

# Oxygen Desaturation Index from Nocturnal Oximetry: A Sensitive and Specific Tool to Detect Sleep-Disordered Breathing in Surgical Patients

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**INTRODUCTION:** It is impractical to perform polysomnography (PSG) in all surgical patients suspected of having sleep disordered breathing (SDB). We investigated the role of nocturnal oximetry in diagnosing SDB in surgical patients.

**METHOD:** All patients 18 years and older who visited the preoperative clinics for scheduled inpatient surgery were approached for study participation. Patients expected to have abnormal electroencephalographic findings were excluded. All patients underwent an overnight PSG at home with a portable device and a pulse oximeter. The PSG recordings were scored by a certified sleep technologist. The oximetry recordings were processed electronically.

**RESULT:** Four hundred seventy-five patients completed the study: 217 males and 258 females, aged  $60 \pm 11$  years, and body mass index  $31 \pm 7$  kg/m<sup>2</sup>. The apnea-hypopnea index (AHI), the average number of episodes of apnea and hypopnea per hour of sleep, was 9.1 (2.8 to 21.4) [median (interquartile range)] and 64% patients had AHI >5. There was a significant correlation between oxygen desaturation index (ODI, hourly average number of desaturation episodes) and cumulative time percentage with SpO<sub>2</sub> <90% (CT90) from nocturnal oximetry, with the parameters measuring sleep breathing disorders from PSG. Compared to CT90, ODI had a stronger correlation and was a better predictor for AHI. The area under receiver operator characteristics curve for ODI to predict AHI >5, AHI >15, and AHI >30 was 0.908 (CI: 0.880 to 0.936), 0.931 (CI: 0.090 to 0.952), and 0.958 (CI: 0.937 to 0.979), respectively. The cutoff value based on the maximal accuracy for ODI to predict AHI >5, AHI >15, and AHI >30 was ODI >5, ODI >15, and ODI >30. The accuracy was 86% (CI: 83%–88%), 86% (CI: 83%–89%), and 94% (CI: 92%–96%), respectively. The ODI >10 demonstrated a sensitivity of 93% and a specificity of 75% to detect moderate and severe SDB.

**CONCLUSIONS:** ODI from a high-resolution nocturnal oximeter is a sensitive and specific tool to detect undiagnosed SDB in surgical patients. (Anesth Analg 2012;114:993–1000)

Obstructive sleep apnea (OSA) is one of the most frequent constituents of sleep disordered breathing (SDB). Growing evidence has implicated OSA as a causal pathway to the development of cardiorespiratory diseases, diabetes mellitus and autoimmune diseases.<sup>1–3</sup> The all-cause mortality is increased in proportion with the severity of SDB.<sup>4</sup> OSA can pose a significant challenge to anesthesiologists in the perioperative period due to the patient's possible difficult airway, increased sensitivity to narcotics, and postoperative upper airway obstruction.<sup>5</sup> It has been shown that OSA patients have an increased incidence of perioperative adverse events.<sup>6–8</sup>

In the general population, OSA was found in 24% of men and 9% of women with apnea-hypopnea index (AHI)

≥5 and in 11.4% of men and 4.7% of women with AHI ≥15 as the OSA diagnostic criteria.<sup>9</sup> It is estimated that nearly 80% of men and 93% of women with moderate to severe sleep apnea are undiagnosed.<sup>10</sup> Identifying the surgical patient with undiagnosed OSA allows the clinician to develop the appropriate perioperative management plan. In-laboratory polysomnography (PSG) is the gold standard for diagnosing OSA.<sup>11</sup> However, in-laboratory PSG is a time-consuming and costly procedure. Referring patients to sleep clinics usually results in postponing surgery. Most portable sleep monitoring devices also require intensive training of patients or assistance from well-trained technicians, and manual scoring of recordings by certified PSG technologists. Lack of convenient and economical diagnostic tools is a major obstacle that prevents anesthesiologists from diagnosing OSA and initiating treatment before surgery.

A high resolution oximeter is a watch oximeter with high sampling frequency and resolution, which requires little training to install properly. It can detect the fluctuations in oxygen saturation caused by episodes of apnea and hypopnea. In addition, the data can be automatically analyzed with commercially available computer programs with an acceptable accuracy. Although oximetry has been studied as a screening tool in sleep clinic patients,<sup>12,13</sup> no study has been published to evaluate the diagnostic performance of oximetry for SDB in the surgical patient. The objective of this study was to evaluate the predictive performance of a high-resolution oximeter in detecting SDB

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in surgical patients. Our hypothesis was that nocturnal oximetry is a simple alternative to PSG in diagnosing SDB in the preoperative clinic.

## METHODS

### Study Subjects

The study was performed at Toronto Western Hospital of University Health Network and Mount Sinai Hospital in Toronto. Approvals from the IRB of both hospitals were obtained. Written informed consent was obtained from all study subjects. Patients 18 years and older were potential candidates for the study. Patients who were unwilling or unable to give informed consent or patients who were expected to have abnormal electroencephalographic (EEG) findings (e.g., brain tumor, epilepsy surgery, patients with deep brain stimulator) were excluded. All eligible patients who visited the preoperative clinics for a scheduled inpatient surgery were approached. The patients giving study consent underwent an entire night of portable PSG and simultaneous oximetry preoperatively at the patients' homes.

In patients with an AHI >5, their family physicians were notified so that the patients could be referred to sleep physicians for further clinical management.

### Oximeter

Simultaneously, SaO<sub>2</sub> monitoring with a high-resolution pulse oximeter wristwatch (PULSOX-300i, Konica Minolta Sensing, Inc., Osaka, Japan) was performed along with portable PSG (Embletta ×100, Embla, Broomfield, CO). Each oxygen probe of the oximeter and PSG were attached to different fingers of the nondominant hand. The sampling frequency of the oximeter PULSOX-300i is 1 Hz on memory interval and an averaging time of 3 seconds. The resolution is 0.1% SpO<sub>2</sub> (information provided by the manufacturer). The data were downloaded into a personal computer the following morning and were processed with a specifically designed computer program, Profox (Profox Associates, Escondido, CA). First, the oximetry recording was visually checked by the technician and obvious artifacts were deleted. The aberrant recording was then scanned and excluded. Next, oxygen desaturation index (ODI), cumulative time percentage with SpO<sub>2</sub> <90% (CT90), lowest and average SpO<sub>2</sub> were extracted from the oximetry data. ODI is the hourly average number of desaturation episodes, which are defined as at least 4% decrease in saturation from the average saturation in the preceding 120 seconds, and lasting >10 seconds.

To minimize the bias from oximetry data recorded while the patient was awake, only oximetry recordings obtained between 00:00 hours and 6:00 hours were processed, although it was not known if patients were actually asleep during this entire period. Data processing was performed by a technician blinded to PSG results.

### Ambulatory Polysomnography

A full-night unattended portable PSG recording with Embletta × 100 was performed at the patient's home preoperatively. Embletta ×100 is a level 2 diagnostic tool for OSA<sup>14</sup> and has been validated against laboratory PSG.<sup>15</sup> The PSG recording montage consisted of 2 EEG channels

(C3 and C4), left or right electroculogram, chin muscle electromyogram, nasal cannula (pressure), thoracic and abdominal respiratory effort bands, body-position sensor, and pulse oximetry. The device measures the oxyhemoglobin saturation at a rate of 3 samples per second. The averaging time is one-third of a second.

At bedtime, the device was connected to the patients by a trained PSG technician at the patients' homes. The overnight recording was unattended. The patients were taught how to disconnect the device, which was retrieved by the same sleep technician the following morning. Patients were asked to keep a sleep diary. The sleep technician retrieving the device ensured that the sleep diary was completed by the patient.

Recordings from the portable PSG were scored by a certified PSG technologist and reviewed by a physician specialized in sleep medicine. Both were blinded to the clinical information and the results of the oximetry. Somnologia Studio 5.0 (Embla, Broomfield, CO) was the platform used for scoring PSG. The PSG recording was manually scored epoch by epoch by the PSG technologist, according to the manual published by the American Academy of Sleep Medicine in 2007.<sup>16</sup> Apnea was defined as a >90% decrease in air flow from baseline, which lasted at least 10 seconds. Apneas were classified as obstructive apnea if respiratory effort was present, central apnea if respiratory effort was absent during the event, or mixed apnea if characteristics of both obstructive or central apnea are present. Hypopnea was defined as a ≥30% reduction in air flow which lasted at least 10 seconds and was associated with a decrease of at least 4% in arterial oxyhemoglobin saturation. The AHI was defined as the number of episodes of apnea and hypopnea per hour of sleep.

## Data Analysis and Statistics

### Sample Size Estimation

The calculation of sample size was performed according to the method reported by Obuchowski.<sup>17</sup> The sample size was calculated based on the sensitivity and specificity of ODI ≥10 for the prediction of AHI ≥5 from a previous study of patients suspected of having OSA<sup>18</sup> and prevalence of OSA.<sup>19,20</sup> When a sensitivity of 0.82, a precision of 0.09, an OSA prevalence rate of 24%, a type I error of 0.05, and a power of 0.8 were used for sample size estimation, the power analysis resulted in 368 patients. Based on the specificity of 0.76 and a precision of 0.09, the sample size was 107 patients. To ensure sufficient power for estimation of sensitivity, a sample size of >368 patients was required.

### Data Analysis

Data were entered into a specifically designed Microsoft Access database (Microsoft Corp., Redmond, WA) and checked for possible errors. SAS 9.1 for Windows (SAS Institute, Cary, NC) was used for data analysis. All the statistical tests were 2-tailed and *P* < 0.05 was accepted as statistically significant.

The demographic data, summary of data from the PSG, and nocturnal oximeter were presented with descriptive statistics. Categorical data were presented as frequency with percentage, and the statistical significance was checked by  $\chi^2$  test or Fisher's exact test. The mean ± SD

was used for continuous data with normal distribution, median (interquartile range [IQR]) for continuous data with skewed distribution. Student's *t*-test was used to check the statistical significance for continuous data with normal distribution. Variances of 2 groups were checked first. If 2 populations had the equal variance, a pooled variance estimator was used. If the variances were unequal, the Satterthwaite's method was used.<sup>21</sup> Wilcoxon 2-sample test was used to check the statistical significance for continuous data with skewed distribution.

ODI and CT90 measured with nocturnal oximetry were compared with AHI simultaneously measured with portable home PSG. The Spearman correlation between variables from PSG with ODI and CT90 from high-resolution oximetry was analyzed. The receiver operator characteristics (ROC) curve and the area under ROC (AUC) for ODI and CT90 to predict OSA at different AHI cutoffs were analyzed with output data from logistic regression. Scatter plot and simple linear regression between ODI and AHI were depicted and explored. Bland-Altman analysis for AHI and ODI, the agreement between AHI and ODI, was also presented. Optimal cutoffs for ODI and CT90 were chosen according to maximal total accuracy (total accuracy = [true positive + true negative]/N). A validation dataset of 475 observations was generated from the original dataset by resampling with bootstrapping. The sensitivity, specificity, positive predictive value, negative predictive value, positive and negative likelihood ratio, and accuracy with 95% confidence intervals of ODI and CT90 at chosen cutoffs were also examined on the validation dataset to evaluate the predictive performance of ODI for SDB at different cutoffs of AHI.

## RESULTS

In 2 years (December 2007 to November 2009), 3760 patients seen in the preoperative clinic were invited to participate in this study, and 503 consented and 475 completed the study satisfactorily. Seventeen patients withdrew from the study because of a change of mind (10), preoperative stress (2), misunderstanding (3), incorrect contact information (1), and participating in another research study (1). Four hundred eighty-six surgical patients underwent portable PSG preoperatively at home. Of them, the PSG quality of 11 patients was technically unreliable. Four hundred seventy-five patients who had recordings of good quality on both PSG and oximetry were included in this report.

Compared to 3285 patients who were approached but not included in this report, the 475 patients included in the final analysis were older ( $60.3 \pm 12.7$  year vs  $58.5 \pm 14.5$  year,  $P = 0.003$ ), had a larger body mass index ( $30.5 \pm 6.8$  vs  $28.6 \pm 6.1$ ,  $P < 0.001$ ) and neck circumference ( $38.7 \pm 4.2$  vs  $37.7 \pm 5.0$ ,  $P < 0.001$ ). The demographic data, type of surgery, and preexisting medical conditions of 475 patients are summarized in Table 1. The most common type of surgery was orthopedic (56%).

The AHI was 9.1 (2.8 to 21.4) [median (IQR)]. There were 171 (36%) patients who had an AHI  $\leq 5$ , 138 (29%) patients who had an AHI  $> 5$  and  $\leq 15$ , 92 (19%) patients who had an AHI  $> 15$  and  $\leq 30$ , and 74 (16%) patients who had an AHI

**Table 1. Demographic Data**

Demographics	
N	475
Age (year)*	60 $\pm$ 13
Gender	
Male	217 (46)
Female	258 (54)
BMI (kg/m <sup>2</sup> )*	31 $\pm$ 7
ASA physical status	
I	13 (2.8)
II	223 (46.9)
III	225 (47.4)
IV	13 (2.8)
Type of surgery	
ENT	4 (0.7)
Orthopedic	266 (56.1)
Spine	60 (12.7)
General	74 (15.7)
Urology	26 (5.5)
Gynecology	21 (4.5)
Plastic	5 (1.0)
Ophthalmology	6 (1.2)
Other	13 (2.7)
Pre-existing co-morbidities	
Hypertension	208 (43.8)
GERD	117 (24.6)
Morbid obesity	188 (39.6)
Diabetes	78 (16.4)
Stroke	15 (3.2)
CAD	22 (4.6)
Myocardial infarction	6 (1.3)
COPD	15 (3.2)
Asthma	56 (11.8)

\*Data presented as mean  $\pm$  SD, all other data presented as frequency (%). CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; GERD = gastroesophageal reflux disease; morbid obesity = body mass index  $\geq 35$ ; ENT = ear, nose, throat.

$> 30$ . Sleep study data are summarized in Table 2. Compared with the patients with ODI  $\leq 5$ , there was a significant frequency of episodes of sleep breathing disorders in patients with ODI  $> 5$ . Although patients with ODI  $> 5$  had more central apnea episodes compared with patients with ODI  $\leq 5$ , the frequency of central apnea in the study population was very low, 0 (0–0) [median (IQR)] or  $0.18 \pm 0.94$  (mean  $\pm$  SD).

The ODI was 9.8 (4.6 to 17.3) [median (IQR)]. There were 128 (27%) patients who had an ODI  $\leq 5$ , 203 (43%) patients who had an ODI  $> 5$  and  $\leq 15$ , 77 (16%) patients who had an ODI  $> 15$  and  $\leq 30$ , and 64 (14%) patients who had an ODI  $> 30$ . Oximeter data are shown in Table 3. Compared with patients with AHI  $\leq 5$ , the patients with AHI  $> 5$  had a higher ODI and CT90, a lower average  $SpO_2$ , and lowest  $SpO_2$ . The correlation between the major parameters of SDB as obtained from the portable PSG and ODI or CT90 from the oximeter is shown in Table 4. There was a strong correlation ( $r_s$ : 0.70 to 0.90) between the ODI and AHI, hypopnea index, nonrapid eye movement AHI, rapid eye movement AHI, respiratory arousal index, and the lowest  $SpO_2$  on PSG. There was a moderate correlation ( $r_s$ : 0.40 to 0.69) between the ODI and obstructive apnea index. There was a weak correlation ( $r_s$ : 0.20 to 0.39) between the ODI and mixed apnea index. There was a very weak correlation ( $r_s \leq 0.19$ ) between the ODI and central apnea index. A correlation between CT90 from the oximeter and the above parameters from PSG was also found (Table 4). However,



**Table 2. Summary of Sleep Study Results**

	Total	ODI ≤5	ODI >5	P*
N	475	128	347	
Total sleep time (min)†	349 (293–396)	358 (316–408)	347 (287–394)	0.063
Sleep efficiency (%)†	86.4 (77.0–91.9)	87.1 (77.9–93.2)	86.3 (76.0–91.6)	0.193
REM latency (min)‡	86.5 (62.2–126.5)	89.3 (64.5–122.8)	85.3 (61.2–127.5)	0.484
REM percent (%)‡	20.8 (15.0–26.1)	21.8 (12.4–26.2)	20.6 (14.5–26.0)	0.115
Sleep stage: 1 (%)‡	4.7 (3.0–8.0)	4.5 (2.4–7.1)	4.9 (5.2–8.7)	0.011
2 (%)‡	61.6 (54.1–69.7)	60.6 (53.7–68.2)	61.8 (54.4–70.3)	0.240
Slow wave sleep (%)‡	9.8 (3.8–16.8)	10.7 (7.3–17.3)	9.3 (2.8–16.8)	0.017
AHI (events/h)‡	9.1 (2.8–21.4)	1.8 (0.8–3.5)	13.5 (7.4–27.8)	<0.001
REM AHI (events/h)‡	14.8 (4.2–39.1)	3.1 (0.7–7.4)	23.7 (9.5–47.1)	<0.001
NREM AHI (events/h)‡	5.9 (1.5–18.5)	1.0 (0.3–2.1)	11.1 (3.9–25.7)	<0.001
Apnea index (events/h)‡	1.5 (0.2–6.8)	0.2 (0–0.9)	3.4 (0.7–10.7)	<0.001
Obstructive apnea index‡	1.3 (0.1–6.3)	0.2 (0–0.7)	3.0 (0.5–10.0)	<0.001
Central apnea index (events/h) ‡	0 (0–0)	0 (0–0)	0 (0–0)	<0.001
Central apnea index (events/h)†	0.18 ± 0.94	0.02 ± 0.10	0.24 ± 1.10	
Mixed apnea index (events/h)‡	0 (0–0.2)	0 (0–0)	0 (0–0.2)	<0.001
Mixed apnea index (events/h)†	0.78 ± 5.01	0.03 ± 0.10	1.06 ± 5.8	
Hypopnea index (events/hr)†	5.9 (1.9–13.0)	1.4 (0.6–2.4)	8.8 (4.6–15.8)	<0.001
Respiratory arousal index (events/h)‡	5.9 (1.7–15.2)	1.1 (0.5–2.2)	9.1 (4.4–19.1)	<0.001

\*P value from Wilcoxon two-sample test.

†data presented as mean ± SD, all other data, except N, presented as median (Interquartile range).

‡AHI = apnea hypopnea index; Apnea index = average hourly number of apnea episodes; hypopnea index = average hourly number of hypopnea episodes; mixed apnea index = average hourly number of apnea episodes with characteristics of both obstructive or central apnea; NREM AHI = apnea hypopnea index during nonrapid eye movement (NREM) sleep; obstructive or central apnea index = average hourly number of obstructive or central apnea episodes; REM AHI = apnea hypopnea index during rapid eye movement (REM) sleep; REM latency = time from light off to first REM sleep; REM percent = percentage of REM sleep in total sleep time; respiratory arousal index = average hourly sleep arousals due to respiratory events; sleep efficiency = percentage of total sleep time in sleep period; sleep stage 1 or 2 = percentage of stage 1 or 2 in total sleep time; slow wave sleep (previous stage 3 and 4) = percentage of slow wave sleep in total sleep time; total sleep time = total time being asleep; ODI = oxygen desaturation index.

**Table 3. Summary of Data from an Oximeter**

	Total	AHI ≤5	AHI >5	P*
N	475	171	304	
Total time analyzed (min) <sup>a</sup>	360 (360–360)	360 (360–360)	360 (360–360)	0.877
Oxygen desaturation index (events/hr) <sup>a</sup>	9.8 (4.6–17.3)	3.8 (2.3–6.0)	14.0 (9.0–24.8)*	<0.001
Average SpO <sub>2</sub> (%) <sup>b</sup>	94.3 ± 2.4	95.1 ± 1.7	93.8 ± 2.6*	<0.001
Lowest SpO <sub>2</sub> (%) <sup>b</sup>	77.3 ± 11.7	80.3 ± 11.2	75.6 ± 11.7*	<0.001
Time percentage with SpO <sub>2</sub> <90% <sup>a</sup>	1.0 (0.3–4.3)	0.3 (0–0.8)	2.3 (0.7–6.9)	<0.001

\*P value from Wilcoxon 2-sample test.

<sup>a</sup> Data presented as median (interquartile range).

<sup>b</sup> Data presented as mean ± SD.

AHI = apnea hypopnea index.

**Table 4. Correlation Between ODI and CT90 with Parameters from Polysomnography**

PSG Parameters	ODI*		CT90	
	r <sub>s</sub>	P	r <sub>s</sub>	P
AHI	0.886 (0.865–0.904)	<0.001	0.604 (0.543–0.658)	<0.001
Apnea index	0.685 (0.634–0.730)	<0.001	0.461 (0.386–0.528)	<0.001
Obstructive apnea index	0.683 (0.631–0.728)	<0.001	0.461 (0.387–0.529)	<0.001
Central apnea	0.189 (0.100–0.275)	<0.001	0.145 (0.055–0.231)	0.007
Mixed apnea	0.398 (0.319–0.472)	<0.001	0.273 (0.187–0.354)	<0.001
Hypopnea index	0.843 (0.815–0.861)	<0.001	0.543 (0.476–0.604)	<0.001
AHI-REM	0.760 (0.718–0.595)	<0.001	0.544 (0.477–0.605)	<0.001
AHI-NREM	0.839 (0.810–0.864)	<0.001	0.560 (0.495–0.619)	<0.001
Respiratory arousal index	0.845 (0.816–0.869)	<0.001	0.539 (0.472–0.600)	<0.001
Lowest SaO <sub>2</sub> on PSG	–0.774 (–0.808–0.734)	<0.001	–0.692 (–0.737–0.641)	<0.001

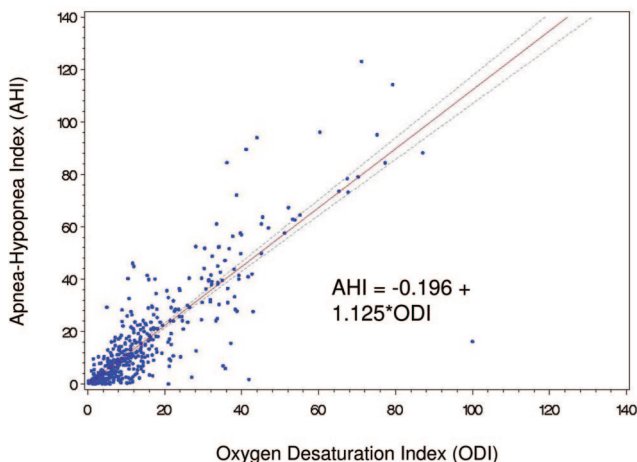
There was a strong correlation between ODI and AHI, hypopnea index, AHI-REM, AHI-NREM, and respiratory arousal index (r<sub>s</sub> >0.7); 78.5% variance of AHI could be explained by ODI.

AHI = apnea-hypopnea index; AHI-NREM = AHI during nonrapid eye movement sleep; AHI-REM = AHI during rapid eye movement sleep; CT90 = cumulative time percentage with SpO<sub>2</sub> <90%; ODI = oxygen desaturation index; r<sub>s</sub> = Spearman correlation coefficient; PSG = polysomnography.

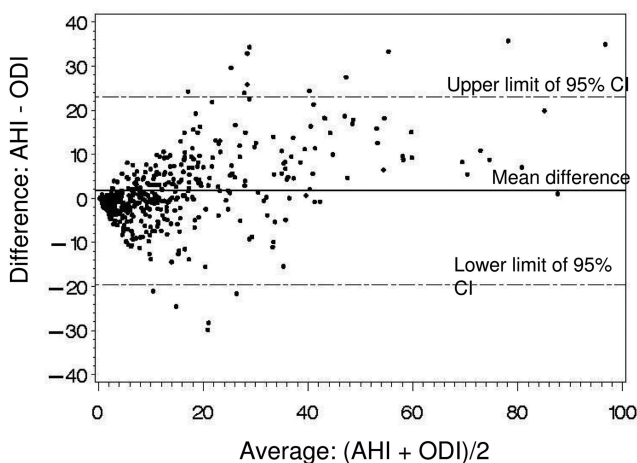
the Spearman coefficients (r<sub>s</sub>) were much smaller. There was no overlapping in 95% confidence intervals of r<sub>s</sub> for ODI with AHI, hypopnea index, nonrapid eye movement AHI, rapid eye movement AHI and respiratory arousal index, and CT90 with the above parameters.

The relationship between the ODI and AHI is further depicted in Figure 1 and Figure 2. The ODI slightly underestimated AHI by 1.6 ± 10 (mean ± SD) or 0 (–2.4 to 4.7) [median (IQR)].

To compare the accuracy of the ODI and CT90 to predict SDB at different AHI cutoffs, the ROC curves and the area



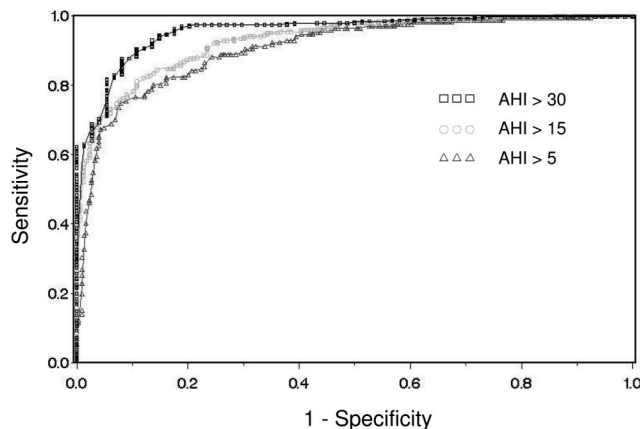
**Figure 1.** Apnea hypopnea index (AHI) from portable polysomnography versus oxygen desaturation index (ODI) from simultaneous oximetry with linear regression and 95% confidence interval.  $R^2 = 0.789$ ,  $AHI = 1.125 \times ODI - 0.196$ .



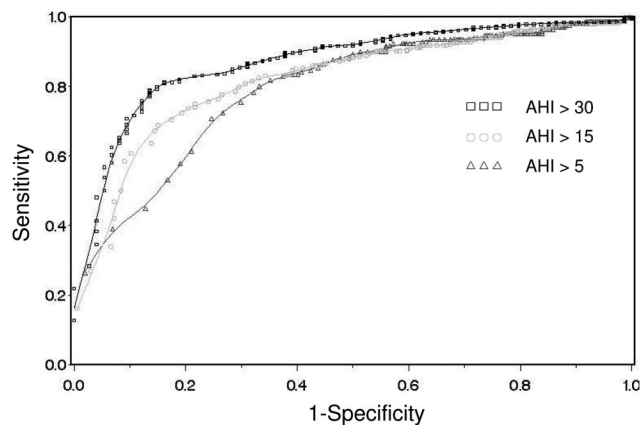
**Figure 2.** Bland-Altman plots of apnea hypopnea index (AHI) from portable polysomnography and oxygen desaturation index (ODI) from simultaneously recorded oximetry. The X axis represents the average of AHI and ODI. The Y axis represents the difference of AHI and ODI (AHI - ODI). When the average of AHI and ODI is low (approximately <18), the ODI tended to overestimate the AHI. When the average is high, ODI tended to underestimate the AHI.

under ROC curves for ODI and CT90 at AHI cutoffs of 5, 15, and 30 are presented in Figure 3 and Figure 4. The accuracy of ODI to predict SDB at different cutoffs was significantly higher than CT90. ODI was a good predictor of SDB at the different AHI cutoffs, where the area under ROC curve (AUC) ranged from 0.908 to 0.958.

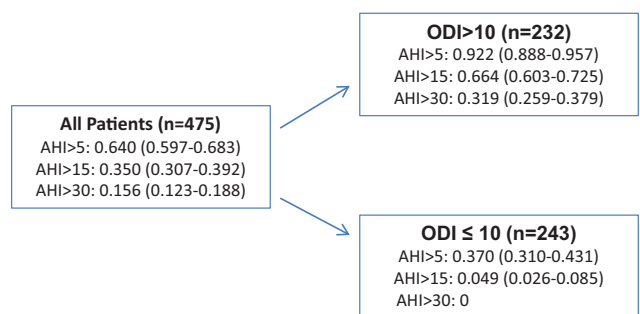
The cutoff based on maximal accuracy for ODI to predict AHI >5 (including all OSA), AHI >15 (including moderate and severe OSA) and AHI >30 (including severe OSA) was ODI >5, ODI >15, and ODI >30. The accuracy was 86% (CI: 83 to 88), 86% (CI: 83 to 89), and 94% (CI: 92 to 96), respectively. The ODI cutoff that ensured a high sensitivity and good specificity to detect moderate and severe OSA (AHI >15) was ODI >10. ODI >10 as a cutoff effectively separated the patients with high probability of moderate and severe OSA (Fig. 5). The predictive performance of ODI



**Figure 3.** Receiver operating characteristic (ROC) curves for oxygen desaturation index (ODI) to predict AHI >5, AHI >15, and AHI >30. The area under ROC curve was 0.908 (CI: 0.880 to 0.936) for AHI >5, 0.931 (CI: 0.909 to 0.952) for AHI >15 and 0.958 (CI: 0.937 to 0.979) for AHI >30.



**Figure 4.** Receiver operating characteristic (ROC) curves for cumulative time percentage with  $SpO_2 < 90\%$  cumulative time (CT90) to predict apnea-hypopnea index (AHI) >5, AHI >15 and AHI >30. The area under ROC curve was 0.792 (CI: 0.750 to 0.834) for AHI >5, 0.818 (CI: 0.779 to 0.858) for AHI >15, and 0.871 (CI: 0.830 to 0.913) for AHI >30.



**Figure 5.** The pretest and posttest probability of obstructive sleep apnea (OSA) at different apnea-hypopnea index (AHI) cutoffs for oxygen desaturation index (ODI) >10. ODI >10 as cutoff effectively identified the patients with high probability of moderate and severe OSA.

at selected cutoffs in the validation dataset was summarized in Table 5. The optimal cutoffs for CT90 to predict AHI >5, AHI >15, and AHI >30 were CT90 >0.7, CT90 >1.7, and CT90 >3.5, respectively (data not presented).

**Table 5. Predictive Value of Oxygen Desaturation Index for Sleep Disordered Breathing at Different AHI Cutoffs**

	ODI >5	ODI >10	ODI >15	ODI >30
N (%)	360 (75.8)	243 (51.2)	148 (31.2)	71 (15.0)
AHI >5/h				
Sensitivity (%)	96.3 (94.2–98.3)	70.5 (65.5–75.5)	45.0 (39.6–50.5)	21.7 (17.2–26.2)
Specificity (%)	67.3 (59.9–74.8)	89.5 (84.7–94.4)	98.0 (95.8–100)	99.4 (98.1–100)
PPV (%)	86.1 (82.5–89.7)	93.4 (90.3–96.5)	98.0 (95.7–100)	98.6 (96.9–100)
NPV (%)	89.6 (84.0–95.2)	59.1 (52.7–65.4)	45.9 (40.5–51.3)	37.6 (32.9–42.4)
Positive LR	2.95 (2.30–4.09)	6.74 (3.96–14.25)	22.97 (6.72–49.47)	33.26 (6.06–29.74)
Negative LR	0.06 (0.03–0.13)	0.33 (0.25–0.41)	0.56 (0.49–0.65)	0.79 (0.72–0.85)
Total accuracy (%)	87.0 (81.5–89.9)	65.5 (60.6–71.5)	62.1 (57.0–68.8)	46.7 (43.0–55.3)
AHI >15/h				
Sensitivity (%)	99.4 (98.4–100)	93.3 (89.7–97.0)	70.0 (63.3–76.7)	38.3 (31.2–45.9)
Specificity (%)	38.6 (33.1–44.2)	74.6 (69.6–79.6)	92.5 (89.5–95.5)	99.3 (98.4–99.9)
PPV (%)	49.7 (44.6–54.9)	69.1 (63.3–74.9)	85.1 (79.4–90.9)	97.2 (93.3–100)
NPV (%)	99.1 (97.4–100)	94.8 (92.0–97.7)	83.5 (79.5–87.5)	72.5 (68.2–76.9)
Positive LR	1.62 (1.51–1.90)	3.67 (3.02–5.22)	9.39 (7.26–23.3)	56.5 (13.05–89.76)
Negative LR	0.03 (0.02–0.08)	0.09 (0.01–0.12)	0.32 (0.20–0.39)	0.62 (0.51–0.71)
Total accuracy (%)	61.7 (56.0–67.4)	81.7 (77.8–86.9)	84.0 (82.3–90.0)	76.2 (72.7–82.9)
AHI >30/h				
Sensitivity (%)	100 (100–100)	100 (100–100)	94.9 (90.1–99.8)	76.0 (66.5–85.4)
Specificity (%)	29.0 (24.6–33.5)	58.6 (53.7–63.4)	81.6 (77.8–85.4)	97.2 (95.6–98.6)
PPV (%)	21.9 (17.7–26.2)	32.5 (26.6–38.4)	50.7 (42.6–58.7)	84.5 (76.1–92.9)
NPV (%)	100 (100–100)	100 (100–100)	98.8 (97.6–100)	95.3 (93.2–97.4)
Positive LR	1.41 (1.36–1.61)	2.42 (2.14–2.98)	5.15 (4.13–7.40)	27.34 (13.85–88.24)
Negative LR	0	0	0.06 (0.02–0.15)	0.25 (0.14–0.40)
Total accuracy (%)	40.8 (37.6–48.8)	65.5 (60.6–71.5)	83.8 (80.1–88.6)	93.7 (90.6–96.3)

The results were from a validation dataset generated from original dataset by re-sampling with bootstrapping. Except N, all data presented as value (lower–upper 95% confidence limit).

LR = negative likelihood ratio; NPV = negative predictive value; positive LR = positive likelihood ratio; ODI = oxygen desaturation index; PPV = positive predictive value; AHI = apnea-hypopnea index.

The agreement between AHI and ODI was evaluated with  $\kappa$  coefficient at different cutoffs. It was 0.666 (CI: 0.596 to 0.731) between AHI >5 and ODI >5, 0.685 (CI: 0.615 to 0.745) between AHI >15 and ODI >15, and 0.775 (CI: 0.694 to 0.856) between AHI >30 and ODI >30.

## DISCUSSION

This prospective cohort study showed that ODI had a strong correlation with the parameters measuring sleep breathing disorders from PSG. Although ODI slightly underestimated AHI by  $1.6 \pm 10$ , ODI had a very high accuracy to predict AHI at cutoffs of 5, 15, and 30 with the area under ROC of 0.908 to 0.958. ODI >5 was a good predictor for AHI >5 with an accuracy of 87%, ODI >15 for AHI >15 with an accuracy of 84%, and ODI >30 for AHI >30 with an accuracy of 93.7%. The cutoff of ODI >10 had a high sensitivity (93.3%) to detect moderate and severe OSA. CT90 was less accurate in detecting SDB.

Because of the lack of a convenient, inexpensive, and accurate tool, it has been a challenge to precisely identify surgical patients with SDB in preoperative clinics. The results from this study suggest that nocturnal ODI from a high-resolution pulse oximetry during sleep can be a very useful tool in assisting anesthesiologists to recognize patients with SDB, which may guide perioperative management. Because the sensitivity of ODI >5 to detect patients with AHI >5, AHI >15, and AHI >30 was 96.3%, 99.4%, and 100.0%, respectively, we would be confident to exclude the possibility of moderate to severe OSA if a patient had ODI  $\leq 5$ . On the other hand, the specificity for ODI >15 to detect AHI >5, AHI >15 m and AHI >30 was 98%, 92.5%, and 81.6%, respectively; and the specificity for ODI >30 to

detect AHI >5, AHI >15, and AHI >30 was 99.4%, 99.4%, and 97.2%, respectively. Therefore, we would be confident to consider a patient with ODI >15 as having moderate to severe SDB, and a patient with ODI >30 as having severe SDB. If one cutoff of ODI needs to be chosen, ODI >10 would be a good choice as it could detect almost all patients with moderate and severe SDB, while still retaining a reasonable specificity.

With ODI from a nocturnal oximeter, it is possible to stratify surgical patients and allocate resources for postoperative monitoring based on OSA-related risk. It could also help us identify patients who might benefit from continuous positive airway pressure treatment. Netzer et al. have suggested that patients with strong clinical suspicion for SDB and ODI >15 on oximetry should have autocontinuous positive airway pressure titration at home.<sup>22</sup>

Although using nocturnal oximetry to detect patients with SDB is not a new idea,<sup>12,13,23–25</sup> there are methodological issues in previously published studies. Several studies used PSG on a different night to evaluate the predictive performance of the oximeter.<sup>13,24,26</sup> Because of the environmental difference between home and sleep laboratory, and night-to-night variability in the frequency of sleep apnea and hypopnea,<sup>27,28</sup> PSG not performed on the same night as oximetry may not be able to accurately evaluate the role of nocturnal oximetry in the diagnosis of OSA. One study in elderly habitual snorers,<sup>24</sup> which also used a decrease in  $\text{SpO}_2$  of >4% to define ODI, showed that the sensitivity and specificity for ODI >10 to predict AHI  $\geq 15$  were 63% and 95%, respectively. The sensitivity is lower than the results in our study (93.3%), but their specificity is higher than in our data (74.6%).

Two previous studies used off-line analysis of pulse oximetry data from PSG as the oximetry data against the PSG data to evaluate the predictive performance of oximetry.<sup>23,29</sup> Because sampling frequency of the oximeter on PSG is usually higher than that of a stand-alone oximeter, and the same pulse oximetry data were also used to define a hypopnea episode of AHI, caution should be exercised when interpreting the results from these studies. Furthermore, no study has been done in surgical patients to evaluate the predictive accuracy for SDB. In our study, PSG and the oximeter were recorded simultaneously at patients' homes, which is a more natural sleeping environment than that in a sleep laboratory.

The different parameters from an oximeter have been investigated to acquire a better predictor for AHI or OSA. The parameters include  $\delta$  index (the average of the absolute differences of oxygen saturation between successive 12-s intervals),<sup>12,23</sup> ODI at the desaturation threshold of 2%, 3%, and 4%,<sup>23,30,31</sup> and CT90.<sup>31,32</sup> CT90 had been shown to have weaker correlation with AHI,<sup>32</sup> which is consistent with our results. Parameters from the more complicated analysis of oximetry recording, such as frequency domain indices,<sup>31</sup> central tendency measure,<sup>33</sup> and other parameters<sup>33–35</sup> were also under investigation. It appears that  $\delta$  index demonstrated a higher predictive accuracy, but there was no significant difference from ODI.<sup>23</sup>

We focused our study on the predictive performance of ODI for two reasons. First, ODI is the most commonly used measurement and can be easily generated with commercially available programs. Second, the definition of ODI is similar to the oxygen desaturation requirement for scoring a hypopnea episode we adopted.<sup>16</sup>

Although high-resolution pulse oximetry is a convenient and inexpensive way to detect SDB, it has some limitations. Because it only measures the oxygen saturation change and does not monitor nasal flow and respiratory effort, it is not able to distinguish OSA from central sleep apnea.

There are also limitations to our study. One is that there was a self-selection of patients involved in the patient recruiting process. Although we approached all patients who met the criteria for inclusion and tried to recruit them for the study, the patients with SDB-related symptoms were more willing to give consent. This may account for the high prevalence of SDB in the study population. This may also affect the positive and negative predictive value of the ODI and CT90. However, the sensitivity and specificity should not be affected.

Another limitation is that we used results from a portable PSG device (Embletta X100) as the standard to evaluate the predictive performance of high-resolution oximetry. Portable monitors are being incorporated into the clinical evaluation of SDB.<sup>36</sup> Embletta X100 is a level 2 diagnostic device for SDB, which is unattended sleep study equipment with a minimum of 7 channels, including EEG, electrooculogram, chin electromyogram, electrocardiogram or heart rate, airflow, respiratory effort, and oxygen saturation.<sup>14</sup> When applied to the patient by a trained technician and scored by a certified PSG technologist, parameters measuring SDB and sleep architecture from Embletta X100 were comparable to in-laboratory, standard PSG.<sup>15</sup> The third limitation of the study is that the predictive performance

was validated in a dataset generated from original dataset by resampling, which might overestimate the predictive performance.

In conclusion, ODI from nocturnal oximetry was strongly correlated with the parameters measuring sleep breathing disorders from PSG. ODI was a good predictor of AHI. Based on maximal accuracy, ODI >5, ODI >15, and ODI >30 were good predictors for AHI >5, AHI >15, and AHI >30, respectively. The ODI >10 demonstrated a high sensitivity (93%) and reasonable specificity (75%) to detect moderate and severe SDB. It effectively identified the patients with moderate and severe OSA. ODI from a high-resolution nocturnal oximeter is a sensitive and specific tool to preoperatively detect undiagnosed SDB in surgical patients. ■■

#### DISCLOSURES

**Name:** Frances Chung, FRCPC.

**Contribution:** This author helped design the study, conduct the study, and write the manuscript.

**Attestation:** Frances Chung has seen the original study data, reviewed the analysis of the data, approved the final manuscript, and is the author responsible for archiving the study files.

**Name:** Pu Liao, MD.

**Contribution:** This author helped design the study, conduct the study, analyze the data, and write the manuscript.

**Attestation:** Pu Liao has seen the original study data, reviewed the analysis of the data, approved the final manuscript, and is the author responsible for archiving the study files.

**Name:** Hisham Elsaid, MD.

**Contribution:** This author helped conduct the study.

**Attestation:** Hisham Elsaid approved the final manuscript.

**Name:** Sazzadul Islam, MSc.

**Contribution:** This author helped conduct the study.

**Attestation:** Sazzadul Islam approved the final manuscript.

**Name:** Colin M. Shapiro, FRCPC.

**Contribution:** This author supervised the sleep study and helped write the manuscript.

**Attestation:** Colin M. Shapiro approved the final manuscript.

**Name:** Yuming Sun, MD.

**Contribution:** This author helped conduct the study.

**Attestation:** Yuming Sun approved the final manuscript.

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#### REFERENCES

1. Hung J, Whitford EG, Parsons RW, Hillman DR. Association of sleep-apnea with myocardial-infarction in men. *Lancet* 1990;336:261–4
2. Doherty LS, Kiely JL, Swan V, McNicholas WT. Long-term effects of nasal continuous positive airway pressure therapy on cardiovascular outcomes in sleep apnea syndrome. *Chest* 2005;127:2076–84
3. Leung RS, Bradley TD. Sleep apnea and cardiovascular disease. *Am J Respir Crit Care Med* 2001;164:2147–65
4. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, Stubbs R, Hla KM. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep* 2008;31:1071–8



5. Chung SA, Yuan H, Chung F. A systemic review of obstructive sleep apnea and its implications for anesthesiologists. *Anesth Analg* 2008;107:1543–63
6. Chung, F, Yegneswaran B, Liao P, Chung SA, Vairavanathan S, Islam S, Khajehdehi A, Shapiro CM. Validation of Berlin Questionnaire and ASA Checklist as screening tools for obstructive sleep apnea in surgical patients. *Anesthesiology* 2008;108:822–30
7. Liao P, Yegneswaran B, Vairavanathan S, Zilberman P, Chung F. Postoperative complications in patients with obstructive sleep apnea: a retrospective matched cohort study. *Can J Anaesth* 2009;56:819–28
8. Flum DR, Belle SH, King WC, Wahed AS, Berk P, Chapman W, Pories W, Courcoulas A, McCloskey C, Mitchell J, Patterson E, Pomp A, Staten MA, Yanovski SZ, Thirlby R, Wolfe B. Perioperative safety in the longitudinal assessment of bariatric surgery. *N Engl J Med* 2009;361:445–54
9. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med* 2002;165:1217–39
10. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep* 1997;20:705–6
11. Practice parameters for the indications for polysomnography and related procedures. Polysomnography Task Force, Am Sleep Disorders Association Standards of Practice Committee. *Sleep* 1997;20:406–22
12. Levy P, Pepin JL, schaux-Blanc C, Paramelle B, Brambilla C. Accuracy of oximetry for detection of respiratory disturbances in sleep apnea syndrome. *Chest* 1996;109:395–9
13. Series F, Marc I, Cormier Y, La FJ. Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome. *Ann Intern Med* 1993;119:449–53
14. Practice parameters for the use of portable recording in the assessment of obstructive sleep apnea. Standards of Practice Committee of the Am Sleep Disorders Association. *Sleep* 1994;17:372–7
15. Chung F, Liao P, Sun Y, Amirshahi B, Fazel H, Shapiro CM, Elsaid H. Perioperative practical experiences in using a level 2 portable polysomnography. *Sleep Breath*. 2010
16. Iber C, Ancoli-Israel S, Quan SF, for the American Academy of Sleep Medicine. *The AASM Manual for the Scoring of Sleep and Associated Events: rules, terminology and technical specifications*, 1st ed.: Westchester, IL: American Academy of Sleep Medicine, 2007
17. Obuchowski NA. Sample size calculations in studies of test accuracy. *Stat Methods Med Res* 1998;7:371–92
18. Chiner E, Signes-Costa J, Arriero JM, Marco J, Fuentes I, Sergado A. Nocturnal oximetry for the diagnosis of the sleep apnoea hypopnoea syndrome: a method to reduce the number of polysomnographies? *Thorax* 1999;54:968–71
19. Netzer NC, Hoegel JJ, Loube D, Netzer CM, Hay B, varez-Sala R, Strohl KP. Prevalence of symptoms and risk of sleep apnea in primary care. *Chest* 2003;124:1406–14
20. Kushida CA, Nichols DA, Simon RD, Young T, Grauke JH, Britzmann JB, Hyde PR, Dement WC. Symptom-based prevalence of sleep disorders in an adult primary care population. *Sleep Breath* 2000;4:9–14
21. Moser BK, Stevens GR. Homogeneity of variance in the two-sample means test. *The Am Statistician* 1992;46:19–21
22. Netzer N, Eliasson AH, Netzer C, Kristo DA. Overnight pulse oximetry for sleep-disordered breathing in adults: a review. *Chest* 2001;120:625–33
23. Magalang UJ, Dmochowski J, Veeramachaneni S, Draw A, Mador MJ, El-Solh A, Grant BJ. Prediction of the apnea-hypopnea index from overnight pulse oximetry. *Chest* 2003;124:1694–701
24. Teramoto S, Matsuse T, Fukuchi Y. Clinical significance of nocturnal oximeter monitoring for detection of sleep apnea syndrome in the elderly. *Sleep Med* 2002;3:67–71
25. Martinez MW, Rodysill KJ, Morgenthaler TI. Use of ambulatory overnight oximetry to investigate sleep apnea in a general internal medicine practice. *Mayo Clin Proc* 2005;80:455–62
26. Williams AJ, Yu G, Santiago S, Stein M. Screening for sleep apnea using pulse oximetry and a clinical score. *Chest* 1991;100:631–5
27. Bittencourt LR, Suchecki D, Tufik S, Peres C, Togeiro SM, Bagnato MC, Nery LE. The variability of the apnoea-hypopnoea index. *J Sleep Res* 2001;10:245–51
28. Davidson TM, Gehrman P, Ferreyra H. Lack of night-to-night variability of sleep-disordered breathing measured during home monitoring. *Ear Nose Throat J* 2003;82:135–8
29. Vazquez JC, Tsai WH, Flemons WW, Masuda A, Brant R, Hajduk E, Whitelaw WA, Remmers JE. Automated analysis of digital oximetry in the diagnosis of obstructive sleep apnoea. *Thorax* 2000;55:302–7
30. Nakano H, Ikeda T, Hayashi M, Ohshima E, Itoh M, Nishikata N, Shinohara T. Effect of body mass index on overnight oximetry for the diagnosis of sleep apnea. *Respiratory Med* 2004;98:421–7
31. Lin CL, Yeh C, Yen CW, Hsu WH, Hang LW. Comparison of the indices of oxyhemoglobin saturation by pulse oximetry in obstructive sleep apnea hypopnea syndrome. *Chest* 2009; 135:86–93
32. Olson LG, Ambrogetti A, Gyulay SG. Prediction of sleep-disordered breathing by unattended overnight oximetry. *J Sleep Res* 1999;8:51–5
33. Alvarez D, Hornero R, Garcia M, del CF, Zamarron C. Improving diagnostic ability of blood oxygen saturation from overnight pulse oximetry in obstructive sleep apnea detection by means of central tendency measure. *Artif Intell Med* 2007;41: 13–24
34. Alvarez D, Hornero R, Abasolo D, del CF, Zamarron C, Lopez M. Nonlinear measure of synchrony between blood oxygen saturation and heart rate from nocturnal pulse oximetry in obstructive sleep apnoea syndrome. *Physiol Meas* 2009;30: 967–82
35. Marcos JV, Hornero R, Alvarez D, del CF, Zamarron C. Assessment of four statistical pattern recognition techniques to assist in obstructive sleep apnoea diagnosis from nocturnal oximetry. *Med Engl Phys* 2009;31:971–8
36. Collop NA, Anderson WM, Boehlecke B, Claman D, Goldberg R, Gottlieb DJ, Hudgel D, Sateia M, Schwab R. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. Portable Monitoring Task Force of the Am Academy of Sleep Med. *J Clin Sleep Med* 2007;3:737–47