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Title

Weak Association between Sleep Bruxism and Obstructive Sleep Apnea. A Sleep Laboratory Study

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The authors have no conflicts of interest to declare concerning the present study.

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All authors made substantial contributions to this study.

Abstract

Purpose

No definitive associations or causal relationships have been determined between obstructive sleep apnea-hypopnea (OSAH) and sleep bruxism (SB). The purpose of this study was to investigate, in a population reporting awareness of both OSAH and SB, the associations between each specific breathing and jaw muscle event.

Methods

Polysomnography and audio-video data of 59 patients reporting concomitant OSAH and SB history were analyzed. Masseteric bursts after sleep onset were scored and classified into three categories: 1) sleep rhythmic masticatory muscle activity with SB (RMMA/SB), 2) sleep oromotor activity other than RMMA/SB (Sleep-OMA), and 3) wake oromotor activity after sleep onset (Wake-OMA). Spearman's rank correlation coefficient analyses were performed. Dependent variables were the number of RMMA/SB episodes, RMMA/SB bursts, Sleep-OMA, and Wake-OMA; and independent variables were apnea-hypopnea index (AHI), arousal index(AI), body mass index(BMI), gender, and age.

Results

Although all subjects had a history of both SB and OSAH, sleep laboratory results confirmed that these conditions were concomitant in only 50.8% of subjects. Moderate correlations were found in the following combinations

($p<0.05$) ; RMMA/SB episode with AI, RMMA/SB burst with AI and age, Sleep-OMA burst with AHI, and Wake-OMA burst with BMI.

Conclusions

The results suggest that 1) sleep arousals in patients with concomitant SB and OSAH are not strongly associated with onset of RMMA/SB; 2) apnea-hypopnea events appear to be related to higher occurrence of other types of sleep oromotor activity, and not SB activity. SB genesis and OSAH activity during sleep are probably influenced by different mechanisms.

Key words:

Obstructive sleep apnea hypopnea, rhythmic masticatory muscle activity, sleep bruxism, tooth grinding, arousal, electromyography

1. Introduction

Some patients report concomitant complaints related to sleep bruxism (SB) and obstructive sleep apnea-hypopnea (OSAH) [1-6]. However, the strength and specificity of this association during sleep remain debated in the literature. [7-8]. In a previous paper, we reported from a nonconsecutive case series our attempt to determine causality between SB and OSAH using a temporal analysis of co-occurrences of jaw muscle activity and apnea-hypopnea [5]. In summary, our results showed that almost half the SB events (i.e., rhythmic masticatory muscle activity – RMMA) occurred after sleep apnea-hypopnea events, suggesting a weak association, and that whereas some SB-related RMMA were concomitant to OSAH, others were probably not related to apnea-hypopnea. Hence, no definite conclusion could be drawn on the strength of the association or on causality between OSAH and SB [9-10]. Various methodological factors may explain these discrepencies.

For example, the use of different methods to assess SB presence (e.g., questionnaire alone vs. advanced sleep recording and scoring methods) is probably a major cause of discrepancy [1-8]. A recent sleep laboratory study in a large general population sample with wide age range failed to show any strong correlations between sleep breathing variables and SB [11]. However, that study was performed without audio-video recording to confirm the specificity of SB. In fact, recording and scoring of jaw muscle activity during sleep revealed that various types of muscle activity are not specific to SB events. These include myoclonic contraction, swallowing, sighing, and idiopathic or natural jaw muscle

activity [12-13]. To assess the specificity of the putative association between OSAH and SB, it is critical to perform a discriminant analysis to distinguish typical RMMA in SB from other types of orofacial motor activity using polysomnography and audio-video recording (PSG-AV).

In this study, because we know that a weak temporal sequence of OSAH triggers RMMA [5], we hypothesized that the number of AH events will correlate with the number of SB events, further supporting a weak association between OSAH and SB. The analysis was performed in subjects who reported concomitant SB-tooth grinding and sleep apnea-hypopnea complaints, in other words, patients with suspected concomitant OSAS and SB.

2. Methods

2.1. Subject characteristics and study design

Fifty-nine Japanese patients (47 male, 12 female) with a mean age of 44.8 (SD 10.8, range 22–72) years reporting awareness of breathing cessation and tooth grinding as well as signs and symptoms of OSAH and SB participated in this study. Subjects spent one night in a sleep clinic to confirm the presence of OSAH and SB. Patients with OSAH reported 1) unintentional sleepiness episodes during daytime, unrefreshing sleep, or poor sleep quality; (2) sudden awakenings with breathing cessation, gasping, or choking during sleep; and (3) bed partner’s reports of loud snoring and/or breathing cessations during sleep. Patients with SB reported 1) awareness of tooth-grinding sounds or tooth clenching during sleep, as reported by sleep partners or family members; 2) jaw muscle discomfort, fatigue, or pain and jaw lock upon awaking; and 3)

clinician-observed tooth wear. Subjects were excluded if they had history of major neurologic, psychiatric, or sleep disorders (e.g., REM behavior disorder, periodic leg movements during sleep) or used psychoactive medication, had a temporomandibular disorder (e.g., orofacial pain, mouth opening limitation), or were edentate.

This study was approved by the ethics committee of Hokkaido University Hospital (No.010-0033). Written informed consent was obtained from all subjects prior to participation. Ten of the 59 subjects also participated in our previous study [5].

2.2. Polygraphic sleep recording

The 59 subjects slept for 1 night in a sleep laboratory, and polysomnographic (PSG) recording and scoring were performed, as described below. The recording variables included electroencephalograms (EEGs) at C_3-M_2, C_4-M_1, O_1-M_2, and O_2-M_1; right and left electrooculogram; electromyogram (EMG) of the submental, masseter, and anterior tibialis muscle; and electrocardiogram. Body positions were detected using a position sensor equipped with a 3D accelerometer. Sleep breathing variables included airflow (with a nasal/oral thermistor), chest and abdominal effort, and SpO_2 measured by pulse oximetry. Recordings were performed with an Alice 5 PSG system (Philips Electronics, Amsterdam, the Netherlands). PSG-AV and masseter recordings were used for SB scoring. The masseter side selected for analysis was randomized across subjects: right side for 30 subjects and left side for 29 subjects.

All recording signals were amplified and analogue-to-digital (A/D)

converted at a 2-kHz sampling frequency. All scorings were performed offline using commercial software (Alice Sleepware, Philips Electronics) and LabChart 5 (ADInstruments, Bella Vista, NSW, Australia). Masseter EMG data were high-pass filtered at 20 Hz and converted to absolute values using LabChart 5. Sleep stages were scored according to standard criteria [14].

2.3. Assessment of breathing and masseter muscle activity

Sleep apnea and hypopnea events were scored according to standard criteria, with apnea-hypopnea index (AHI) threshold of 5 events/h or more [14-15].

EMG bursts of sleep rhythmic masticatory muscle activity with SB (RMMA-SB) and other oromotor activities were assessed according to published criteria [13,14-16,17-19]. The amplitude threshold was set at twice the baseline activity [13]. Bursts with a greater amplitude than the value with duration exceeding 0.25 seconds were selected after onset of sleep until wake-up in the morning.

Other oromotor activities observed during wake stage were classified as wake oromotor activity after sleep onset (Wake-OMA). The sleep related oromotor activity (OMA) (e.g., swallowing, coughing, or face scratching), other than RMMA/SB, were classified as sleep oromotor activity (Sleep-OMA) based on PSG and audio-video (AV) scoring. Unidentified bursts, i.e., face hidden by a blanket, were excluded from the analysis.

SB events were estimated by localizing rhythmic masticatory muscle activity (RMMA) in masseter EMG traces. EMG events separated by 3-second intervals were recognized as RMMA/SB episodes 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). Then EMG bursts included in RMMA/SB episodes were scored as RMMA/SB bursts (Fig. 1) .

Inclusion criteria for SB diagnosis based on PSG-AV recoding was the presence of >2 RMMA/SB episodes/h and/or >25 RMMA/SB bursts/h [15,17].

2.4. Statistical analysis

Spearman's rank correlation coefficient analyses were performed on data from the 59 subjects. Dependent variables were the number of RMMA/SB episode, RMMA/SB bursts, Sleep-OMA bursts, and Wake-OMA bursts per hour. Independent variables were 1) the apnea-hypopnea index (AHI), or total number of apnea and hypopnea events(AH events) per hour of sleep; 2) the arousal index (AI) = (total number of wake arousals during sleep period including sleep related micro arousals (3–15 sec))/hr) [20]; 3) body mass index (BMI); 4) gender, with male–female classification treated as a categorical factor using dummy variables (female=0, male=1); and 5) age.

For the correlation analysis, statistical significance was set at $p<0.05$. Correlations with AHI, AI, BMI, age, and gender were investigated for each number of bursts. Analyses were performed using Microsoft Office Excel 2007 (Microsoft Co.) and Statcel 2 (OMS Publishing Inc., Tokorozawa, Japan).

3. Results

3.1. Polysomnographic sleep data analysis

Sleep macrostructure data are presented in Table 1.

3.2. Sleep breathing data

Most detected and scored AH events were obstructive apnea-hypopnea (91.5%). The mean AHI was 16.2 (SD 16.3). Forty-one (69%) of the subjects were diagnosed with OSAH using AHI >5/h (range 0–60) (Fig. 2). Although all subjects had a history compatible with sleep apnea, one subject showed no AH event during PSG recording.

3.3. Masseter EMG data

Overall masseter EMG bursts (regardless of type) had a mean index of 65.8/h (Table 2). Scorable bursts were classified into RMMA/SB bursts within RMMA/SB episodes, Sleep-OMA bursts, or Wake-OMA bursts. Mean number for each burst index per category was 20.6/h (31.3%), 22.1/h (33.6%), and 17.5/h (26.6%), respectively. Of all masseter EMG traces, 7.8% were scored as unidentified bursts (mean=5.1/h) due to the face hidden by a blanket.

The mean RMMA/SB episode index (episodes/h) was 5.1/h. Forty six subjects presented more than 2 episodes/h with RMMA/SB bursts, and only 13 subjects presented more than 25 bursts/h. In all, 46 subjects (78.0%) were diagnosed with SB, having both >2 episodes/h and/or 25 bursts/h [15,17-18].

3.4. Concomitant conditions

Of the 46 SB patients with high RMMA/SB frequency, 30 showed AHI>5/h and were considered as having concomitant OSAH and SB (Fig.2). The distributions

of the number of each EMG burst type and the AHI for the 59 participants are shown in Fig. 3. Although all 59 subjects reported a concomitant history of both SB and OSAH, only 57 had at least one or both conditions confirmed by sleep laboratory data. Of the total sample, 50.8% had both diagnoses, 18.6% had OSAH diagnosis alone, and 27.1% had SB diagnosis alone. As a negative finding, 3.3% had neither diagnosis.

3.5. Correlation analysis

Correlation coefficients and p-values for each number of bursts and episodes with AHI, AI, BMI, age, and gender are shown in Table 3. AHI did not show significant correlation with RMMA/SB episodes nor RMMA/SB bursts. RMMA/SB episodes were significantly correlated with AI. RMMA/SB bursts were significantly correlated with AI and age. Sleep-OMA bursts were significantly correlated with AHI. Wake-OMA bursts were significantly correlated with BMI. However, these correlations were moderate.

4. Discussion

Self-reported and clinical diagnoses of concomitant SB and OSAH are poorly confirmed by sleep laboratory recordings. These results also suggest that: 1) sleep arousals in patients with concomitant SB and OSAH are weakly associated with RMMA/SB onset; and 2) apnea-hypopnea events appear to be related to a higher occurrence of other types of sleep oromotor activity, and are not dominant with RMMA-SB activity. We therefore did not confirm our

hypothesis: the number of AH events will correlate with number of SB events, and RMMA-SB and OSAH activity genesis during sleep are probably influenced by different mechanisms as discussed below (e.g., not arousal-related to RMMA/SB but to other types of oromotor activity that may be due to oral dryness-mouth breathing, fluctuation in oxygen level, etc).

4.1. Discrepancy between self-reports and clinical diagnosis and sleep laboratory diagnosis

Previous studies suggested that fewer than 50% of subjects with awareness of tooth grinding, based on questionnaires, were confirmed as SB patients in a sleep laboratory [11,17]. In the present sleep laboratory study using the above criteria (see Methods), SB was confirmed in 78.0% of self-reported and clinically observed SB patients, and OSAH was confirmed in the sleep laboratory in 69% of patients.

The discrepancy between self-reports and clinically observed conditions and sleep laboratory diagnosis was acceptable in the present study. However, this discrepancy would be critical when co-occurring AH-OSAH and RMMA-SB are investigated. We found concomitant activity in only half the subjects, regardless of self-reported awareness and history.

4.2. Relationship between RMMA/SB, Sleep-OMA, and AH event

Previous studies suggested that sleep arousal could be associated with the onset of both sleep OSAH and SB [21,22]. In previous reviews, RMMA-related arousal with autonomic sympathetic activation was speculated to be part of

sleep preservation, to oropharyngeal lubrication and re-opening of the upper airway [9,23-24]. In a young population of SB subjects, an association was found between sleep arousal and RMMA genesis for about 80% of events, and in a general population of all ages, this percentage dropped to about 50% [11] and [25]. Other mechanisms therefore appear to be involved in RMMA/SB genesis in SB subjects. In the present study, a low correlation was found between the number of RMMA/SB bursts and the arousal index, which does not support a predominant role of sleep arousal in the presence of concomitant AH and RMMA.

Considering the positive association between OMA and AHI as well as previous findings that most AH events and arousals occur in a close temporal relationship [26], it is possible that the genesis of co-occurring SB and OSAH in this subset of patients may be due to different mechanisms during sleep.

4.3. Association between SB and OSAH

We previously investigated the causality of the genesis of RMMA/SB and AH events in a temporal association study [5]. We found that although most RMMA/SB events occurred after AH events in patients with concomitant OSAH and SB, some also occurred before. However, the cause should precede the effect in a strong causality assessment. This temporal association does not support a direct cause-and-effect relationship, but rather an indirect association. Therefore, mechanisms other than a dominant role of sleep arousal might explain the co-occurrence. The presence of other movements that were not specific to SB could provide an alternative explanation [9,12-13]. SB may have

co-occurred with periodic limb movements in a subgroup of individuals [27]. Furthermore, masseter activity was frequently observed with sleep-related respiratory activity in patients with OSAH, but in only about 1/3 of events associated with sleep arousal [28]. These findings suggest that when EMG bursts of masticatory muscle are observed during sleep, activity such as swallowing, yawning, or body movements may influence the probability of observing oromotor activity that is non-specific to RMMA/SB during sleep in OSAH patients [12-13]. In the present study, a low association was found between SB incidence and AH (i.e., concomitant in only half the subjects), along with a low association with sleep arousal. We therefore concluded that these OSAH patients presented a secondary form of SB [5], and that the SB mechanism may differ between OSAH patients and normal healthy subjects.

4.4. Limitations

Whereas this study provides new evidence to challenge the frequent clinical observation that OSAH and SB co-occur,, several limitations should be noted to avoid misleading interpretations or extrapolations. First, we did not consider autonomic heart rate variability or sympathetic nerve activity. Second, the cross-sectional study design did not allow drawing definite conclusions about cause-and-effect relationships among the variables. To overcome these limitations, a longitudinal intervention study design is required. Third, the weak association might be partially due to the inclusion of subjects who self-reported signs and symptoms of OSAH and/or SB but who were not diagnosed as OSAS and /or SB by PSG-AV in our single night of sleep analysis. Fourth, all subjects

were patients in a sleep clinic, and no control subjects were included to assess the strength and specificity of the association. Fifth, influence of female–male distribution needs to be further evaluated. Sixth, in this study, we did not analyze AHI or EMG bursts for each sleep stage (N1, N2, N3, REM). The addition of this data in future studies could increase the accuracy of the findings. Seventh, we did not assess upper airway resistance syndrome (UARS) or snoring. Future studies should include UARS to better challenge the relationship between breathing and SB. These limitations should be controlled for in future studies.

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Conflicts of interest:

All authors certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

GL holds governmental grant on pain, sleep and trauma plus free access to oral appliances for research purposes (Narval, Somnomed) and this not in conflict with the present paper content.

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Figure legends

Fig.1
Flow chart of selection and classification of each burst group
RMMA: Rhythmic Masticatory Muscle Activity

SB: Sleep Bruxism
Sleep-OMA: sleep oromotor activity other than RMMA/SB
Wake-OMA: wake oromotor activity after sleep onset

Fig.2
A Venn diagram representation of subject distribution based on RMMA/SB and OSAH sleep laboratory diagnosis

NB: 41patients were diagnosed as OSAH, with an AHI exceeding 5/h.
46 patients were diagnosed as SB, with an RMMA/SB episode index exceeding 2/h and/or number of RMMA/SB bursts exceeding 25/h.
Dx: Diagnosis; OSAH: Obstructive Sleep Apnea Hypopnea; RMMA: Rhythmic Masticatory muscle Activity; SB: Sleep Bruxism

Fig.3
Scatter diagrams of AHI and each RMMA burst

AHI: Apnea Hypopnea Index
RMMA: Rhythmic Masticatory Muscle Activity
SB: Sleep Bruxism
Sleep-OMA: sleep oromotor activity other than RMMA/SB
Wake-OMA: wake oromotor activity after sleep onset

*****Significant differences are indicated in **bold**.

Table1 Data of sleep and apnea-hypopnea event (n=59)

	Mean	S.D.
Physical data		
Age	44.8	10.8
BMI	25.8	4.2
Sleep data		
SPT(min)	382.5	43.2
TST (min)	337.2	57.9
TIB (min)	402.0	54.0
Sleep efficiency TST/TIB (%)	87.0	32.7
Sleep stage distribution		
N1/SPT(%)	7.0	10.7
N2/SPT(%)	54.1	5.7
N3/SPT(%)	15.0	12.0
R/SPT(%)	12.1	7.6
W/SPT(%)	11.7	13.7
Arousal		
Number of total arousal	52.7	52.7
Arousal Index (number of total arousal	9.2	9.3
Numbers of apnea-hypopnea event		
Obstructive apnea and hypopnea	83.7(91.5%)	87.1
Central apnea	4(4.4%)	9.4
Mixed apnea	3.8(4.2%)	14.2
AHI	16.2	16.3

SPT: Sleep period time
TST: Total sleep time
TIB: Time in bed asleep and awake
R: REM
W: Wake

Table2 Type and Frequency of masseter muscle (n=59)

	Mean	S.D.
Total number of RMMA/SB episode	32.5	29.3
Number of phasic episode	11.6	18.1
Number of tonic episode	8.5	8.7
Number of mixed episode	12.4	12.0
RMMA/SB episode (/h)	5.1	4.5
RMMA/SB burst (/h)	20.6	31.8
Phasic burst (/h)	16.7	31.4
Tonic burst (/h)	3.9	4.7
Sleep-OMA burst (/h)	22.1	30.3
Wake-OMA burst (/h)	17.5	18.2
Excluded burst(/h)	5.1	9.9
Total number of burst (/h)	65.8	57.9

Total burst: all of bursts after onset of sleep until wake-up in the morning
(/h): sleep period time hour
Excluded burst: Unidentified bursts with face hidden by blanket
Sleep-OMA: Other sleep oromotor activity
Wake-OMA: Wake stage oromotor activity
RMMA: Rhythmic Masticatory Muscle Activity
SB: Sleep Bruxism

Table 3 correlation analyses

	R	P-value
RMMA/SB episode (/h)		
AHI	0.0945	0.472
AI	0.2666	**0.042***
BMI	0.1433	0.275
M/F	0.1731	0.187
Age	-0.1832	0.163
RMMA/SB burst (/h)		
AHI	0.0685	0.602
AI	0.2595	**0.048***
BMI	0.0950	0.470
M/F	0.1311	0.318
Age	-0.2785	**0.034***
Sleep-OMA burst (/h)		
AHI	0.4151	**0.002***
AI	0.2333	0.076
BMI	0.1755	0.181
M/F	0.1582	0.228
Age	-0.1189	0.365
Wake-OMA burst (/h)		
AHI	-0.1587	0.227
AI	0.2461	0.061
BMI	-0.2603	**0.047***
M/F	-0.0668	0.611
Age	-0.1320	0.315

RMMA/SB: Rhythmic Masticatory Muscle Activity/ Sleep Bruxism
Sleep-OMA: Other sleep oromotor activity
Wake-OMA: Wake stage oromoter activity
AHI: Apnea hypopnea index
AI: Arousal index
BMI: Body mass index
***=p<0.05**

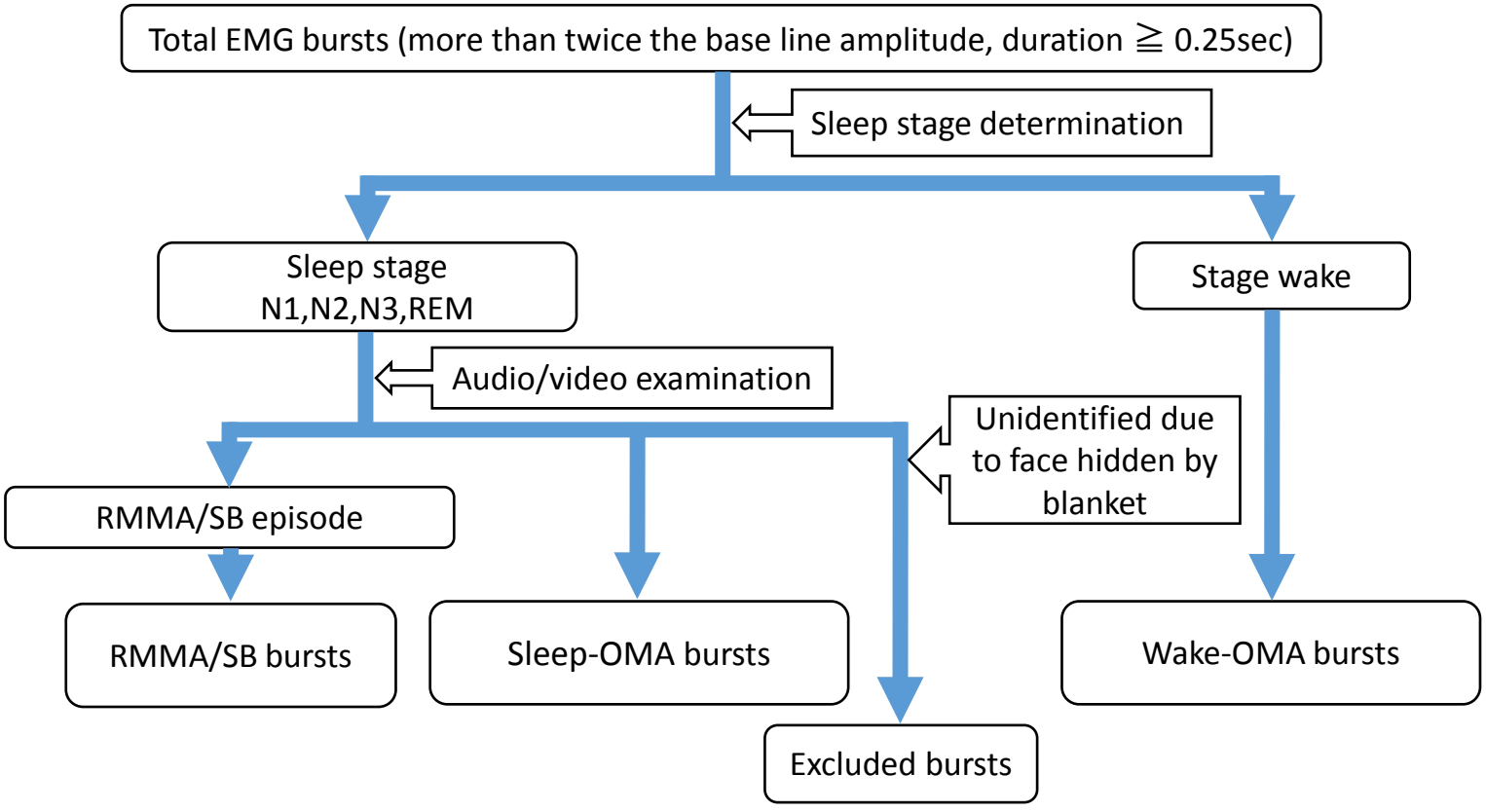

Total EMG bursts (more than twice the base line amplitude, duration ≧ 0.25sec)
Sleep stage determination
Sleep stage
N1,N2,N3,REM
Stage wake
Audio/video examination
Unidentified due to face hidden by blanket
RMMA/SB episode
RMMA/SB bursts
Sleep-OMA bursts
Wake-OMA bursts
Excluded bursts

Fig.1

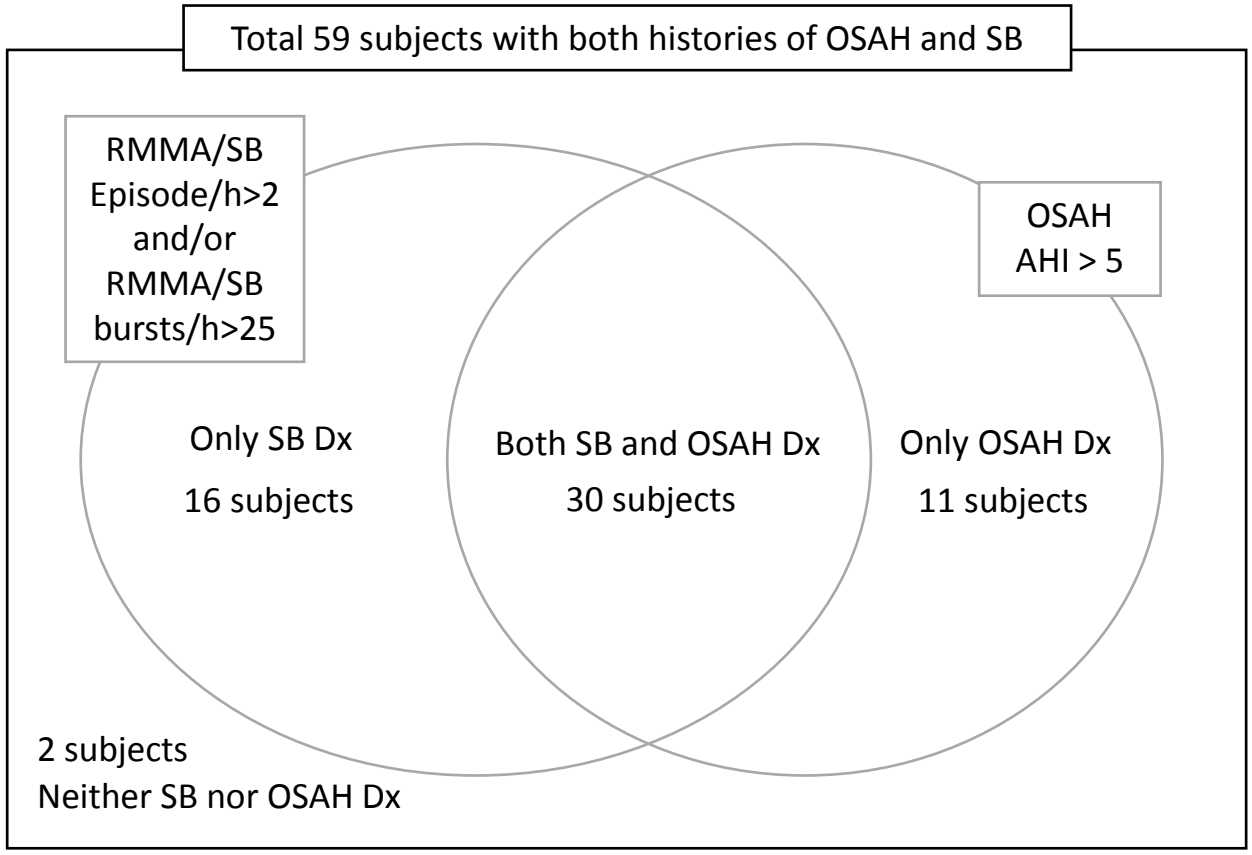

Total 59 subjects with both histories of OSAH and SB
RMMA/SB Episode/h>2 and/or RMMA/SB bursts/h>25
OSAH AHI > 5
Only SB Dx
16 subjects
Both SB and OSAH Dx
30 subjects
Only OSAH Dx
11 subjects
2 subjects
Neither SB nor OSAH Dx

Fig.2

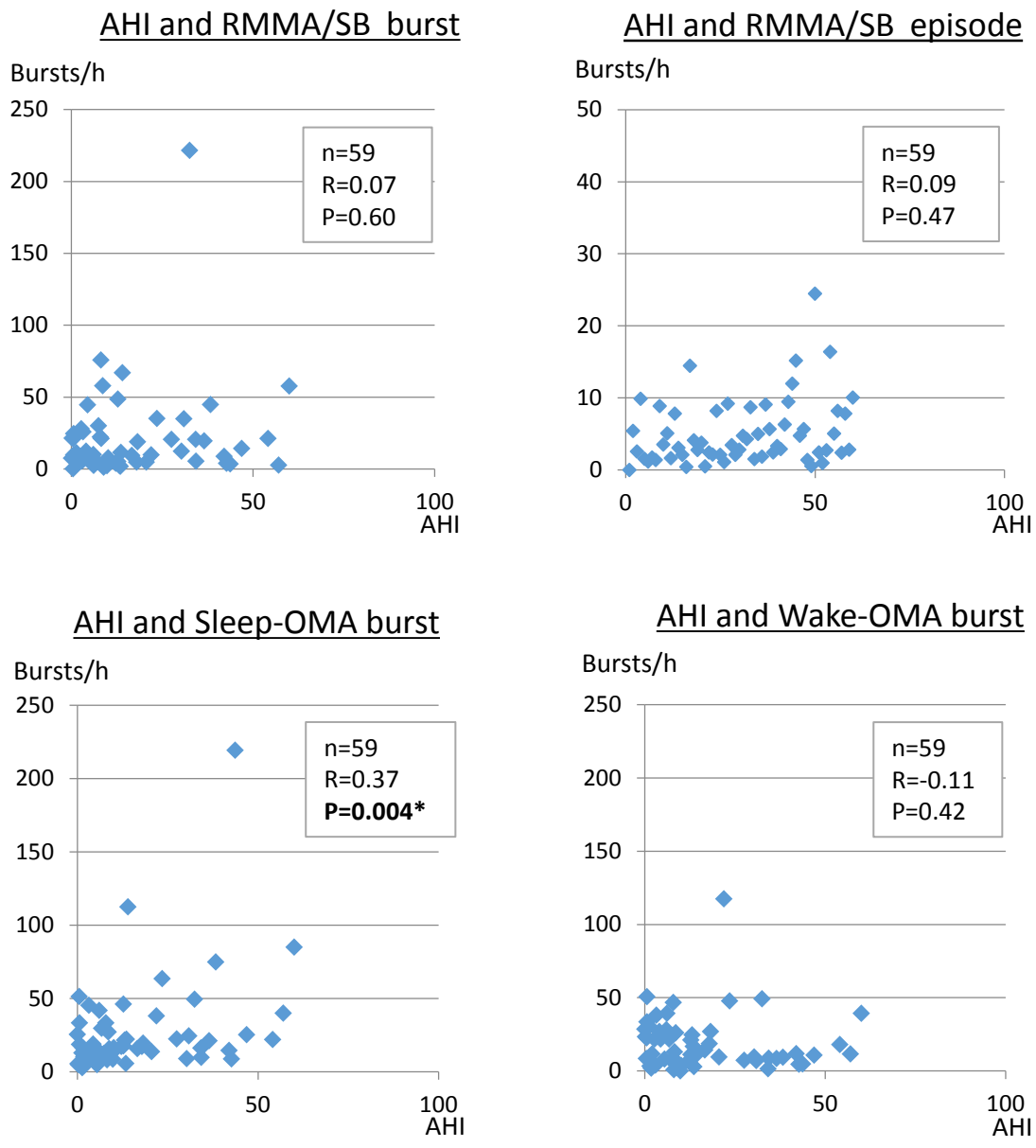

AHI and RMMA/SB burst
Bursts/h
250
200
150
100
50
0
0
50
100
AHI
n=59
R=0.07
P=0.60
AHI and RMMA/SB episode
Bursts/h
50
40
30
20
10
0
0
50
100
AHI
n=59
R=0.09
P=0.47
AHI and Sleep-OMA burst
Bursts/h
250
200
150
100
50
0
0
50
100
AHI
n=59
R=0.37
P=0.004*
AHI and Wake-OMA burst
Bursts/h
250
200
150
100
50
0
0
50
100
AHI
n=59
R=-0.11
P=0.42

Fig.3