non-stabilization-splint group and stabilization-splint group at 10% MVC intervals.

Results

1　Number of bruxism events

Fig. 1 shows the frequency of sleep bruxism events (events/hour). The frequency in the bruxism group (20.49 events/hour) was significantly higher (p<0.01) than in the non-bruxism group (8.19 events/hour).

2　Electromyographic level

Fig. 2 shows the electromyographic level at event onset at 10% MVC intervals, for each subject. In the non-bruxism group (Fig. 2a), the level at 20% MVC (18) was significantly higher (p<0.01) than at 30% MVC (9.3), 40% MVC (7.4), 50% MVC (4.3), 60% MVC (2), 70% MVC (1.2), and 80% MVC (0.3), and the level at 30% MVC was significantly higher (p<0.01) than at 80% MVC.

In the bruxism group (Fig. 2b), the level at 20% MVC (40.1) was significantly higher (p<0.01) than at 40% MVC (17.2), 50% MVC (10.6), 60% MVC (3.8), 70% MVC (2.3), and 80% MVC (0.89), and the level at 30% MVC (26) was significantly higher (p<0.01) than at 60% MVC, 70% MVC, and 80% MVC.

Comparison of the non-bruxism and bruxism groups showed significant differences at 20% MVC and 30% MVC (Fig. 2c).

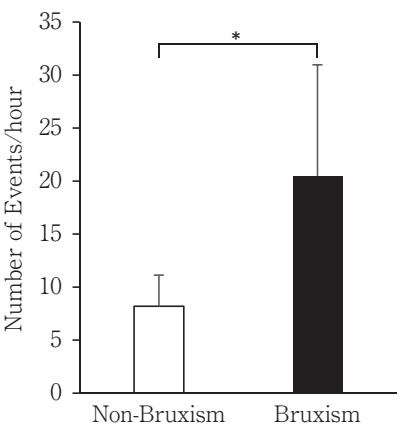

Fig.1 Number of events per hour during sleep bruxism in Non-Bruxism group and Bruxism group *:p<0.01

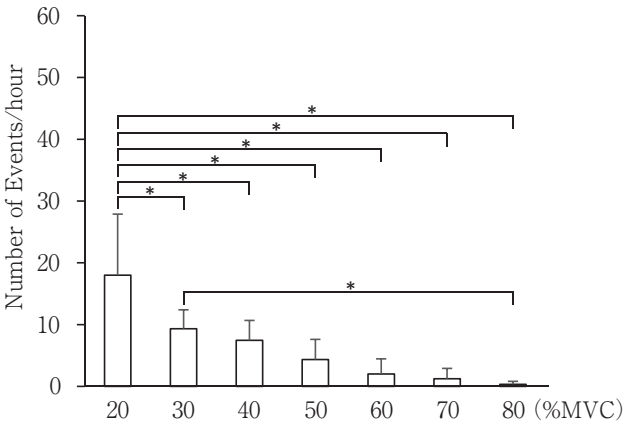

Fig.2(a) Non-Bruxism group

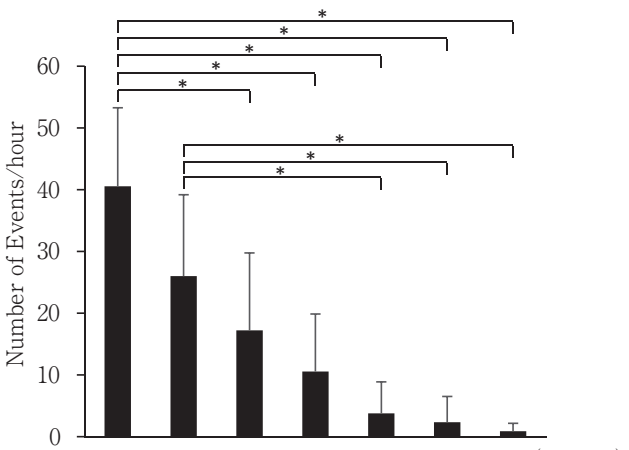

Fig.2(b) Bruxism group

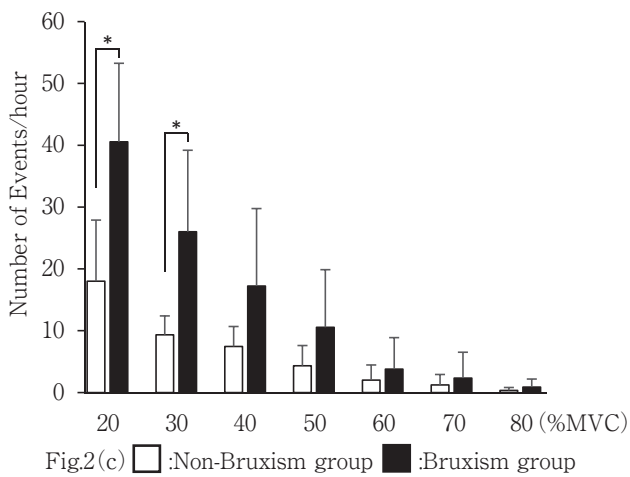

Fig.2(c)

Fig.2 EMG level at Bruxism event (10% MVC interval) Non-Bruxism group (a), Bruxism group (b), Compared Non-Bruxism group with Bruxism group (c) *:p<0.01

3 Event frequencies before and after fitting stabilization splints

Fig. 3 shows the event frequencies before and after fitting stabilization splints. The frequency in the non-stabilization-splint group (29.3 events/hour) was significantly higher than in the stabilization-splint group (18.9 events/hour).

4 Electromyographic levels before and after fitting stabilization splints

Fig. 4 shows the electromyographic levels at 10% MVC intervals before and after fitting stabilization splints. In the non-stabilization splint group (Fig. 4a), the level at 20% MVC (60.5) was significantly higher ($p<0.01$) than at 30% MVC (17.5), 40% MVC (8.0), 50% MVC (3.5), and 60% MVC (1.75), and the level at 30% MVC was significantly higher ($p<0.01$) than at 40% MVC, 50% MVC, and 60% MVC. In the stabilization splint group (Fig. 4b), the level at 20% MVC (34.75) was significantly higher ($p<0.01$) than at 30% MVC (15.0), 40% MVC (7.25), and 50% MVC (4.0), and the level at 30% MVC (15.0) was significantly higher ($p<0.01$) than at 50% MVC (4.0).

A comparison of the non-stabilization splint and stabilization-splint groups is shown in Fig. 4c, and the levels were significantly different at 20% MVC.

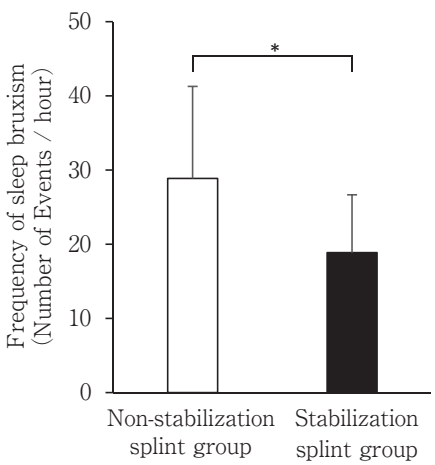

Fig.3 Non-stabilization splint group and Stabilization splint group sleep bruxism Events per hour. Stabilization splint group is wearing of stabilization splint. Non-stabilization splint group is not wearing of stabilization splint. *:$p<0.01$

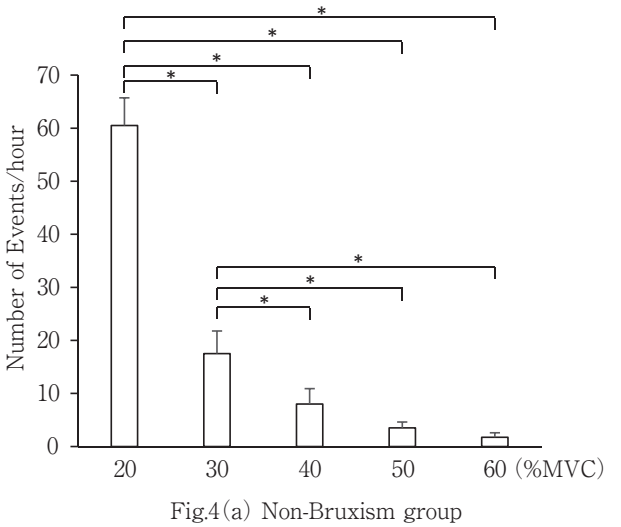

Fig.4(a) Non-Bruxism group

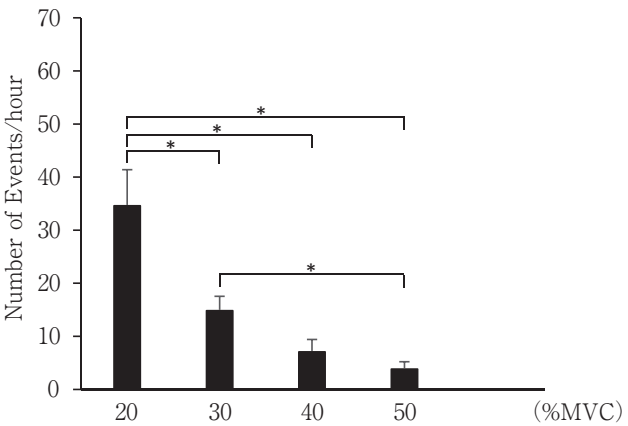

Fig.4(b) Bruxism group

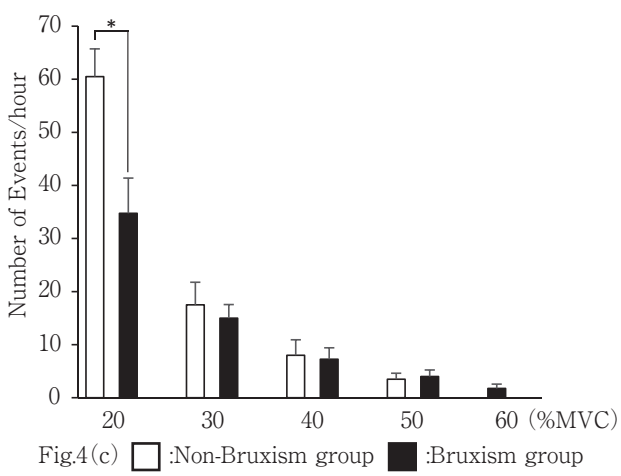

Fig.4(c)

Fig.4 EMG level at Bruxism event (10% MVC interval) Non-stabilization splint (a), Stabilization splint (b), Compared Non-Stabilization splint with Stabilization splint (c). Stabilization splint group is wearing of stabilization splint. Non-stabilization splint group is not wearing of stabilization splint. *:$p<0.01$

Discussion

Sleep bruxism is thought to have a close relationship with psychological stress[5]. With the aim of enabling alleviation of damage to the oromandibular system by sleep bruxism, subjects in the sleep bruxism group were fitted with stabilization splints, which were found to show efficacy in suppressing sleep bruxism.

Bruxism occurs due to the activation of the mouth-opening muscle, so the site of measurement of bruxism, that is, muscle activity, was the central region of the masseter muscle. In addition, there were concerns that swallowing might result in masseter muscle activity being incorrectly judged to be bruxism events, but the masseter muscle activity level at the time of swallowing in a preliminary study did not exceed the 20% MVC muscle activity level in any of the subjects. However, Ikeda et al.[6] were concerned about the effects on sleep caused by the affixed electrodes for the transmission of biological information at event onset to the recording device as well as the leads for those, so with each subject, the site of affixing each electrode was ascertained, and the operating method for the measurement device was explained. The subject then practiced using it twice before measurement.

With respect to the sleep bruxism occurrence modes, sleep bruxism events were found even in the non-bruxism group. The non-bruxism group consisted of subjects who, in the interview before measurement, completed a questionnaire stating that they had no subjective awareness of bruxism. Lavigne et al.[4] have reported that patients with clenching-type bruxism often do not have the characteristic signs of bruxism. In other words, subjects in the non-bruxism group are not subjectively aware of sleep bruxism, but in fact sleep bruxism does occur, albeit significantly less often than in the bruxism group.

The electromyographic level at bruxism event onset was significantly higher at 20% MVC in both the non-bruxism and bruxism groups. Then, with increasing MVC, bruxism events become less frequent, and it is therefore suggested that sleep bruxism involves repeated contraction of the masseter muscle at an occlusion strength of 20% to 30% of the maximum occlusion strength.

In the bruxism group, the bruxism event frequency after fitting stabilization splints was significantly lower than before fitting them. In different randomized controlled trials relating to sleep bruxism, bruxism has been found to be both alleviated[7-10] and not alleviated after fitting stabilization splints[11, 12], but there have been more reports of a tendency toward a positive relationship between fitting of stabilization splints and alleviation of bruxism[7-10]. However, findings that stabilization splints are effective against bruxism, but with efficacy that decreases with time, have also been reported[13].

The bruxism treatment method in clinical practice generally involves covering the occlusion surface with a splint, and as to the efficacy of such methods, Huynh et al.[14] have reported that use of stabilization splints suppresses sleep bruxism. Various opinions have been expressed about the efficacy of splints, but it can at least be said that splints are recommended for preventing dental attrition, and dealing with denture disconnection and all-ceramic crown fracture[15].

In summary, it is suggested that the stabilization splint is effective against sleep bruxism.

Conclusions

The bruxism event frequency in the bruxism group was significantly higher than in the non-bruxism group. The bruxism event frequency was significantly lower in the stabilization-splint group than in the non-stabilization-splint group. The electromyographic levels were significantly lower at 20% MVC. Thus, it is suggested that stabilization splints are effective for preventing dental attrition and denture disconnection and for suppressing sleep bruxism.

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