Real-Time Automatic Apneic Event Detection Using Nocturnal Pulse Oximetry

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Abstract*—*Objective: **Nocturnal pulse oximetry has been proposed as a simpler alternative to polysomnography in diagnosing sleep apnea. However, existing techniques are limited in terms of inability to provide time information on sleep apnea occurrence. This study aimed to propose a new strategy for near real-time automatic detection of apneic events and reliable estimation of apnea–hypopnea index using nocturnal pulse oximetry.** ***Methods:*** **Among 230 polysomnographic recordings with apnea–hypopnea index values ranging from 0 to 86.5 events/h, 138 (60%) and the remaining 92 recordings (40%) were categorized as training and test sets, respectively. By extracting the quantitative characteristics caused by the apneic event for the amount and duration of the change in blood oxygen saturation value, we established the criteria to determine the occurrence of apneic event. Regression modeling was used to estimate the apnea–hypopnea index from the apneic event detection results.** ***Results:*** **The minute-by-minute apneic segment detection exhibited an average accuracy of 91.0% and an average Cohen's kappa coefficient of 0.71. Between the apnea–hypopnea index estimations and reference values, the mean absolute error was 2.30 events/h. The average accuracy of our diagnosis of sleep apnea was 96.7% for apnea–hypopnea index cutoff values of $\geq$5, 10, 15, and 30 events/h.** ***Conclusion:*** **We developed an effective strategy to detect apneic events by using morphometric characteristics in the fluctuation of blood oxygen saturation values.** ***Significance:*** **Our study could be potentially useful in home-based multinight apneic event monitoring for purposes of therapeutic intervention and follow-up study on sleep apnea.**

***Index Terms*—Apnea-hypopnea index, blood oxygen saturation, overnight pulse oximetry, sleep apnea.**

I. Introduction

SLEEP apnea is a sleep-related breathing disorder characterized by repetitive reductions or cessations of respiration during sleep. Sleep apnea is known to have deleterious effect on daytime functioning by causing excessive somnolence, tiredness, irritability, and inattention [1]. Undiagnosed and untreated sleep apnea is a risk factor for hypertension [2], coronary artery disease [3], cardiac arrhythmias [4], stroke [5], diabetes mellitus [6], cognitive dysfunction [7], and depression [8]. The cost of treating the aforementioned various complications of sleep apnea imposes a huge economic burden on the health care system [9].

A nocturnal in-laboratory polysomnography (PSG) is the standard method for accurate diagnosis of sleep apnea. However, the requirements for PSG, including the well-controlled facility/equipment, trained personnel, and expensive fee, have caused the under-diagnosis of 80% of women and 90% of men with moderate to severe sleep apnea [10], [11]. Therefore, reliable sleep apnea diagnostic alternatives that use fewer physiological signals and provide a high level of usability should be developed.

Pulse oximetry is a noninvasive method to measure the percentage of hemoglobin saturated with oxygen. In sleep research, pulse oximetry is an essential measure for tracking the fluctuations in arterial blood oxygen saturation (SpO_2), which are signs of unstable ventilation in patients with sleep apnea. Overnight pulse oximetry has been proposed as a simpler alternative to PSG in diagnosing sleep apnea because it is readily available and relatively inexpensive, which could potentially meet the large demand for diagnostic testing [12]–[15]. Overnight pulse oximetry allows for easier long-term monitoring of patients with sleep apnea in the home environment, which is not the case with PSG. Moreover, the technological advances in pulse oximetry devices (pulse oximeters) with respect to their size and communication features (most of which support Bluetooth communications) facilitate their connection to mobile devices. Therefore, simple monitoring systems can be provided as cheaper alternatives to the current PSG in the diagnosis of sleep apnea.

Many quantitative indexes derived from the analysis of overnight SpO_2 recordings in the time [14], [16]–[24] and frequency domains [25]–[29] have been used to diagnose sleep apnea. Several nonlinear parameters extracted from overnight SpO_2 recordings have also been proposed to identify sleep apnea [30]–[33]. Multivariate models developed from time- and frequency-domain, and nonlinear analyses of overnight SpO_2 recordings have also demonstrated their effectiveness as sleep apnea diagnostic models [34]–[37]. However, all the aforemen-

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tioned methods are based on the delayed (offline) analysis of entire overnight SpO_2 recordings and thus could not provide time information on sleep apnea occurrence in real time.

Our study aimed to develop an SpO_2-based real-time automatic algorithm for detection of apneic events, defined to connote both apnea and hypopnea episodes. Based on the demonstrated causal relationship between an apneic event and oxygen desaturation, we hypothesized that identifying and characterizing SpO_2 change patterns related to apneic events could be effective to achieve our aim. Providing a reliable estimation of the apnea-hypopnea index (AHI), a commonly used measure for diagnosing sleep apnea, from the detected apneic events was also the aim of our study.

II. Materials and Methods

A. Subjects and Polysomnography

The institutional review board (IRB) of Seoul National University Hospital approved this retrospective study and waived the patient consent requirement (IRB No. 1412-091-634). We collected 455 polysomnographic recordings of subjects who underwent overnight PSG at the Center for Sleep and Chronobiology of Seoul National University Hospital between January 2013 and December 2013. All subjects were of Mongoloid origin.

According to the standard in-laboratory overnight PSG routine, the following physiological parameters were recorded using a bipolar Physiodata Amplifier System (Model 15LT; Grass Technologies, West Warwick, RI) and collected with a digital computer system equipped with a software developed in-house (Xomnia, 2005): electroencephalogram (EEG) at the F4-M1, C4-M1, and O2-M1 positions based on the International 10-20 System; bilateral electro-oculogram (EOG); electromyogram (EMG) at the chin and anterior tibialis muscles; electrocardiogram (ECG) at lead II; body posture using a tri-axis accelerometer; nasal pressure using a nasal cannula/pressure transducer; oronasal airflow using a thermistor; thoracic and abdominal volume changes using piezoelectric-type belts; snoring sound using a microphone; and SpO_2 using a pulse oximeter (MARS, type 2001; Respironics Novametrix, Murrysville, PA). The pulse oximeter was worn on the index finger of the non-dominant hand and provided digitalized SpO_2 values every 1 s with 1% resolution. Each SpO_2 value displayed on the pulse oximeter was obtained by calculating the average of the SpO_2 values acquired from the photoplethysmographic signals collected for 8 s after reducing the effect of artifacts, including motion, using the Motion Artifact Rejection System (MARS) algorithm [38], [39]. Because the MARS algorithm embedded in the pulse oximeter was effective to provide undistorted SpO_2 values by eliminating effects of missing samples, which are common in oximetry signals, no additional post-processing strategy was applied for artifact rejection.

In accordance with the 2012 American Academy of Sleep Medicine (AASM) manual [40], all of the PSG recordings were scored by certified sleep technologists and verified by sleep physicians. The rules used to determine apneas are as follows: drop in peak thermal sensor excursion by $\geq$90% of pre-event baseline; and the duration of the event lasts $\geq$10 s [40]. Hypopneas were determined in accordance with the following rules: deceases in nasal pressure signal excursions by $\geq$30% in airflow from pre-event baseline, which last $\geq$10 s; and $\geq$3% oxygen desaturation or event is associated with an arousal [40]. The numbers of apneas and hypopneas per hour of sleep were defined as the apnea index (AI) and hypopnea index (HI), respectively. The AHI reference value was determined by summing the AI and HI.

TABLE I
Summary of Subject Characteristics and Polysomnography Results

	Non-sleep apnea	Mild sleep apnea	Moderate sleep apnea	Severe sleep apnea
Sample size (male/female)	46 (30/16)	48 (32/16)	50 (33/17)	86 (56/30)
Age (years)	36.2 ± 12.8	43.7 ± 13.4	47.5 ± 13.2	49.4 ± 15.6
BMI (kg/m^2)	22.3 ± 2.7	24.8 ± 2.9	26.7 ± 3.0	27.5 ± 4.2
AHI (events/h)	1.4 ± 1.3	9.8 ± 2.7	22.3 ± 4.4	58.0 ± 16.2
TRT (min)	494.6 ± 42.5	492.8 ± 41.4	493.5 ± 39.7	494.1 ± 43.3
SE (%)	87.9 ± 9.6	86.3 ± 10.0	83.9 ± 12.3	82.6 ± 9.6

BMI, body mass index; AHI, apnea-hypopnea index; TRT, total recording time; SE, sleep efficiency.
Subjects were classified according to their AHI values into non-sleep apnea (AHI $<$ 5 events/h), mild sleep apnea (5 $\leq$ AHI $<$ 15 events/h), moderate sleep apnea (15 $\leq$ AHI $<$ 30 events/h), and severe sleep apnea (AHI $\geq$ 30 events/h) groups.
Data are presented as the mean ± SD.

In our study, the inclusion criterion was age of $\geq$18 years. The exclusion criteria were as follows: 1) the presence of periodic limb movements during sleep; 2) the presence of parasomnia (e.g., bruxism, sleep paralysis, sleep enuresis, confusional arousal, sleepwalking, nightmares, night terrors, nocturnal sleep-related eating disorder, and rapid eye movement sleep behavior disorder); 3) the presence of lung disease (e.g., chronic obstructive pulmonary disease, idiopathic pulmonary fibrosis, emphysema, bronchitis, pneumonia, and pulmonary edema); 4) the presence of chronic chest wall disease; 5) the presence of ischemic heart disease or heart failure; and 6) the presence of anemia. Among the 455 polysomnographic recordings, 230 recordings (51%) that met the inclusion and exclusion criteria were classified according to their AHI reference values into non-sleep apnea (AHI $<$ 5 events/h), mild sleep apnea (5 $\leq$ AHI $<$ 15 events/h), moderate sleep apnea (15 $\leq$ AHI $<$ 30 events/h), and severe sleep apnea (AHI $\geq$ 30 events/h) groups. In each group, 46 (non-sleep apnea), 48 (mild sleep apnea), 50 (moderate sleep apnea), and 86 (severe sleep apnea) subjects were included. The demographic and anthropometric characteristics and sleep parameters of the four groups are summarized in Table I.

B. Study Design

The primary output of this study was the apneic events detected in seconds. From the primary output, two subsequent outputs were produced. One output was a minute-by-minute sequence of annotations that identified the segment with an apneic event; the other output was an estimate of the AHI.

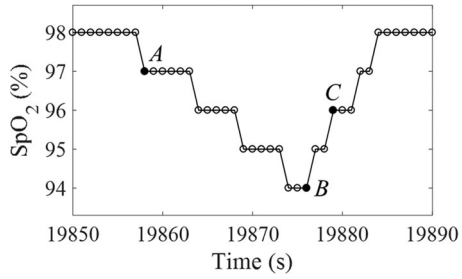

Fig. 1. Pattern of blood oxygen saturation (SpO_2) changes caused by an apneic event. *A* indicates the point at which a decrease in SpO_2 value of ≥1% and ≤3% is first achieved. *B* denotes the point at which SpO_2 value reaches a minimum of at least 3% below *A*. *C* is the point at which SpO_2 value returns to a level either 1% below *A* or 3% above *B*. Between *A* and *B*, all differences in successive SpO_2 values should be <1%. All differences in successive SpO_2 values should be >-1% between *B* and *C*. The length of time between *A* and *C* must be ≥10 s and ≤90 s.

After sorting the 230 recordings in ascending order of AHI reference value, odd-numbered recordings (115 recordings) were categorized as a training set. Among the remaining 115 recordings, sorted in ascending order of AHI reference value, the recordings in the order corresponding to multiples of 5 (23 recordings) were also included in the training set. Thus, the training set consisted of 138 recordings, and the remaining 92 recordings were categorized as a test set. The AHI reference values (mean ± SD) were $28.60 \pm 25.58\,\mathrm{events/h}$ and $29.27 \pm 26.02\,\mathrm{events/h}$ for the training and test sets, respectively. No bias existed for the AHI reference value between the training and test sets (independent samples *t*-test, $P = 0.85$).

The training set was used to extract morphometric characteristics in the fluctuation of SpO_2 values caused by an apneic event and then to determine numerical values that compose the rules for apneic event detection. As the direct estimation of the AHI was not feasible because of the difficulty of obtaining total sleep time using only SpO_2 values, a regression model that can provide AHI estimates was developed with the training set. The test set was used for independent validation of the performance on minute-by-minute apneic segment detection and AHI estimation.

1) Minute-by-Minute Apneic Segment Detection: Fig. 1 shows a typical pattern of change in SpO_2 value caused by an apneic event. Three points, denoted by *A*, *B*, and *C* in Fig. 1, were defined to determine the occurrence of an apneic event without false detection of artifactual fluctuation in the SpO_2 value. *A* is the point at which SpO_2 value decreases by $\geq 1\%$ and $\leq 3\%$; at *B*, SpO_2 value reaches a minimum of at least 3% below *A*; and at *C*, SpO_2 value returns to a level either 1% below *A* or 3% above *B*, whichever occurs sooner. Between *A* and *B*, all differences in successive SpO_2 values should be $<1\%$. All differences in successive SpO_2 values should be $>-1\%$ between *B* and *C*. The length of time between *A* and *C* must be ≥ 10 s and ≤ 90 s. Using these quantitative criteria, we developed a real-time automatic apneic event detection algorithm represented by the flow chart shown in Fig. 2. The "a," "b," and "c," and SpO_2(a), SpO_2(b), and SpO_2(c) determined to be related with the apneic event in Fig. 2 correspond to the *x*- and *y*-coordinate values of *A*, *B*, and *C* in Fig. 1, respectively.

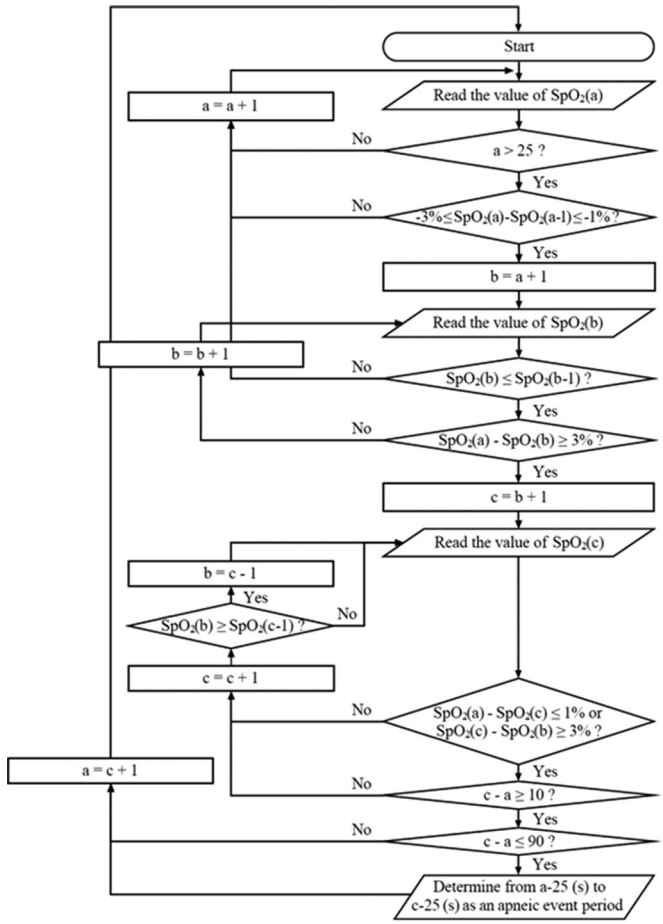

Fig. 2. Flow chart of the real-time automatic algorithm for apneic event period detection. $SpO_2(x)$ (*x* is "a," "b," or "c") indicates the SpO_2 value obtained at the time of "a," "b," or "c" (in seconds).

Considering the slow response of SpO_2 to the apneic event, a time advance of 25 s was applied in the final determination of the apneic event period. For this reason, the value of "a" in Fig. 2 should be >25.

Each minute was labeled with an annotation of "apneic segment" when at least one apneic event was in progress during the associated minute. Otherwise, the minute was labeled with an annotation of "non-apneic segment."

2) Apnea-Hypopnea Index Estimation: CurveExpert Professional software (v.2.0.4, www.curveexpert.net) was used for the regression analysis. The regression modeling was performed based on a Hill function with a significant probability level of 95% [41]. The dependent and explanatory variables were the AHI reference value and the ratio of the number of estimated apneic events to the total recording time (time difference between the start and end of the recording), respectively, in the regression analysis.

III. Results

A. Apneic Segment Detection Performance

The comparison between the sequence of annotations ("apneic segment" or "non-apneic segment") determined by using

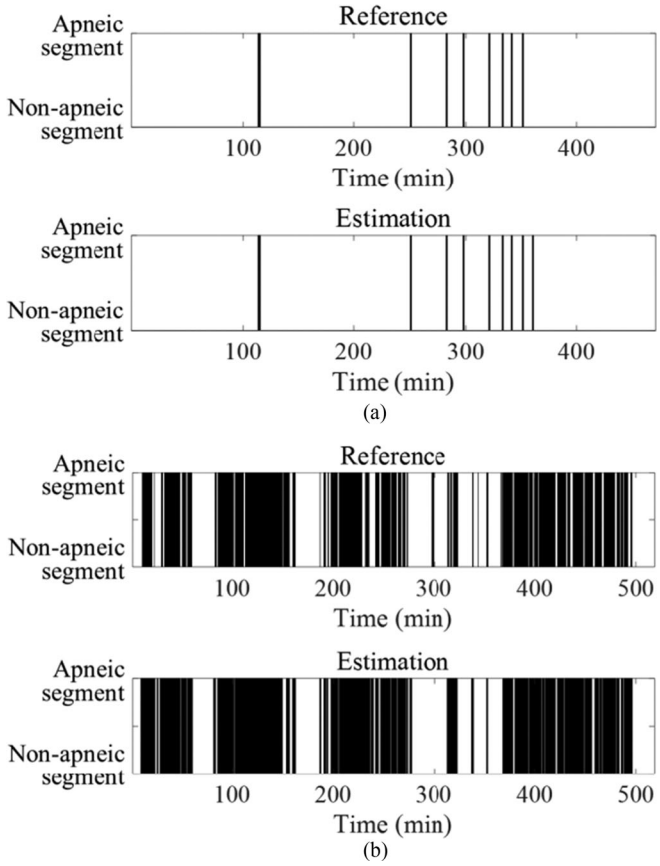

Fig. 3. Comparison between the reference apneic segments derived from the polysomnography result and the estimated apneic segments by using our method. (a) The best estimation result (Cohen's kappa coefficient = 0.95) observed in the non-sleep apnea group, and (b) that (Cohen's kappa coefficient = 0.80) reported in the severe sleep apnea group. Each bar in the plots indicates an apneic segment.

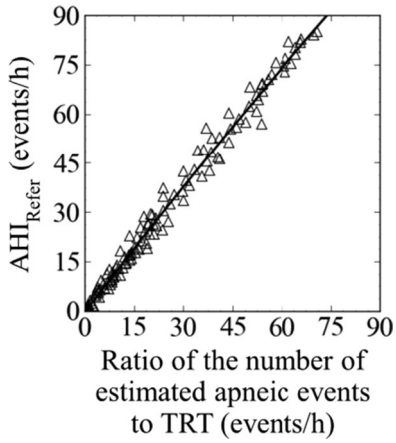

Fig. 4. Scatter plot showing the relationship between the apnea-hypopnea index reference values measured with polysomnography ($\mathrm{AHI}_{\mathrm{Refer}}$) and the ratios of the number of estimated apneic events to the total recording time (TRT) for the training set. The best-fitting curve (solid line) to the data of the training set (hollow triangles) was derived from the Hill function.

TABLE II
REGRESSION MODEL TO ESTIMATE APNEA-HYPOPNEA INDEX

	Variable	Regression function
y	$\mathrm{AHI}_{\mathrm{Estim}}$ (events/h)	$y = 0.07 + \frac{1254.06 \times x^{1.01}}{936.64^{1.01} + x^{1.01}}$
x	Ratio of the number of estimated apneic events to TRT (events/h)	

$\mathrm{AHI}_{\mathrm{Estim}}$, apnea-hypopnea index estimate; TRT, total recording time.

our method and that derived from the PSG result exhibited a sensitivity (mean ± SD) of 83.5 ± 11.8%, a specificity of 89.1 ± 10.5%, a positive predictive value (PPV) of 84.8 ± 13.0%, a negative predictive value (NPV) of 89.3 ± 12.5%, an accuracy of 91.3 ± 5.6%, and a Cohen's kappa coefficient of 0.71 ± 0.09 for the training set. For the test set, a sensitivity (mean ± SD) of 82.8 ± 11.7%, a specificity of 88.6 ± 10.5%, a PPV of 83.8 ± 12.4%, a NPV of 89.9 ± 11.0%, an accuracy of 91.0 ± 5.7%, and a Cohen's kappa coefficient of 0.71 ± 0.08 were obtained. With regard to the computation of the reliability parameters, true positives were the apneic segments correctly annotated as apneic segments by using our method, false positives were the non-apneic segments erroneously annotated as apneic segments, true negatives were the non-apneic segments correctly annotated as non-apneic segments, and false negatives were the apneic segments erroneously annotated as non-apneic segments.

Fig. 3 displays two illustrations that show the highest agreement between the sequence of annotations derived from the PSG result and that determined by using our method in the non-sleep apnea group (Fig. 3(a); AHI reference value = 1.02 events/h, Cohen's kappa coefficient = 0.95) and severe sleep apnea group (Fig. 3(b); AHI reference value = 46.87 events/h, Cohen's kappa coefficient = 0.80). Each bar in Fig. 3 indicates an apneic segment.

B. Apnea-Hypopnea Index Estimation Performance

Fig. 4 shows the regression model developed to estimate AHI from the ratio of the number of estimated apneic events to the total recording time. The best-fitting curve (Fig. 4, solid line) to the data of the training set (Fig. 4, hollow triangles) was derived from the Hill function depicted in Table II with a coefficient of determination (R^2) value of 0.98. Between the AHI estimates and reference values, an absolute error (mean ± SD) of 1.89 ± 1.88 events/h and an intra-class correlation coefficient (ICC) value of 0.99 ($P < 0.01$) were reported from the training set.

Fig. 5 displays the scatter plot of the AHI estimates, obtained by applying the developed regression model to the test set, and reference values. Between the estimates and reference values of AHI, an absolute error (mean ± SD) of 2.30 ± 2.19 events/h and an ICC value of 0.99 ($P < 0.01$) were exhibited.

Fig. 6 represents the Bland-Altman plot of the differences between the AHI estimates and reference values against the averages for the test set. For the differences between the estimates and reference values of AHI, the mean value (Fig. 6, solid line) was −0.09 events/h, and the 95% limits of agreement (Fig. 6, dashed lines) ranged from −6.33 to 6.15 events/h.

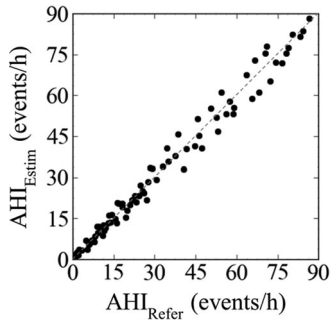

Fig. 5. Scatter plot showing the relationship between the apnea-hypopnea index estimates obtained by using our method ($\mathrm{AHI_{Estim}}$) and the apnea-hypopnea index reference values measured with polysomnography ($\mathrm{AHI_{Refer}}$) for the test set.

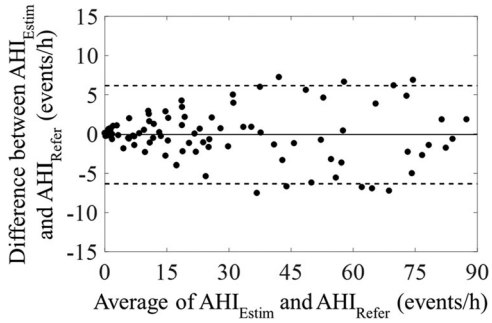

Fig. 6. Bland-Altman plot of the differences between the apnea-hypopnea index estimates obtained by using our method ($\mathrm{AHI_{Estim}}$) and the apnea-hypopnea index reference values measured with polysomnography ($\mathrm{AHI_{Refer}}$) against the averages for the test set. The solid line represents the mean of the differences between $\mathrm{AHI_{Estim}}$ and $\mathrm{AHI_{Refer}}$, and the dashed lines denote the 95% limits of agreement ($\pm 2 * \mathrm{SD}$ of the differences).

TABLE III
SLEEP APNEA DIAGNOSTIC PERFORMANCE FOR TEST SET

	AHI cutoff (events/h)			
	≥5	≥10	≥15	≥30
Sensitivity (%)	98.6	98.4	96.4	97.1
Specificity (%)	94.4	92.9	94.6	96.5
PPV (%)	98.6	96.9	96.4	94.4
NPV (%)	94.4	96.3	94.6	98.2
Accuracy (%)	97.8	96.7	95.7	96.7
Cohen's kappa coefficient	0.93	0.92	0.91	0.93
ROC-AUC	0.995	0.990	0.987	0.997

AHI, apnea-hypopnea index; PPV, positive predictive value; NPV, negative predictive value; ROC-AUC, area under the receiver-operating characteristics curve.
Cohen's kappa coefficients exceeding 0.90 indicate almost perfect agreement.

Table III summarizes the sleep apnea diagnostic performance assessed with the test set at AHI cutoff values of ≥5, 10, 15, and 30 events/h. For the four different AHI cutoff values, the mean sensitivity, specificity, PPV, NPV, accuracy, Cohen's kappa coefficient, and area under the receiver-operating characteristics curve were 97.6%, 94.6%, 96.6%, 95.9%, 96.7%, 0.92 corresponding to almost perfect agreement, and 0.992.

TABLE IV
APNEIC SEGMENT DETECTION PERFORMANCE IN LOW- AND HIGH-SLEEP EFFICIENCY GROUPS

	Low-SE group	High-SE group	*P*-value
Sensitivity (%)	82.9 ± 11.9	82.7 ± 11.6	0.94
Specificity (%)	87.2 ± 11.4	90.0 ± 9.5	0.20
PPV (%)	86.3 ± 10.5	81.4 ± 13.8	0.08
NPV (%)	88.6 ± 11.2	91.3 ± 10.8	0.24
Accuracy (%)	90.6 ± 5.5	91.4 ± 6.0	0.52
Cohen's kappa coefficient	0.72 ± 0.07	0.70 ± 0.09	0.39

SE, sleep efficiency; PPV, positive predictive value; NPV, negative predictive value.
Data are presented as the mean ± SD.
P-values were obtained by using the independent samples *t*-tests.

IV. DISCUSSION

New approaches for simplified identification of sleep apnea have commonly focused on analyzing a reduced set of data. The physiological data that received attention in our approach was SpO_2 because oxygen desaturation is a concomitant of incomplete breathing. We examined the changes in SpO_2 value caused by apneic events and then extracted the quantitative characteristics of the pattern. The empirically determined criteria for the amount of change in SpO_2 value and the duration of decreased SpO_2 values were effectively used for the real-time automatic detection of apneic events.

To confirm the independence of the performance of our method from sleep efficiency (SE), we classified the subjects in the test set into low- and high-SE groups. In the low- and high-SE groups, 46 subjects with SE <85% (SE [mean ± SD] = 76.0 ± 8.3%) and 46 subjects with SE ≥85% (SE [mean ± SD] = 92.0 ± 3.6%) were included, respectively. Table IV summarizes the values of the reliability parameters for the apneic segment detection, reported from the two groups, along with the *P*-values obtained by using the independent samples *t*-tests. The differences in sensitivity, specificity, PPV, NPV, accuracy, and Cohen's kappa coefficient were not significant between the two groups (all $P > 0.05$).

Table V shows the SpO_2-based sleep apnea diagnostic sensitivities and specificities reported from the studies published since 2006. For comparison, the sleep apnea identification results of our study are summarized in the last row of each AHI cutoff value in Table V. Based on the hypothesis that stronger SpO_2 dynamics would be observed in patients with more severe sleep apnea expressed as higher AHI values, previous studies in Table V focused on extracting various features to quantify the dynamics of SpO_2 values (e.g., oxygen desaturation indexes, second- and fourth-order moments, spectral features, approximate entropy, central tendency measure, Lempel-Ziv complexity, etc.). Alvarez *et al.* (2006) and Hornero *et al.* found the optimum threshold values of the extracted features to assess sleep apnea [30], [32]. Sanchez-Morillo *et al.* developed the hierarchical sleep apnea diagnosis model by applying classifiers to the datasets of the extracted features [37]. Each study conducted by Lin *et al.*, Hang *et al.*, and Alvarez *et al.* (2010) used the extracted features as explanatory variables in the regression analyses and then developed the AHI estimation model

TABLE V
COMPARISON OF SLEEP APNEA DIAGNOSTIC PERFORMANCE WITH EXISTING STUDIES

AHI cutoff (events/h)	Author	Year	Validation method	Se (%)	Sp (%)
≥5	Sanchez-Morillo *et al*. [37]	2014	LOOCV with 115 subjects	97.8	88.0
	Jung *et al*. (this study)	2017	Training and test with 138 and 92 subjects	98.6	94.4
≥10 or >10	Alvarez *et al*. [30]	2006	Training and test with the identical group including 187 subjects	90.1	82.9
	Hornero *et al*. [32]	2007	Training and test with 74 and 113 subjects	82.1	87.0
	Alvarez *et al*. [35]	2010	LOOCV with 148 subjects	92.0	85.4
	Jung *et al*. (this study)	2017	Training and test with 138 and 92 subjects	98.4	92.9
≥15 or >15	Lin *et al*. [22]	2009	Training and test with 257 and 279 subjects	85.3	85.3
	Hang *et al*. [29]	2012		78.9	92.0
	Jung *et al*. (this study)	2017	Training and test with 138 and 92 subjects	96.4	94.6
≥30 or >30	Lin *et al*. [22]	2009	Training and test with 257 and 279 subjects	88.2	89.6
	Hang *et al*. [29]	2012		73.3	99.3
	Sanchez-Morillo *et al*. [37]	2014	LOOCV with 115 subjects	86.7	95.3
	Jung *et al*. (this study)	2017	Training and test with 138 and 92 subjects	97.1	96.5

AHI, apnea-hypopnea index; Se, sensitivity; Sp, specificity; LOOCV, leave-one-out cross-validation.
The sensitivities and specificities are the results for the test sets.

[22], [29], [35]. In comparison with existing studies presented in Table V that achieved effective identification of sleep apnea using overnight SpO_2 recording, it is worthy of notice that our study, which equally used nocturnal pulse oximetry, provided time information on apneic events as well as diagnostic information on sleep apnea with acceptable performance.

Our study could be potentially useful in home-based multi-night apneic event monitoring for purposes of therapeutic intervention and follow-up study. Applying our method to a wrist-worn pulse oximeter with an embedded accelerometer and stimulator such as a vibrator and an alarm could be helpful to provide positional therapy for obstructive sleep apnea (OSA). Our method may also be used in positive airway pressure therapies to keep an OSA patient's airway open and prevent snoring. If used as a supplementary measure, our method could contribute to improving the predictive performance of existing alternative methods that use ECG or respiratory signal for real-time apneic event detection.

One limitation of our study is that the three types of sleep apnea were not classified. The morphometric characteristics in the fluctuation of SpO_2 values observed in obstructive, central, and mixed sleep apneas were not significantly different. Therefore, subcategorization of sleep apnea was difficult to achieve in this study. Another limitation is related to the incorporation of apnea and hypopnea episodes in an apneic event. Based on the greater clinical usefulness of AHI rather than AI and HI in diagnosing sleep apnea, this study focused on acquiring AHI. Developing an SpO_2-based algorithm with the ability to discriminate between apnea and hypopnea episodes is important to study in future work.

Further research is needed to achieve generalizability and verify the reproducibility of our method. Because we analyzed an internal dataset corresponding to in-laboratory recordings, further validation of our method with external datasets, including at-home multi-night recordings, is required.

V. CONCLUSION

The aim of this study was to propose a new strategy for near real-time automatic detection of apneic events and reliable estimation of AHI using nocturnal pulse oximetry. Based on the morphometric characteristics in the fluctuation of blood oxygen saturation values, we established the effective criteria to determine the occurrence of apneic events.

Although many studies have suggested the applicability of nocturnal pulse oximetry for sleep apnea, our study is an innovation in that it provided not only diagnostic information on sleep apnea but also time information on apneic events, which is not available with the approaches used in previous studies.

Our study has the potential to be helpful in therapeutic intervention for OSA by facilitating automatic home-based multi-night apneic event monitoring. Long-term monitoring of AHI variability in out-of-sleep laboratory environments, which is useful for follow-up study on sleep apnea, could be potentially enabled by utilizing our study.

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