

Detection of Sleep Disordered Breathing and Its Central/Obstructive Character Using Nasal Cannula and Finger Pulse Oximeter

Dirk Sommermeyer, Ph.D.^{1,2,3}; Ding Zou, M.D., Ph.D.¹; Ludger Grote, M.D., Ph.D.¹; Jan Hedner, M.D., Ph.D.¹

¹Center for Sleep and Wake Disorders, Institute of Medicine, University of Gothenburg, Sweden; ²Institute for Monitoring, Diagnosis and Assistance (IMDA), SRH University of Applied Science Heidelberg, Germany; ³Institute of Biomedical Engineering, Karlsruhe Institute of Technology (KIT), Karlsruhe, Germany

Study Objective: To assess the accuracy of novel algorithms using an oximeter-based finger plethysmographic signal in combination with a nasal cannula for the detection and differentiation of central and obstructive apneas. The validity of single pulse oximetry to detect respiratory disturbance events was also studied.

Methods: Patients recruited from four sleep laboratories underwent an ambulatory overnight cardiorespiratory polygraphy recording. The nasal flow and photoplethysmographic signals of the recording were analyzed by automated algorithms. The apnea hypopnea index (AHI_{auto}) was calculated using both signals, and a respiratory disturbance index (RDI_{auto}) was calculated from photoplethysmography alone. Apnea events were classified into obstructive and central types using the oximeter derived pulse wave signal and compared with manual scoring.

Results: Sixty-six subjects (42 males, age 54 ± 14 yrs, body mass index 28.5 ± 5.9 kg/m²) were included in the analysis. AHI_{manual} (19.4 ± 18.5 events/h) correlated highly significantly with AHI_{auto} (19.9 ± 16.5 events/h) and RDI_{auto} (20.4 ± 17.2 events/h); the correlation coefficients were r = 0.94 and 0.95,

respectively (p < 0.001) with a mean difference of -0.5 ± 6.6 and -1.0 ± 6.1 events/h. The automatic analysis of AHI_{auto} and RDI_{auto} detected sleep apnea (cutoff AHI_{manual} ≥ 15 events/h) with a sensitivity/specificity of 0.90/0.97 and 0.86/0.94, respectively. The automated obstructive/central apnea indices correlated closely with manually scoring (r = 0.87 and 0.95, p < 0.001) with mean difference of -4.3 ± 7.9 and 0.3 ± 1.5 events/h, respectively.

Conclusions: Automatic analysis based on routine pulse oximetry alone may be used to detect sleep disordered breathing with accuracy. In addition, the combination of photoplethysmographic signals with a nasal flow signal provides an accurate distinction between obstructive and central apneic events during sleep.

Keywords: Central sleep apnea, finger photoplethysmography, home sleep test, obstructive sleep apnea, sleep disordered breathing

Citation: Sommermeyer D; Zou D; Grote L; Hedner J. Detection of sleep disordered breathing and its central/obstructive character using nasal cannula and finger pulse oximeter. *J Clin Sleep Med* 2012;8(5):527-533.

Sleep disordered breathing (SDB) is highly prevalent in the general population. It is estimated that 20% of the general population has at least mild obstructive sleep apnea (OSA) and moderate-to-severe OSA occurs in 1 of 15 adults.¹ Central sleep apnea (CSA) is less common but has been reported in 40% of patients with chronic heart failure.² The gold standard to monitor and diagnose SDB is polysomnography (PSG).³ However, PSG is a costly, labor-intensive, and technically demanding procedure, which in many healthcare systems limits the accessibility of SDB diagnosis and treatment. Hence, a large number of portable monitoring devices including simplified cardiorespiratory polygraphy and limited-channel recording devices have been introduced.⁴

The limited-channel device in the current context uses a continuous single or dual bio-parameter recording, typically oxygen saturation and/or nasal airflow.⁵ Despite the advantages in terms of applicability and cost-effectiveness, the usefulness of such a device for SDB diagnosis has generally been limited by insufficient accuracy.⁶ For instance, depending on the cutoff, the sensitivity and specificity of a pulse oximeter to detect OSA can range from 31% to 98% and 41% to 100%, respectively.⁷ A

BRIEF SUMMARY

Current Knowledge/Study Rationale: The usefulness of limited channel devices for sleep disordered breathing (SDB) diagnosis has generally been restricted by limited sensitivity and specificity. This study describes a novel automated algorithm that uses a combination of nasal flow and several features of photoplethysmographic pulse wave signals to detect and differentiate obstructive and central apneas.

Study Impact: It was demonstrated that the combination of photoplethysmographic signals and a nasal flow signal provides an accurate distinction between obstructive and central apneic events during sleep. Information from a finger pulse oximeter alone may be used for an accurate diagnosis of sleep disordered breathing.

high sensitivity but low specificity was reported with a single-channel device that measures airflow through a nasal cannula using an apnea/hypopnea index (AHI) ≥ 5 as OSA cutoff.⁸⁻¹² Moreover, without respiratory effort recording, this device does not discriminate obstructive from central events.⁸ Several approaches have been applied to improve the diagnostic accuracy of SDB using single oximetry or a combined Holter

oximeter.^{13,14} However, distinction between obstructive and central events using oximetry alone is an issue that remains to be solved, especially in patients with chronic heart failure.¹⁵

Intrathoracic pressure changes during spontaneous breathing are mirrored in the peripheral pulse wave.¹⁶ Whether this feature can be used to distinguish obstructive and central apnea events has not been tested. We have developed a novel finger photoplethysmography pulse oximeter sensor for the assessment of cardiovascular risk in patients with suspected OSA.¹⁷ Several parameters including oxygen saturation, pulse rate and a finger pulsatile wave signal can be derived from the oximeter sensor. In the current study, we aimed to validate an automatic algorithm using signals derived from the oximeter probe and nasal cannula to detect and differentiate obstructive and central apnea. In addition, the accuracy of the oximeter sensor signal for quantification of SDB was evaluated alone or in combination with a nasal airflow signal.

METHODS

Study Subjects

Seventy-six subjects with suspected SDB were recruited from the sleep laboratory at Sahlgrenska University Hospital, Gothenburg, Sweden (n = 46) and 3 German sleep centers in Ulm, Berlin, and Nuernberg (n = 30). Subjects were informed regarding their participation in the study.

Sleep Study

All patients underwent an ambulatory overnight cardiorespiratory polygraphy recording (SOMNOcheck2, WEINMANN, Germany) including nasal airflow, thoracic and abdominal respiratory effort, heart rate, oxygen saturation (SpO₂) and body position. Patients received oral/written instructions for proper application during the sleep clinic visit. The device was self-applied by the patients at home before bedtime.

Sleep related respiratory events were manually scored by sleep technicians who were blind to the study. In brief, an apnea was scored as $\geq 90\%$ flow reduction compare to baseline ≥ 10 seconds. An obstructive apnea was classified by continued/increased inspiratory effort during the event and a central apnea was characterized by absent inspiratory effort during the event. A hypopnea was defined by $\geq 50\%$ nasal flow reduction compared to baseline for ≥ 10 sec together with $\geq 3\%$ oxygen desaturation. AHI_{manual} was calculated as the total number of apnea/hypopnea events divided by the recording time.

Automatic Algorithm Description

Signal Handling

A subset of the polygraphic channels (pulse oximeter and optional nasal cannula) was applied for autonomic analyses. The pulse wave signal was extracted from the oximeter recording. Pulse wave amplitude (PWA) and pulse rate were calculated from the pulse wave signal.

A minimum ≥ 3 -h artifact-free analysis periods of the flow signal were considered acceptable for computation of validity data in the study. In general, a signal decomposition algorithm based on a dictionary of time-frequency atoms (matching pur-

suit method)¹⁸ was modified in order to analyze specific patterns in the oximeter signals (see appendix for further detail of method). Thus, well-defined template functions were correlated through the original signals and the template functions with the highest correlation coefficient were saved for further analysis. The forms of these template functions were predefined by designed mathematical functions which reflect the typical patterns of interest (e.g., desaturations). These functions were then fitted to the particular, real signal pattern by varying function parameters to generate the best fit in terms of amplitude and frequency characteristics. By doing this, chronological coherences as well as morphological parameters of the used signals were considered in the calculation process. Separate algorithms were applied to automatically quantify respiratory disturbance index (RDI_{auto}), AHI_{auto}, obstructive apnea index (OAI), and central apnea index (CAI).

Autonomic Arousal Detection

An autonomic arousal, which indicates an activation of the autonomic nervous system, was used to confirm the sleep disrupting character of mild respiratory events like hypopneas. It was defined as: (1) pulse rate increase $\geq 20\%$ compared to baseline; or (2) PWA attenuation $\geq 40\%$ compared to baseline; or (3) PWA attenuation $\geq 35\%$ with pulse rate increase $\geq 15\%$ compared to baseline.

Respiratory Event Detection

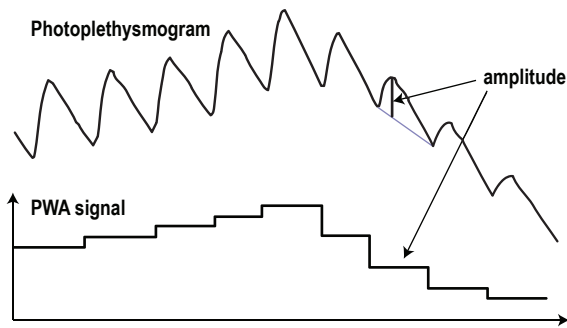
RDI_{auto} was calculated based on oximeter signal alone. SpO₂, pulse rate, and PWA were used to classify respiratory disturbance events. The definition of an automatically scored respiratory disturbance event was: (1) SpO₂ drop $\geq 4\%$ or (2) SpO₂ drop $\geq 3\%$ with an autonomic arousal. RDI_{auto} was calculated as the number of automatically scored respiratory disturbance events divided by the recording time.

AHI_{auto} was automatically calculated using nasal flow and oximeter signals. The definition of an automatically scored apnea was: (1) $\geq 90\%$ drop in the flow amplitude compare to baseline; (2) duration of the event ≥ 10 sec; (3) $> 90\%$ of the event's duration met the amplitude reduction criteria for apnea. The definition of an automatically scored hypopnea was: (1) $\geq 50\%$ drop in the flow amplitude compared to baseline together with 3% oxygen desaturation or an autonomic arousal; (2) duration of the event ≥ 10 sec; (3) more than 90% of the event's duration met the amplitude reduction criteria for hypopnea, which conforms to the alternative criteria of the AASM scoring manual.¹⁹ AHI_{auto} was calculated as the number of automatically scored apnea/hypopnea events divided by the recording time.

Obstructive/Central Apnea Differentiation

Identification of apneas as well as determination of their duration was based on the assessment of the inspiratory and expiratory phases of the nasal flow signal. The type of the apnea (obstructive vs. central) was determined by analysis of the pulse waveform. In detail, respiratory effort was derived by analyzing fluctuations of the PWA signal caused by intrathoracic pressure changes during spontaneous breathing cycles. The PWA signal was derived from the photoplethysmographic pulse wave signal by computing and plotting subsequently the amplitude value of each pulse wave for the duration of each single pulse wave

Figure 1—Visualization of the computation of the PWA signal



Shown is the raw signal (top) and the derived amplitude value of each pulse wave (bottom).

(**Figure 1**). In order to detect the presence or absence of respiratory effort, baseline drifts of the PWA signal were eliminated as a first analysis step. Next, the signal power in the typical frequency band of breathing (0.15-0.4 Hz) was determined during the apnea phase and compared with the signal power of the same frequency range 15 sec before the apnea event (**Figure 2**). An *effort ratio* index was defined as:

$$\text{effort_ratio} = \frac{\text{signal_power_pwa_periodic}_{\text{During Apnea}}}{\text{signal_power_pwa_periodic}_{\text{Before Apnea}}}$$

Since the intrathoracic pressure changes may increase during obstructive events, effort ratios > 1.0 are possible. An effort ratio < 0.56 was required for classification of a central event.

Post Hoc Analysis On Flow Signal Quality

In order to evaluate the influence of signal quality to the algorithms, flow data quality was rated according to the proportion of recording time with an offset signal, mouth leak, or reduced signal amplitude (positioning of the sensor). A 3-level scale was used with cutoff of more than 80%, 60% to 80%, and < 60% of artifact-free recording time.

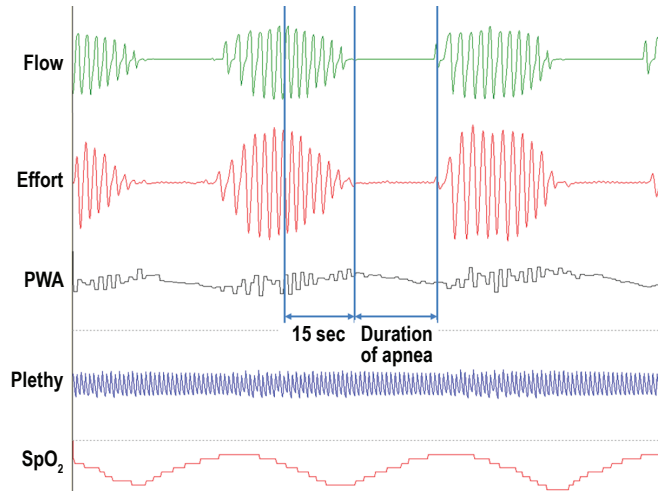
Statistical Analysis

Pearson correlation analysis was used to assess the association between $\text{AHI}_{\text{manual}}$ and $\text{AHI}_{\text{auto}}/\text{RDI}_{\text{auto}}$. The intraclass correlation coefficient (ICC) was used to assess the overall agreement between $\text{AHI}_{\text{manual}}$ and $\text{AHI}_{\text{auto}}/\text{RDI}_{\text{auto}}$. Differences between the methods were analyzed using the Bland-Altman plot. Receiver operator characteristic (ROC) curves were applied to assess the diagnostic accuracy of the algorithm using different AHI cutoff points. Sensitivity and specificity, positive predictive value (PPV), negative predictive value (NPV), as well as positive and negative likelihood ratio were calculated. The data were presented as mean and standard deviation. A *p*-value of 0.05 or less was considered statistically significant.

RESULTS

Ten of 76 subjects were excluded due to poor data quality (7 with poor flow or effort signals, 3 with insufficient recording

Figure 2—Illustration of the principle of the algorithm for obstructive/central apnea distinction



The respiratory induced component of the high pass filtered PWA signal during the apnea event is considered in relation to the accordant signal component 15-sec window before the apnea event.

Table 1—Patient characteristics of the study cohort

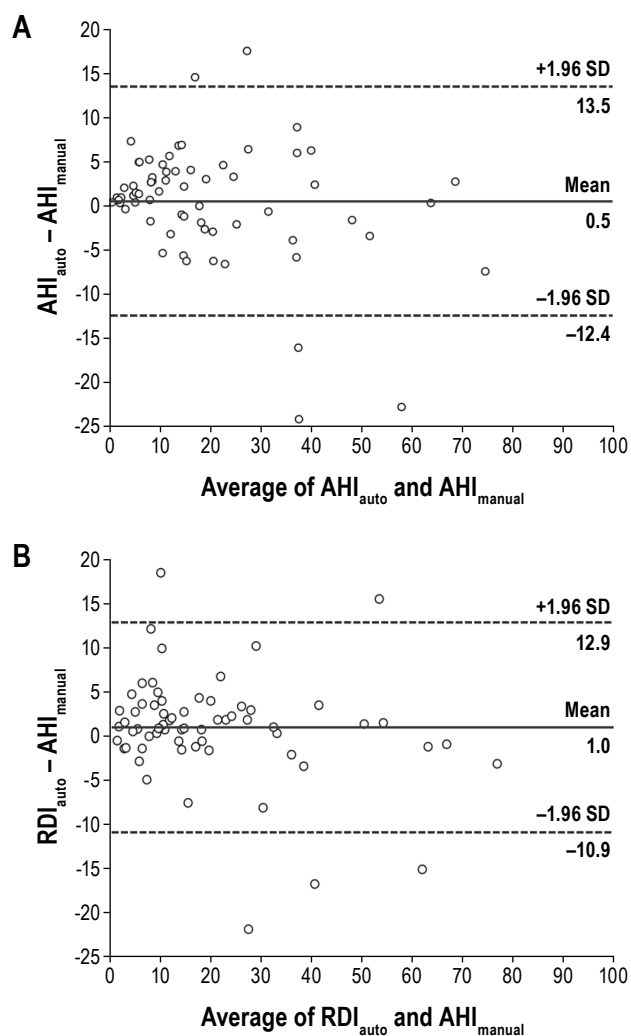
	Mean (SD)
Age (yrs)	54 (14)
Body mass index (kg/m ²)	28.5 (5.9)
Heart rate (bpm)	68.2 (10.3)
Systolic BP (mm Hg)	133.6 (25.3)
Diastolic BP (mm Hg)	78.2 (12.1)
Apnea/hypopnea index (events/h)	19.3 (18.5)
Apnea index (events/h)	10.4 (15.4)
4% oxygen desaturation index (events/h)	19.3 (17.2)
ESS	10 (5)
	Number (percentage)
Subjects	66
Males	42 (63%)
Hypertension	30 (46%)
Post myocardial infarction	7 (11%)
Congestive heart failure	5 (8%)
Stroke	3 (5%)
Diabetes	9 (14%)
COPD	4 (6%)

time due to loss of finger sensor signal). Sixty-six subjects (42 males, age 54 ± 14 years, body mass index 28.5 ± 5.9 kg/m², recording time 6.9 ± 1.2 h) were included in the final analysis. The most frequent comorbidities included hypertension (46%) and diabetes (14%). See **Table 1** for patient characteristics.

Accuracy of the Automated AHI/RDI Scoring Algorithm

The recordings were manually scored by 2 experienced sleep technicians according to the standard criteria. The AHI_{auto} vs. $\text{AHI}_{\text{manual}}$ difference did not systematically differ between the 2 scorers in a post hoc analysis (-1.1 ± 5.7 events/h vs. 0.2 ± 8.1 events/h, *p* = 0.47).

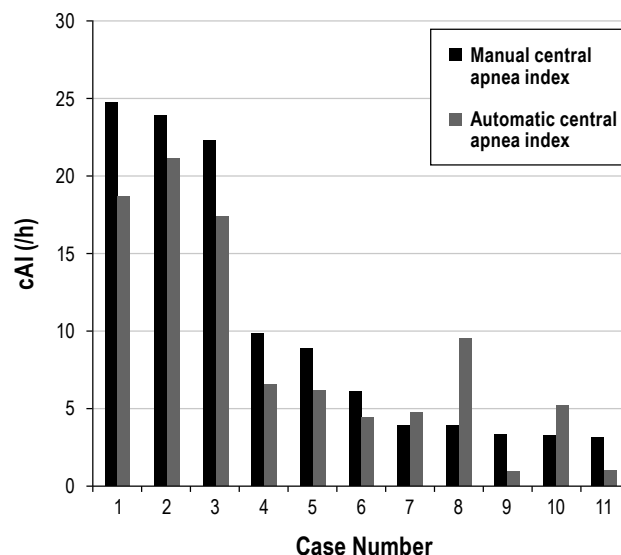
Figure 3—Bland-Altman plots



(A) AHI_{manual} and AHI_{auto} comparison; (B) AHI_{manual} and RDI_{auto} comparison.

In total, 8,804 apnea/hypopnea events (4,715 apneas) were manually scored, and 9,055 apnea/hypopnea events were detected by the autonomic algorithm. Among 2,260 hypopnea events automatically detected, 74% were based on 3% oxygen desaturation criteria and 26% were based on autonomic arousal criteria (approximately 9 events per subject, generating an index of 1.4/h). There was a very good agreement between AHI_{manual} and AHI_{auto} ($r = 0.94$, ICC 0.93, $p < 0.001$). The mean difference between the methods was -0.5 ± 6.6 events/h (Figure 3A). The correlation between the AHI_{manual} and the oximeter based RDI_{auto} was $r = 0.95$ ($p < 0.001$). The Bland-Altman analysis showed a mean difference between the methods of -1.0 ± 6.1 events/h (Figure 3B); the ICC was 0.94. The variability of the AHI/RDI differences suggested an underestimation of the automated algorithm in some patients with higher AHI_{manual} . Flow signal quality analysis showed that 36 of the 66 recordings contained $> 80\%$ artifact-free time. An additional 20 recordings were in the 60% to 80% range. Finally, 10 recordings showed $< 60\%$ of recording time without artifacts. The correlation between AHI_{manual} and AHI_{auto} in the 3 categories was 0.98, 0.90, and

Figure 4—Case-by-case comparison of subjects with central apnea index $\geq 3/h$



Subject 8 has mixed apneas.

0.88, respectively, indicating that flow signal quality strongly influenced the agreement between the methods.

Accuracy of the Automated Obstructive/Central Apnea Scoring Algorithm

The analysis of the algorithm for differentiation of obstructive ($n = 3,673$) and central ($n = 1,042$) apneas was made on the complete data set irrespective of the flow signal quality. Mixed apnea in the manual scoring was classified as obstructive apnea in the comparison. The correlation coefficients between manually and automatically scored CAI and OAI were 0.95 and 0.87, respectively. The mean difference between the methods for central apnea and obstructive apnea detection was 0.3 ± 1.5 and -4.3 ± 7.9 events/h, respectively. BMI did not systematically influence the accuracy of the detection algorithm, although only a limited number of subjects ($n = 6$) had $BMI > 35 \text{ kg/m}^2$ (data not shown). Some of the obstructive apnea events were scored as hypopnea in the algorithm. The difference between manually and automatically scored hypopnea indices was 4.1 ± 7.2 events/h. A case-by-case comparison in subjects with central apnea index ≥ 3 events/h is shown in Figure 4.

Diagnostic Accuracy

Different cutoff values were used to validate the diagnostic capacity of the AHI_{auto} . The sensitivity, specificity, positive and negative predictive values (PPV, NPV), positive and negative likelihood ratios (+LR, -LR), and the area under the ROC curve value for identification of sleep apnea are shown in Table 2.

The AHI_{auto} , based on photoplethysmography and flow signals, showed a sensitivity of 0.90 and a specificity of 0.86 when the AHI cutoff of 15 was used. The area under the ROC curve was 0.96. When the assessment was purely based on the pulse oximetry derived RDI, the sensitivity and specificity were 0.97 and 0.94, respectively, with the area under the ROC curve of 0.98 (Figure 5).

Table 2

AHI	Sensitivity	Specificity	PPV	NPV	+LR	-LR	AUC
≥ 5/h	1.00	0.60	0.82	1.00	2.50	0.00	0.99
≥ 10/h	0.97	0.75	0.84	0.96	3.88	0.04	0.95
≥ 15/h	0.90	0.86	0.84	0.91	6.43	0.12	0.96
≥ 20/h	0.82	0.93	0.86	0.91	11.71	0.19	0.97
≥ 30/h	0.87	0.96	0.87	0.96	21.75	0.14	0.99

Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (+LR), negative likelihood ratio (-LR), and area under the ROC curve (AUC) of AHI_{auto} using different cutoff values of AHI_{manual}.

When using CAI_{manual} ≥ 5 events/h as the diagnostic cutoff for patient with a component of CSA (n = 6), the automated algorithm detected CSA patients with sensitivity, specificity, PPV, NPV, +LR, -LR, and area under the ROC curve of 0.83, 0.98, 0.83, 0.83, 25, 0.17, and 0.98, respectively.

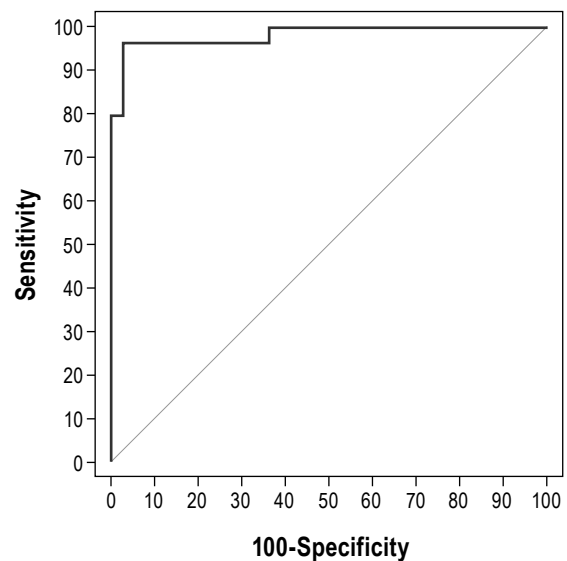
DISCUSSION

The current study demonstrated for the first time that a novel computer algorithm based on a combination of nasal air flow and photoplethysmographic pulse wave signals may be used to accurately detect and differentiate obstructive and central sleep apnea events. In addition, an algorithm based on the pulse oximeter signal alone provided a good quantitative estimate of SDB severity.

OSA, the most common type of SDB, has been associated with cardiovascular, metabolic, and pulmonary comorbidities.²⁰ In the light of a growing need for diagnosis and treatment of SDB patients, unattended home sleep studies using portable monitoring devices have been widely applied in the clinical setting. For instance, ambulatory cardiorespiratory polygraphy have been used for CSA/Cheyne-Stokes respiration detection in heart failure patients.²¹ According to the AASM, a minimum of heart rate, oxygen saturation, and respiratory analysis (e.g., by airflow or peripheral arterial tone) is required for out-of-center sleep testing.²² Finger PWA derived from peripheral arterial tone has been used for respiratory disturbance event detection in combination with a pulse oximeter at home.²³ In this study, we were interested in the combination of airflow and signals derived from a common pulse oximeter sensor, including PWA. Recently, we were able to derive a PWA signal from pulse oximetry and evaluated its relevance for cardiovascular risk assessment.¹⁷ Thus, we wanted to further test the validity of such a signal for SDB diagnostics in the ambulatory setting. The diagnostic capacity was tested in two separate conditions: One included application of a nasal cannula and the other was based on signals derived from the pulse oximeter sensor alone.

It is known that respiratory movements cause variation in the peripheral circulation. Changes in intrathoracic pressure modulate central venous pressure and alter venous return to the heart which can be detected by photoplethysmographic sensor attached to skin.¹⁶ The respiratory induced frequency component of the photoplethysmographic signal has been closely associated with respiratory volume.²⁴ Although the underlying physiological mechanisms are not fully understood, intrathoracic pressure changes and autonomic nervous activity oscilla-

Figure 5—Receiver operator characteristic curve of RDI_{auto} and AHI_{manual} (cut-off AHI_{manual} ≥ 15), AUC = 0.98



tions seem to play a role in this variation.^{24,25} Central apnea is characterized by an absence of inspiratory effort during airflow cessation which is associated with lower signal power in the 0.15-0.40 Hz frequency band of the PWA signal. This was indeed the case in our finger PWA signal analysis which provided a possibility to distinguish between obstructive and central apnea. We also found that in subjects with dominant central apneas, the algorithm tended to slightly underestimate the number of central events. In cases with mixed apneas, which were classified as “obstructive apnea” in the analysis, there was a trend towards overestimation of the central events, and the degree depended on the length of the central apnea component.

The pathogenesis of CSA and OSA is different, but there may be similar clinical complications in terms of cardiovascular morbidity and mortality. In a large community-based cohort, increments of obstructive and central apnea indices were both associated with increased incidence of cardiovascular events.²⁶ In patients with congestive heart failure, severe SDB provided a 2-fold increase risk for death compared with those without severe SDB.²⁷ In the post hoc analysis comparing severe and mild SDB groups, mortality was only significantly higher in the group with predominant CSA but not in those with predominant OSA. Hence, the distinction between different types of respiratory events may have important implications for the

classification and treatment of patients with SDB. According to the AASM guidelines, the preferred technique for detection of respiratory effort is either esophageal manometry, or calibrated or uncalibrated inductance plethysmography. However, both methods provide an obvious limitation in terms of applicability during unattended recordings in the home environment. To the best of our knowledge, the current available diagnostic instruments using nasal flow and pulse oximeter can detect apnea/hypopnea events but are unable to differentiate obstructive versus central apnea event. In the current study we have shown that our automatic algorithm can differentiate central/obstructive events with high accuracy. Hence, the algorithm may provide an important additional benefit in screening programs, particularly in populations with high likelihood of central events (e.g., patients with heart failure, stroke, or opioid intake).

Using $AHI \geq 15$ as the diagnostic cutoff, oximeter combined with nasal cannula has been shown to detect sleep apnea with a high accuracy. However, when $AHI \geq 5$ was applied as the cutoff, high sensitivity but low specificity of the automatic analysis was found in the current study. This was not unexpected, as previous studies using similar signals and cutoff levels showed comparable results.⁸⁻¹¹ Hence, limited-channel devices may be sensitive to rule in SDB patients but seem to be less robust to rule out patients, especially when lower thresholds are applied. The diagnostic accuracy of these devices could be improved if recordings are manually reviewed. A study comparing automatic analysis to manual scoring from a limited channel device found an increase in specificity from 0.6 to 0.87 when a cutoff $AHI \geq 5$ was used.¹²

The quantification of RDI using information derived from oximeter yielded also a good agreement with the manually scored AHI. The diagnostic accuracy of using oximeter signal alone was found to be similar to nasal cannula/oximeter combination in this cohort. One explanation could be that there were few respiratory disturbance events based on 3% desaturation and autonomic arousal in the analyzed material (~2.5 events/h). On the other hand, finger pulse wave attenuations reflecting sympathetic nervous activity have been shown to associate with apnea/hypopnea events and to coincide with arousals.²⁸ A composite criterion including PWA attenuation, pulse rate increase, and oxygen desaturation has been shown to accurately detect respiratory disturbance events during sleep.²⁹ Although information from the oximeter alone did not assess the severity of respiratory events (apnea/hypopnea/flow limitation), it may serve as a standalone screening tool or alternative method in SDB diagnostics when the focus is directed towards the general assessment of SDB or when technical problems (e.g., mouth breathing, poor nasal airflow signal, patients intolerance of the nasal cannula) occur during the night.

Several study limitations need to be addressed. In order to reflect the clinical setting, unattended ambulatory recordings were applied in the study. However, we did not use PSG as the comparator which limited the capacity for scoring arousals and may, in some patients (delayed sleep onset or low sleep efficiency), lead to overestimation of the validity of the algorithm. Inter- and intra-score variability testing was not conducted prior to the analysis and event-by-event concordance was not performed. Nasal flow, but not oronasal thermistor, was used to classify an apnea event; this is not in accordance

with current AASM guidelines as remaining mouth flow may be left undetected.³⁰ The hypopnea event definition in the study was adapted from the alternate AASM guidelines, but automatic arousal was used rather than arousal determined in electroencephalographic (EEG) recording. Although the number of hypopnea events detected solely by the autonomic arousal criterion was low in the current study, we consider that these events may provide novel information on apnea/hypopnea related autonomic activation and it is important to maintain it in the algorithm. PWA attenuations and pulse rate accelerations were used to classify autonomic arousal events instead of changes in electroencephalography activity used for standard arousal classification. It has been shown that a drop of PWA is a sensitive marker for changes in cortical activity during sleep.³¹ Acoustically induced arousal from NREM sleep could induce biphasic changes of finger PWA fall and an increase of heart rate.³² Indeed, the reduced PWA alone or in combination with an increase in heart rate was found to be closely correlated with PSG-scored electroencephalography arousals.³³ In the current study, we did not use a separate nasal airflow and oximeter device for comparison with the polygraphic recording. Rather, we tested the algorithm using signals derived from the polygraphic recording itself. Hence, the reproducibility of such algorithms in a two-channel screening device for SDB recognition remains to be determined. Study subjects were selected among patients referred to the sleep laboratory. Although the cohort included some patients with predominantly central apneas, the study population was not ideally balanced in terms of proportion of central or obstructive sleep apnea. The applicability of the algorithms to identify high risk SDB patients in the general population needs also to be further studied. Finally, the classification of mixed apneas as “obstructive” apneas might not be accurate in all the cases.

CONCLUSIONS

There is an unmet need for simple diagnostic devices with documented accuracy for the differentiated classification of SDB. This study describes a novel automated algorithm that uses a combination of nasal flow and different features of the photoplethysmographic signals to detect and differentiate obstructive and central apneas. It was also demonstrated that information from the finger pulse oximeter alone could be used for more advanced SDB diagnostics. This finding has a potential implication in the era of shift from attended laboratory monitoring towards ambulatory home testing.³⁴

REFERENCES

1. 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.
2. Oldenburg O, Lamp B, Faber L, Teschler H, Horstkotte D, Topfer V. Sleep-disordered breathing in patients with symptomatic heart failure: a contemporary study of prevalence in and characteristics of 700 patients. *Eur J Heart Fail* 2007;9:251-7.
3. Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep* 2005;28:499-521.
4. Flemons WW, Littner MR, Rowley JA, et al. Home diagnosis of sleep apnea: a systematic review of the literature. An evidence review cosponsored by the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society. *Chest* 2003;124:1543-79.

5. Ferber R, Millman R, Coppola M, et al. Portable recording in the assessment of obstructive sleep apnea. ASDA standards of practice. *Sleep* 1994;17:378-92.
6. Executive summary on the systematic review and practice parameters for portable monitoring in the investigation of suspected sleep apnea in adults. *Am J Respir Crit Care Med* 2004;169:1160-3.
7. Netzer N, Eliasson AH, Netzer C, Kristo DA. Overnight pulse oximetry for sleep-disordered breathing in adults: a review. *Chest* 2001;120:625-33.
8. Erman MK, Stewart D, Einhorn D, Gordon N, Casal E. Validation of the ApneaLink for the screening of sleep apnea: a novel and simple single-channel recording device. *J Clin Sleep Med* 2007;3:387-92.
9. Chen H, Lowe AA, Bai Y, Hamilton P, Fleetham JA, Almeida FR. Evaluation of a portable recording device (ApneaLink) for case selection of obstructive sleep apnea. *Sleep Breath* 2009;13:213-9.
10. Ragette R, Wang Y, Weinreich G, Teschler H. Diagnostic performance of single airflow channel recording (ApneaLink) in home diagnosis of sleep apnea. *Sleep Breath* 2010;14:109-14.
11. Nigro CA, Serrano F, Aimaretti S, Gonzalez S, Codinardo C, Rhodius E. Utility of ApneaLink for the diagnosis of sleep apnea-hypopnea syndrome. *Medicina (B Aires)* 2010;70:53-9.
12. Nigro CA, Dibur E, Aimaretti S, Gonzalez S, Rhodius E. Comparison of the automatic analysis versus the manual scoring from ApneaLink device for the diagnosis of obstructive sleep apnoea syndrome. *Sleep Breath* 2011;15:679-86.
13. Alvarez D, Hornero R, Garcia M, del Campo F, 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.
14. Heneghan C, Chua CP, Garvey JF, et al. A portable automated assessment tool for sleep apnea using a combined Holter-oximeter. *Sleep* 2008;31:1432-9.
15. Series F, Kimoff RJ, Morrison D, et al. Prospective evaluation of nocturnal oximetry for detection of sleep-related breathing disturbances in patients with chronic heart failure. *Chest* 2005;127:1507-14.
16. Murray WB, Foster PA. The peripheral pulse wave: information overlooked. *J Clin Monit* 1996;12:365-77.
17. Grote L, Sommermeyer D, Zou D, Eder DN, Hedner J. Oximeter-based autonomic state indicator algorithm for cardiovascular risk assessment. *Chest* 2011;139:253-9.
18. Mallat S, Zhang Z. Matching Pursuit in time-frequency dictionary. *IEEE Trans Signal Process* 1993;41:3397-415.
19. Iber C, Ancoli-Israel S, Chesson AL, Quan SF. *The AASM manual for the scoring of sleep and associated events - rules, terminology and technical specifications*. Westchester, IL: American Academy of Sleep Medicine, 2007.
20. Hedner J, Grote L, Bonsignore M, et al. The European Sleep Apnoea Database (ESADA): report from 22 European sleep laboratories. *Eur Respir J* 2011;38:635-42.
21. Sinha AM, Skobel EC, Breithardt OA, et al. Cardiac resynchronization therapy improves central sleep apnea and Cheyne-Stokes respiration in patients with chronic heart failure. *J Am Coll Cardiol* 2004;44:68-71.
22. Standards for Accreditation of Out of Center Sleep Testing (OCST) in Adult Patients. In: American Academy of Sleep Medicine: <http://www.aasmnet.org/OCSTstandards.aspx>.
23. Zou D, Grote L, Peker Y, Lindblad U, Hedner J. Validation a portable monitoring device for sleep apnea diagnosis in a population based cohort using synchronized home polysomnography. *Sleep* 2006;29:367-74.
24. Johansson A, Oberg PA. Estimation of respiratory volumes from the photoplethysmographic signal. Part I: Experimental results. *Med Biol Eng Comput* 1999;37:42-7.
25. Nilsson L, Johansson A, Kalman S. Macrocirculation is not the sole determinant of respiratory induced variations in the reflection mode photoplethysmographic signal. *Physiol Meas* 2003;24:925-37.
26. Chami HA, Resnick HE, Quan SF, Gottlieb DJ. Association of incident cardiovascular disease with progression of sleep-disordered breathing. *Circulation* 2011;123:1280-6.
27. Jilek C, Krenn M, Sebah D, et al. Prognostic impact of sleep disordered breathing and its treatment in heart failure: an observational study. *Eur J Heart Fail* 2011;13:68-75.
28. Zou D, Grote L, Eder DN, Peker Y, Hedner J. Obstructive apneic events induce alpha-receptor mediated digital vasoconstriction. *Sleep* 2004;27:485-9.
29. Bar A, Pillar G, Dvir I, Sheffy J, Schnall RP, Lavie P. Evaluation of a portable device based on peripheral arterial tone for unattended home sleep studies. *Chest* 2003;123:695-703.
30. Collop NA, Anderson WM, Boehlecke B, et al. 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 American Academy of Sleep Medicine. *J Clin Sleep Med* 2007;3:737-47.
31. Delessert A, Espa F, Rossetti A, Lavigne G, Tafti M, Heinzer R. Pulse wave amplitude drops during sleep are reliable surrogate markers of changes in cortical activity. *Sleep* 2010;33:1687-92.
32. Catchside PG, Chiong SC, Mercer J, Saunders NA, McEvoy RD. Noninvasive cardiovascular markers of acoustically induced arousal from non-rapid-eye-movement sleep. *Sleep* 2002;25:797-804.
33. Pillar G, Bar A, Shlitner A, Schnall R, Sheffy J, Lavie P. Autonomic arousal index: an automated detection based on peripheral arterial tonometry. *Sleep* 2002;25:543-9.
34. McNicholas WT, Levy P. Portable monitoring in sleep apnoea: the way forward? *Eur Respir J* 2011;37:749-51.

ACKNOWLEDGMENTS

This work was supported by the German Ministry for Education and Science (BMBF), the Swedish Heart and Lung Foundation and the Royal Society of Arts and Science in Göteborg.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication July, 2011

Submitted in final revised form February, 2012

Accepted for publication March, 2012

Address correspondence to: Dirk Sommermeyer, Center for Sleep and Wake Disorders, University of Gothenburg, Box 421, SE 405 30, Gothenburg, Sweden; Tel: +46 31 3423741; Fax: +46 31 825207; E-mail: dirk.sommermeyer@lungall.gu.se

DISCLOSURE STATEMENT

This study was supported by a research grant from Weinmann GmbH. Dr. Sommermeyer was an employee of MCC which was under contract to Weinmann GmbH. Dr. Grote has served as a medical advisor of Weinmann and has participated in clinical studies sponsored by MSD, Philips Respironics, Weinmann and Mundipharma. Dr. Hedner has participated in clinical trials sponsored by MSD, Philips Respironics and Weinmann. The other author have indicated no financial conflicts of interest.