Obstructive sleep apnoea syndrome: is the "half-night polysomnography" an adequate method for evaluating sleep profile and respiratory events?

F. Fanfulla, V. Patruno, C. Bruschi, C. Rampulla


ABSTRACT: Recently, to reduce the costs of polysomnography, split-night studies have been introduced into routine practice: the first part of the night is used to make the diagnosis of obstructive sleep apnoea syndrome (OSAS) and the second part to achieve an appropriate level of continuous positive airway pressure. Since this split-night protocol has not yet been validated by the comparison of polysomnographic pictures obtained in the first and second parts of the night, the aim of this study was to evaluate sleep profile and respiratory disturbances in the first part (PSG1) and second (PSG2) portion of a standard full-night polysomnographic examination (PSGtot) in a group of OSAS patients.

Twenty nine consecutive OSAS patients, aged 54±10 yrs; body mass index (BMI) 40±6 kg·m⁻² (mean±SD values), were studied by separate analyses of PSG1, PSG2 and PSGtot.

PSG1 was found to have a low sensitivity value (66%). A significant difference was found between apnoea-hypopnoea indices (AHI) recorded in PSG1, PSG2 and PSGtot (mean±SD, AHI1 33±27, AHI2 45±28, AHItot 40±25 events·h⁻¹, respectively; p<0.01). A strong correlation was observed between AHItot and AHI1 (r=0.89) and between AHItot and AHI2 (r=0.92), but a weaker correlation between AHI1 and AHI2 (r=0.66). These correlations became weaker when patients were subdivided into two different classes on the basis of disease severity. PSG1 was representative of PSGtot and similar to PSG2 only in those patients with rapid eye movement (REM) phase sleep in the first part of the night.

We conclude that split-night protocols are not appropriate for evaluating sleep-disordered breathing in obstructive sleep apnoea syndrome patients when rapid eye movement phase sleep does not occur in the first part of the night.


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Full-night polysomnography is currently considered the standard method for establishing a diagnosis of obstructive sleep apnoea syndrome (OSAS), but these studies are time-consuming and expensive. Furthermore, in patients who need nasal continuous positive airway pressure (nCPAP), a second study is required to achieve an appropriate level of continuous positive airway pressure (CPAP).

Recently, several authors have proposed a simplified protocol, usually known as a "split-night" study, for evaluating OSAS patients. In this protocol, the first part of standard polysomnography (2 h) is spent making the diagnosis of OSAS, while the second part is used to establish an adequate level of CPAP [1–3].

However, the reliability of this method has only been evaluated in a few studies [1, 2]. In a retrospective study, Iber et al. [1] found that a single-night study was sufficient to establish effective CPAP therapy in 78% of their patients, and offered considerable conservation of resources compared to routine multiple-night studies. Sanders and co-workers [2] concluded that the split-night study is an appropriate method for evaluating sleep-disordered breathing (SDB), having noted a strong correlation of respiratory disturbance indices between data from the first part of the study and the whole recording. According to this method, treatment is started only when clinically significant sleep-disordered breathing (usually apnoea-hypopnoea index (AHI) >10 events·h⁻¹) is recorded during the initial period of polysomnography. The shortness of the recording period, although sufficient to diagnose OSAS, may not be adequate for the evaluation of disease severity; thus, the diagnosis may be unreliable and the titration of CPAP level required for its correction not correctly defined. In fact, up to now, the single-night protocol has not been validated by the comparison of polysomnographic pictures obtained in the first and second parts of the recording.

Thus, the aim of this study was to evaluate the distribution of polysomnographic indices and respiratory events in the first (PSG1) and second (PSG2) portions of a standard full-night polysomnographic examination (PSGtot) in a group of OSAS patients.
Materials and methods

We studied 29 consecutive patients (27 males and 2 females), mean (±SD) age 54±10 yrs, referred to our clinic because of suspected obstructive sleep-apnoea, according to clinical symptoms and overnight monitoring of arterial oxygen saturation (SaO2) [4–6]. All patients had symptoms related to sleep disorders (i.e. somnolence, snoring, sleep disturbance). In order to avoid bias related to a nonhomogeneous sample, patients with concomitant diseases or therapy that could affect sleep-disordered breathing or sleep quality (i.e. obstructive lung diseases or chronic heart failure) were excluded from the study.

All patients performed pulmonary function tests, consisting of at least body plethysmography and flow-volume curves (Masterlab; Jaeger, Würzburg, Germany) according to the European Respiratory Society (ERS) statement [7]. Respiratory function data were compared with predicted normal values, obtained by the European Coal and Steel Community (ECSC) 1983 regression equation and expressed as a standard deviation score according to the ERS statement [7, 8]. No patient had airway obstruction: in all patients forced expiratory volume in 1 second/vital capacity ratio (FEV1/VC) was above obstructive lung diseases or therapy that could affect sleep-disordered breathing or sleep quality (i.e. obstructive lung diseases or chronic heart failure) were excluded from the study.

All patients underwent a standard full-night polysomnography, performed in a quiet, partially sound-proofed room with stable humidity and temperature. The procedure usually started at 21.30 h and the operator switched off the light on request from the patient. The procedure was stopped at 06.00 h. The following indices were monitored by standard methods: electroencephalogram (EEG); electro-oculogram (EOG); submental electromyogram (EMG); oronasal airflow; electrocardiogram (ECG); SaO2 by means of a pulse-oximeter (Sat-trak; Sensormedics, Anheim, CA, USA); and respiratory movements by inductive plethysmography [10, 11]. All signals were recorded and stored on the optical disk of a semi-automated scoring system (Somno-star; Sensormedics, Anheim, CA, USA). Using this system, sleep and breathing data were played back on the video terminal, and scoring was performed visually on 30 s epochs by the same physician (F.P.), who did not know the aim of the study.

The PSG file for each patient was divided into two periods of equal duration: PSG1, defined as the initial half of the total sleep period time (SPT); and PSG2, defined as the second half of SPT. Sleep and breathing data from PSG1 and PSG2 were then separately computed and compared with data collected over the total PSG (PSGtot). Respiratory events were identified and classified by standard methods [10, 11]. In particular, apnoea was defined by absence of inspiratory airflow for at least 10 s. Hypopnoea was defined as a reduction in airflow signal by >50% from the level measured before the event, lasting at least 10 s. Arousals were defined as alpha activity or increased EEG frequency lasting at least 3 s [12, 13].

OSAS was defined as the presence on polysomnography of >10 obstructive or mixed apnoeas/hypopnoeas per hour of sleep, associated with a history of loud snoring and excessive daytime sleepiness.

The total sleep time (TST), apnoea index (AI), hypopnoea index (HI), apnoea-hypopnoea index (AHI), desaturation event frequency (DEF), and mean duration apnoea (MDA) were determined separately for PSG1, PSG2 and PSGtot.

Values of TST, wake stage (WS, expressed as percentage of SPT), AI, HI, AHI and MDA, both in non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, recorded in the first and second parts of the night, were compared using the paired t-test. The different distributions of respiratory disturbances in PSGtot, PSG1 and PSG2 were analysed by analysis of variance (ANOVA).

The relationships between indices of breathing pattern during the three study periods were evaluated using Pearson’s product-moment correlation coefficient.

In order to identify different clusters of patients on the basis of OSAS severity, the distribution of AHI was analysed. The patients were then subdivided into two different classes of severity: patients with AHI between 10 and 13.35 (corresponding to the lower quartile of distribution); and patients with AHI >13.35 (2nd, 3rd and 4th quartiles).

The distribution of respiratory events was also analysed by a two-way ANOVA test, comparing the patients with and without REM phase sleep in the first part of the night. A post-hoc multiple comparison test was then performed.

In order to identify a predictive model for AHItot, a multiple regression analysis was performed considering as independent variables AH1 and the presence or absence of REM phase sleep in the first part of registration:

$$\text{AHItot}= \alpha + \beta_1 \times \text{AHI1} + \beta_2$$

where $\beta_1$=1 when REM is present in AHI1, and $\beta_2$=0 when REM is absent in AHI1.

All the analyses were performed using the STATISTICA/w statistical package (StatSoft inc., Tulsa, OK, USA). A p-value of less than 0.05 was considered significant.

Results

Table 1 presents the demographic, daytime respiratory function and polysomnographic data of the study population.

TST was sufficiently long to obtain evaluable data. A wide range of OSAS severity was found (AHI 10.2–94.9 events·h⁻¹). The sleep profile was classically altered, being characterized by a high number of arousals and a low percentage of slow-wave sleep.

Whereas REM phase sleep was not observed at any time during the recording in only two subjects, 14 subjects (48% of the sample) did not have REM phase sleep in the first part of the night. Because of this bias, no comparison was made between respiratory indices recorded during REM phase in the first and second parts of polysomnography.

In table 2, sleep and breathing indices are reported separately for PSG1 and PSG2. The TSTs observed in PSG1 and PSG2 were similar (p>0.05). A difference
was observed between wake stage in PSG1 and PSG2 (WS1 and WS2) (p<0.01), and this result explains the difference in TST observed between PSG1 and PSG2.

Differences were observed between AHI1 and AHItot (p<0.01), as well between AHI1 and AHI2 (p<0.01) and between AHI2 and AHItot (p<0.01). Similar differences were observed among the three different indices (ANOVA 0.0012).

A similar trend was observed for the AHI during NREM phases of sleep (ANOVA 0.009). The arousals-awakenings index was higher in PSG2 than PSG1 (p=0.01). MDA was longer in PSG2 than PSG1 (p=0.015); and SaO2 nadir, was lower in the second part of the night than in the first part (p<0.001).

A strong correlation was observed between AHItot and AHI1 (r=0.892; p<0.01) (fig. 1a and b). However, the correlation between AHI1 and AHI2 was weaker, although still significant (r=0.66; p<0.01); figure 1c shows clearly that individual AHI1 data were lower than AHI2.

In order to evaluate whether the severity of the disease might influence the difference between data recorded in the first and second parts of PSG, two different analyses were performed: the first on patients with milder disease (AHI within the 1st quartile of distribution); and the second on the remaining patients. In less severely diseased patients a difference was found between AHItot, AHI1 and AHI2 (11.5±1.3, 4.0±3.4, 16.3±4.5 events·h−1, respectively; p<0.01). Similar results were

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</tr>
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<td>Age yrs</td>
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<tr>
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</tr>
<tr>
<td>54±10</td>
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Data are presented as mean±sd. BMI: body mass index; VC: vital capacity; sd score: standard deviation score; FEV1: forced expiratory volume in one second; Pao2: arterial oxygen tension; SaO2: arterial oxygen saturation; SPT: sleep period time; TST: total sleep time; REM: rapid eye movement; AHI: apnoea-hypopnoea index; DEF: desaturation events frequency.

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<td>TST min</td>
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<td>---------</td>
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<tr>
<td>139±45.9</td>
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<td>150.6±40</td>
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Data are presented as mean±sd. WS: wake stage; MDA: mean duration apnoea; NREM: non-rapid eye movement (sleep); PSG1 and PSG2: first and second portion of a standard full-night polysomnographic examination, respectively. For further definitions see legend to table 1.
Table 3. – Distribution of apnoea/hypopnoea index, separately for the three study periods, in patients with or without REM phase sleep in PSG1

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Data are presented as mean±SD. AHItot: apnoea/hypopnoea index in the full-night polysomnographic examination; AHI1: AHI2: apnoea/hypopnoea index in the first and second portion of the first night polysomnographic examination, respectively.

For further definitions see legends to tables 1 and 2.

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To the best of our knowledge, only two validation studies have been published in the literature [1, 2]. SANDERS and co-workers [2] concluded that PSG during 2 h of sleep is an appropriate method for evaluating sleep-disordered breathing. In their study, a strong correlation was observed between the indices obtained in the first part of the night and those of the entire night. However, data obtained in PSG1 represent a significant part of the whole night recording (about 50%), thus significantly costing the results: in other words, PSG1 and PSGtot should not be treated as independent variables. No analyses have been performed between the first and the second parts of the study. With this design, we found that the disease is more manifest in the second part of the night. The failure to study this part of the night by polysomnography could result in an underestimation of disease severity. As a consequence, patients or their referring clinicians could be less aware of the disease and therapeutic treatment might be delayed.

In the present study, the severity of the disease did not seem to have a significant influence on the different distribution of respiratory events indices, although the difference between the first and the second parts of the night was more evident in patients with milder disease.

In their paper, SANDERS and co-workers [2], did not separately analyse the trend of breathing disorder indices in patients who did not have REM phase sleep in PSG1. In the present study, we found that the reliability of the diagnosis of OSAS by a split-night study is fundamentally linked to the occurrence of REM phase sleep in the first part of the night, which, when it occurs, permits as accurate a diagnosis as that made with full standard polysomnography. In fact, in patients who experienced REM phase sleep during the first part of the night, the AHItot, AHI1 and AHI2 were all quite similar; in contrast, in patients without REM phase sleep in PSG1, the same parameters were significantly different, with lower values of AHI in the first part of the night. The multiple linear model used in the present study did not improve the predictive power of the single regression model. Thus, the role of REM phase sleep in inducing pathological respiratory events does not seem to be predictable in the individual case by this statistical model.

This limitation of the split-night method may account for the discouraging level of accuracy of CPAP-titration found by different authors. IBER et al. [1] validated...
the efficacy of CPAP treatment, titrated by a single-night study. The authors demonstrated in a short follow-up that an effective CPAP level was established in 78% of their patients. Unfortunately, a control PSG evaluation was not provided in the design of this study. In this way, CPAP could be titrated only on the basis of PSG1.

In another paper, Sanders and co-workers [21] analysed the adequacy of CPAP treatment prescribed on the basis of a partial-night trial. The results seem discouraging. In fact, in 44% of the sample a change of pressure ventilator level was necessary, and in another 14% of patients a change of the modality of treatment was needed. More recently, similar results were obtained by Yamashiro and Kryger [13], who conducted a study on the effectiveness of CPAP titration by a split-night protocol in 107 OSAS patients. The authors concluded that this procedure may be sufficient to determine the effective CPAP pressure, especially in patients with an AHI >20 events h⁻¹, but 69.1% of the sample required a change of the previously identified CPAP level, and 7.5% of the sample required a change of treatment modality. These problems were more evident in the less diseased group (AHI <20 events h⁻¹) of patients. Furthermore, the gap in pressure level between the split-night and the control study was significantly greater in the group of patients whose PSG1 lasted less than 3 h. We consider that this high frequency of changes may be due to incorrect evaluation of the severity of the disease during the partial-night study [22].

In summary, in this study we found that a falsely high number of patients may be classified as normal on the basis of data obtained in the first portion of a standard full-night polysomnographic examination. Independently of the severity of the disease, pathological events are more manifest in the second portion of the night. We found that in some patients neither full-night polysomnography nor the second portion of a full-night polysomnographic examination could be adequately predicted from the observation of data obtained during the first 2–3 h of the night: the presence of rapid eye movement phase sleep in the first part of the night would have been the discriminant event responsible for the accuracy of the diagnosis of obstructive sleep apnoea syndrome. In our opinion this is an important result because it identifies a method of linking the pragmatic demand of reducing the costs of sleep laboratories and the clinical need for correct assessment of the severity of disease.

References

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