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Original article

Assessment of a wrist-worn device in the detection of obstructive sleep apnea

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Abstract

Objective: To assess the accuracy of a wrist-worn device (Watch_PAT100) to diagnose obstructive sleep apnea (OSA).

Methods: Thirty adult subjects with and without suspected OSA simultaneously had a standard in-laboratory polysomnogram (PSG) and wore the Watch_PAT100 during a full-night recording. PSG sleep and respiratory events were scored according to standard criteria. Watch_PAT data were analyzed with an automated computerized algorithm which calculated the frequency of respiratory events per hour of actigraphy measured sleep using a combination of peripheral arterial tonometry (PAT) signal attenuation, desaturation on pulse oximetry, and changes in heart rate. This yielded a PAT apnea hypopnea index (AHI).

Results: Mean age was 47.0 ± 14.8 years, mean body mass index 31.0 ± 7.6 kg/m^2, mean PSG AHI 23 ± 23.9 events per hour, and mean PAT AHI 23 ± 15.9 events per hour. There was a significant correlation between PAT AHI and AHI by PSG ($r = 0.87$, $P < 0.001$). To assess sensitivity and specificity of Watch_PAT, we constructed receiver operator characteristic curves using a variety of AHI threshold values (10, 15, 20, and 30 events per hour). Optimal combinations of sensitivity and specificity for the various thresholds were 82.6/71.4, 93.3/73.3, 90.9/84.2, and 83.3/91.7, respectively.

Conclusions: The Watch_PAT is a device that can detect OSA with reasonable accuracy. Thus, the Watch_PAT may be a useful method to diagnose OSA.

Keywords: Sleep apnea syndromes; Sympathetic nervous system; Diagnostic techniques and procedures; Arousal; Peripheral arterial tonometry; Vasoconstriction

1. Introduction

Obstructive sleep apnea (OSA) is a common disorder estimated to affect approximately 4% of men and 2% of adult women [1]. The disease is characterized by repetitive episodes of upper airway collapse during sleep leading to sleep fragmentation, daytime sleepiness, cognitive dysfunction, automobile and industrial accidents, and possibly adverse cardiovascular sequelae [2,3]. Treatment of OSA leads to improvements in many of these adverse outcomes, and may reduce health care costs [4–6]. Consequently, early diagnosis of this disorder is important.

Presently, the diagnosis of OSA predominately depends on overnight in-laboratory polysomnography (PSG), which includes the continuous recording of a number of physiologic variables including airflow, chest/abdominal movements, electroencephalography, electromyography, and oxygen saturation [7]. This allows quantification of hypopneas and apneas according to standard criteria [8]. However, full PSG is expensive, cumbersome, and difficult to perform on an ambulatory basis. As a result, there have been a number of attempts to develop simpler and cheaper methods to diagnose OSA. Many such devices rely on similar physiologic signals as are used in full PSG, but with a reduced number of such signals making it cheaper and more feasible in an ambulatory setting [9].

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One new technology that may be useful in the ambulatory diagnosis of OSA is the Peripheral Arterial Tonometer (PAT) [10,11]. This technology uses a finger mounted optic/pneumatic sensor that eliminates venous pulsations and continuously measures the pulse volume of the digit. Episodic vasoconstriction of digital vascular beds from sympathetic stimulation (mediated by alpha receptors) results in attenuation of the signal. Because discrete obstructive airway events (e.g. apneas, hyponeas, upper airways resistance) cause arousal from sleep, sympathetic activation, and peripheral vasoconstriction [12], these events are associated with attenuations of the PAT signal. Schnall et al. actually demonstrated a strong correlation ($r = 0.92$) between the frequency of PAT attenuations and the apnea hypopnea index (AHI) when tested simultaneously in the laboratory [10].

In this study, we assessed the accuracy of a wrist-worn device which combines this PAT technology with actigraphy and arterial oxygen saturation to diagnose OSA.

2. Materials and methods

We recruited adult subjects to participate in our study, including patients suspected of having sleep apnea and subjects without suspected sleep apnea who were recruited through advertisements. This allowed us to obtain a broad range of sleep-disordered breathing. Individuals who were using home oxygen, medically unstable, or using medications that block the alpha receptor were not eligible to participate. Informed consent was obtained from all participants after the protocol was approved by the Human Subjects Committee at Brigham and Women's Hospital.

All subjects completed a comprehensive sleep and health survey that included an Epworth Sleepiness Scale [13]. All underwent a standard in-laboratory full overnight PSG. Signals recorded included: electrooculography (EOG), electrocardiography (ECG), submental and tibial electromyography (EMG), electroencephalography (EEG, C2-A1, C3-A2, O1-A2, O2-A1), chest and abdominal motion, airflow [nasal–oral thermistor and nasal pressure (PTAF2, Pro-Tech Services, Woodinville, WA)], arterial oxygen saturation (Model 930 Pulse-Oximeter, Respironics, Pittsburgh, PA), and snoring intensity. All physiologic data were collected and stored using the ALICE3 digital PSG system (Respironics Inc., Pittsburgh, PA).

All subjects simultaneously wore the Watch_PAT100 during the night (Itamar Medical LTD, Caesarea, Israel). This is a self-contained device worn around the wrist (Fig. 1). Two finger probes extend from the main body of the device; one is the optico-pneumatic sensor that detects the PAT signal, the other measures arterial oxygen saturation. The body of the device also contains an actigraph (three-axis accelerometer for detection of limb activity), which was used to differentiate sleep time from wake time.

PSGs were all scored by a single technologist who was blinded to the Watch_PAT signals. Sleep plus apneas and hypopneas were scored from the PSG according to standard criteria (Rechtschaffen and Kales, Chicago criteria) [8,14]. In particular, an apnea was scored if airflow was absent for 10 s, and a hypopnea if airflow was reduced by 50% or a lesser extent in association with a desaturation of 3% or an arousal. Arousals were identified using ASDA criteria [15].

The Watch_PAT continuously recorded four physiologic signals throughout the night (PAT, oxygen saturation, heart rate, actigraphy). These data were then analyzed with an automated computerized algorithm that calculated the frequency of respiratory events per hour of actigraphy-determined sleep. Respiratory events were detected during sleep (per actigraphy) using a combination of PAT signal attenuation, desaturation on pulse oximetry, and changes in heart rate. In particular, a respiratory event was scored if one of three criteria were met: (1) a 30% or greater PAT amplitude reduction together with a pulse rate acceleration of 10%; (2) a 30% or greater PAT amplitude reduction together with a 3% oxyhemoglobin desaturation; or (3) a 4% oxyhemoglobin desaturation.

2.1. Data analysis

The PSG was considered the gold standard for identifying and quantifying OSA. The utility of the Watch_PAT in detecting OSA (i.e. AHI) was based on summary data for the nights and was assessed in a number of ways. This included evaluations for correlation (using linear regression), agreement (using Bland–Altman plots), and by constructing receiver operator characteristic (ROC) curves using ANALYSE-IT, Clinical Laboratories Software. Also, given that there is no well-defined AHI cut-off value for defining OSA, we also assessed diagnostic agreement using a clinical approach described previously to assess the diagnostic utility of another ambulatory device to detect OSA [9]. According to this analysis, the Watch_PAT and PSG were considered in agreement if: (1) both AHI from the PSG and PAT AHI were greater than 40 events per hour; or (2) if the AHI was <40 per h on PSG, the Pat AHI was within 10 events per h of the AHI.

3. Results

We studied a total of 30 subjects (19 males, 11 females—Table 1). Twenty-five of these were recruited from patients with suspected sleep apnea, and five were subjects who responded to a flyer. The mean age of our subjects was 47.0 ± 14.8 (range = 24.3–66.6), and mean body mass index was 31.0 ± 7.6 kg/m^2. Mean AHI per PSG was 23 ± 23.9 events per hour, with a broad range of sleep apnea severity (range of AHI = 1–94). The mean PAT AHI was 23 ± 15.9 events per hour (range = 6–69).

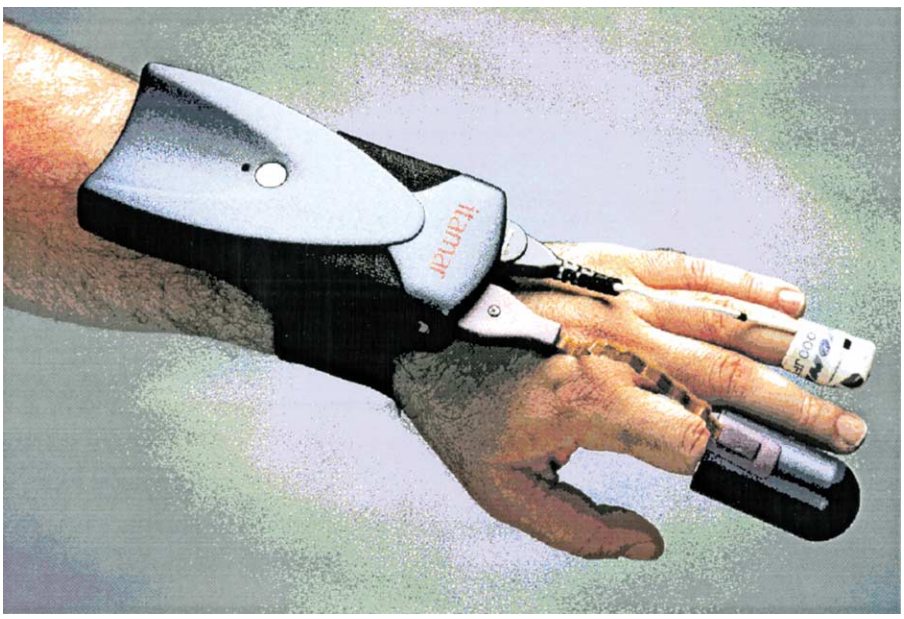

Fig. 1. The Watch_PAT100. The instrument on the fourth digit is the oximeter and on the second digit the PAT probe. The actigraph is contained in the wrist device.

Table 1
Characteristics of subjects

Subject no.	Age (years)	Gender	BMI (kg/m^2)	ESS	PSG AHI (events per hour)	PAT AHI (events per hour)	PLM index (events per hour)
1	32.2	F	24.1	0	4	6	0
2	56.2	M	24.5	14	59	33	4
3	56.2	M	22.1	11	5	15	42
4	61.6	M	25.8	4	11	15	2
5	66.6	M	25.1	11	21	26	0
6	26.6	M	30.8	16	32	42	0
7	54.6	M	33.2	7	20	26	0
8	73.4	M	26.0	7	19	17	3
9	37.7	M	33.8	3	13	15	23
10	48.6	F	37.6	7	12	20	0
11	36.7	F	38.6	9	11	9	0
12	48.8	M	36.6	7	81	45	0
13	49.8	F	37.8	6	18	17	18
14	32.5	M	33.5	14	27	34	0
15	34.8	F	33.0	8	13	23	0
16	33.0	M	30.0	8	14	9	0
17	74.8	M	23.0	13	28	27	60
18	37.2	M	41.0	24	94	69	0
19	55.2	M	33.0	16	15	24	0
20	24.3	M	21.0	11	1	9	0
21	58.8	F	51.0	5	34	19	0
22	22.4	F	44.0	13	11	35	0
23	57.0	M	36.0	5	79	68	12
24	59.5	M	25.0	5	28	20	0
25	70.7	F	34.0	15	10	7	0
26	31.5	F	20.0	6	3	11	0
27	38.8	F	37.0	6	11	13	0
28	50.9	M	24.0	9	4	14	23
29	35.7	M	23.0	8	7	19	1
30	44.3	M	25.0	9	15	9	3
Mean	47.0		31.0	9.2	23	23	6

BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hyponea index; PSG, polysomnography; PAT, peripheral arterial tonometer; PLM, periodic limb movement.

As shown in Fig. 2, there was a significant correlation between PAT AHI and PSG AHI (Pearson's coefficient = 0.87, $P < 0.001$). However, the slope of the best-fit line between PSG and PAT AHI was 30% steeper than the line of identity. A Bland–Altman plot of PAT AHI and PSG AHI is shown in Fig. 3. At lower levels of AHI, PAT tended to overestimate disease severity, while at higher levels of AHI, Watch_PAT underestimated severity.

In order to assess the sensitivity and specificity of Watch_PAT, we constructed a number of ROC curves. Because there is no threshold value for AHI that definitively differentiates patients with and without OSA, we constructed different curves using a variety of AHI threshold values (10, 15, 20, and 30 events per hour). One of these ROC curves is shown in Fig. 4. For all thresholds of AHI, the area under the ROC was greater than 0.86. For AHI thresholds of 10, 15, 20, and 30, optimal combinations of sensitivity and specificity were 82.6/71.4, 93.3/73.3, 90.9/84.2, and 83.3/91.7, respectively.

When using the previously described clinical approach to assess agreement, concordance was found in 26 of 30 subjects (87%).

Based on preliminary data that suggested that PAT indices can predict daytime sleepiness [16], we calculated the correlation coefficient between PAT AHI and subjective sleepiness as measured by the Epworth Sleepiness Scale. In this study, PAT AHI was a significant predictor of Epworth score (Pearson's coefficient = 0.46, $P = 0.01$), with a coefficient similar to PSG AHI ($r = 0.34$, $P = 0.07$) and the PSG arousal index ($r = 0.33$, $P = 0.08$).

4. Discussion

In this study, we assessed the ability of a wrist-worn device to detect OSA. This device is unique in that it relies predominately on the detection of episodic vasoconstriction of digital vascular beds to detect obstructive airway events, rather than measurements of flow and/or chest movements. We demonstrated that the Watch_PAT AHI is significantly correlated with the AHI as measured by PSG. This compares favorably with oximetry alone. The pooled sensitivity of oximetry is approximately 87% with a specificity of 68%; however, these values vary widely (36–100%, 23–99%, respectively) [17]. The PAT device also has an accuracy similar to ambulatory devices currently used in clinical practice. For instance, White et al. assessed the accuracy of NightWatch (Respironics Inc.), an ambulatory device to detect OSA. In their study, NightWatch had a sensitivity of 91% and a specificity of 70%, with a correlation between PSG and NightWatch detected events of 0.92 [9]. Thus, despite recording substantially more physiologic information (eye movements, leg movements, oxygen saturation, chest/abdominal motion, airflow, and heart rate)

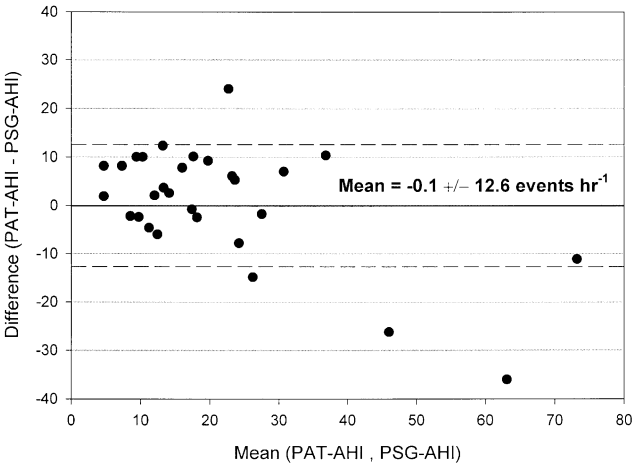

Fig. 3. A Bland–Altman plot between PAT AHI and PSG AHI. At lower levels of AHI, Watch_PAT overestimated PSG AHI. At higher levels, Watch_PAT underestimated PSG AHI.

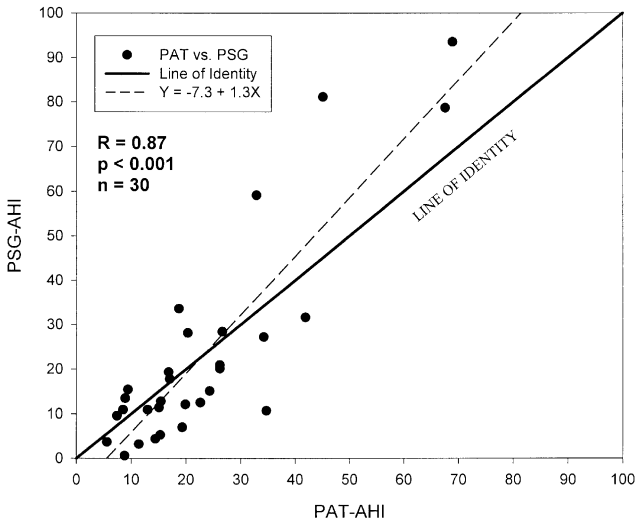

Fig. 2. Pearson's correlation coefficient between PAT AHI and PSG AHI was 0.87.

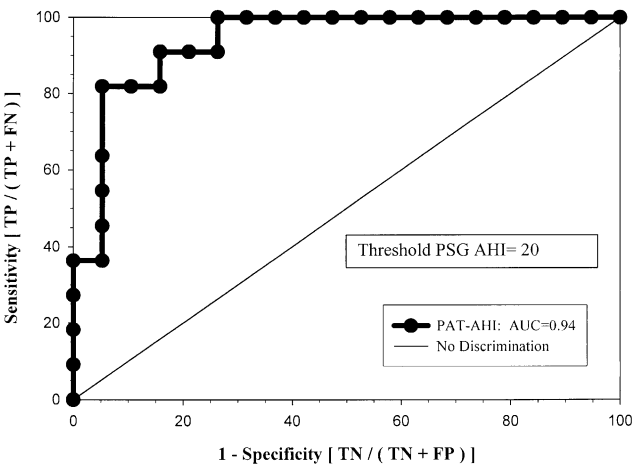

Fig. 4. A representative ROC curve (using a PSG AHI threshold value of 20 events per hour).

NightWatch was not substantially better than the Watch_ PAT. Of note, however, we did not directly test the Watch_PAT in the home, and its performance under these conditions is not known. Nevertheless, one could speculate that the Watch_ PAT may eventually become a useful tool in the ambulatory diagnosis of sleep apnea.

Our results are consistent with and extend those of Schnall et al. [10]. Although these investigators did not use a self-contained wrist-worn device, they were able to collect simultaneous PAT signals and overnight PSG in 42 subjects. In their study, they defined a PAT event when: (1) the PAT signal amplitude decreased by 33% or greater from baseline, and (2) heart rate increased by 15% or greater. The correlation between the frequency of PAT attenuations and AHI was 0.92. This value is very similar to the correlation we found in our study. They also found that PAT overestimated events when PSG AHI was in the lower range but underestimated events when AHI was high.

In our study, the Watch_PAT tended to overestimate events when PSG AHI was in the lower range. According to our linear regression equation (PSG AHI $= -7.3 + 1.3 \times$ PAT AHI) relating PAT and PSG AHI, a PSG AHI of 0 would result in a PAT AHI of 5.6, a PSG AHI of 5 would result in a PAT AHI of 9.5, and a PSG AHI of 10 would result in a PAT AHI of 13.3. One could speculate that this discordance was partly because the Watch_PAT detected 'events' that were missed by standard scoring criteria. For instance, some subjects had discrete episodes of decreased flow or flow limitation followed by subtle EEG changes plus signs of sympathetic activation (i.e. increases in heart rate and vasoconstriction) (Fig. 5). However, these episodes did not have adequate desaturation or EEG changes (e.g. burst of K complexes or alpha of less than 3 s) to meet either ASDA or Chicago criteria, and thus could not be scored as events. It is possible that these events represented 'upper airway resistance syndrome' events or 'respiratory effort related arousals'; however, this cannot be confirmed, as esophageal pressure was not monitored during the study.

Another explanation for the overestimation of PAT at lower levels of AHI may relate to sleep fragmentation (arousals) other than those induced by apneas. Because PAT detects episodic sympathetic activation, other disorders that cause arousal from sleep may also result in PAT attenuation. An example of this was found in patient number three. This patient had moderately severe periodic limb movements of sleep (index of 42 events per hour) that caused arousals from sleep. When full arousals occurred, the PAT signal was attenuated and, on some occasions, was incorrectly scored by the automated analysis algorithm as obstructive airway events (Fig. 6). It is possible that modifications of the current algorithm may enable it to better distinguish apneas from limb movements, thereby decreasing this source of error. However, at the present time, we conclude that the accuracy of the Watch_PAT may be reduced in patients

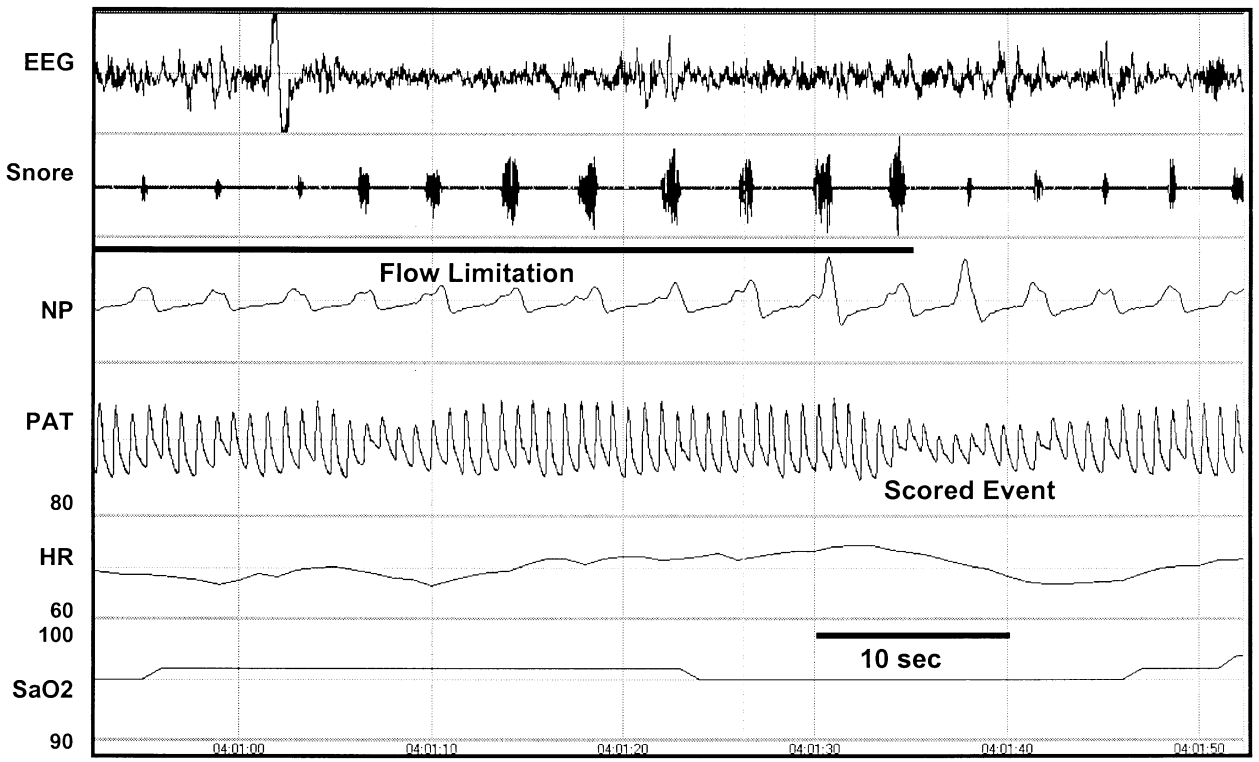

Fig. 5. This represents a 60 s sample of a subject's PSG. One can see an episode of crescendo snoring followed by a PAT attenuation and increase in heart rate; this was detected by the device as a respiratory event. However, this could not be scored as a hypopnea according to standard criteria given the lack of an ASDA arousal (i.e. 3 s of alpha) or sufficient oxygen desaturation. NP, nasal pressure; HR, heart rate; SaO_2, arterial oxygen saturation.

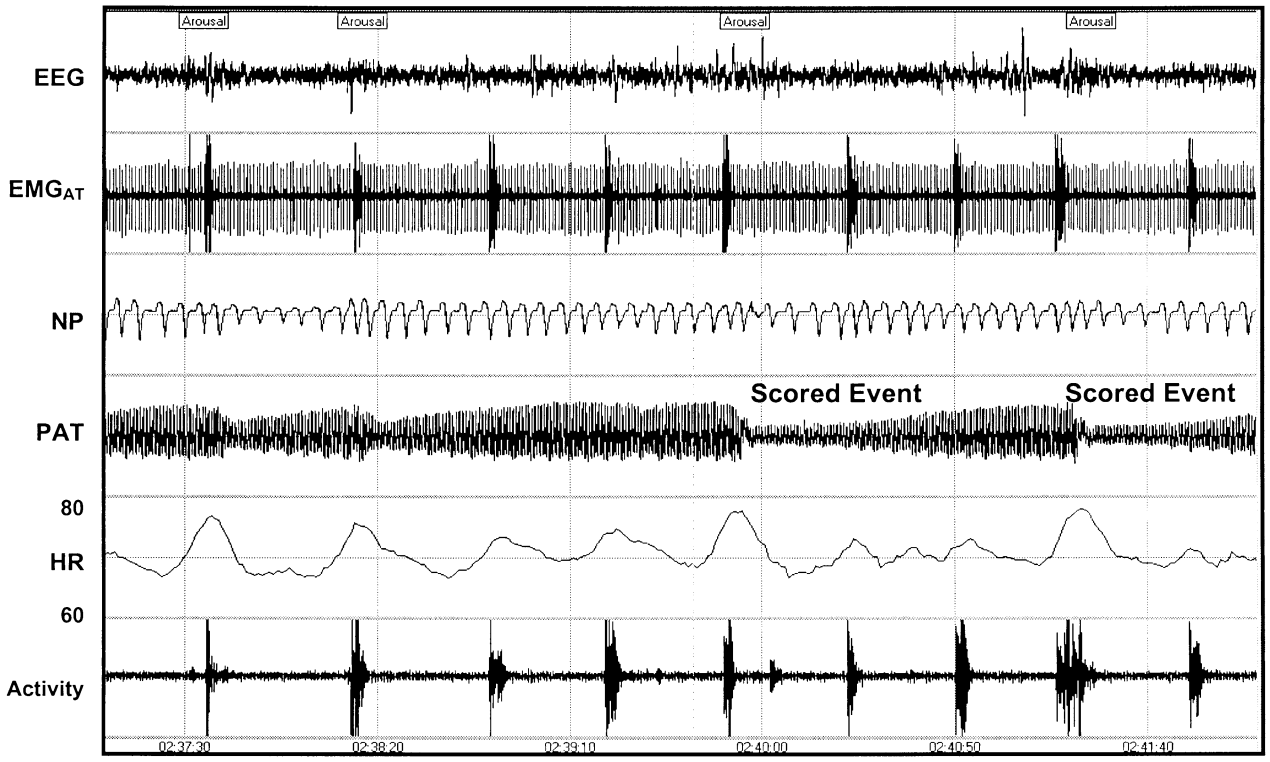

Fig. 6. This represents a 5-min sample of a PSG from a subject with periodic limb movements of sleep. These movements can be well seen in both the actigraphy and leg EMG channels. Some of these events were associated with arousal and resulted in increases in heart rate and PAT attenuation. These were incorrectly scored as respiratory events by the device. EMG_{AT}, anterior tibialis EMG; NP, nasal pressure; HR, heart rate; Activity, actigraphy signal.

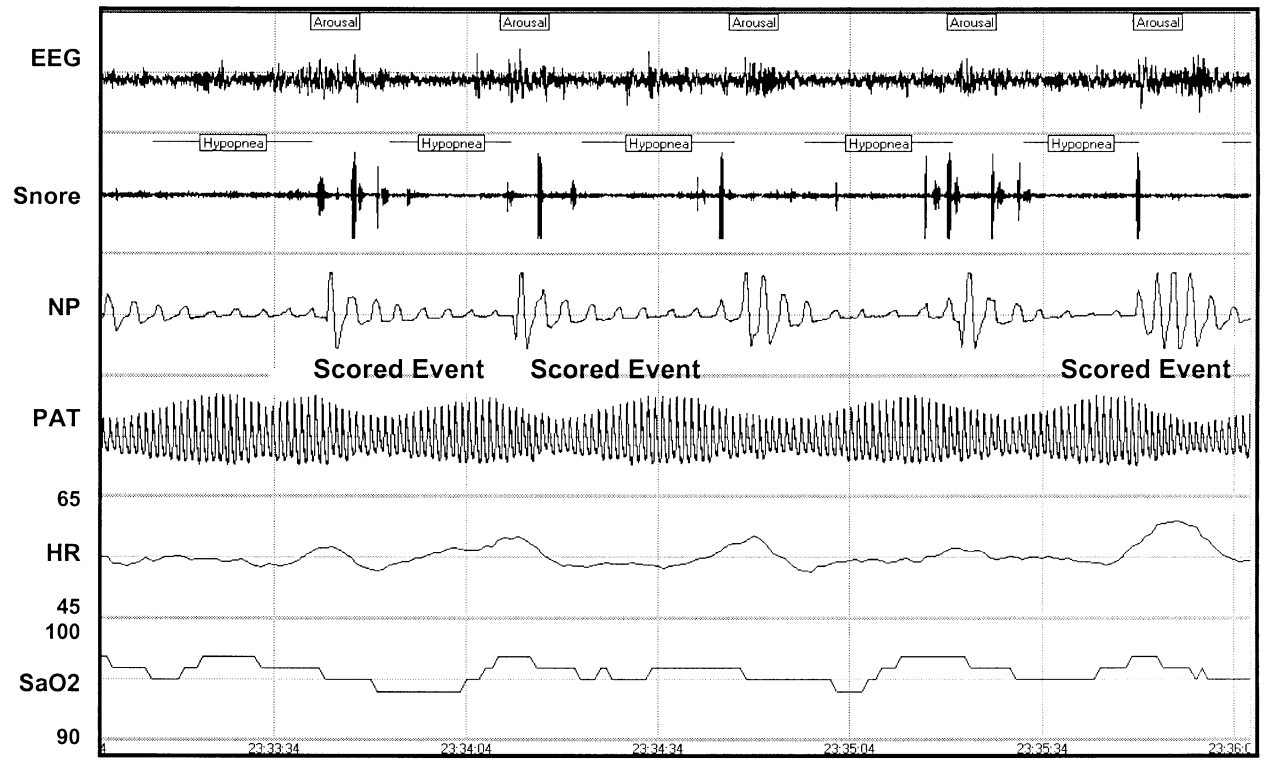

Fig. 7. This represents a three-minute sample from a PSG of a patient with severe sleep apnea. Five hypopneas are shown (events indicated by the word 'Hypopnea' in boxes). However, only three of these events were detected by the Watch_PAT ('Scored Event') due to the high frequency of the events. NP, nasal pressure; HR, heart rate; SaO_2, arterial oxygen saturation.

with other disorders of sleep that lead to substantial sleep fragmentation and arousals.

In contrast, as PSG AHI increased, Watch_PAT tended to underestimate AHI. Using our regression equation, a PSG AHI of 30 would correlate with a PAT AHI of 28.7 and a PSG AHI of 60 would result in a PAT AHI of 51.8. This resulted, in part, because the Watch_PAT had more difficulty detecting each individual event when such multiple events occurred over a brief time period. Under these conditions, there is insufficient time for the PAT signal to return to baseline; consequently, only one event is detected even though multiple events occurred. An example is shown in Fig. 7 (patient number 18) where PAT AHI was 69, but PSG AHI was 94.

There are a number of limitations to our study. First, accuracy of the device may be affected if another disorder yields substantial sleep disruption. Improved analysis algorithms may eventually resolve this, but at the current time the presence of non-apneic disorders yielding sleep fragmentation, such as periodic limb movements of sleep or restless insomnia, may compromise the accuracy of the device. Second, the device does not measure airflow and thus cannot differentiate hypopneas from apneas. This is likely of little clinical significance, as it is generally accepted that both apneas and hypopneas have similar clinical consequences. Indeed, severity of OSA is defined according to the sum of apneas and hypopneas rather than the frequency of solely apneas or hypopneas [8]. Third, although this device is designed to be used in an ambulatory setting, we assessed it only in the laboratory. It is conceivable that the device may not function as well at home as patients will need to place the device accurately on the fingers/wrist by themselves. Although we did not collect data concerning ease of use in our study, given the simplicity of the device, we speculate that most patients will be able to use it at home. Nevertheless, a study designed to specifically assess home use of the Watch_PAT should be conducted to verify that this is the case. Fourth, this study included a small number of subjects at only one sleep center. We cannot exclude the possibility that performance will be different in other locations. Thus, further studies of the device at other laboratories should be performed to address this issue. Finally, we did not perform an event-by-event analysis of Watch_PAT versus PSG AHI. This would have been difficult to accomplish as the two systems have different epoch lengths and time recorders. Thus, this was not attempted.

In this study, the Watch_PAT was useful in detecting OSA in patients in the sleep laboratory with a reasonable degree of sensitivity and specificity. Thus, the Watch_PAT could become a useful tool in diagnosing and assessing OSA in a variety of potential ways. First, it could be a convenient screening device for detecting OSA in high-risk populations such as morbidly obese patients and severe hypertensives [18]. Second, the Watch_PAT, in conjunction with home autotitrating CPAP technology, may eventually make complete home diagnosis and therapy of OSA a viable option [19]. Patients with suspected OSA could have their diagnosis verified with an ambulatory Watch_PAT device, followed by home determination of ideal CPAP pressure using an autotitrating device. Given that no laboratory time would be required, costs would likely be reduced. However, we do not believe the Watch_PAT will completely replace PSG. Because the device in not 100% sensitive, some patients with OSA may have a negative study. Therefore, in patients in whom OSA is highly suspected, a negative Watch_PAT should be followed by full PSG. In addition, one could consider a full sleep study in patients with a positive Watch_PAT in whom a non-respiratory disorder of sleep suspected.

Prior to widespread use of the device, further studies are needed. These include verification of accuracy and ease of use in an ambulatory setting, studies in other medical centers, and studies including more patients with non-respiratory causes of sleep fragmentation. Nevertheless, the Watch_PAT may become a useful technology to diagnose and manage patients with OSA.

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