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

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. ©ERS Journals Ltd 1997.

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 (PSG_{tot}) 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 $\pm$ SD values), were studied by separate analyses of PSG1, PSG2 and PSG_{tot}.

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 PSG_{tot} (mean $\pm$ SD, AHI₁ 33 ± 27 , AHI₂ 45 ± 28 , AHI_{tot} 40 ± 25 events·h⁻¹, respectively; $p < 0.01$). A strong correlation was observed between AHI_{tot} and AHI₁ ($r = 0.89$) and between AHI_{tot} 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 PSG_{tot} 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.

Eur Respir J 1997; 10: 1725–1729.

S. Maugeri Foundation I.R.C.C.S., Rehabilitation Institute of Montescano, Respiratory Function Laboratory, Montescano, Italy.

Correspondence: F. Fanfulla
Fondazione S. Maugeri
Centro Medico di Montescano
27040 Montescano (PV),
Italy

Keywords: Obstructive sleep apnoea
polysomnography
split-night studies

Received: March 19 1996

Accepted after revision May 5 1997

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 (PSG_{tot}) in a group of OSAS patients.

Materials and methods

We studied 29 consecutive patients (27 males and 2 females), mean ($\pm$ 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 (S_{a,O_2}) [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 one second/vital capacity ratio (FEV_1/VC) was above the 5th percentile of normal distribution.

Arterial blood gas values were analysed using an automated, computerized gas analyser (ABL 500; Radiometer, Copenhagen, Denmark), and were compared to new reference values for the middle-aged and elderly [9].

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 0.600 h. The following indices were monitored by standard methods: electroencephalogram (EEG); electro-oculogram (EOG); submental electromyogram (EMG); oronasal airflow; electrocardiogram (ECG); S_{a,O_2} by means of a pulse-oximeter (Sat-trak; SensorMedics, Anaheim, 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, Anaheim, 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 (V.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 (PSG_{tot}). 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 PSG_{tot}.

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 PSG_{tot}, 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 AHI_{tot}, a multiple regression analysis was performed considering as independent variables AHI1 and the presence or absence of REM phase sleep in the first part of registration:

$$AHI_{tot} = \alpha + \beta_1 \times AHI_1 + \beta_2$$

where $\beta_2=1$ 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

Table 1. — Demographic, daytime respiratory function and polysomnographic data of the study population

Age yrs	54±10
BMI kg·m ⁻²	40±6
VC SD score	-0.91±0.98
FEV ₁ /VC SD score	0.25±1.59
P _{a,O₂} kPa	8.6±1.1
mmHg	64.7±8.2
P _{a,O₂} SD score	-1.24±0.84
Sa _{a,O₂} awake %	93±3
SPT min	339±85
TST min	293±82
% TST	
Stage 1	18±6
Stage 2	58±16
Stage 3	7±7
Stage 4	2±5
REM sleep	15±8
Sleep efficiency %	84±14
AHI events·h ⁻¹	39.8±24.8
Arousal-awakenings index	41.6±25.4
DEF events·h ⁻¹	29.7±22
Sa _{a,O₂} nadir %	65±12
% TST spent at Sa _{a,O₂} <90%	41±33

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; P_{a,O₂}: arterial oxygen tension; Sa_{a,O₂}: arterial oxygen saturation; SPT: sleep period time; TST: total sleep time; REM: rapid eye movement; AHI: apnoea-hypopnoea index; DEF: desaturation events frequency.

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 AHI_{tot} ($p<0.01$), as well between AHI₁ and AHI₂ ($p<0.01$) and between AHI₂ and AHI_{tot} ($p<0.01$). 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 Sa_{a,O₂} nadir, was lower in the second part of the night than in the first part ($p<0.001$).

A strong correlation was observed between AHI_{tot} and AHI₁ ($r=0.892$; $p<0.01$) and between AHI_{tot} and

Table 2. — Sleep and breathing indices separately for PSG₁ and PSG₂

	PSG ₁	PSG ₂	p-value
TST min	139±45.9	150.6±40	0.053
WS % of SPT	18±15	11±13	<0.01
AHI events·h ⁻¹	33.4±27.4	44.9±27.7	<0.01
AHI in NREM events·h ⁻¹	30.1±32.3	41.0±38	0.004
Arousals-awakenings events·h ⁻¹	34.5±25.8	49.2±30.3	0.01
MDA s	21.3±6.1	25.8±9.7	0.016
Sa _{a,O₂} nadir %	84±9	76±9	<0.0001

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.

AHI₂ ($r=0.92$; $p<0.01$) (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 AHI_{tot}, 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

Fig. 1. — Correlations between: a) AHI₁ and AHI_{tot}; b) AHI₂ and AHI_{tot}; and c) AHI₁ and AHI₂. —: the line of identity; —: regression line;: 95% confidence limits of the regression. AHI_{tot}: apnoea/hypopnoea index in the full-night polysomnographic examination; AHI₁ and AHI₂: apnoea/hypopnoea index in the first and second portion of the full-night polysomnographic examination, respectively.

Table 3. – Distribution of apnoea/hypopnoea index, separately for the three study periods, in patients with or without REM phase sleep in PSG1

Occurrence of REM phase sleep in PSG1	AHI _{tot} events-h ⁻¹	AHI ₁ events-h ⁻¹	AHI ₂ events-h ⁻¹
Present (p=NS)	52.1±23.0	51±24.1	53.3±23.4
Absent (p=0.003)	26.5±19.8	14.6±16.0	36.1±30.0

Data are presented as mean±SD. AHI_{tot}: apnoea/hypopnoea index in the full-night polysomnographic examination; AHI₁: AHI2: apnoea/hypopnoea index in the first and second portion of the full-night polysomnographic examination, respectively. For further definitions see legends to tables 1 and 2.

found in the group of more severely affected patients (50.5±20.5, 44.6±23.9, 55.9±24.8 events-h⁻¹, respectively; p=0.03). Furthermore, in the more mildly affected patients, only the correlation between AHI_{tot} and AHI₂ remained significant, albeit weaker (r=0.76; p<0.05). In the patients with more severe disease, the correlation between AHI₁ and AHI₂ disappeared (r=0.41; NS) whilst the other correlations became weaker (AHI_{tot} vs AHI₁; r=0.79; p<0.01) (AHI_{tot} vs AHI₂; r=0.87; p<0.001).

Another important finding regards the low sensitivity of PSG1 (true positive/true positive + false negative = 66%) in making the diagnosis of OSAS.

To identify the possible cause of the discordance between the first and second parts of PSG data, patients who did not have REM phase sleep during PSG1 were analysed. Whilst in patients with REM phase sleep in the first part of the night, AHI_{tot}, AHI₁ and AHI₂ were quite similar, in patients without REM phase sleep in the first part of the night the AHI_{tot}, AHI₁ and AHI₂ were different (ANOVA p=0.003) (table 3).

The multiple regression analysis did not identify a strong predictive model for AHI_{tot}. The multiple correlation coefficient did not improve from that obtained by the single regression model: r=0.897 with r²=0.805 cf r=0.892 and r²=0.796, respectively. The p-value of the β₂-factor included in the model (presence or absence of REM phase sleep in PSG1) is not significant (p=0.24).

Discussion

The most important finding of this study was that, in a population of patients referred for evaluation of obstructive sleep apnoea with a wide ranging severity of sleep-disordered breathing, a reliable diagnosis of OSAS could not be made with certainty by using the split-night study. Only when REM phase sleep occurred in the first part of the study could an adequate estimation of sleep disorders be made using such a split-night protocol.

All but seven (24%) patients had normal arterial oxygen tension (P_aO₂) values, although the standard deviation scores were negative. The patients were quite obese and, thus, the expected values of P_aO₂ were lower than in subjects of normal weight [9]. Nonetheless, it is known that patients with OSAS can have low values of P_aO₂ related to the development of pulmonary hypertension. The development of pulmonary hypertension cannot be predicted by OSAS severity, FEV₁ values, BMI or smoking habit [14].

The gold standard for diagnosis of sleep-apnoea syndrome (obstructive or not) is widely considered to be

full-night polysomnography [15]. A significant number of patients can require another evaluation for accurate assessment of disease severity. Indeed, several reports have found that even full-night studies can be subject to significant diagnostic inaccuracy [15–18].

The growing interest in sleep-disordered breathing has increased the number of polysomnographic studies, with consequent congestion of sleep laboratories and rising costs. These considerations have stimulated efforts to develop other techniques for the diagnosis of sleep disordered breathing, for example the diurnal sleep or nap recording [19].

Recently, the split-night method has been introduced into routine practice. This method has some advantages over the traditional techniques: it allows diagnosis and treatment identification in the same diagnostic session. It was expected that there would be considerable savings in the resources of sleep laboratories [20].

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 conditioning the results: in other words, PSG1 and PSG_{tot} 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 AHI_{tot}, 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

- Iber C, O'Brien C, Schluter J, Davies S, Leatherman J, Mahowald M. Single-night studies in obstructive sleep apnea. *Sleep* 1991; 14: 383–385.
- Sanders MH, Black J, Costantino JP, Kern N, Studnicki K, Coates J. Diagnosis of sleep-disordered breathing by half-night polysomnography. *Am Rev Respir Dis* 1991; 144: 1256–1261.
- Orr WC. Sleep studies in OSA patients before CPAP treatment: one night or two?. *Chest* 1993; 103: 652–653.
- Williams AJ, Yu G, Santiago S, Stein M. Screening for sleep apnea by pulse oximetry and a clinical score. *Chest* 1991; 100: 631–635.
- Douglas NJ, Thomas S, Jan M. Clinical values of polysomnography. *Lancet* 1992; 339: 347–350.
- Fleury B, Rakotonanahary D, Tehindrazanarivelo AD, Hausser-Hauw C, Lebeau B. Long-term compliance to continuous positive airway pressure therapy (nCPAP) set up during a split-night polysomnography. *Sleep* 1994; 17: 512–515.
- Quanjer PhH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault J-C. Lung volumes and forced ventilatory flows. Report Working Party "Standardized of Lung Function Tests". *Eur Respir J* 1993; 6 (Suppl. 16): 5–40.
- Quanjer PhH, Dalhuijsen A, Van Zomeren BC. Summary equations of reference values. *Bull Eur Physiopathol Respir* 1983; 19 (Suppl. 5): 45–51.
- Cerveri I, Zoia MC, Fanfulla F, *et al.* Reference values of arterial oxygen tension in the middle-aged and elderly. *Am J Respir Crit Care Med* 1995; 152: 934–941.
- Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Bethesda, US Government Printing Office, 1968 (NIH publication No. 204).
- Martin RJ (Chairman). Indications and standards for cardiopulmonary sleep studies. *Sleep* 1985; 8: 371–379.
- Atlas task Force. EEG arousals: scoring rules and examples. *Sleep* 1992; 15: 174–184.
- Yamashiro Y, Kryger MH. CPAP titration for sleep apnea using a split-night protocol. *Chest* 1995; 107: 62–66.
- Sajkov D, Cowie RJ, Thornton AT, Espinoza HA, McEvoy RD. Pulmonary hypertension and hypoxemia in obstructive sleep apnea syndrome. *Am J Respir Crit Care Med* 1994; 149: 416–422.
- Jamieson AO. Split-night studies: a new standard? Forcing the examination of outcome. *Sleep* 1991; 14: 381–382.
- Kramer M. Obstructive sleep apnea: one night is not enough! *Sleep Res* 1988; 17: 205–206.
- Bliwise DL, Benkert RE, Ingham RH. Sleep apnea nightly variability in aged subjects and related factors. *Sleep Res* 1990; 19: 191–194.
- Mosko SS, Dickel MJ, Ashurst J. Night-to-night variability in sleep apnea and sleep-related periodic leg movements in the elderly. *Sleep* 1988; 11: 340–348.
- Series F, Cormier Y, La Forge J. Validity of diurnal sleep recording in the diagnosis of sleep apnea syndrome. *Am Rev Respir Dis* 1991; 143: 947–949.
- Sanders MH, Kern NB, Costantino JP, *et al.* Prescription of positive airway pressure for sleep apnea on the basis of a partial-night trial. *Sleep* 1993; 16: S106–S107.
- Sanders MH, Kern NB, Costantino JP, *et al.* Adequacy of prescribing positive airway pressure therapy by mask for sleep apnea on the basis of a partial-night trial. *Am Rev Respir Dis* 1993; 147: 1169–1174.
- Findley LJ, Wilhoit SC, Suratt PM. Apnea duration and hypoxemia during REM sleep in patients with obstructive sleep apnea. *Chest* 1985; 87: 432–436.