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Daytime polysomnography and portable recording device for diagnosis and CPAP therapy in patients with obstructive sleep apnea syndrome

(閉塞性睡眠時無呼吸症候群の診断および
CPAP 療法のための検査に関する研究)

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Daytime polysomnography for early diagnosis and treatment of patients with suspected sleep-disordered breathing

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Abstract

Excessive daytime sleepiness (EDS) is a common complaint among patients with sleep disordered-breathing (SDB). Population-based studies on traffic and industrial accidents suggest a relationship between EDS and life-threatening events, and adults with EDS have cognitive and memory problems. Nocturnal polysomnography (nPSG) is essential for diagnosing SDB but it is time and energy consuming. We examined the usefulness of daytime polysomnography (dPSG) for the early diagnosis and treatment of patients with suspected SDB.

We studied 108 consecutive patients, aged 51.9 ± 13.5 years (mean ± SD). All patients underwent dPSG and nPSG. The number of apnea/hypopnea episodes per hour (apnea/hypopnea index; AHI) and the number of 3% desaturation episodes per hour (desaturation index; DSI) were calculated. All patients were classified into two groups. The REM group consisted of subjects who had an AHI ≤ 25/h, $AHI_{REM}/AHI_{NREM} > 2$, and $AHI_{NREM} < 15$/h. Those who did not satisfy these criteria were placed in the NREM group. Continuous positive airway pressure (CPAP) titration was performed for patients whose AHI was ≥ 20/h on dPSG.

Using the international classification of sleep disorders, 96 patients were diagnosed as obstructive sleep apnea (including five upper airway resistance syndrome patients), six patients were snoring, four had idiopathic hypersomnia due to a medical condition, and two had circadian rhythm sleep disorders. The sensitivity of dPSG for AHI was 81.0%, specificity was 100%, and accuracy was 83.5%. The sensitivity and accuracy of dPSG for AHI in the REM group were considerably lower than in the NREM group. There was no significant difference for optimal CPAP between dPSG and nPSG. In the

five patients with UARS, their AHI, DSI, and arousal index on dPSG were 0.92 ± 1.2/h, 2.9 ± 3.4/h, and 29.3 ± 3.5/h, respectively, and their AHI and DSI on nPSG were 3.2 ± 2.5/h and 2.8 ± 2.4/h, respectively. However, their respiratory effort-related arousals were 37.9 ± 7.4/h and arousal index 33.2 ± 6.3/h. The five patients with UARS were also treated with CPAP, and their daytime sleepiness was improved.

Although dPSG has limitations, these results indicate that dPSG recording is clinically useful for the diagnosis of and determination of types of treatment in patients with suspected SDB.

Key words sleep disordered breathing, upper airway resistance syndrome, excessive daytime sleepiness, daytime polysomnography, continuous positive airway pressure titration,

Introduction

Excessive daytime sleepiness (EDS) is a common complaint among patients with sleep disordered-breathing (SDB). Population-based studies on traffic and industrial accidents suggest a relationship between EDS and life-threatening events [1, 2], and adults with EDS have cognitive and memory problems [3]. Therefore, there is a social demand for the early and proper diagnosis and treatment of these patients. Nocturnal polysomnography (nPSG) is essential for evaluating the quantity and quality of sleep [4]; however, the recording and analysis of nPSG are time consuming, which means that patients with suspected SDB cannot immediately undergo nPSG, thereby delaying the detection of SDB and the implementation of any associated therapy.

Portable monitoring instruments have been recognized as useful tools for screening for obstructive sleep apnea (OSAS) [5, 6]. According to the American Academy of Sleep Medicine Association guidelines, a portable monitoring device in an unattended setting is not recommended to rule in, rule out, or both rule in and rule out a diagnosis of OSAS [7]. Portable monitoring cannot provide information on total sleep time and sleep structure, which results in lower reliability in recording the number of apnea/hypopnea episodes per hour (apnea/hypopnea index: AHI). Moreover, it is not adequate in some types of SDB, such as upper airway resistance syndrome (UARS), in which the AHI is often < 5/h despite frequent arousals and subsequent poor quality sleep [4]. Consequently, the limitations of portable monitoring to detect SDB other than OSAS should be recognized.

The American Academy of Sleep Medicine recommended that continuous positive airway pressure (CPAP) titration must be performed during the night under full

polysomnography (PSG) and a manual protocol [4]. Alternative protocols have been studied for early initiation of treatment, such as split-night diagnostic/titration studies [8] and home-attended manual or unattended automated CPAP devices [9, 10].

In this study, we compared daytime and nighttime evaluations of sleep architecture and abnormal respiratory events to investigate whether daytime polysomnography (dPSG) is clinically useful in the diagnosis and CPAP titration of patients with suspected SDB.

Patients and methods

Patients

We recruited consecutive patients who were referred to our sleep clinic from 2000 to 2002. All patients underwent dPSG, nPSG, and routine physical examination. These patients were diagnosed by sleep physicians and pulmonologists based on their symptoms, routine examination, and PSG. Patients with narcolepsy, severe chronic obstructive pulmonary disease, and neuromuscular diseases were excluded from this study. Finally, we studied 108 patients—99 men and nine women—who all gave written informed consent. The Nagoya University ethical committee approved all procedures associated with this study.

Study design

All patients underwent dPSG prior to nPSG, with a minimum interval of a week between the two studies. No treatment was initiated between the two studies. The duration of dPSG was approximately two hours (recorded from 1 p.m.). The nPSG was started from 9 p.m. and ended at 6 a.m. After natural sleep was recorded during the dPSG and nPSG, an optimal pressure for the CPAP device was determined for patients whose AHI was ≥ 20/h.

Polysomnography

Standard PSG (ALICE3, Respironics Inc., Murrysville, PA) with pulse oximetry was performed in all patients during both daytime and nighttime. C3–A2, C4–A1, O1–A2, and O2–A1 electroencephalograms, electrooculograms, electromyograms (mentalis, legs, and diaphragm), and electrocardiograms (bipolar CM_5 and standard V_5 lead positions) were recorded, and respiration was monitored using an oronasal thermistor and a thoracoabdominal piezo sensor in both dPSG and nPSG. The esophageal pressure was measured using an esophageal intraluminal pressure catheter (GMS, Tokyo, Japan) in nPSG.

Scoring

All recordings were double checked by experienced scorers. The technicians who scored both dPSG and nPSG were blinded to patient information. The sleep stage was scored by visual analysis according to the criteria of Rechtshaffen and Kales [11]. Sleep efficiency was calculated as the total sleep time divided by the time spent in bed. Apnea was defined as cessation of airflow through the mouth and nose for ≥ 10 seconds, and hypopnea was defined as an obvious reduction in airflow accompanied with either an oxygen desaturation of ≥ 3% or arousal, also for ≥ 10 seconds. Arousal was defined by a modification of the three-second rule of the American Sleep Disorders Association: an abrupt shift in EEG frequency including theta, alpha, and/or frequencies greater than 16

Hz and, during REM, arousal was scored according to three-second EEG changes with submental EMG amplitude increases [12]. AHI, the number of episodes of oxygen desaturation ≥ 3% per hour (desaturation index: DSI), lowest oxygen saturation (lowest SpO_2), and the number of arousals per hour (arousal index) were calculated. Patients with an AHI ≥ 5/h were diagnosed as having OSAS [13]. To be considered as UARS patients, patients must have had a complaint of daytime sleepiness, with a score on the Epworth Sleepiness Score (ESS) [14] of > 9, an AHI of < 5/h, and AHI and respiratory effort-related arousals (RERAs) of ≥ 5/h with an additional measurement of esophageal pressure on nPSG [15]. The sensitivity, specificity, and accuracy of AHI, lowest SpO_2, and DSI were calculated from the dPSG and nPSG results. We classified all patients into two groups according to the rule suggested by O'Connor and colleagues [16]. One group was the REM group, with mild SDB that occurred almost exclusively during REM sleep, and the other was the NREM group, with mild to severe SDB that occurred during both REM and non-REM sleep. The REM group consisted of subjects with an AHI ≤ 25/h, $AHI_{REM}/AHI_{NREM} > 2$, and $AHI_{NREM} < 15$/h. Those who did not satisfy these criteria were allocated to the NREM group.

CPAP titration procedure

Different technicians, blinded from patients' information, carried out CPAP titration on dPSG and nPSG. CPAP was administered via machine (Tranquility, Respironics Inc., Murrysville, PA) with remote controlled adjustment. The initial CPAP setting was 4 cm H_2O. The pressure was progressively increased in increments of 1 cm

H_2O to eliminate respiratory events, arousals, and oxyhemoglobin desaturation below 90%, and then 1 cm H_2O adjustments (up and down) were made with sleep stage and position until the minimum CPAP necessary to eliminate respiratory arousals (obstructive apneas and hypopneas, and repetitive snoring-associated arousals) had been carefully defined. On dPSG, CPAP titration was performed in the same manner as nPSG.

Daytime sleepiness

For the ESS [14], patients scored themselves on a scale from 0 (not at all likely to fall asleep) to 3 (very likely to fall asleep), according to how easily they would fall asleep in eight different situations, with a possible overall score of 0 to 24. Daytime sleepiness was evaluated for all patients before their study and after treatment with CPAP for three months.

Data analysis

Data are presented as means ± standard deviation (SD). The mean AHI, lowest SpO_2, DSI, and arousal index obtained with dPSG were compared to those obtained with nPSG using a paired t-test. Probability (P) values < 0.05 were considered statistically significant. Bland–Altman plots [17] were used to investigate the agreement between nPSG and dPSG for AHI, lowest SpO_2, DSI, and arousal index. Each patient

was included in one of the following categories: true positive (TP), patients with abnormal respiratory data in both dPSG and nPSG; false positive (FP), patients with abnormal respiratory data only in the dPSG; false negative (FN), patients with abnormal respiratory data only in the nPSG; and true negative (TN), patients with normal respiratory data in both dPSG and nPSG. The sensitivity, specificity, and accuracy were calculated according to the following equations: sensitivity = TP / (TP + FN); specificity = TN / (FP + TN); and accuracy = TP + TF / (TP + FP + FN + TN).

Results

The baseline characteristics of all patients are shown in Table 1. According to the international classification of sleep disorders [18], 96 patients were diagnosed as OSAS (including five UARS patients), six patients were snoring, four had idiopathic hypersomnia due to a medical condition, and two had circadian rhythm sleep disorders.

There was no significant difference for sleep efficiency between dPSG and nPSG, but it tended to be worse on dPSG (68.1 ± 27.6 vs. 73.7 ± 20.5%). There were no significant differences between dPSG and nPSG for AHI, DSI, and arousal index (AHI: 29.4 ± 29.0 vs. 31.1 ± 23.5/h; DSI: 29.0 ± 28.4 vs. 26.5 ± 23.6/h; arousal index; 40.0 ± 19.5 vs. 42.5 ± 17.6/h). The lowest SpO_2 on dPSG was significantly higher than that on nPSG (83.5 ± 9.8 vs. 77.3 ± 10.3%, $p < 0.05$) (Table 2). The mean difference in AHI between dPSG and nPSG was 4.0/h, giving limits of agreement of –41.5 to 49.5/h. The mean difference in DSI between dPSG and nPSG was 2.9/h and the SD of the differences was –45.9 to 51.9/h. The mean difference in the lowest SpO_2 between dPSG and nPSG was –7.0%, giving limits of agreement of –33.1 to 19.0%. The mean difference in arousal index between dPSG and nPSG was –2.6/h and the SD of the differences were –45.0 and 39.6/h (Fig. 1). The sensitivity of dPSG for AHI was 81.0%, specificity was 100%, and accuracy was 83.5%. For the lowest SpO_2 with dPSG, the sensitivity was 77.5%, specificity 77.7%, and accuracy 70.4%. The sensitivity, specificity, and accuracy of dPSG for DSI were 84.9%, 61.1%, and 80.2%, respectively (Table 3).

Thirteen patients (12.0%) were classified into the REM group (mean age 47.0 ± 11.9 years, BMI 26.3 ± 3.9 kg/m^2, ESS 10.2 ± 4.6) and the other 95 (88.0%) patients

into the NREM group (mean age 51.5 ± 13.9 years, BMI 26.5 ± 5.0 kg/m^2, ESS 9.3 ± 5.4). There were no significant differences in age, BMI, and ESS between the two groups (Table 4). There were no significant differences between dPSG and nPSG for AHI in the NREM group (30.3 ± 29.7 vs. 31.7 ± 22.9/h). However, AHI on dPSG was significantly lower than that on nPSG in the REM group (2.8 ± 3.7 vs. 7.6 ± 3.9/h, $p < 0.05$; Table 4). The mean difference in AHI between dPSG and nPSG was 1.4/h, giving limits of agreement of –41.6 to 44.4/h in the NREM group. The mean difference in AHI between dPSG and nPSG was 4.8/h and the SD of the differences was –2.9 to 12.5/h in the REM group (Fig. 2). The sensitivity and accuracy of dPSG for AHI in the REM group were considerably lower than those in the NREM group (sensitivity: 20.0 vs. 81.9%; accuracy: 15.3 vs. 84.1%, respectively). The specificity of dPSG for AHI in both REM and NREM groups was 100% (Table 5). There was no significant difference in the arterial carbon dioxide tension between the REM and NREM groups. In contrast, arterial oxygen tension (PaO_2) in the REM group was significantly decreased compared to the NREM group (83.6 ± 12.1 vs. 90.5 ± 9.8 mmHg, $P < 0.05$) (Table 4).

Sixty-seven patients satisfied the criteria for CPAP titration on dPSG. There was no significant difference in optimal pressure during dPSG compared with that during nPSG (13.6 ± 1.9 vs. 14.2 ± 1.9 cmH_2O). However, 13 patients refused or did not tolerate CPAP therapy. In the final analysis, 54 of the 108 patients (50.0%) were prescribed CPAP, 10 (9.2%) underwent surgery, 29 (26.8%) used oral appliances, one (0.9%) was prescribed medications, and 14 (13.1%) were untreated. The ESS of patients after treatment with CPAP for three months was significantly improved over that before the study (9.6 ± 5.6 vs. 6.1 ± 6.0, $p < 0.05$) (Table 1).

In the five patients with UARS, their AHI, DSI, and arousal index on dPSG were 0.92 ± 1.2/h, 2.9 ± 3.4/h, and 29.3 ± 3.5/h, respectively, and their AHI and DSI on nPSG were 3.2 ± 2.5/h and 2.8 ± 2.4/h. However, their RERAs were 37.9 ± 7.4/h and arousal index was 33.2 ± 6.3/h (Table 2). The five patients with UARS were also treated with CPAP, and their daytime sleepiness was improved (ESS: 12.0 ± 5.5 to 3.2 ± 1.0) (Table 1).

Discussion

We showed that, according to the Bland–Altman plots, considerable agreement was obtained between nPSG and dPSG for AHI, DSI, lowest SpO_2, and arousal index in all patients. The sensitivity, specificity, and accuracy for AHI, DSI, lowest SpO_2, and arousal index were considerably high. There was no significant difference in optimal CPAP between dPSG and nPSG. In five patients, AHI was < 5/h and the arousal index was 34.2 ± 10.1/h on dPSG, and they were diagnosed on nPSG as having UARS. These results suggest that dPSG allows accurate diagnosis and successful determination of optimal CPAP in patients with suspected SDB.

The sensitivity, specificity, and accuracy of dPSG were considerably high. Our findings were supported by previous studies that showed the usefulness of dPSG [19, 20]. Our finding that the lowest SpO_2 differed significantly between nPSG and dPSG was identical with a previous study [21]. The dPSG was recorded over a shorter time compared with nPSG. Moreover, patients were not as able to sleep sufficiently during dPSG as they are in nPSG; almost all of their sleep structures were stages 1 and 2, and stages REM 3 and 4 were not observed. Oxygen desaturation is more severe during REM sleep than during NREM sleep in patients with OSAS [22]. Therefore, in this study, sleep time and quality might have influenced the differences in the lowest SpO_2 between dPSG and nPSG.

The REM stage is rarely observed during dPSG. Consequently, the agreement of measurement between dPSG and nPSG for AHI in the REM group was worse than in the NREM group. However, the PaO_2 was significantly lower in the REM group than in

the NREM group. Assessment of PaO_2 and home screening tests might be useful in patients with moderate SDB that occurs during REM sleep.

UARS is described as a form of SDB in which repetitive increases in resistance to airflow within the upper airway lead to brief arousals, of which the patient is unaware, and daytime somnolence [23]. Screening for SDB and sleep disorders using portable devices is limited [9]. The screening device does not record the EEG, and repetitive microarousals during the night cannot be detected, so patients who are suspected of UARS might fail to be detected. In our study, AHI on dPSG was not enough to meet the criteria for OSAS in five patients. However, we observed repeated arousal on dPSG, and these patients were diagnosed as having UARS on nPSG including esophageal pressure measurement. Their daytime sleepiness was improved by CPAP therapy. These facts suggest that dPSG-recorded airflow, thoracoabdominal movements, and EEG to observe microarousals during sleep are useful in the detection of UARS.

In Japan, the cost of CPAP for SAS patients is covered by health insurance only when their AHI is ≥ 20/h on nPSG. The observation of airflow, thoracoabdominal movements, and sleep stage is essential for CPAP titration. We performed CPAP titration when a patient's AHI was higher than 20/h on dPSG. There was no significant difference in CPAP pressure between nPSG and dPSG. Furthermore, previous studies showed no significant differences in terms of treatment compliance, symptom improvement, or side effects between pressures selected using daytime or nighttime PSG [24]. These results indicate that dPSG is also useful for early initiation of CPAP therapy.

The subjects who enrolled in this study were referred from general practitioner clinics with suspected SDB. The prevalence of SDB in our study was greater than that

in the general population [25]. This fact may have influenced the calculated sensitivities, specificities, and accuracies. Performing esophageal pressure manometry is invasive and may disturb a patient's sleep. According to previous studies examining the usefulness of dPSG, a daytime nap is different from sleep during nighttime. In this study, we did not perform esophageal pressure manometry in order to allow the patients a good sleep.

We suggest that dPSG is an accurate diagnosis tool for patients with suspected SDB. However, the absence of or shortened REM sleep may influence the AHIs obtained by dPSG [15]. The dPSG may not be as valid as nPSG in patients who are moderately overweight or not overweight [20]. Furthermore, despite the adequate titration of CPAP, pressure changes will be required following dPSG titration [26]. Despite these limitations of dPSG, this method is still reliable for identifying patients with moderate to severe OSAS [19–21] and daytime CPAP titration is an appropriate procedure for the effective titration of CPAP [24, 26].

In summary, dPSG recording is a practical method for evaluating respiratory status and sleep architecture, can be performed in a few hours, and can simultaneously identify the types of apnea. We conclude that dPSG is clinically useful for early and low-cost diagnosis, and for determining treatment strategies in patients with SDB.

References

1. Broman JE, Lundh LG, Hetta J. (1996) Insufficient sleep in the general population. Neurophysiol Clin 26:30-39.
2. Lindberg E, Carter N, Gislason T, Janson C. (2001) Role of snoring and daytime sleepiness in occupational accidents. Am J Respir Crit Care Med 164: 2031-2035.
3. Blagrove M, Alexander C, Horne JA. (1994) The effect of chronic sleep reduction on the performance of cognitive tasks sensitive to sleep deprivation. Appl Cogn Pshycol 9:21-40.
4. Kushida CA, Litner MR, Morgenthaler T, et al. (2005) Practice parameters for the indications for polysomnography and related procedures: An update for 2005. Sleep 28: 499-521.
5. Man GCW, Kang BV. (1995) Validation of a portable sleep apnea monitoring device. Chest 108:388-393.
6. Olson LG, Ambrogetti A, Gyulay SG. (1999) Prediction of sleep-disordered breathing by unattended overnight oximetry. J Sleep Res 8:51-55.
7. Chesson LS, Berry RB, Pack A. (2003) Practice parameters for the use of portable monitoring devices in the investigation of suspected sleep apnea in adults. Sleep 26: 907-913.
8. Sanders MH, Constantino JP, Strollo PJ, et al. (2000) The impact of split-night polysomnography for diagnosis and positive airway pressure therapy titration on treatment acceptance of adherence in sleep apnea/hypopnea. Sleep 23:17-24.

9. Juhasz J, Schillen J, Urbigkeit A, et al. (1996) Unattended continuous positive airway pressure tiration. Clinical relevance and cardiorespiratory hazards of the method. Am J Respir Crit Care Med 154:359-365.
10. Waldhorn RE, Wood K. (1993) Attended home titration of nasal continuous positive airway pressure therapy for obstructive sleep apnea. Chest 104:1707-1710.
11. Rechtschaffen A, Kales A. (1968) A manual of standardized techniques and scoring system for sleep stages of human subjects. Brain Information Service and Brain Research Institute, Los Angeles.
12. American Academy of Sleep Medicine. (1992) EEG arousals: scoring rules and samples. Sleep 15:173-184.
13. American Academy of Sleep Mediciene. (1999) Sleep-related breathing disorders in adults. Recommendations for syndrome definition and measurement techniques in clinical research. Sleep 22:667-689.
14. Johns MW. (1991) A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. Sleep 14:540-545.
15. Guilleminault C, Bassiri A. (2005) Clinical features and evaluation of obstructive sleep apnea-hypopnea syndrome and upper airway resistance syndrome. In: Kryger MH, Roth T, Dement WC (eds) Principles and practice of sleep medicine. Elsvier Saunders, Philadelphia. pp 1043-1052.
16. O'connor C, Thornlet KS, Hanly PJ. (2000) Gender differences in the polysomnographic features of obstructive sleep apnea. Am J Respir Crit Care Med 161:1465-1472.
17. Bland JM, Altman DG. (1986) Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1:307-310.

18. American Academy of sleep medicine. (2005) The international classification of sleep disorders, Diagnostic & coding manual, Second edition. Westchester, IL, USA.
19. Van Keimpema ARJ, Rutgers SR, Strijers RLM. (1993) The value of one hour daytime sleep recording in the diagnosis of sleep apnoea syndrome. J Sleep Res 2:257-259.
20. Series F, Cormier Y, Forge JLA. (1991) Validty of diurnal sleep recording in the diagnosis of sleep apnea syndrome. Am Rev Respir Dis 143:947-949.
21. Sergi M, Rizzi M, Greco M, et al. (1998) Validity of diurnal sleep recording performed by an ambulatory device in the diagnosis of obstructive sleep apnea. Respir Med 92:216-220.
22. Sampol G, Sagales MT, Roca A, et al. (1996) Nasal continuous positive airway pressure with supplemental oxygen in coexistent sleep apnoea-hypopnoea syndrome and severe chronic obstructive pulmonary disease. Eur Respir J 9:111-116.
23. Exar EN, Collop NA. (1999) The upper airway resistance syndrome. Chest 115:1127-1139.
24. Rudkowski JC, Verschelden P, Kimoff RJ. (2001) Efficacy of daytime continuous positive airway pressure titration in severe obstructive sleep apnoea. Eur Respir J 18:535-541.
25. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. (1993) The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 328: 1230-1235.

26. Hoekema A, Stegenga B, van der Aa JG, Meinesz AF, van der Hoeven JH, Wijkstra PJ. (2006) Nap-titration: An effective alternative for continuous positive airway pressure titration. Repir Med 100:705-713.

Figure legend

Fig. 1. Bland-Altman plot for AHI, DSI, lowest Spo_2 and arousal index with dPSG compared with that with nPSG.

The line represents the mean of difference, and dash lines represent the standard deviation of difference. Upper left: the mean difference between AHI obtained with dPSG and nPSG was 4.0 /h giving limits of agreement of –41.5 to 49.5 /h, upper right: the mean difference in DSI between dPSG and nPSG was 2.9 /h and SD of differences was –45.9 to 51.9 /h, lower left: the mean of difference between lowest Spo_2 obtained with dPSG and nPSG was –7.0 % giving limits of agreement of –33.1 to 19.0 % and lower right: the mean of difference in arousal index between dPSG and nPSG was –2.6 /h and SD of difference was –45.0 and 39.6 /h. AHI: the total number of apnea/hypopnea per hour (apnea/hypopnea index), dPSG: daytime polysomnography, nPSG: nocturnal polysomnography, DSI: the total number of 3% oxygen desaturation per hour (oxygen desatiration index), Spo_2: oxygen saturation, and arousal index: the number of arousal per hour.

Fig. 2. Bland-Altman plot for AHI with dPSG compared with that with nPSG in group REM and group NREM.

The line represents the mean of difference, and dash lines represent the standard deviation of difference. Left: the mean difference between AHI obtained with dPSG and nPSG was 1.4 /h giving limits of agreement of –41.6 to 44.4 /h in group NREM and Right: the mean difference in AHI between dPSG and nPSG was 4.8 /h and SD of differences was –2.9 to 12.5 /h in group REM. The group REM: patients who satisfied

with the criterion; AHI ≤ 25 /h, AHI_{REM}/AHI_{NREM} >2, and AHI_{NREM} <15 /h, group NREM: patients who did not satisfy with this criterion, AHI: the total number of apnea/hypopnea per hour (apnea/hypopnea index), dPSG: daytime polysomnography, nPSG: nocturnal polysomnography.

Table 1. The characteristics of patients.

	before the study	after treatment with CPAP for 3 months
Age (years)	51.9 ± 13.5	-
BMI (kg/m^2)	26.4 ± 4.8	-
ESS		
All patients (n=108)	9.6 ± 5.6	6.1 ± 6.0*
UARS patients (n=5)	12.0 ± 5.5	3.2 ± 1.0*

BMI: body mass index, ESS: Epworth Sleepiness Scale, UARS: upper airway resistance syndrome, *: significant between before the study and after treatment with CPAP for 3 months ($p<0.05$).

Table 2. The comparison of sleep efficiency, AHI, lowest Spo_2, DSI and arousal index between nPSG and dPSG in all patients

	nPSG	dPSG
Sleep efficiency (%)	73.3±20.5	68.1±27.6
All patinets (n=108)		
AHI (/h)	31.1±23.5	29.4±29.0
Lowest SpO_2 (%)	77.3±10.3	83.5±9.8*
DSI (/h)	26.5±23.6	29.0±28.4
Arousal index (/h)	42.5±17.6	40.0±19.5
UARS patinets (n=5)		
AHI (/h)	3.2 ± 2.5	0.92 ± 1.2
DSI (/h)	2.8 ± 2.4	2.9 ± 3.4
Arousal index (/h)	33.2 ± 6.3	29.3 ± 3.5
RERAs (/h)	37.9 ± 7.4	-

AHI: the number of apnea/hypopnea per hour (apnea/hypopnea index), arousal index: the number of arousal per an hour, dPSG: daytime polysomnography, DSI: the number of 3% desaturation per hour (desaturation index), lowest SpO_2: the lowest oxygen saturation, nPSG: nocturnal polysomnography, RERAs: respiratory effort-related arousals, UARS: upper airway resistance syndrome, *: significant between dPSG and nPSG ($p<0.05$).

Table 3. The sensitivity, specificity and accuracy in all patients

	sensitivity (%)	specificity (%)	accuracy (%)
AHI (/h)	81.0	100	83.5
Lowest SpO_2 (%)	77.5	77.7	77.5
DSI (/h)	84.9	61.1	80.2

AHI: the number of apnea/hypopnea per hour (apnea/hypopnea index), DSI: the number of 3% desaturation per an hour (desaturation index), lowest SpO_2: the lowest oxygen saturation

Table 4. The comparison of AHI, arterial carbon dioxide tension and arterial oxygen tension between group REM and group NREM

	group REM (n=13)	group NREM (n=95)
Age (years)	47.0 ± 11.9	51.5 ± 13.9
BMI (kg/m^2)	26.3 ± 3.9	26.5 ± 5.0
ESS	10.2 ± 4.6	9.3 ± 5.4
AHI (/h)		
nPSG	7.6±3.9	31.7±22.9
dPSG	2.8±3.7*	30.3±29.7
$Paco_2$ (mmHg)	45.6±5.5	43.6±3.7
Pao_2 (mmHg)	79.6±15.5$^{\#}$	88.4±10.7

AHI: the number of apnea/hypopnea per an hour (apnea/hypopnea index), group REM: patients who satisfied with the criterion; AHI ≤ 25 /h, AHI_{REM}/AHI_{NREM} >2, and AHI_{NREM} <15 /h, group NREM: patients who did not satisfy with this criterion, nPSG: nocturnal polysomnography, dPSG: daytime polysomnography, $Paco_2$: arterial carbon dioxide tension, Pao_2: arterial oxygen tension, *: significant between nPSG and dPSG ($p<0.05$), $^{\#}$: significant between group REM and group NREM. ($p<0.05$)

Table 5. The sensitivity, specificity and accuracy on AHI in groups REM and NREM

	sensitivity (%)	specificity (%)	accuracy (%)
group REM (n=13)	20.0	100	15.3
group NREM (n=95)	81.9	100	84.1

AHI: the number of apnea/hypopnea per an hour (apnea/hypopnea index), group REM: patients who satisfied with the criterion; AHI $\leq$ 25 /h, AHI_{REM}/AHI_{NREM} >2, and AHI_{NREM} <15 /h, group NREM: patients who did not satisfy with this criterion