博 士 論 文

Temporal association between sleep apnea-hypopnea and sleep bruxism events

（睡眠時無呼吸イベントと睡眠時ブラキシズムの時間的関係）

平成26年3月申請

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Title

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Running title

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This is the accepted version of the following article:
Saito M, Yamaguchi T, Mikami S, Watanabe K, Gotouda A, Okada K, Hishikawa R, Shibuya E, Lavigne G. J. Temporal association between sleep apnea–hypopnea and sleep bruxism events. J Sleep Res 2014;23:196-203, which has been published in final form at http://onlinelibrary.wiley.com/doi/10.1111/jsr.12099/full.

Summary

There is some evidence suggesting that obstructive sleep apnea-hypopnea syndrome (OSAHS) is concomitant with sleep bruxism (SB). The aim of this study was to investigate the temporal association between sleep apnea-hypopnea events and SB events (AHEs & SBEs).

In an open observational study, data were gathered from 10 male subjects with confirmed OSAHS and concomitant SB. Polysomnography and audio-video recordings were performed for one night in a sleep laboratory. Breathing, brain, heart, and masticatory muscle activity signals were analyzed to quantify sleep and sleep stage duration and number and temporal distribution of AHEs and SBEs. AHEs were collected within a 5-minute time window before and after SBEs, with the SBE as the pivotal reference point. Two temporal patterns were analyzed: 1) the interval between AHE termination and SBE onset, called T1, and 2) the interval between SBE termination and AHE onset, called T2.

Of the intervals between SBEs and the nearest AHE, 80.5% were scored within 5 minutes. Most intervals were distributed within a period of less than 30 seconds, with peak at 0 to 10 seconds. The T1 interval had a mean length of 33.4 seconds and was significantly shorter than the T2 interval (64.0 seconds; $p<0.05$). Significantly more SBEs were scored in association with the T1 than the T2 pattern ($p<0.05$).

Thus, in patients with concomitant obstructive sleep apnea-hypopnea syndrome and sleep

bruxism, most sleep bruxism events occurred after sleep apnea-hypopnea events, suggesting that sleep bruxism event occurring close to sleep apnea-hypopnea event is a secondary form of sleep bruxism.

Key words: sleep-disordered breathing, sleep apnea-hypopnea syndrome, sleep bruxism, tooth grinding, masseter muscle, rhythmic masticatory muscle activity

1. Introduction

Sleep bruxism (SB) is a common sleep-related movement disorder characterized by clenching or grinding of the jaws or teeth (American Academy of Sleep Medicine, 2005). In a new, revisited definition, bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (sleep bruxism) or during wakefulness (awake bruxism) (Lobbezoo *et al.*, 2013). Clinical consequences associated with SB include damage to the teeth and dental prostheses, possible temporomandibular disorder exacerbation, and headache (Camparis and Siqueira, 2006; Johansson *et al.*, 2011; Lobbezoo and Lavigne,1997; Pergamalian *et al.*,2003). Cumulative evidence supports that the genesis of some SB-related motor activity is secondary to autonomic and central nervous system co-activation. However, because approximately 20% of SB motor event genesis has not yet been clearly explained, this explanation is not exclusive (Lavigne *et al.*, 2011; Carra *et al.*, 2012b). SB may also occur concomitantly or secondary to other sleep disorders (Carra *et al.*,2012a; Carra *et al.*, 2012b, Kato 2013a). Obstructive sleep apnea-hypopnea syndrome (OSAHS) may be a sleep disorder that is concomitant with SB (Inoko *et al.*, 2004; Kato *et al.*, 2013b; Okeson *et al.*, 1991; Phillips *et al.*, 1986; Sjőholm *et al.*, 2000).

An epidemiological study suggested that patients with sleep-disordered breathing had

higher risk for SB, with a 1.8 odds ratio (Ohayon *et al.*, 2001). However, despite a low but significant association between SB and OSAHS, the cross-sectional telephone survey design did not allow concluding causality between the two conditions. Based on polysomnographic jaw muscle recordings in patients with OSAHS, masticatory muscle activity tended to occur around the termination of apnea or hypopnea events (AHEs) (Inoko et al., 2004; Okeson et al., 1991; Phillips et al., 1986; Sjőholm et al., 2000). However, these studies used electromyography (EMG) to measure masseter and temporalis muscle activity, a method that does not allow determining the specificity of jaw muscle activity to SB, which is typically identified by scoring rhythmic masticatory muscle activity (RMMA) (Lavigne *et al.*, 1996; Lavigne *et al.*, 2011). Other types of non-specific EMG activity could occur in the masticatory muscles during sleep, including swallowing, coughing, or face rubbing. (Dutra *et al.*, 2009; Yamaguchi *et al.*, 2012). To assess SB events (SBEs) with higher specificity, polysomnography with audio-video (PSG-AV) recordings is the gold standard (American Academy of Sleep Medicine, 2005). In a recent study on OSAHS patients without SB diagnosis, nonspecific muscular activity, discriminated from SB using PSG-AV, tended to occur at OSAHS event termination (Kato et al., 2013c). In the above-mentioned studies, except for the latter study on OSAHS patients without SB diagnosis, simultaneous audio-video recording was not performed, and masticatory muscle activity around the termination of apnea or hypopnea events was not definitively identified as SBE.

A case report (Oksenberg and Arons, 2003) showed that most tooth grinding events

appeared at the termination of apnea/hypopnea events. We presented an OSAHS case with severe SB that supported a potential temporal association between AHEs and SBEs (Saito et al., 2011). However, these studies were case reports, with limited assessment of temporal and causal associations between OSAHS and SB.

It was further suggested that little or no solid temporal relationship is present between OSAHS and SB (Okeson et al., 1991; Sjöholm et al., 2000). Few differences were found in the number of SB-scored events in subjects with sleep-disordered breathing compared to controls (Okeson et al., 1991). Masseter muscle events were associated with AHE termination for only 3.5% to 14.4% of scored events. Moreover, rhythmic jaw movement, a proxy for RMMA, was rarely directly associated with apnea events (Sjöholm et al., 2000).

Given these contradictory findings, we performed PSG-AV analysis to investigate for the presence of a temporal association between SBEs and AHEs in patients with concomitant OSHAS and SB. If SBEs occur mainly after AHEs, this would support that SBEs in OSAHS patients may be a secondary form of SB.

2. Methods

Subject characteristics and study design

OSAHS patients with suspected SB were selected based on: 1) reports of tooth-grinding sounds or tooth clenching awareness during sleep; 2) jaw muscle discomfort, fatigue, or pain and jaw lock upon awaking; and 3) clinician-observed tooth wear. Subjects visited a sleep

clinic for an examination and to confirm OSAHS diagnosis. Subjects were included if both OSAHS and SB were diagnosed. Subjects were excluded for major neurologic, psychiatric, or sleep disorders (e.g., REM behavior disorder, periodic leg movements during sleep); psychoactive medication intake, which may increase the risk of limb or orofacial activity; and absence of natural dentition. OSAHS and SB were definitively diagnosed from a one-night sleep study, as described below, using a threshold apnea hypopnea index (AHI) defined as >5 events/h for OSAHS and a threshold SB index (SBE/h) of >4 events/h for SB (AASM 2005). The final patient sample comprised ten male OSAHS patients with SB with a mean age of 46.7 (SD: 11.5) years. The mean body mass index (BMI) was 27.7 (3.9) and mean score on the Epworth sleepiness scale was 8 (5).

This study was approved by the ethical committee of Hokkaido University Hospital, and written informed consent was obtained from all subjects prior to participation in the study.

Polygraphic sleep recording

Subjects slept for one night in a sleep clinic. The recording variables included electroencephalograms (EEGs) at C3-M2, C4-M1, O1-M2, and O2-M1; right and left electrooculogram (EOGs); electromyograms (EMGs) of the sub-mental and masseter muscle and anterior tibialis muscle (five channels); and an ECG (one channel). Body positions were detected using a body position sensor equipped with a 3D accelerometer.

Sleep breathing variables included airflow (with a nasal/oral thermistor), chest and abdominal effort (two channels), and SaO2 measured by pulse oximetry (one channel). Recordings were performed with an Alice 5 PSG system (Philips Electronics, Amsterdam, The Netherlands). For SB recognition, PSG recordings included masseter EMG with audio-video. Masseter recording side was randomized across subjects: masseter EMG electrodes were attached on the right side for seven subjects and on the left side for three subjects. All recording signals were amplified and analog-to-digital (A/D) converted at a 2-kHz sampling frequency. All scoring was performed offline using commercial software (Alice Sleepware, Philips Electronics, Amsterdam, The Netherlands). Chart5 (ADInstruments Ltd., Bella Vista, NSW, Australia) was also used for offline processing of masseter EMG waveforms. Masseter EMG data were high-pass filtered at 20 Hz and converted to absolute values using Chart5.

Sleep stage was scored according to standard criteria (Iber et al., 2007). AHEs were scored according to AASM criteria (Iber et al., 2007). Arousal events within 5 seconds of AHE termination were scored, and AHEs were classified into events with and without arousal.

Assessment of SB events

SBEs were assessed according to published criteria and masseter events were identified as RMMA (Lavigne, 1996; AASM, 2005). The amplitude threshold was set at twice the baseline

activity (Yamaguchi et al., 2012). Bursts with greater amplitude than the value with duration exceeding 0.25 seconds were selected. RMMA EMG events separated by 3-second intervals were recognized as SBEs if they corresponded to one of the three following patterns: phasic (3 or more masseter EMG bursts, each lasting 0.25 to 2.0 sec), tonic (at least one masseter EMG burst longer than 2.0 sec), or mixed (both masseter burst types).

Events other than RMMA typical of SBEs (e.g., swallowing, coughing, and face scratching), unidentified events with face hidden by blanket, and events during wake stage were excluded by audio-visual (AV) scoring.

Classification of temporal associations between SBEs and AHEs

The nearest AHE before and after each SBE was selected, with the SBE as the pivotal reference point. Regardless of whether SBEs and/or AHEs occurred in clusters or in close sequence, only the nearest AHE was scored for each SBE. Two temporal patterns were analyzed: 1) the interval between AHE termination and SBE onset, called T1 (AHE to SBE), and 2) the interval between SBE termination and AHE onset, called T2 (AHE to SBE) (Fig. 1). Overlapping AHE termination and SBE onset was considered T1, and overlapping SBE termination and AHE onset was considered T2. SBE temporal distribution was scored within a 5-minute period after (T1) or before (T2) an AHE. Considering a possible sudden rise in autonomic sympathetic nerve activity at 4 to 8 minutes before SBE onset (Huynh et al., 2006; Khoury et al., 2008; Lavigne et al., 2011), we examined a 5-minute time window

around AHEs and SBEs. Subjects were divided into two subgroups according to AHI. The moderate–severe OSAHS group included subjects with AHI more than 15 and the mild OSAHS group included subjects with AHI from 5 to 15.

Statistical analysis

Distributions of temporal intervals, or T1 and T2 within the 5-minute windows, were expressed as a histogram. Mean distributions of the two intervals were calculated for all subjects. The numbers of SBEs associated with T1 and T2 were statistically compared. The intervals T1 and T2 were also statistically compared. All data are presented as means (SD).

The paired *t*-test was used for the statistical analysis. Statistical significance was set at $p<0.05$. Microsoft Office Excel 2007 (Microsoft Co.) and Statcel 2 (OMS Publishing Inc., Tokorozawa, Japan) were used for statistical analyses.

3. Results

Subjects' sleep data are presented in Table 1. The mean AHI was 24.0 (17.1) (range: 7.4–60.0) and mean number of SBEs/h was 13.5 (5.0) (range: 8.2–24.4). Most detected AHEs were obstructive apnea-hypopnea and mixed apnea (Table 1). Similarly, most AHEs nearest to SBEs were obstructive sleep apnea-hypopnea and mixed apnea. Very few central apneas were scored (Fig. 2).

For the total sample, mean percentage of SBEs occurring within 5 minutes of AHEs

was 80.5%, with 19.5% of SBEs occurring at longer than 5 minutes from AHEs (Table 2). A significantly higher percentage of SBEs (54.9%) showed the T1 pattern (AHE to SBE) than the T2 pattern (SBE to AHE) ($P<0.05$), with the nearest AHE tending to occur before the SBE. This trend was also shown for the moderate-severe OSAHS group with significant difference between T1 and T2, while no significant difference in percentage of SBEs between T1 and T2 was shown for the mild OSAS group (Table 2).

Within the 5-minute windows, most T1 and T2 patterns were distributed within a period of less than 30 seconds, with 86.8% of T1 peaking at from 0 to 10 seconds and 65.8% of T2 peaking at from 0 to 10 seconds (Fig. 3, Table 3). Particularly strong convergence was found in the 0- to 10-second category for T1. Mean interval for T1 and T2 within 5 minutes was 33.4 seconds and 64.0 seconds, respectively, and T1 was significantly shorter than T2 ($p<0.05$) (Fig. 4).

The numbers of apnea-hypopnea events classified in relation to arousals and SBEs are presented in Table 4. For the total 10 subjects, the mean number of the nearest AHE before an SBE within 5 minutes (AHE(T1)) was 40.8 (30.0% of total AHEs), and the mean number of the nearest AHE after an SBE within 5 minutes (AHE(T2)) was 16.8 (12.3% of total AHEs). The ratio of AHE(T1) with arousal to total AHEs with arousal was significantly larger than the ratio for AHE(T2) ($p<0.05$). Similarly, the ratio of AHE(T1) without arousal to total AHEs without arousal was significantly larger than the ratio for AHE(T2) ($p<0.05$). In comparison between AHE(T1) with arousal and that without arousal, mean ratio of AHE(T1)

with arousal to total AHE with arousal was slightly larger than that without arousal, but no significant difference was shown.

4. Discussion

A detailed investigation was performed to assess the temporal associations between SBEs and AHEs. In patients with concomitant OSAHS and SB, most SBEs occurred after AHEs. Such finding is suggesting that SBE occurring close to AHE is a secondary form of SB.

Owing our study limitation, we recognize that not all SBEs are secondary to AHEs, suggesting that other concomitant influences may be present in the temporal relationship between AHEs and SBEs.

Masseter EMG in SBE, AHE, and other activities

PSG-AV was used to confirm the presence of both OSAHS and SB, a major strength of the present study. When EMG bursts of masticatory muscle during sleep are scored, various types of bursts are observed, including myoclonic contraction, swallowing, and sighing (Dutra et al., 2008; Yamaguchi et al., 2012). In wake periods during sleep, various types of masticatory muscle activity are also observed. Accordingly, when analyzing nocturnal EMG data, RMMA burst events typical of SB should be distinguished from other masseter activity. In the present study, other motor activity and bursts during wake periods were excluded, and more reliable data concerning SB was obtained with simultaneous audio-video recording.

Sleep arousal in relation to OSAHS and SB

The association between OSAHS and SB has been attributed to several potential causes: comorbid sleep disorders, re-opening of upper airways, lubrication of the oropharynx, and arousal reaction (Carra et al., 2012a; Lavigne, 2003).

RMMA may be an oromotor activity that helps reinstate airway patency following a disrupted respiratory event during sleep, including airway resistance (Carra et al., 2012a; Khoury et al., 2008). RMMA may also act as a physiological motor event that is required to lubricate the oropharyngeal structures during sleep (Carra et al., 2012a; Thie et al., 2002). These hypotheses remain to be confirmed.

Masticatory muscle bursts may also arise from body movements as an arousal reaction to AHEs or from saliva swallowing to lubricate a dry oropharynx due to AHEs. Previous studies have suggested that some masticatory EMG bursts occur at AHE termination (Inoko et al., 2004; Okeson et al., 1994; Phillips et al., 1986). In addition, non-specific masseter contractions, which are distinct from RMMA, also tended to occur at AHE termination in OSAHS patients without SB diagnosis (Kato, in press, published online). These results showed masseter muscle activity-SBEs at AHE termination as well as non-specific masseter muscle contractions.

It may also be suggested that both arousal reactions during sleep and autonomic sympathetic activation can be involved in the association between OSAHS and SB. Recently,

it was found that SBE was accompanied by a specific ascending sequence of physiological activity: at 4 minutes before SBE onset (i.e., minus 4 minutes), a rise in autonomic sympathetic cardiac dominance with a withdrawal of cardiac parasympathetic dominance occurs; at minus 4 seconds a rise in rapid-frequency cortical activity (EEG activity) occurs; and at minus 1 second a change in heart rate, suprahyoid muscle tone, and breathing occurs with a modest but significant rise in blood pressure (Huynh et al., 2006; Khoury et al., 2008; Lavigne et al., 2011; Nashed et al., 2012). Considering a potential sudden rise in autonomic sympathetic nerve activity and the results of the above-mentioned study (Huynh et al., 2006), indicating a rise in the sympathetic nerve 4 to 8 minutes before SBE onset, we postulated that AHEs occurring within a few minutes of SBEs had some association with SBEs. To assess this association, we performed a 5-minute time window analysis around AHEs and SBEs.

Due to the presence of arousals and hypoxia with AHEs and a concomitant rise in sympathetic activity (Bradley et al., 2003), we also speculated that an arousal reaction to an AHE would acutely increase the probability of SBE occurrence. Since the sympathetic system in OSAHS patients is considered to be chronically hypersensitized (Gilmartin et al., 2010), SBEs in OSAHS patients would tend to occur more frequently in conjunction with AHEs. In subgroup analyses, the nearest AHE tended to occur before the SBE for the moderate-severe OSAHS group, with a significant difference between T1 and T2, whereas no significant difference was found for the mild OSAS group. Due to the small size of the

subgroup samples, we cannot draw any firm conclusions at this time. Further studies are needed to confirm whether the association between AHE and SBE depends on the severity of OSAHS and chronic hypersensitization of the sympathetic system.

In the above-mentioned study by Kato (Kato et al., 2013c), the non-specific contractions of masseter muscles after an AHE also depended on arousal duration. The data suggest that activation of the masseter muscle can be accelerated via an arousal reaction to an AHE. Similarly to this non-specific masticatory motor activity, we speculated that SBEs found in association with AHEs could be associated with sleep arousals. However, the AHE-arousal scoring in the present study showed no significant difference in the post-AHE occurrence of SBEs with and without arousal. Our hypothesis that OSAHS-related sleep instability may contribute to increase post-AHE occurrence of SBEs is therefore not supported. Further studies with larger sample sizes are needed to clarify the putative association with sleep arousal.

Cause and effect: not a unique temporal sequence pattern

Although there is no clear consensus on the temporal association between SBEs and AHEs, as described above, recent findings support our hypothesis (Inoko et al., 2004; Okeson et al., 1991; Phillips et al., 1986; Sjőholm et al., 2000; Saito et al., 2011; Kato in press, published online).

There are three possible cause-and-effect relationships between SB and OSAHS:

OSAHS induces SB, SB induces OSAHS, or some other factors coincidently induce both SB and OSAHS. The present study focused on a potential temporal association, namely that a specific order of onset time between SBEs and AHEs would support a cause-and-effect relationship between the two events. One main finding was a greater number of T1 intervals (AHE to SBE), with peak occurrence within a 0–10-second period. This suggests that in patients with OSAHS, RMMA is frequently secondary to AHE. However, because 25% of SBEs preceded AHEs and 20% had no close temporal association with an AHE, we cannot exclude the possibility of other concurrent factors (e.g., SBE with airway resistance and SBE without arousal triggering AHE).

Changes in SBE occurrence in association with a reduction in AHEs through OSAHS treatments (e.g., continuous positive airway pressure – CPAP; mandibular advancement appliance) could provide further insight into the cause-and-effect issue. A case report using a CPAP demonstrated a reduction in the respiratory disturbance index (RDI) from 47.6 to 4.1 and in the number of audible bruxism events from 73 to 0 after CPAP treatments, although SB was assessed by sound recording only (Oksenberg and Arons, 2003).

Both SBE and AHE in clusters or in single events were included in the analyses. In a cluster event, the presence of several AHEs might affect SBE occurrence. In the future, more detailed analyses of multiple associations among SBEs and AHEs are needed to clarify the associations between AHEs, SBEs, and arousals.

Limitations

Whereas this study provided significant new information, several limitations should be noted. First, PSG-AV data were recorded for only one night. The PSG-AV equipment and unfamiliar circumstances might have affected the subjects' sleep state, known as first-night effects. Second, the results cannot be claimed as exclusive to general OSAHS patients, because some OSAHS patients may not exhibit SB (Sjöholm et al., 2000), and the present study examined a small sample of patients with both OSAHS and SB. In addition, because the subjects were selected among OSAHS patients, we could not determine the temporal association between SBEs and central AHEs. Third, we did not investigate the temporal association in relation to autonomic-cardiac activation. More detailed analyses in OSAHS patients are needed, with larger samples, including patients suffering from central sleep apnea-hypopnea syndrome. Relationships to arousal events as well as changes in sympathetic nerve activity should be examined in order to determine an intermediary factor between AHEs and SBEs. Fourth, only middle-aged OSAHS male subjects were examined. To improve the generalization of findings, this study should be replicated in larger samples of mixed OSAHS and SB populations.

Acknowledgments

We would like to thank Yoshie Shibuya, Natsue Yokoyama, Mari Niimi, and Miku Matsumura for their invaluable cooperation. This study was supported in part by a Grant-in-Aid for Scientific Research from the Japan Society for the Promotion of Science (No. 24390427). GL is a Canada Research Chair. We also thank Margaret McKyes for English editing.

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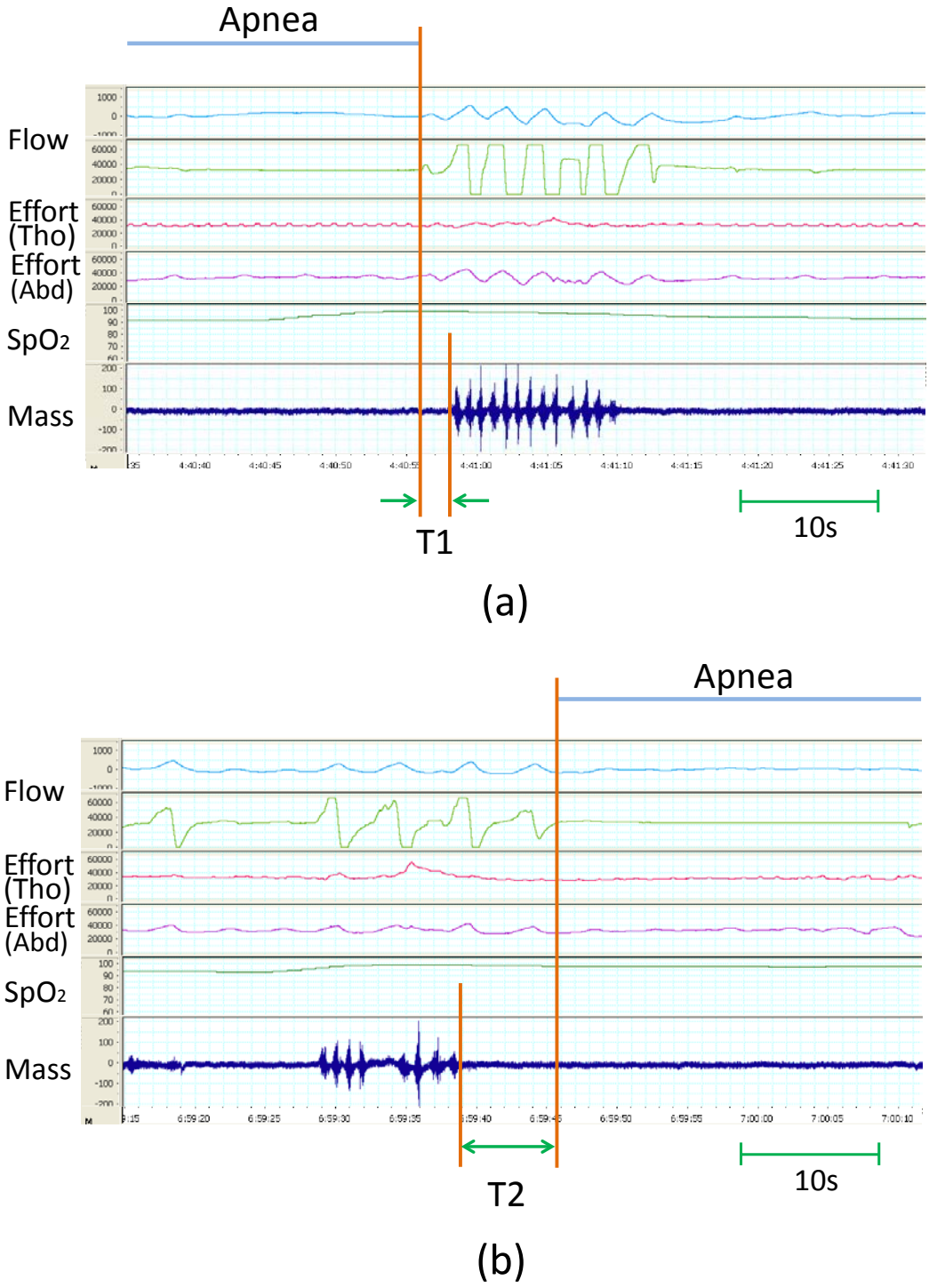

Fig.1 Temporal pattern of association between sleep apnea-hypopnea event and sleep bruxism event within a 5-minute scoring window

T1 = temporal interval between AHE termination and SBE onset (a).

T2 = temporal interval between SBE termination and AHE onset (b).

AHE = sleep apnea-hypopnea event; SBE = sleep bruxism event; Flow = airflow (with a nasal/oral thermistor); Effort (Tho) = chest effort; Effort (Abd) = abdominal effort; Mass = electromyogram (EMG) of the masseter muscle.

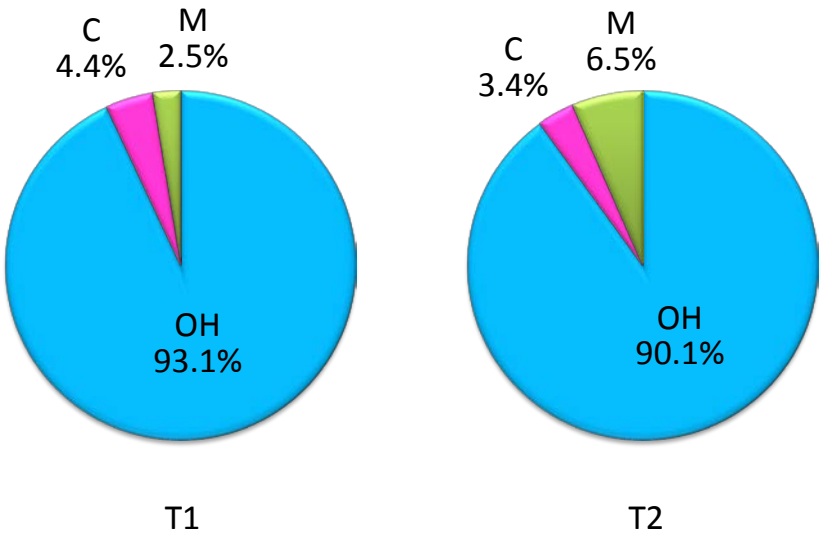

Fig.2 Classification of sleep bruxism events based on the nearest sleep apnea-hypopnea event type (n=10)

Averages for the ten subjects are shown.

AHE = sleep apnea-hypopnea event; SBE = sleep bruxism event.

OH = SBE with the nearest AHE being obstructive apnea-hypopnea; M = SBE with the nearest AHE being mixed apnea; C = SBE with the nearest AHE being central apnea

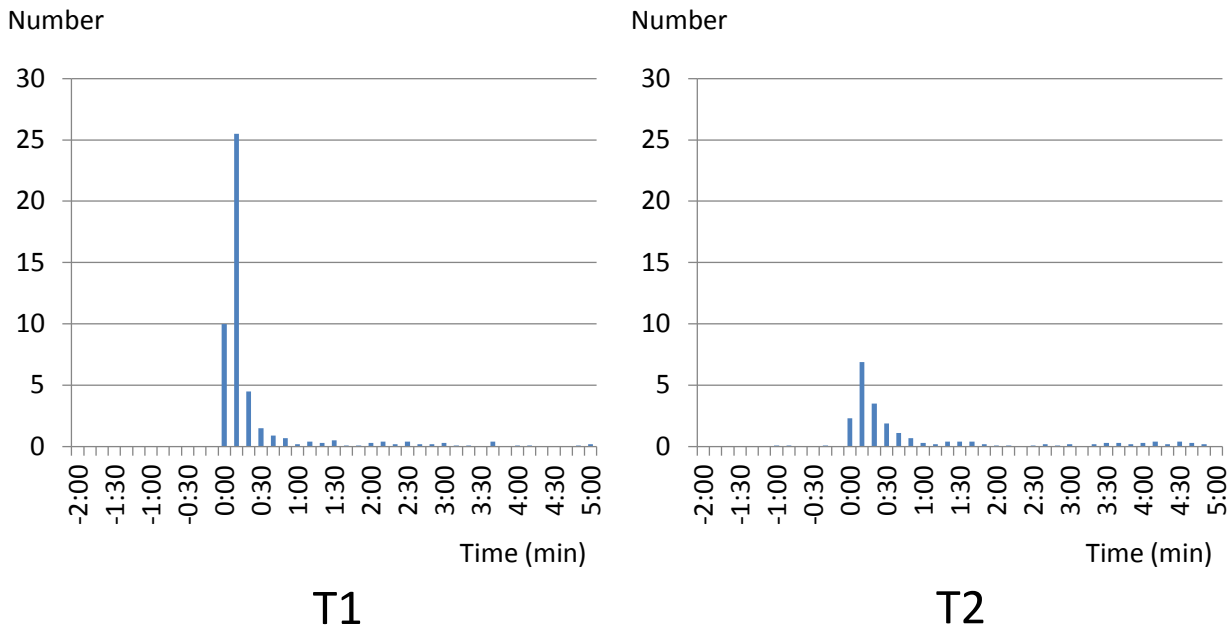

Fig.3 Distributions of temporal intervals from sleep bruxism events to the nearest sleep apnea-hypopnea event within a 5-minute scoring window (n=10)

The value of the vertical axis is the number of SBEs in each interval class. Averages for the ten subjects are shown.

T1 = temporal interval between AHE termination and SBE onset (25.5 events, peak at 0–10 sec).

T2 = temporal interval between SBE termination and AHE onset (6.9 events, peak at 0–10 sec).

AHE = sleep apnea-hypopnea event; SBE = sleep bruxism event.

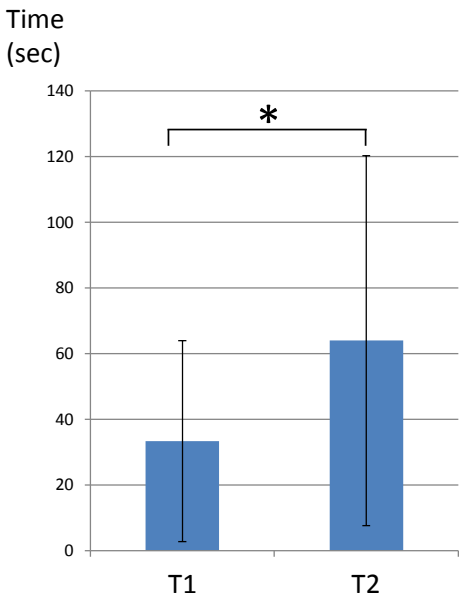

Fig.4　Mean intervals for T1 and T2 within 5 minutes (n=10)

Vertical lines on the bars indicate standard deviations.

*p<0.05

T1 = temporal interval between AHE termination and SBE onset.

T2 = temporal interval between SBE termination and AHE onset.

Table1 Sleep and apnea-hypopnea event (AHE) data of ten subjects

	Mean	SD
Sleep data		
TST (min)	350.3	45.8
TIB (min)	399.6	30.1
Sleep efficiency TST/TIB (%)	87.5	7.8
Sleep stage distribution		
N1/TST(%)	30.3	17.1
N2/TST(%)	54.5	13.5
N3/TST(%)	4.1	5.2
R/TST(%)	10.7	4.5
Numbers of apnea-hypopnea event		
Obstructive apnea and hypopnea	131.2 (95.5%)	96.8
Central apnea	1.2 (1.8%)	1.5
Mixed apnea	3.8 (2.4%)	9.8

TST=total sleep time
TIB=time in bed

Table 2 Number of SBE classified by temporal interval from AHE according to OSAHS severity

	Moderate-severe OSAHS (n=5)		Mild OSAHS (n=5)		Total (n=10)	
	Mean	SD	Mean	SD	Mean	SD
Total number of SBE	94.6	37.8	79.4	23.9	87.0	30.9
SBE with T1						
N1	31.2	34.7	16.6	13.5	23.9	26.0
N2	35.6	15.8	9.6	11.8	22.6	19.0
N3	0.2	0.4	0	0.0	0.1	0.3
REM	1.2	2.7	1.2	1.8	1.2	2.1
Total	68.2*	27.6	27.4	20.4	47.8**	31.4
(%)	72.0		30.9		54.9	
SBE with T2						
N1	11.4	13.5	14.8	8.3	13.1	10.7
N2	8.8	6.8	7.6	7.8	8.2	6.9
N3	0.0	0.0	0.4	0.5	0.2	0.4
REM	1.2	2.7	0.2	0.4	0.7	1.9
Total	21.4*	12.9	23.0	10.8	22.2**	11.3
(%)	22.8		28.5		25.5	
SBE without AHE						
N1	4.8	9.7	18.2	8.7	11.5	11.2
N2	0.2	0.4	10.4	6.1	5.3	6.7
N3	0.0	0.0	0.4	0.9	0.2	0.6
REM	0.0	0.0	0.0	0.0	0.0	0.0
Total	5.0	9.6	29.0	9.4	17.0	15.5
(%)	5.2		40.7		19.5	

*,** $p<0.05$ between SBE with T1 and SBE with T2
SBE without AHE =SBEs occurred at longer than 5-min intervals from AHEs
T1=temporal interval between cessation of an AHE and onset of a SBE
T2=temporal interval between end of a SBE and onset of an AHE
AHE=sleep apnea-hypopnea event
SBE=sleep bruxism event
Moderate-severe OSAHS=subjects with AHI more than 15
Mild OSAHS=subjects with AHI from 5 to 15

Table 3 Cumulative number of SBE scored within 5min from the nearest AHE (n=10)

Temporal interval	Cumulative number	%
T1 (AHE to SBE)		
<10 sec	35.5	74.3
<20 sec	40	83.7
<30 sec	41.5	86.8
<60 sec	43.3	90.6
Total (<5 min)	47.8	100
T2 (SBE to AHE)		
<10 sec	9.2	41.4
<20 sec	12.7	57.2
<30 sec	14.6	65.8
<60 sec	16.7	75.2
Total (<5 min)	22.2	100

The numbers are mean of the ten subjects

T1=temporal interval between cessation of an AHE and onset of a SBE

T2=temporal interval between end of a SBE and onset of an AHE

Table 4 Numbers of apnea-hypopnea event classified in relation to arousal and SBE

	Moderate-severe OSAHS (n=5)		Mild OSAHS (n=5)		Total (n=10)	
	Mean	SD	Mean	SD	Mean	SD
Arousal						
Number of total arousal	105.2	67.9	35.2	26.5	70.2	61.0
Arousal Index (number of total arousal /h)	18.7	11.8	5.9	4.5	12.3	10.8
AHE						
AHI	37.8	13.0	10.2	3.0	24.0	17.1
Total AHE	210.6	51.2	61.8	21.5	136.2	86.7
AHE without arousal	140.0	68.6	51.8	23.0	95.9	67.0
AHE with arousal	70.6	60.6	10.0	11.8	40.3	52.1
AHE (T1) without arousal	31.0	15.5	12.4	6.4	21.7	14.8
AHE (T1) without arousal / total AHE without arousal (%)	23.5%	6.2%	30.7%	26.8%	27.1%*	18.7%
AHE (T1) with arousal	33.0	42.0	5.2	7.9	19.1	32.1
AHE (T1) with arousal / total AHE with arousal (%)	38.1%	18.2%	27.5%	29.1%	32.8%**	23.6%
AHE (T2) without arousal	11.2	5.8	10.4	3.7	10.8	4.6
AHE (T2) without arousal / total AHE without Arousal (%)	10.9%	8.2%	25.6%	17.7%	18.2%*	15.2%
AHE (T2) with arousal	8.4	12.3	3.6	6.1	6.0	9.5
AHE (T2) with arousal / total AHE with Arousal (%)	11.0%	11.1%	15.3%	22.6%	13.2%**	16.9%

*,** p<0.05 between AHE (T1) and AHE (T2)

AHE (T1) =the nearest AHE before SBE within 5 min

AHE (T2) =the nearest AHE after SBE within 5 min

T1=temporal interval between cessation of an AHE and onset of a SBE

T2=temporal interval between end of a SBE and onset of an AHE

AHE=sleep apnea-hypopnea event

SBE=sleep bruxism event

Moderate-severe OSAHS=subjects with AHI more than 15

Mild OSAHS=subjects with AHI from 5 to 15