Respiratory Event Detection by a Positive Airway Pressure Device

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Study Objectives: Compare automatic event detection (AED) of respiratory events using a positive airway pressure (PAP) device with manual scoring of polysomnography (PSG) during PAP treatment of obstructive sleep apnea (OSA).

Design: Prospective PSGs of patients using a PAP device.

Setting: Six academic and private sleep disorders centers.

Patients: A total of 148 PSGs from 115 participants with OSA (apnea-hypopnea index [AHI] ≥ 15 events/hr) were analyzed.

Interventions: A signal generated by the PAP device identifying the AED of respiratory events based on airflow was recorded during PSG.

Measurements and Results: The PSGs were manually scored without visualization of the AED signal and scoring of a hypopnea required a ≥ 4% oxygen desaturation. The apnea index (AI), hypopnea index (HI), and AHI by manual score and PAP AED were compared. A customized computer program compared individual events by manual scoring and AED to determine the true positive, false positive, false negative, or true negative events and found a sensitivity of 0.58 and a specificity of 0.98. The AHI, AI, and HI by the two methods were highly correlated. Bland-Altman analysis showed better agreement for AI than HI. Using a manually scored AHI of ≥ 10 events/hr to denote inadequate treatment, an AED AHI ≥ 10 events/hr had a sensitivity of 0.58 and a specificity of 0.94.

Conclusions: An AHI < 10 events/hr by PAP AED is usually associated with good treatment efficacy. Differences between manually scored and AED events were primarily due to different criteria for hypopnea detection.

Keywords: CPAP, autoCPAP, APAP

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INTRODUCTION

Positive airway pressure (PAP) devices are the primary treatment for patients with obstructive sleep apnea (OSA). Continuous, autoadjusting, and bilevel PAP devices can measure airflow and pressure in the circuit to detect and store in memory the mask-on time and the occurrence of respiratory events. The algorithms for event detection used by these devices are based on measurements of airflow, vibration, and airflow profile flattening. Clinicians use the event detection information recorded by these PAP devices to assess treatment efficacy. Event detection is also used in autotitrating positive airway pressure devices (APAP) to regulate changes in delivered pressure over a set pressure range.[1-3]

The event detection downloads of the PAP devices report the same variables obtained on polysomnogram (PSG), including the apnea-hypopnea index (AHI), apnea index (AI), and hypopnea index (HI). However, PAP devices and PSG differ significantly in the methods used to generate these measures. PAP units use airflow to detect apneas and hypopneas. In contrast, the abnormal respiratory events on PSG are scored using not only airflow but also the arterial oxygen saturation level by pulse oximetry, respiratory effort, and, depending on the scoring criteria, electroencephalographic (EEG)/electromyographic (EMG) arousals.[4,5] PAP units estimate the AHI as the mean number of apneas and hypopneas per hour of device use,

whereas the AHI on PSG is calculated as the mean number of apneas and hypopneas per hour of sleep. Given the differences between how PAP units and PSG detect respiratory events and generate respiratory event indices, it is critically important that providers understand how these different measurements compare with each other.

A substantial amount of literature exists concerning the efficacy of commercially available APAP devices to reduce obstructive apneas and hypopneas on manually scored PSG to clinically acceptable levels.[1-3] However, relatively few studies have documented the accuracy of the event detection algorithms.[6-9] Two studies published more than 15 years ago tested PAP units that used automatic event detection (AED) algorithms that have since undergone extensive modification.[6,7] Two more recent studies using current AED algorithms compared the manually scored AHI on PSG with the AHI exported from an APAP device used during the PSG. Both studies report that the AHI information obtained from the devices was reasonably accurate.[8,9] One study did not distinguish apneas from hypopneas, and neither study performed an event-by-event or epoch-by-epoch comparison of the two methods of measurement.

The purpose of this study was to compare the AED algorithm used in a PAP device with manually scored events on PSG. The PAP device was modified to produce a square wave voltage output identifying when apneas, hypopneas, and snoring events were detected. Recording this event signal on the PSG performed with the patient using the PAP device allowed an event-by-event comparison between manually scored PSG events and AED events. In addition, the AHI, AI, and HI derived from the manually scored PSG were compared with the respective measures reported by the PAP device used during the PSG.
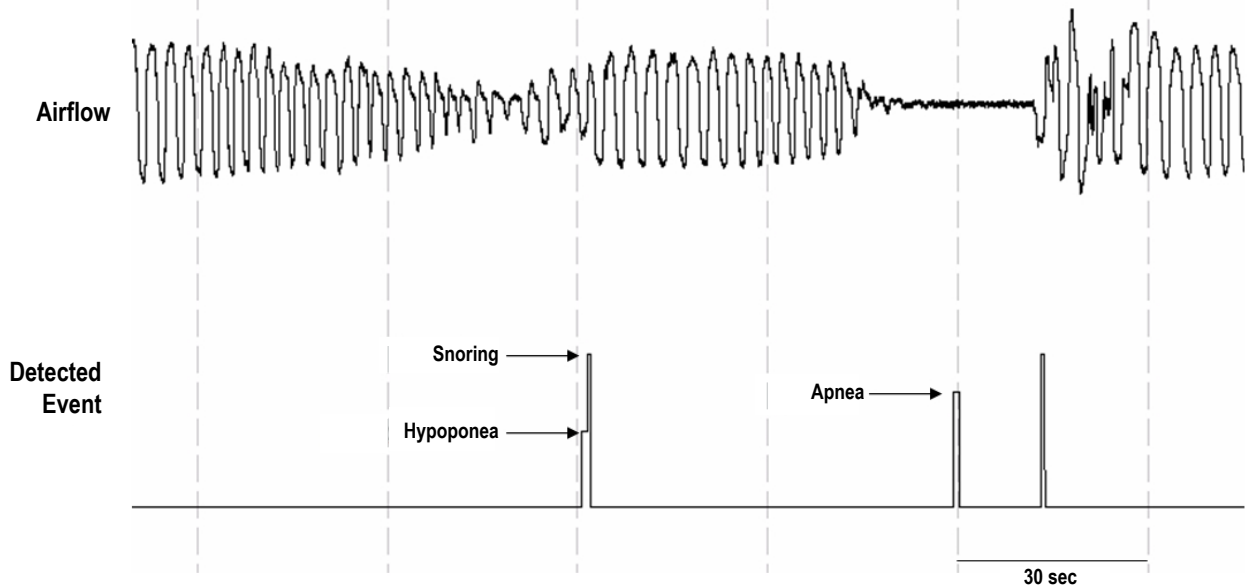
Figure 1—Airflow (PAP device flow signal) and automatic event detection signals from a PSG in a patient on subtherapeutic CPAP during nonrapid eye movement sleep. The signals for the snoring, hypopnea, and apnea events detected by the PAP unit were of different voltage and duration. Detected events (amplitude in volts, duration in sec): hypopnea (2 v, 1.5 sec), apnea (3 v, 1 sec), snoring (4 v, 0.5 sec).

METHODS

The PSGs used for this study were obtained from a multi-center, randomized, double-blind trial comparing three modes of positive pressure delivery.[10] This study will be referred to as the parent study. Inclusion criteria of the parent study included age 21-65 yrs, diagnosis of OSA (AHI ≥ 15 events/hr), adequate clinical CPAP titration PSG within 2 wk of enrollment, and agreement to try PAP treatment. Exclusion criteria included prior prescription for, or exposure to, PAP therapy within the previous year; surgery of the upper airway, nose, sinus, or middle ear within the previous 90 days; surgery at any time for the treatment of OSA, such as uvulopalatopharyngoplasty; presence of untreated sleep disorders not related to OSA (e.g., moderate to severe periodic limb movements, restless leg syndrome, or chronic insomnia), use of medications with hypnotic or sedative effects or regular use of nighttime sedatives or sleeping aids on 3 or more nights per wk, consumption of ethanol more than 4 nights per wk, an unstable medical condition, moderate to severe chronic obstructive pulmonary disease, and shift work.

Subjects enrolled in the parent study were randomized to one of three treatment arms: CPAP at the pressure determined by the titration PSG; APAP using a form of flexible positive pressure (A-Flex™), or APAP for 2 wks followed by CPAP treatment based on the results of the 2 wks of APAP treatment (pressure below which the patient spent 90% of the time). The same study device was used for all participants (REMstar Auto M-Series, Philips-Respironics, Murrysville PA) functioning in the CPAP, APAP, or AFlex™ modes[10]. The pressure range on the APAP units was set at 4 cm H_2O to 20 cm H_2O.

A PSG with standard signal montage[5] was performed on the assigned positive pressure treatment at study entry (n = 164) and after 6 months of treatment (n = 140). The PAP device used during the PSGs was modified to produce square wave outputs of different voltage and duration that identified when an apnea, hypopnea, or snoring event was detected (Figure 1). Delivered pressure, airflow, and the event detection signal from the PAP device were recorded during the PSGs.

Because the PSGs were performed with the participants on effective PAP settings, the resulting manually scored AHI values were low. To obtain a greater range of AHI values, 29 patients with OSA (AHI ≥ 15 events/hr) were recruited to undergo a PSG using the PAP device in CPAP mode set at a subtherapeutic pressure. These patients had undergone a previous manual CPAP titration PSG with a CPAP prescription pressure from 8 cm H_2O to 18 cm H_2O. Four patients participated in both the parent study and the sub-therapeutic study. The sub-therapeutic pressures selected were approximately 50% of the optimal pressure setting determined on the previous titration PSG. The protocols were approved by the local institutional review boards of the participating institutions. All participants signed an informed consent before participation.

Automatic Event Detection

The AED algorithm used the following criteria to identify respiratory events. An apnea was detected when there was an 80% or greater reduction in the airflow for 10 sec or longer in comparison with the average airflow over the previous 2 minutes. A hypopnea was detected when there was a device-estimated 40% reduction in airflow for ≥ 10 sec but < 60 sec compared with the average airflow over the previous 2 minutes. The hypopnea detection algorithm required the presence of two recovery breaths that nominally were at least 75% to 80% of the baseline airflow. The algorithm also looked for evidence of

flow limitation to detect hypopneas. The algorithm monitored the flow signal for changes in peak flow, and the shape of the inspiratory airflow signal that would be associated with flow limited breathing. The algorithms used in the current study did not have the ability to determine if apneas were central or obstructive. The PAP device generated an event marker signal 10 sec from the start of a detected apnea and within 2 breaths of the end of a detected hypopnea.

Manual PSG Scoring

The PSGs were manually scored with the aid of computer software by a PSG technologist at a central scoring facility. The technologist was blinded to the event signal during manual scoring (the event signal was not visible in the montages used for scoring). Sleep staging and respiratory events were scored using 2007 American Academy of Sleep Medicine (AASM) guidelines.[5] The scoring of a hypopnea required that the event be associated with a $\geq 4\%$ oxygen desaturation.[5]

Studies Analyzed

A total of 148 studies, collected from 115 participants, were included in this analysis. A total of 304 studies were available from the parent study. Of these, 119 studies had a technically adequate recording of the event detection signal and were collected from 90 patients (29 patients contributed two studies from the parent study). These 119 studies were pooled with another 29 studies from the subtherapeutic trial for a total of 148 studies. Four patients participated in both trials, and three of these patients were among those in the parent study who contributed two recordings. Therefore, 85 patients contributed only 1 PSG, 27 patients contributed exactly 2 PSGs, and 3 participants contributed 3 PSGs.

Individual Event Analysis

To facilitate the comparison of the AED events to the manually scored events, a customized computer program was developed to identify whether a device-detected event was a true positive (identified on both the manually scored PSG and by the device), false positive (identified by the device but not on the manually scored PSG), or false negative (identified on the manually score PSG but not the device). For the event-by-event analysis, no distinction was made between apneas and hypopneas (combined as "respiratory event").The customized computer program generated a time-ordered list of manually scored events and events identified by the device (event marks in the event detection channel, Figure 1). True negative events were estimated by determining the number of 30-sec epochs during the PSG when neither method identified an event.

To validate the customized computer program, the 29 PSGs performed at sub-therapeutic pressure were reviewed manually by a single skilled PSG technologist at the central scoring facility. For this validation, only device-detected events during sleep or associated with a manually scored event were considered. All of the manually scored events were successfully detected by the customized computer program (2,234 events). The customized computer program identified 2,229 of the 2,234 AED events. The AED events on the PSG signal that were not detected by the customized computer program were due to five couplets of AED events occurring very close together (essentially superim-

posed). The computer determinations of event type (false positive, true positive, false negative) were then checked manually by the technologist for accuracy according the pre-established rules for each category. Except for the five AED events that were missed by the program, all computer-generated classifications of the relationship between all PSG and AED events were correct. In summary, the manual review confirmed that the customized computer program accurately noted the occurrence of both the manually scored and device events, listed all events (100% of PSG events and 0.99% of AED events), and correctly classified events as true positive, false positive, and false negative.

After its validation, the customized computer program was applied to all 148 studies for an event-by-event comparison of AED versus manually scored events. After each manually scored event, a 30-sec window was analyzed for the occurrence of an AED event. This accounted for any AED delay in reporting the event (e.g., the AED does not report a hypopnea until at least 2 breaths after the end of the actual event). Specifically, if at least one AED-detected event (apnea or hypopnea) occurred sometime between the start of a manually scored event (apnea or hypopnea) and 30 sec after the end of the same manually scored event, then the detected event was considered a true positive. If the automatic event marker preceded the manually scored event, it was considered a false positive. The absence of an event marker during or within 30 sec after a manually scored event was considered a false negative.

Statistical Methods

The respiratory event indices for manually scored events were computed as the total number of events divided by the hours of sleep. The indices for AED events were the number of events per hour of therapy time. The agreement in AHI, AI, and HI between the two detection methods was assessed using the nonparametric Wilcoxon signed-rank test due to the asymmetric distribution of the data. Intraclass correlation was performed using a two-way random model measuring absolute agreement for single measures. In addition, Bland-Altman plots of the AHI, AI, and HI data were generated for visualization of the bias and limits of agreement.[11] The sensitivity, specificity, positive predictive value, and negative predictive value for given AED cutoff values were calculated, and receiver operating characteristic (ROC) curves were constructed. The data were analyzed using computer programs: SPSS (version 15; Chicago, IL) and Analyze-it (version 2.21; UK) computer programs. Statistical trends were considered to be significant at $P < 0.05$.

As noted previously, 30 subjects contributed 2 or 3 PSGs to our data set. To determine if these contributions may have affected our results, we repeated our analysis by defining a smaller group in which each subject contributed only one PSG (n = 115). When a given subject contributed more than 1 PSG, the study to be included was selected using a random number generator.

RESULTS

Demographics and Sleep Apnea Severity

The demographic data for the subjects are listed in Table 1. The subjects were obese and had, on average, severe

Table 1—Demographics and summary statistics

Variables	Mean ± SD	n
Age (yr)	49.5 ± 11.3	115*
BMI (kg/m^2)	36.2 ± 7.6	115*
AHI from diagnostic PSG	45.3 ± 30.3	119**
Sex Distribution		
Male (n, %)	84 (73%)*	
Female (n, %)	31 (27%)*	

*In summarizing the age, body mass index (BMI), and sex statistics, the four participants who completed both trials were only counted once. **The four participants who contributed results from the parent and subtherapeutic studies underwent a separate diagnostic polysomnogram (PSG) for each of the trials. Therefore, both apnea-hypopnea index (AHI) values were included in calculating the population's average.

Table 2—Apnea-hypopnea index (AHI), apnea index (AI) and hypopnea index (HI) on manually scored PSG and AED (n = 148)

Variable	Manual score*	AED*	P value**
AHI	5.6 ± 8.0 (2.8)	5.8 ± 6.3 (4.1)	0.007
AI	2.4 ± 4.8 (0.80)	3.2 ± 5.25 (1.8)	< 0.001
HI	3.2 ± 5.2 (1.15)	2.6 ± 2.3 (2.1)	0.301

*Values are mean ± SD (median). **Analysis by Wilcoxon signed-rank test.

Table 3—Intra-class correlation (ICC)*

Variable	Coefficient	P value	n
AED AHI versus PSG AHI	0.789	< 0.001	148
AED AI versus PSG AI	0.825	< 0.001	148
AED HI versus PSG HI	0.350	< 0.001	148

*Two-way random model assessing absolute agreement, for single measures.

OSA (AHI ≥ 30) on their diagnostic PSG. The subjects in whom PSGs were performed at subtherapeutic PAP settings had similar demographics as those in the parent trial. For the studies with PAP treatment, the mean AHI was 5.6 ± 8.0 events/hr on the manually scored PSG and 5.8 ± 6.3 by AED (P = 0.007; Table 2). The mean AI was slightly higher by AED than manual scoring (P < 0.001). The mean HI by manual scoring and AED were not significantly different.

Correlation and Bland-Altman Analysis

The AHI, AI, and HI values from manually scored PSG and AED were highly correlated (Table 3, Figure 2). There was considerably more scatter for hypopneas than apneas.

The difference between AHI, AI, and HI values (AED versus manually scored PSG) were plotted against their respective averages (Figure 3). The bias (mean difference) and limits of agreement (95%) are shown. The AED tended to overestimate the AHI at low AHI values and underestimate the AHI at higher AHI values. The agreement for AI was quite good. The limits of agreement were larger for HI than AI and the difference between the two methods of HI determination increased with increasing values of the HI. Therefore, most of the scatter in AHI comparisons was due to differences in hypopnea detection by the two methods.

Event-by-Event Analysis

The event-by-event analysis (for apneas and hypopneas combined) found that the AED had a sensitivity of 0.58 and a specificity of 0.98 (n = 148 studies). For the smaller data set of 115 studies, the results were almost identical, with a sensitivity of 0.55 and a specificity of 0.98. Review of the individual tracings found that false-positive AED events were often due to reductions in flow that were not manually scored as hypopneas because they were not associated with at least a 4% decrease in oxygen saturation (Figure 4) or the reductions in flow occurred during wakefulness. False-negative events were often characterized by reductions in flow associated with at least a 4% oxygen desaturation but without sharp demarcations in flow reduction (gradual reductions) and/or absence of distinct recovery breaths at the end of the event (Figure 5).
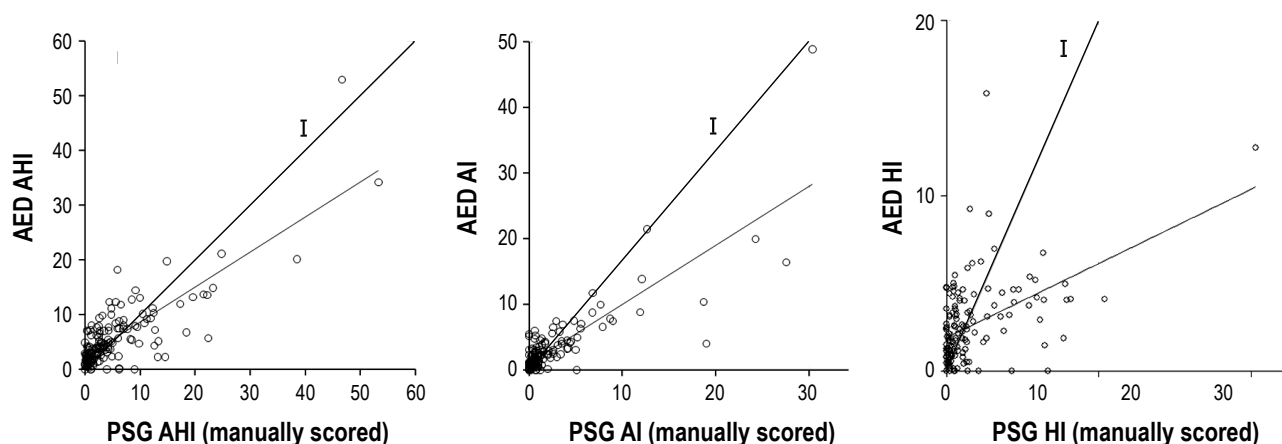

Figure 2—Plots of the apnea-hypopnea index (AHI), apnea index (AI), and hypopnea index (HI) determined by manual scoring (events/hr of sleep) versus the corresponding values determined by AED (events/hr of recording). Each point represents the results of one sleep study. The lines are the linear regression by least squares analysis and line of identity is denoted by I.
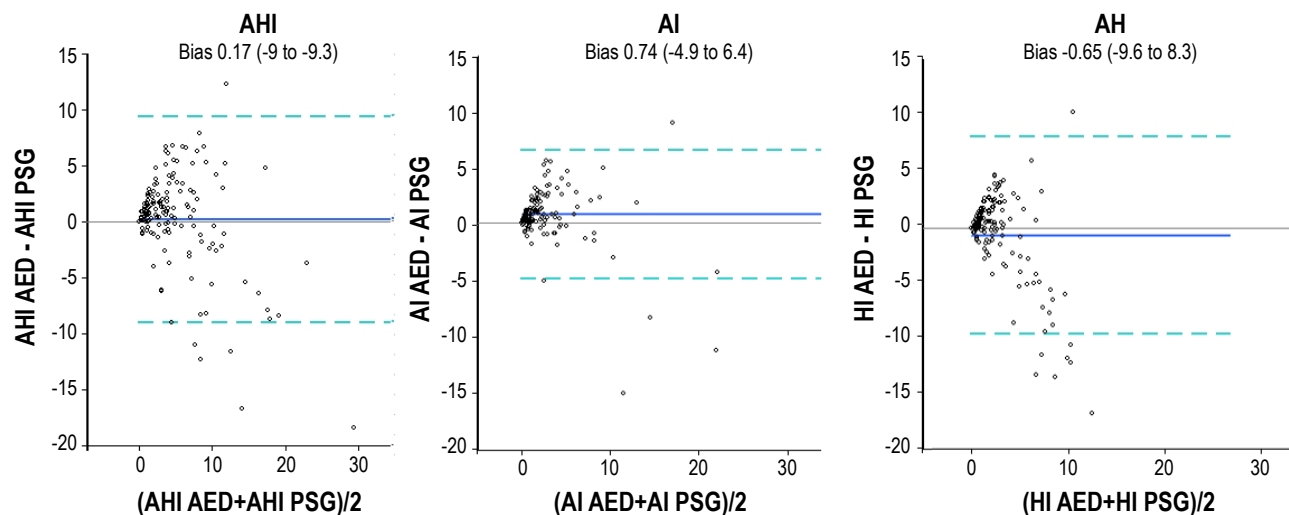
AHI
Bias 0.17 (-9 to -9.3)

AI
Bias 0.74 (-4.9 to 6.4)

AH
Bias -0.65 (-9.6 to 8.3)

Figure 3—Bland-Altman plots of apnea-hypopnea index (AHI), apnea index (AI), and hypopnea index (HI) for each sleep study with the difference between the respective AED and manually scored measures plotted against the average of the values. Outliers (x,y) for AHI (49.3,6.23; 43.7,-19.1) and AI (39.6, 18.5) are not shown to improve visualization. Note that the scatter tended to increase with higher AHI and that the difference between the two methods of HI determination increased with increasing values of HI.

Figure 4—In this 120-sec PSG tracing in Stage N2, the period of reduced airflow is detected as a hypopnea (H) event by AED but was not scored manually because it was not associated with a ≥ 4% decrease in oxygen saturation. Inspiration for the PAP flow signal (CFlow) is downward. The frequent narrow events on the AED signal are snoring. Here (H) marks the event detection signal for hypopnea and the corresponding airflow tracing event. The background "2" refers to stage N2.

Figure 5—A 120-sec PSG tracing in which a reduction in airflow with an associated 4% oxygen desaturation is scored manually as a hypopnea but did not trigger an AED hypopnea detection as the decrease in airflow during the event and increase in recovery airflow were not very large. In the event channel, the frequent narrow square waves were snoring events. Inspiration for the PAP flow signal (CFlow) is downward. The background "R" refers to stage R.

ROC Analysis

The corresponding AHI values (PSG and AED) for each study were analyzed using the manually scored PSG results as the gold standard. True-positive and true-negative pairs as well as false-positive and false-negative pairs could then be determined. A ROC curve was generated using a PSG AHI cutoff of 10 events/hr and various cutoffs of the AED AHI and their associated sensitivity and specificity (n = 148 studies). Figure 6 shows the res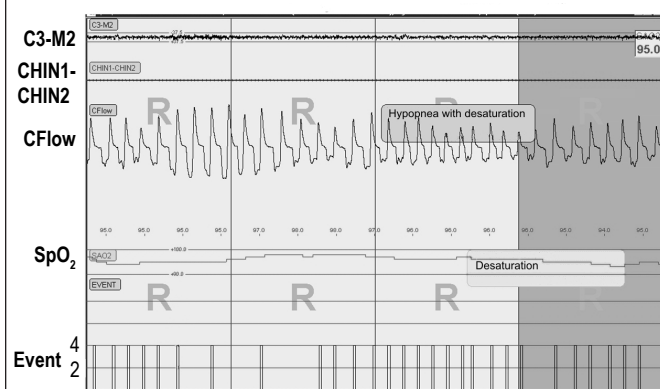ulting ROC curve with an area under the curve of 0.86. Using an AHI AED cutoff of 10 events/hr, the sensitivity was 0.58 and specificity was 0.94. The positive predictive value was 0.67 and negative predictive value was 0.92. For the smaller data set of n = 115 studies, the sensitivity was 0.55 and the specificity was 0.94.

The positive predictive value is the true positives divided by the sum of true positives and false positives. Therefore, the positive predictive value was reduced by a substantial number of false positives. Thus, a significant proportion of the AHI AED values > 10 events/hr were associated with an AHI PSG < 10 events/hr (false-positive values by AED). As noted previously, most of the false positives occurred when the AED detected a hypopnea based on a change in flow that was not associated with a ≥ 4% arterial oxygen desaturation (no hypopnea by manually scored PSG).
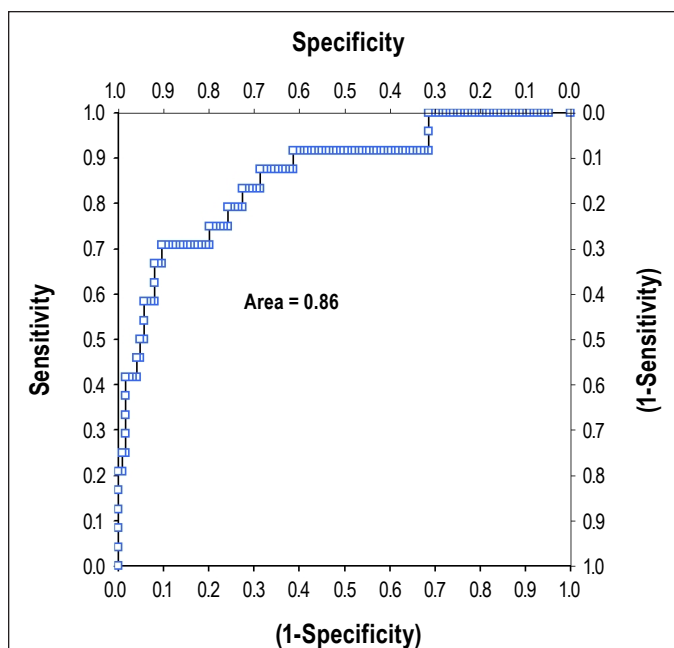
Figure 6—ROC curve for an AHI PSG = 10 events/hr created by determining sensitivity and specificity values for various AHI AED values.

DISCUSSION

This is the first event-by-event comparison of PAP device respiratory event detection to PSG results scored manually using the recommended 2007 AASM scoring criteria for apneas and hypopneas.[5] The results of our study show that the AED tended to overestimate the AHI when the manually scored AHI was low and underestimate the AHI when the manually scored AHI was high. An AHI < 10 events/hr by AED was highly predictive that the manually scored PSG AHI was < 10 events/hr. On the other hand, an AHI ≥ 10 events/hr by AED was only moderately predictive that the PSG AHI was ≥ 10 events/hr (positive predictive value = 67%). The event-by-event analysis showed that AED detection had a high specificity but only modest sensitivity (high number of false negatives). There was relatively good agreement between AED and manual scoring for apnea detection, but there was less agreement between the two methods with respect to detecting hypopneas.

The respiratory event information provided by PAP devices has the potential to help clinicians alter the prescribed pressure settings and make decisions about the need for an additional attended PAP titration PSG. The AASM practice parameters state that a PAP titration PSG is not indicated for patients apparently doing well on PAP treatment.[4] However, studies have found that a surprisingly large fraction of patients thought to be well treated still have a considerable number of residual events.[12,13] Therefore, the availability of accurate residual AHI information could be of great clinical utility in delivering effective PAP treatment.

A limited number of studies have compared the respiratory event detection algorithms of current-generation PAP devices with manually scored events on PSG. Ueno et al.[8] compared the AHI from manually scored PSG to the event detection by APAP (S8 auto-CPAP, ResMed, Poway, CA). Although the unit tested in that study used a different algorithm to detect respiratory events than that used by the unit in the current study,

similar results were obtained. The AHI was slightly higher than that by manually scored PSG, although individual AHI pairs were highly correlated. Similar to our study there was a weaker relationship between hypopnea indices (PSG versus device). Desai and coworkers[9] compared the AHI by APAP (RemStar AutoPAP, Philips-Respironics) versus manually scored PSG. A device cutoff AHI of 8 events/hr predicted a PSG AHI > 10 events/hr with a sensitivity of 0.94 and a specificity of 0.90. The PSG criteria for hypopnea in their study included a decrease in airflow with either a ≥ 4% decrease in the arterial oxygen saturation *or an arousal*. The reason for higher sensitivity and specificity in their study is not known, but could be related to more inclusive criteria for a manually scored hypopnea.

A number of factors likely explain the differences between AED-detected respiratory events and manually scored events. The AED is entirely dependent on the PAP airflow signal, whereas PSG scoring using AASM-recommended criteria depends on both the PAP airflow signal and changes in arterial oxygen saturation. Figure 4 shows an example of an event scored as a hypopnea by AED but not by manual PSG scoring due to the absence of a ≥ 4% desaturation. On the other hand, some respiratory events associated with arterial oxygen desaturation were manually scored with only a modest decrease in flow (30% from baseline) that was not large enough to meet AED detection criteria (Figure 5). The event detection algorithms were designed to be conservative to avoid overtitration and therefore require greater and distinct changes in flow.

Another factor that accounts for the observed differences between AED and manual scoring is the different methods used to calculate the respiratory event indices. The current study sought to mimic real clinical practice and determine how well the device AHI reflects an AHI that would be determined with PSG. Therefore, the AED respiratory indices used in the analyses represented the number of detected events per hour of therapy time (run time associated with fluctuating flow), whereas the respiratory events on PSG were only scored during sleep. As a result, some of the false-positive AED events were due to events scored by the AED algorithm during wakefulness.

A limitation to our study is that, even with the addition of patients on suboptimal PAP, the overall AHI values were relatively low (reflecting good treatment). In the event-by-event analysis, it could be argued that the low AHI values may have improved the negative predicted values (low prevalence). However, the situation where the residual AHI is most often of clinical use is in patients who are fairly well treated. Another limitation to our study is that our patients did not have a substantial number of apneas on PAP. Apnea classification by manual scoring does not depend on the presence or absence of arterial oxygen desaturation. The apnea index of our patients was approximately 50% of the AHI. Agreement between AED and manual scoring was actually better for apneas than hypopneas. Thus, it might be expected that AED based on the current algorithms would be reasonably accurate in patients with numerous residual apneas.

Ambulatory pulse oximetry has been used to help adjust fixed CPAP settings.[14] One possible approach to improve the sensitivity of PAP device event recognition is to couple the current algorithms with information from oximetry. Oximetry modules connecting to PAP devices are commercially avail-

able. Perhaps it would be possible to integrate information from airflow and measured oximetry to produce a better estimate of the residual AHI. If oximetry information was available, then it might be possible to develop an AED algorithm that more closely resembles the AASM PSG scoring criteria for hypopneas. This type of information might also help clinicians detect periods of hypoxemia due to low ventilation without discrete respiratory events.

Our study used the proprietary event detection algorithm of one manufacturer's device. Therefore, the results of the current study may not generalize to devices made by other manufacturers. When using the residual AHI from a given device, the clinician should keep in mind the studies validating that algorithm and the types of patients studied. Ueno et al.[8] evaluated the event detection algorithm of a different manufacturer than the one used in the current study and found similar results.

It is also important to note that the subject group we studied did not include patients with prior upper airway surgery, Cheyne-Stokes breathing, or significant non-respiratory sleep disorders. Such patients were excluded from the protocol. Therefore, our results cannot be generalized to these patient populations. Furthermore, if a patient has not had a good clinical response to PAP treatment, a device estimate of AHI is not a substitute for a PSG on positive pressure treatment. A PSG may detect frequent unsuspected arousals from mask or mouth leak as well as identify nonrespiratory causes of sleep disturbance.

In summary, the current study suggests that the AED algorithm has the greatest clinical utility when the residual AHI is low. An AHI < 10 events/hr by AED is likely to indicate efficacious treatment. High AHI values by AED may represent inadequate treatment especially if the reported events are apneas. Differences between manually scored and AED events were primarily due to the detection of hypopneas and can be explained by the different criteria used by the two methods to detect these events. Clinicians need to interpret the AED results obtained from PAP downloads with the realization that the criteria used by AED to detect the respiratory events are different from those used to manually score PSGs. Importantly, the AED results must be combined with clinical assessment of the patient to determine appropriate management decisions.

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DISCLOSURE STATEMENT

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