

# First-night-effect on polysomnographic respiratory sleep parameters in patients with sleep-disordered breathing and upper airway pathology

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**Abstract** The objective of this study is to test whether there is a difference between the polysomnographic (PSG) values of Apnea–hypopnea index (AHI), minimal oxygen saturation (SpO<sub>2</sub>), oxygen desaturation index (ODI) and arousal index recorded on two consecutive nights (so-called “first night effect”) in patients with sleep-disordered breathing (SDB) and concomitant upper airway pathology. Retrospective case–control study of polysomnographical recordings of 130 patients (112 males, 18 females, age range 23–80 years) with SDB and upper airway pathology who were tested on two consecutive nights in a hospital sleep laboratory was conducted. Only patients with upper airway pathology without other medical conditions causing SDB were included. AHI, minimal SpO<sub>2</sub>, ODI and arousal index values of the first night were compared to those of the second night using Wilcoxon’s test. There were no significant statistical differences between AHI, SpO<sub>2</sub>, ODI and arousal index values ( $P = 0.130$ ,  $P = 0.640$ ,  $P = 0.052$ , and  $P = 0.692$ , respectively) between the two nights. However, 15% of the patients showed significant variability in the AHI between the two recordings and in 6% of the patients, a diagnosis of severe OSA (AHI > 10/h) would have been missed if only one night of sleep study had been performed. In general, one night of sleep study is sufficient to lead to a clear diagnosis of severe OSA in patients with sleep-disordered breathing and upper airway pathology but may still not diagnose 6% of the patients

with severe OSA. Additionally, 15% of the patients showed a significant variability in the AHI between the two nights.

**Keywords** Polysomnography · Apnea · Obstructive · Sleep · Respiratory · Diagnosis

## Introduction

Awareness for the existence of sleep-related breathing disorders is continuously rising in the general population. As a result, otorhinolaryngologists are increasingly consulting patients with sleep disorders or sleep-disordered breathing and are getting referrals from other physicians in order to exclude or ascertain the existence of obstructive sleep apnea (OSA) in their patients. Obstructive sleep apnea is marked by a repeated partial or complete obstruction of the upper respiratory tract during the inspiratory phase of the respiratory cycle [1]. Severe forms of OSA are associated with a significantly increased morbidity and mortality rate due to their proven association with cardiovascular diseases [2].

The gold standard for diagnosis of OSA is the cardio-respiratory polysomnography (CR-PSG) [1, 3]. Oxygen saturation (SpO<sub>2</sub>), electrocardiography (ECG), electromyography (EMG), electrooculography (EOG), electroencephalography (EEG), body position, abdominal and thoracic respiratory movements, oronasal airflow as well as optical and acoustical recordings of events during sleep are recorded.

In previous studies, a remarkable variability between the recordings of the first and those of the second night of sleep study could be noticed in patients with sleep-related breathing disorders. This phenomenon, commonly known as the “first night effect” (FNE), has been described by

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many authors and involved such phenomena as variability in the latency of sleep induction, a recorded delay in the emergence of the stages of deep sleep, a variability in the proportion of REM sleep as part of total sleep time and a variability in the duration of nightly awaking [4, 5].

Additionally, the results concerning sleep-related respiratory parameters such as the apnea–hypnopnea index (AHI), the minimal SpO<sub>2</sub>, the ODI, and the arousal index are still quite controversial [6, 7].

Given that otorhinolaryngologists are confronted with patients with sleep disorders and upper airway pathology, the aim of this study was to test whether there is a FNE on the recorded AHI, the minimal SpO<sub>2</sub>, ODI, and the arousal index in a selected population of patients with sleep-disordered breathing and upper airway pathology.

## Patients and methods

In this retrospective study, the charts of 130 consecutive patients suspected of having OSA who had undergone cardiorespiratory polysomnography on two consecutive nights in the sleep laboratory of our department were reviewed. All patients were snoring and/or had breathing cessation during sleep reported by their life partners. Among the tested persons, 112 were men (86.1%) and 18 were women (13.9%), ranging in age between 23 and 80 years. The average age was 52.6 years ( $\pm 10.7$  = standard deviation). Only patients with upper airway pathology as the sole possible cause of sleep-related breathing disorder were eligible to be included in the study. Upper airway pathology included deviation of the nasal septum, inferior nasal turbinate hypertrophy, soft palate webbing, tonsillar hypertrophy, elongated uvula, macroglossia, hypertrophy of the tongue base, and unfavorable palate position relative to the tongue base. Patients suspected of suffering from isolated or concomitant to the upper airway pathology narcolepsy, hypersomnolence, chronic fatigue syndrome, periodic limb movement disorder, and/or diurnal rhythm disorders as well as patients with an established diagnosis of a psychiatric or neurologic (peripheral or central) disorder were excluded from the study. Additionally, patients with congestive heart failure, chronic obstructive pulmonary disease or any severe pulmonary disorder requiring standard medication were excluded from consideration.

In the accredited sleep disorders laboratory of our department, polysomnography was performed in all patients. An electroencephalogram (EEG), an EOG, an EMG and an ECG were simultaneously recorded. The oronasal airflow was measured by thermal sensors which registered the difference in temperature between inspiration and expiration. The measurements of the thoracic and

abdominal excursions were effected with use of elastic bands equipped with piezoelectric tension sensors. The oxygen saturation was photometrically measured using pulse oxymetry. The snoring noises were recorded by a pre-laryngeally attached microphone.

The polysomnographical recordings were initially automatically computer-assisted evaluated using software of the Alice-4-Sytem (Heinen und Löwenstein, Bad Ems, Germany) which contains an automatic evaluation system determining the stages of sleep, the respiratory parameters and the oxygen saturation. The definitive evaluation of the recordings was performed manually.

In the morning following each sleep study night, the sleep-related respiratory parameters were manually analyzed in 30-s-epochs by physicians, meeting the criteria set by Rechtschaffen and Kales [8] as well as the ICSD-2 guidelines [9].

The manual evaluation of the respiratory disorders and the oxygen saturation was carried out in epochs of 5 min, in compliance with the recommendations of the American Academy of Sleep Medicine Task Force [1] and the ICSD-2 [9]. The decline of the airflow amplitude and the decline of the oxygen saturation have been assessed. A reduction of oronasal airflow amplitude of more than 90%, lasting for at least 10 s has been defined as apnea. As hypopnea has been defined as a reduction of the airflow of 50–90% with a 4% reduction of the SpO<sub>2</sub>. The classification in obstructive, central or mixed respiratory obstruction during sleep resulted from the simultaneous evaluation of the thoracic and abdominal movements. An obstructive incident is characterized by diminished oronasal airflow while the thoracic and abdominal excursions are increased.

Using the polysomnographical records and the patient's chart, the age, sex, AHI, the minimal SpO<sub>2</sub>, ODI, and the arousal index of both nights as well as the difference of the recorded parameters between the first and the second night were evaluated. The physician who evaluated manually the recordings of the second night was different from the physician who evaluated the recordings of the first night.

The patients have been divided into two groups according to OSA severity in order to test the night-to-night variability. Regarding the AHI difference between both nights of measurement, in the first group patients who had an AHI <10/h and the second group patients with an AHI > 10/h were included. An AHI cut-off value of 10/h on PSG is frequently used to differentiate between patients with mild OSAs and patients with more severe forms of OSAs [10].

All relevant data of the reviewed patient charts have been collected electronically and evaluated statistically using the program SPSS (Statistical Package for the Social Sciences), Version 12.0.

The nonparametric Wilcoxon's test has been applied to compare the recorded values of the first and those of the second night.

## Results

The median AHI was 26.8/h at the first night (interquartile range 14.8–48.4/h) and was 26.2/h at the second night (interquartile range 14.7–51.6/h) (Table 1; Fig. 1). The median displaying the difference between the AHI of the first night and the AHI of the second night is  $-2.2$ . A  $P$  value of 0.130 resulted from the Wilcoxon's test, thus expressing that the measurement results of both nights do not differ strikingly from each other.

Nevertheless, in a subgroup of patients there was a high intra-individual variability of the AHI. When comparing both nights, 20 out of 130 patients (15% of the patients of our cohort) showed a difference in their AHI values of more than 10/h between the first and the second night. Eight out of 130 patients had an AHI of  $< 10$ /h in the first night and an AHI of  $> 10$ /h in the second night.

As a general trend, women showed lower AHI-values than men. For women the median AHI was 24.1/h at both the first and second night, for men the median AHI was 26.8/h (interquartile range 14.7–49.2/h) at the first night and at the second night the median was 26.5/h (interquartile range 15.1–51.4/h).

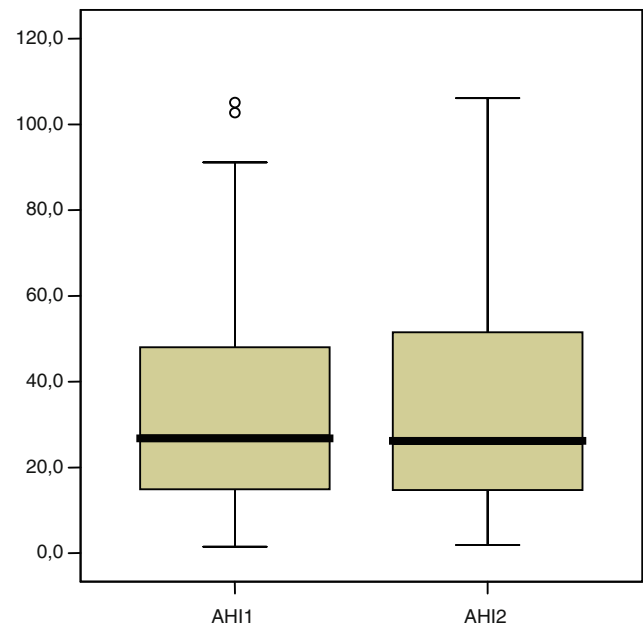
The minimal  $\text{SpO}_2$  value was almost identical for both nights (median 82%, interquartile range 76–86%). The resulting  $P$  value, when the Wilcoxon's test was applied to compare findings of the first to those of the second night, was 0.640.

The first night ODI had a median value of 19.5/h (interquartile range 10.4–33.3/h) and the second night, the median value was 16.0/h (interquartile range 8.8–34.4/h)

**Table 1** Statistical data on AHI (in episodes/hour) during the first (AHI1) and second (AHI2) night of polysomnographic sleep study (data were present in all 130 patients)

	AHI1	AHI2
Average	33.97	32.72
Median	26.75	26.20
Standard deviation	23.13	23.10
Minimum	1.50	1.90
Maximum	105.10	106.10
Percentile		
25	14.80	14.65
50	26.75	26.20
75	48.40	51.63

AHI is defined as the mean number of apneas and hypopneas per hour of sleep



**Fig. 1** Box plots of the AHI values (in episodes/hour) recorded at the first (AHI1) and second (AHI2) night of polysomnographic sleep study

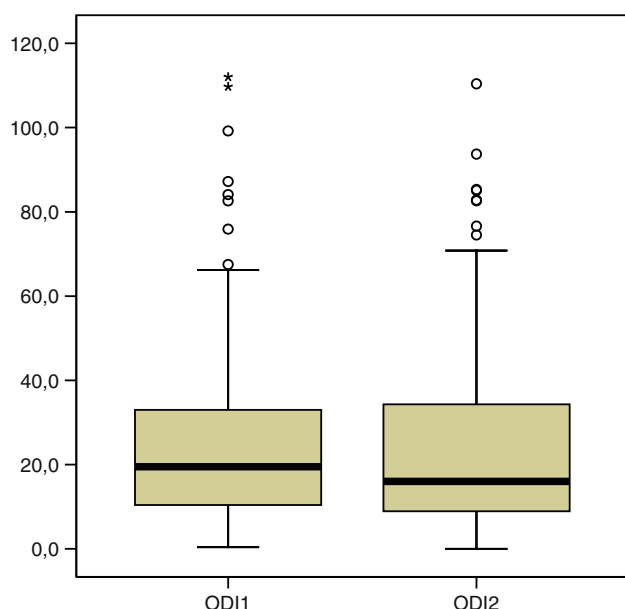
**Table 2** Statistical data on ODI during the first (ODI1) and second (ODI2) night of polysomnographic sleep study (data were present in all 130 patients)

	ODI1	ODI2
Average	26.88	25.00
Median	19.50	16.00
Standard deviation	23.78	22.62
Minimum	0.40	0
Maximum	112.00	110.40
Percentile		
25	10.40	8.78
50	19.50	16.00
75	33.33	34.43

ODI is defined as the number of oxyhaemoglobin desaturation events per hour of sleep at desaturations  $\geq 4\%$  from the baseline

(Table 2; Fig. 2). The difference of the ODI comparing the first and the second night was in median  $-1.3$ . The Wilcoxon test calculated a  $P$  value of 0.052. The results of measurement of both nights did not differ largely from each other, when the level of significance was set at  $P < 0.05$ .

The arousal index had a median value of 27.5/h in the first night of measurement (interquartile range 19.5–37.9/h) and in the second night it was 28.8/h (interquartile range 18.7–40.4/h). The difference of the arousal index comparing the first and the second night was in median 0.15/h. The  $P$  value of the Wilcoxon test was 0.692. Again, the results of measurement of both nights did not differ



**Fig. 2** Box plots of the ODI values (in episodes/hour) recorded at the first (ODI1) and second (ODI2) night of polysomnographic sleep study

considerably from each other, when the level of significance was set at  $P < 0.05$ .

## Discussion

The existence of a FNE on the AHI is a matter of controversy in the sleep literature. A few authors were able to provide evidence for a classical FNE on the AHI [6, 7]. Nonetheless, most studies could not prove the existence of a FNE [5, 11, 12]. Therefore, the term FNE could not be established for the AHI, and the term “night-to-night variability” is rather being used.

Accordingly, in the present study the AHI values of both nights do not differ significantly from each other. The results of this study, which involved only individuals with upper airway pathology without other medical conditions associated with sleep-disordered breathing, support the hypothesis that there is no considerable FNE concerning the AHI in this particular group of patients. This finding lends support to the currently prevailing opinion in the literature, which—nonetheless—resulted from studies in patient populations different from ours. Additionally, different authors have found minimal differences between two different nights of PSG recordings, even when introducing a pharyngoesophageal catheter the second night [13], thus supporting the results of the actual paper.

Although a classical FNE of the AHI could not be proved for the majority of the studies, almost all authors report intra-individual variability of the AHI [5–7, 11, 12].

The same authors point out that in some patients an OSA would not have been diagnosed if only one night of measurement had taken place. In the present study, a remarkable intra-individual variability of the AHI could be determined in 15% of the patients. Twenty out of 130 patients had a difference in the AHI of  $> 10/h$  between both nights.

Many authors suggest that a number of patients with OSA would have been misdiagnosed concerning severity of disease or not diagnosed at all if there was only one night of measurement with rates varying between 8 and 55% [5, 7, 12, 14].

The necessity of a second night of measurement has been a matter of controversy for a long time already. In 1989 the American Thoracic Society decided that one night of measurement should be sufficient to diagnose a clinical relevant OSA [3]. Yet, most authors advocated the necessity of a second night of sleep study to make a clear diagnosis of an OSA [7, 12, 14].

In the present study, in 6% of the particular group of OSA patients with upper airway pathology the diagnosis would have been missed if only one night of sleep study was performed, given that the cut-off limit for diagnosis of (severe) OSA required the AHI being  $> 10/h$ . It should be mentioned that many authors classify OSAs with  $5/h < AHI < 15/h$  as mild OSAs. In the latter case, use of a cut-off at  $AHI = 15/h$  in the present study would have been more pertinent. With the criteria used in this study, the data of the present study illustrate the fact that for a considerable number of patients a second PSG-night is essential for a clear diagnosis of a clinical relevant OSA.

In this study a FNE could neither be proved for the minimal  $SpO_2$  nor for the ODI nor the arousal index in patients with sleep-disordered breathing and upper airway pathology. This is in concert with results of the majority of other studies in which different populations of patients were included than in ours' [5, 7, 11, 12].

Given that interpersonal differences in PSG interpretation are frequently occurring, the evidence generated by the present study would have been strengthened if the same physician (or two physicians) had done both nights' scorings, blinded.

Over the last years, sleep-related breathing disorders attracted more attention as important clinical syndromes. Especially the severe forms of OSA demonstrate a significantly increased morbidity and mortality from cardiovascular diseases [2]. The typical cardinal symptoms are excessive day sleepiness and loud snoring with respiratory obstructions, although some patients only feature nonspecific syndromes like sleep-onset or sleep-maintenance insomnia, reduced achievement potential, depressive episodes or erectile dysfunction. Due to the numerous secondary complications of an untreated severe OSA, the

resulting risks, e.g. falling asleep while driving [15], awareness for OSA among physicians and among patients with unspecific discomfort, should rise.

In times of tremendous cost pressure on health care systems worldwide the necessity of a second PSG night is a matter of debate. The results of the present clinical study show that if only one PSG night had been realized, for 6% of the patients (8 out of 130) a severe OSA would not have been diagnosed. The given facts highlight that a second PSG night is compellingly necessary to preclude an OSA for sure.

## Conclusions

In general, one night of sleep study is sufficient to lead to a clear diagnosis of severe OSA in patients with sleep-disordered breathing and upper airway pathology, but may still not diagnose severe OSA in 6% of these patients. Additionally, 15% of this particular group of patients with upper airway pathology showed a significant variability in the AHI between the two nights.

**Conflict of interest** None.

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