

SLEEP MEDICINE

Sleep Medicine 3 (2002) 67-71

www.elsevier.com/locate/sleep

Original article

Clinical significance of nocturnal oximeter monitoring for detection of sleep apnea syndrome in the elderly

Shinji Teramoto^{a,*}, Takeshi Matsuse^b, Yoshinosuke Fukuchi^c

^aSan-no Hospital, International University of Health and Welfare, 8-10-16, Akasaka, Minato-ku, Tokyo, 107-0052, Japan

^bDepartment of Pulmonary Medicine, Yokohama City University Medical Center, Yokohama, 232-0024, Japan

^cDepartment of Respiratory Medicine, Juntendo University, Tokyo, 113-8421, Japan

Received 5 May 2000; received in revised form 7 February 2001; accepted 29 May 2001

Abstract

Objectives: The sensitivity and specificity of overnight monitoring of arterial oxygen saturation (SaO₂) using an oximeter were evaluated in elderly subjects who are being investigated for possible sleep apnea syndrome (SAS).

Methods: Seventy-five consecutive elderly subjects with habitual snores (47 men, 28 women; mean (\pm SE) age 75.5 \pm 0.9 years (range 65–94 years)) were studied. The SaO₂ was measured with an oximeter and a chart recorder during the night immediately before detailed polysomnographic studies. The SaO₂ recordings were classified by two observers as positive or negative using a number of significant oxyhemoglobin desaturation (SDS) of more than 2, 4, and 6%. The sensitivity of the oximeter alone for the recognition of the SAS was calculated as the number of true positive SaO₂ records divided by the total number of positive definitive (polysomnographic) records. The specificity was defined as the number of true negative SaO₂ records divided by the total number of negative definitive records.

Results: Of the 75 subjects, 24 had moderate SAS (apnea index (AI) > 15) and 55 had mild to moderate SAS (AI > 5). The sensitivity and specificity of the dosimeter as a screening test were determined with the two diagnostic thresholds of the AI. For AI exceeding 5 or 15, the respective sensitivity by using the criterion of SDS of more than 4% was 85.5 or 91.7%, with corresponding specificity of 85.0 or 92.2%.

Conclusions: The nocturnal oximeter monitoring allows recognition of elderly subjects with a mild to moderate SAS, and 4% desaturation of SaO_2 is a candidate index to detect a significant number of apneas in elderly SAS patients with an oximeter. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Oximeter; Sleep apnea; Elderly; Polysomnography

1. Introduction

Because sleep disorders are extremely common in the elderly, complaints of insomnia and daytime sleepiness have been found to increase within the aging sample in surveys [1–3]. Sleep apnea syndrome (SAS) is known to increase with advancing age, contributing to excessive daytime sleepiness, cardiovascular dysfunction and the impairment of health-related quality of life (HRQoL) [4–12]. Increased mortality rates reported in patients with severe SAS are probably due to the greater magnitude of cardiovascular complications [13–15]. Although SAS itself and SAS-related symptoms may be hazardous for elderly persons, elderly patients with SAS are often overlooked and undiagnosed. It is reasonable to assume that earlier detection of SAS in elderly persons is important for successful treatment. In principle, polysomnographic studies are

* Corresponding author. Tel.: +81-3-3402-3151; fax: +81-3-3404-3652. *E-mail address:* shinjit-tky@umin.ac.jp (S. Teramoto).

1389-9457/02/\$ - see front matter 0 2002 Elsevier Science B.V. All rights reserved. PII: S1389-9457(01)00129-0

needed for the detection and classification of sleep apnea and associated disorders [16], but these studies are relatively expensive in cost and time, and often inconvenient for both subjects and observers. The explosive growth of the aged population prevents the availability of polysomnography for all elderly patients, especially those who live far from our laboratory. From this perspective, a simple method of screening SAS would be beneficial. Although oximetry is a candidate to diagnose SAS, the results of the previous studies have been variously reported. The sensitivity of oximetry alone for the recognition of SAS ranges from 40 to 100% [17–25]. In addition, the clinical significance of oximeter monitoring for detecting SAS in elderly subjects has not been extensively reported.

The purpose of this study is to assess the value of single overnight oximeter monitoring in predicting whether significant sleep apnea is present in the elderly.

2. Method

2.1. Subjects

Seventy-five consecutive elderly subjects (47 men, 28 women; mean (\pm SE) age 75.5 \pm 0.9 years (range 65–94 years)) were studied during the night after obtaining informed consent. All were habitual snorers; other demographic and anthropometric data are shown in Table 1. We observed the arterial oxygen saturation (SaO₂) recordings of these subjects referred for investigation of possible sleep apnea, which were obtained the night immediately preceding more detailed nocturnal polysomnographic studies on the second and third nights. In subjects who slept for less than 6 h, as determined by EEG and EOG, repeat oximetry or polysomnography was performed to assess whether poor sleep led to a misdiagnosis or inaccurate estimation of disease severity. Sixty of 75 patients were free of cardiopulmonary diseases. They did not have abnormal spirometric results or histories of cardiopulmonary diseases. Five patients with reduced left ventricular function were stable clinically (belonging to Class 2 or more according to the New York Heart Association functional classification) and in sinus rhythm. Ten patients had stable chronic obstructive pulmonary disease (COPD) which was determined by the patients' history, the usual radiographic and pulmonary criteria, and exertional dyspnea with signs of over-distended lung volume. No patients with COPD had a forced expiratory volume in 1 s (FEV₁) to more than 60% predicted. We chose the best of three maximal flow-volume curves using a dry rolling seal spirometer (DRS-360, Fukuda Sangyo, Japan) before the sleep study. The best index is defined as the curve with the highest sum of forced vital capacity (FVC) and FEV₁ [26].

2.2. Study design

All patients were admitted for three or more consecutive nights. SaO₂ was recorded with an oximeter (953 Finger Flex Sensor; Healthdyne Technologies) with an output analog signal connected to a chart recorder (Multicorder MC 6716, Graph Tec Corp., Japan). The oxygen saturation

Table 1	
Anthropometric and pulmonary function data ^a	

n	75
Male/female	18/6
Age (years)	75.0 ± 8.7
Height (cm)	155.8 ± 7.6
Weight (kg)	53.5 ± 9.2
BMI	22.1 ± 4.1
FVC (L)	2.37 ± 0.49
%VC (%)	80.9 ± 1.8
FEV_1 (L)	1.94 ± 0.34
FEV ₁ /FVC (%)	81.8 ± 4.1

 a Data are presented as the mean \pm SD. BMI, body mass index; FVC, forced vital capacity (L); FEV1 (L), forced expiratory volume in 1 s.

signal was digitally sampled at 1 Hz. The recorder speed was set at 6 cm/h and the subject recorded the start time. The finger probe was attached with the assistance of a nurse or doctor before the subject went to sleep. Nursing staff intermittently checked the placement of the probe during the night, but the subject was not under constant supervision. To minimize the oximeter artifact, the nurses checked the chart records every 5 min and re-checked the patients' movements and the probe position when accelerated dips of oximeter recordings were found. Polysomnography was performed on the second night. Polysomnography on the second night consisted of 8 h of overnight monitoring using a standard technique. Respiratory effort was measured using respiratory inductance plethysmography (Respitrace Crop., USA), and airflow at the nose and mouth was measured with thermistors [27]. Surface electrodes were applied to obtain EEG, EOG, and ECG, and we also recorded the heart rate. SaO₂ was recorded with the pulse oximeter (953 Finger Flex Sensor; Healthdyne Technologies). The oxygen saturation signal was digitally sampled at 1 Hz. A polygraph recorded data on paper using a 12channel chart recorder (Nihonkoden, Japan) and on floppy disk via an IBM-compatible personal computer data acquisition system (NEC 9801, NEC, Japan). The recording included four channels of EEG, two channels of EOG, one channel of ECG, one channel of airflow by thermistor, three channels of effort by respiratory inductance plethysmography, and one channel of SaO₂.

Apnea was defined as the cessation of oronasal airflow for more than 10 s. Desaturation was not a criterion for scoring apnea. In this study mild SAS was determined by an apnea index (AI) greater than 5/h; moderate SAS was determined by an AI greater than 15/h. Patients were diagnosed on the basis of an AI exceeding 5 or 15 events per hour, and the sensitivity and specificity of oximeter monitoring for the detection of SAS were examined for both mild and moderate SAS.

2.3. Definition of significant oxyhemoglobin desaturation on oximeter recording

The number of significant oxyhemoglobin desaturation (SDS) was determined by drops in oxyhemoglobin saturation of more than 2, 4, and 6%. Drops in SaO_2 lasting more than 5 s were counted.

2.4. Analysis using the number of SDS

The observers were unaware of the aim of the study. Recordings of both nights were examined separately, without knowledge of the other night's recording. Oximetry records from the first night were coded and analyzed 'blind' by two experienced observers and classified in one of two categories: positive, SAS present; negative, SAS not present. A second observer checked samples of the record.

The criteria for positive scores were >7.5, >10, and >15 SDS/h for moderate SAS and >1, >2.5, >3, and >5 SDS/h

er of $\begin{bmatrix} 100\\ 00 \end{bmatrix}$ (SDS>7.5

for mild SAS. The sensitivity of oximetry alone for the recognition of the SAS was calculated as the number of true positive SaO_2 records divided by the total number of positive definitive (polysomnographic) records. The specificity was defined as the number of true negative SaO_2 records divided by the total number of negative definitive records.

To test the reproducibility of our findings, observers unfamiliar with the aim of the study re-examined the SaO_2 data and also compared the second night's data with the third night's polysomnographic data in 61 subjects to examine night-to-night variability. All values are expressed as the mean \pm SE.

3. Results

Of the 75 elderly patients studied, 20 were normal with AI < 5. Twenty-four subjects had moderate SAS (AI > 15) and 31 had mild SAS with AI < 15 but >5. Thus, a total of 55 subjects had mild to moderate SAS (AI > 5). When the SaO₂ recordings were classified as positive or negative, using a number of SDS of more than 2% desaturation from baseline SaO₂, the sensitivity was 92-98.7% with corresponding specificity of 56-30.7% for AI > 5, and 85.3–98.7% with corresponding specificity of 70.7–52% for AI > 15. Sensitivity was very high, but there were many cases of false positive results (Fig. 1). When we judged SDS as a drop in SaO₂ of more than 4%, the sensitivity was 61.8-96.4% with corresponding specificity of 100–40.0% for AI > 5 and 37.5–100% with corresponding specificity of 100-72.5% for AI > 15. Sensitivity is increased by reducing the frequency of SDS events required to count a positive result, but the corresponding specificity

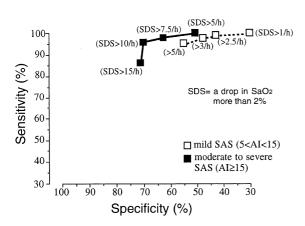


Fig. 1. The sensitivity and specificity for various criteria of a significant number of oxyhemoglobin desaturations to differentiate patients with an AI of more than 5 (open squares) or 15 (closed squares) from normal subjects (AI < 5). Open squares represent frequencies of desaturation of 2% or more from baseline from bottom to left: 5/h, 3/h, 2.5/h, 1/h. Closed squares represent frequencies of desaturation of 2% or more from baseline from bottom to left: 15/h, 10/h, 7.5/h, 5/h. Mild SAS, subjects with an AI of more than 5; moderate SAS, subjects with an AI of more than 15; SDS, 2% or more desaturation.

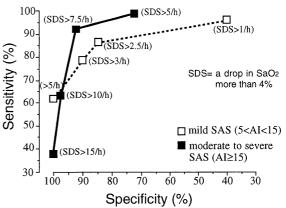


Fig. 2. The sensitivity and specificity for various criteria of a significant number of oxyhemoglobin desaturations to differentiate patients with an AI of more than 5 (open squares) or 15 (closed squares) from normal subjects (AI < 5). Open squares represent frequencies of desaturation of 2% or more from baseline from bottom to left: 5/h, 3/h, 2.5/h, 1/h. Closed squares represent frequencies of desaturation of 4% or more from baseline from bottom to left: 15/h, 10/h, 7.5/h, 5/h. Mild SAS, subjects with an AI of more than 5; moderate SAS, subjects with an AI of more than 15; SDS, 4% or more desaturation.

decreases. The balance between sensitivity and specificity can be acceptable (Fig. 2). When we used the number of SDS defined as a drop in SaO₂ of more than 6%, the sensitivity was 37.3-62.7% with corresponding specificity of 100-70.7% for AI > 5, and 30.7-78.7% with corresponding specificity of 100-85.3% for AI > 15. Although specificity seemed reasonable, sensitivity was not high enough to exclude SAS from normal subjects (Fig. 3).

Because apneas and SDS in one patient are not always similar to those in another, a scatter plot with a regression

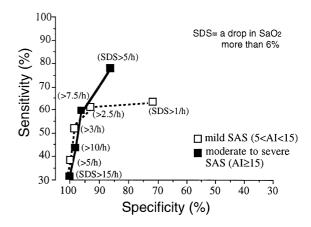


Fig. 3. The sensitivity and specificity for various criteria of a significant number of oxyhemoglobin desaturations to differentiate patients with an AI of more than 5 (open squares) or 15 (closed squares) from normal subjects (AI < 5). Open squares represent frequencies of desaturation of 2% or more from baseline from bottom to left: 5/h, 3/h, 2.5/h, 1/h. Closed squares represent frequencies of desaturation of 6% or more from baseline from bottom to left: 15/h, 3/h, 2.5/h, 1/h. Closed squares represent frequencies of desaturation of 6% or more from baseline from bottom to left: 15/h, 10/h, 7.5/h, 5/h. Mild SAS, subjects with an AI of more than 5; moderate SAS, subjects with an AI of more than 15; SDS, 6% or more desaturation.

line for the number of SDS vs. the number of apneas is shown in Fig. 4.

The difference among observers in the interpretation of records (number of SDS) of oximeter monitoring was less than 1%. The night (day 2) to night (day 3) variability of the numbers of apneas in polysomnographic studies was less than 11% ($5.8 \pm 0.4\%$). These results do not affect the analysis of sensitivity and/or specificity of oximeter monitoring for SAS.

4. Discussion

Among patients with hypersomnolence, SAS is the most common diagnosis made in sleep disorder centers. Sleep disordered breathing (SDB), including SAS, is often observed in the elderly population and may impair psychological as well as cardiovascular functions, sometimes causing potentially life-threatening events [4–15]. Detection of SDB among elderly subjects is important, not only to treat SAS but to detect and avoid risk factors. Although polysomnography is required to reveal and characterize SAS [16], it is expensive, time-consuming, and unavailable to patients living far from the sleep laboratory. In addition, some elderly persons cannot tolerate repeated polysomnographic studies. It would be desirable to have a simple screening test for sleep apnea in order to examine a greater number of elderly people.

Recently, non-invasive oxygen saturation monitoring, pulse oximeter, has been developed and used for screening and assessing SAS [17–25]. Séries et al. [23] reported a 98% sensitivity in oximetry diagnosis for obstructive sleep apnea, but a specificity of only 48% in 240 consecutive patients. On the other hand, Issa et al. [24] reported that the combination of snoring and oximetry monitoring for diagnosing SAS had an 84–90% sensitivity with a specificity of 95–49%. The difference may depend on the apnea hypopnea index (AHI) diagnostic criterion employed. A recent prospective study reported that the respiratory disturbance index (RDI), determined by an automated algorithm for analyzing digital oximetry, showed an excellent correla-

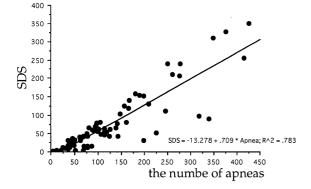


Fig. 4. A scatter plot with a regression line for the number SDS vs. the number of apneas in the patients.

tion of AHI determined by polysomnography [28]. Thus, the analytical use of oximetry data may also be important. However, the value of oximetry monitoring has not been extensively evaluated in the elderly.

An ideal screening test should have a high sensitivity with a reasonable specificity. Oximetry monitoring alone may identify many elderly patients with mild to moderate SAS, confirming the results of previous studies of the young and adults [17-25]. However, appropriate criteria of oxyhemoglobin desaturation for detection of SAS have not been determined. In the current study, we chose three different criteria for SDS for SAS. Our overall results suggest that 2% desaturation from baseline SaO₂ is sensitive, but not specific to differentiate SAS from normals, whereas 6% desaturation from baseline SaO₂ has high specificity but low sensitivity. There were many false positive cases judged by the 2% desaturation criterion and many false negatives judged by the 6% desaturation criterion. Compared with the two previous desaturation criteria, 4% desaturation from baseline SaO₂ was able to detect elderly subjects with SAS with a good sensitivity and a reasonable specificity. The 4% desaturation of baseline SaO₂ may be an appropriate index to detect elderly subjects with SAS by using an oximeter.

There is still controversy in published papers about the defining criteria for SAS [29]. The originally proposed criterion of more than five episodes of apnea per hour of sleep is now generally regarded as too strict, and an AI above ten or 15 is considered more realistic [30,31]. When we used the original criteria for the elderly, numerous subjects could be diagnosed with SAS. In the present study, we found 55 subjects with mild SAS, determined by an AI of greater than five per hour. These numbers reached 73.3% of the total sample in this study. However, some of the patients did not exhibit any symptoms and were not considered to have clinically significant SAS. When we defined moderate SAS as an AI of greater than 15 per hour, 24 subjects out of a total of 75 (32%) were found to have moderate SAS. These patients exhibited some of the clinical features of SAS, and were considered to have significant SAS. The relatively high prevalence of significant SAS in the elderly with habitual snoring is in agreement with previous studies [32,33].

Because a great number of elderly subjects should be examined to exclude SAS, a useful screening test is necessary to reduce the need for polysomnography. The current study indicates that home oximeter monitoring may be useful to exclude elderly patients with SDB from normal breathing elderly, with a relatively high sensitivity. It would be reasonable to speculate that oximeter monitoring is effective in identifying SAS, but not suitable for assessing disease severity. Thus, we should carefully consider the clinical limitations of nocturnal oximeter monitoring for detecting/assessing SAS. First, oximeter dips during the night are not always consistent with apneic events. The number and duration of dips are affected by many factors, including incorrect placement of the probe, impaired blood flow in the hand with the probe, line trouble in the oximeter and body movements which create artifacts in the oximeter recordings. Second, some cases of obstructive sleep apnea syndrome (OSAS) with predominantly short apneas may have few SDSs, resulting in misdiagnosis. Third, employment of oximetry monitoring alone implies a risk of both false positive and false negative results. Fourth, SaO₂ recordings alone cannot measure sleep time. Thus, the number of significant oxyhemoglobin desaturations during the night may not be the same as the number of oxyhemoglobin desaturations during sleep. Fifth, selection criteria for subjects may affect the results of sensitivity and specificity profiles. Sensitivity is inflated when the majority of subjects have sleep apnea. A larger sample with a wide range of apneas, providing more accurate sensitivity and specificity profiles of overnight oximeter monitoring, may be needed for further study.

In conclusion, nocturnal oximeter monitoring can be a useful tool to identify SAS in the elderly, and a 4% desaturation from baseline can be useful to predict significant sleep apnea in elderly persons.

Acknowledgements

This study was aided in part by grants from the Mitsui Life Social Welfare Foundation of Japan and Health Science Research Grant (Comprehensive Research on Aging and Health), Ministry of Health and Welfare of Japan.

References

- Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230–1351.
- [2] Bixler EO, Vgontzas AN, Ten Have T, et al. Effect of age on sleep apnea in men: 1. prevalence and severity. Am J Resp Crit Care Med 1998;157:144–148.
- [3] Foley DJ, Monjan AA, Masaki KH, et al. Associations of symptoms of sleep apnea with cardiovascular disease, cognitive impairment, and mortality among older Japanese-American men. J Am Geriatr Soc 1999;47:524–528.
- [4] Berry D, Phillips B, Cook Y, et al. Sleep-disordered breathing in healthy aged persons: possible daytime sequelae. J Gerontol 1987;42:620–626.
- [5] Ziegler MG, Nelesen R, Mills P, et al. Sleep apnea, norepinephrine release rate, and daytime hypertension. Sleep 1997;20:224–231.
- [6] Wali SO, Bahammam AS, Massaeli H, et al. Susceptibility of LDL to oxidative stress in obstructive sleep apnea. Sleep 1998;21:290–296.
- [7] Hung H, Whitford EG, Parsons RW, Hillman DR. Association of sleep apnea with myocardial infarction in men. Lancet 1990;336:261–264.
- [8] Yang EH, Hla KM, McHorney CA, et al. Sleep apnea and quality of life. Sleep 2000;23:535–541.
- [9] Teramoto S, Ohga E, Ouchi Y. Obstructive sleep apnoea (letter). Lancet 1999;354:1213–1214.
- [10] Bennett LS, Barbour C, Langford B, et al. Health status in obstructive sleep apnea. Relationship with sleep fragmentation and daytime sleepiness, and effects of continuous positive airway pressure treatment. Am J Resp Crit Care Med 1999;159:1884–1890.

- [11] Jenkinson C, Davies RJO, Mullins R, Stradling JR. Comparison of therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnea. Lancet 1999;353:2100–2105.
- [12] Kingshott RN, Vennelle M, Hoy CJ, et al. Predictors in improvements in daytime function outcomes with CPAP therapy. Am J Resp Crit Care Med 2000;161:866–871.
- [13] He J, Kryger M, Zorick F, et al. Mortality and apnea index in obstructive sleep apnea. Chest 1988;94:9–14.
- [14] Sajkov D, Cowie RJ, Thornton AT, et al. Pulmonary hypertension and hypoxemia in obstructive sleep apnea syndrome. Am J Resp Crit Care Med 1994;149:416–422.
- [15] Peker Y, Kraiczi H, Hedner J, et al. An independent association between obstructive sleep apnoea and coronary artery disease. Eur Resp J 1999;14:179–184.
- [16] Guilleminault C, Van den Hoed J, Milter MM. Clinical overview of the sleep apnea syndrome. In: Guilleminault C, Dement WC, editors. Sleep apnea syndromes. New York: Alan R. Liss, 1978:1–12.
- [17] Farney RJ, Walker LE, Jensen RL, et al. Ear oximetry to detect apnoea and differentiate rapid eye movement (REM) and non-REM (NREM) sleep. Screening for the sleep apnea syndrome. Chest 1986;89:533–539.
- [18] Cooper BG, Veale D, Griffiths CJ, Gibson GJ. Value of nocturnal oxygen saturation as a screening test for sleep apnoea. Thorax 1991;46:586–588.
- [19] Svanborg E, Larsson H, Carlsson-Nordlander B, Pirskanen R. A limited diagnostic investigation for obstructive sleep apnea syndrome. Chest 1990;98:1341–1345.
- [20] Williams AJ, Yu GF, Santiago S, et al. Screening for sleep apnea using pulse oximetry and a clinical score. Chest 1991;100:631–635.
- [21] Douglas NJ, Thomas S, Jan MA. Clinical value of polysomnography. Lancet 1992;339:347–350.
- [22] Gyulay S, Olson LG, Hensley MJ, et al. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. Am Rev Resp Dis 1993;147:50–53.
- [23] Séries F, Marc I, Cormier Y, et al. Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea syndrome. Ann Intern Med 1993;119:449–453.
- [24] Issa FG, Morrison DL, Hajduk E, et al. Digital monitoring of sleep disordered breathing using snoring sound and arterial oxygen saturation. Am Rev Resp Dis 1993;148:1023–1029.
- [25] Lévy P, Pépin JL, Deschaux-Blanc C, et al. Accuracy of oxymetry for detection of respiratory disturbances in sleep apnea syndrome. Chest 1996;109:395–399.
- [26] Teramoto S, Fukuchi Y, Orimo H. Effects of inhaled anticholinergic drug on dyspnea and gas exchange during exercise in patients with chronic obstructive pulmonary disease. Chest 1993;103:1774–1782.
- [27] Teramoto S, Sudo E, Matsuse T, et al. Impaired swallowing reflex in patients with obstructive sleep apnea syndrome. Chest 1999;116:17– 21.
- [28] Vázquez J-C, Tsai WH, Flemons WW, et al. Automated analysis of digital oximetry in the diagnosis of obstructive sleep apnoea. Thorax 2000;55:302–307.
- [29] Berry D, Webb W, Block AJ. Sleep apnea syndrome. A critical review of the apnea index as a diagnostic criterion. Chest 1984;86:529–531.
- [30] Berry DTR, Phillips BA, Cook YR, et al. Geriatric sleep apnea syndrome. A preliminary description. J Gerontol 1990;45:M169– M174.
- [31] Gould GA, Whyte KF, Rhind GB, et al. The sleep hypopnea syndrome. Am Rev Resp Dis 1988;137:895–898.
- [32] Ancoli-Israel S, Kripke DF, Mason W, Kaplan OJ. Sleep apnea and periodic movements in an aging sample. J Gerontol 1985;40:419– 425.
- [33] Ancoli-Israel S, Kripke DF, Mason W. Characteristics of obstructive and central sleep apnea in the elderly. An interim report. Biol Psychiatry 1987;22:741–750.