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SCIENTIFIC INVESTIGATIONS

Impact of Arterial Stiffness on WatchPAT Variables in Patients With Obstructive Sleep Apnea

Taku Kinoshita, MD[1]; Misuzu Yahaba, MD, PhD[1]; Jiro Terada, MD, PhD[1]; Takuma Matsumura, MD[1]; Yoriko Sakurai, MD, PhD[1]; Kengo Nagashima, PhD[2]; Seiichiro Sakao, MD, PhD[1]; Koichiro Tatsumi, MD, PhD[1]

[1]Department of Respirology, Graduate School of Medicine, Chiba University, Chiba, Japan; [2]Department of Global Clinical Research, Graduate School of Medicine, Chiba University, Chiba, Japan

Study Objectives: The WatchPAT is a wrist-worn portable device that creates integration data regarding peripheral arterial tone (PAT), oxyhemoglobin saturation, heart rate, and actigraphy to diagnose or screen for obstructive sleep apnea (OSA). Previous studies have demonstrated the efficacy and validity of respiratory variables measured by the WatchPAT compared to those using polysomnography (PSG). However, the effects of arterial stiffness or atherosclerosis on WatchPAT parameters remain to be elucidated.

Methods: Sixty-one consecutive patients with suspected OSA who underwent home-based testing with the WatchPAT 200, standard in-laboratory overnight polysomnography (PSG), and pulse wave velocity (PWV) as an index of arterial stiffness were studied. All PSG recordings were scored manually using the American Academy of Sleep Medicine criteria, whereas WatchPAT data were analyzed by an automatic algorithm. We evaluated how arterial stiffness affected respiratory event index data in WatchPAT (WP-AHI), because WP-AHI could be partly influenced by PAT, comparing WP-AHI and the apnea-hypopnea index measured by PSG (PSG-AHI) in consideration of PWV result.

Results: Overall, WP-AHI was moderately correlated to PSG-AHI, but WP-AHI was significantly lower than PSG-AHI (28.4 ± 19.2 versus 53.6 ± 30.2 events/h, $P < .0001$). For the lower PWV group, there was a significant correlation and good agreement between the WP-AHI and PSG-AHI, but as the PWV increased, there was low correlation between the WP-AHI and PSG-AHI.

Conclusions: Arterial stiffness may affect the respiratory variables measured by WatchPAT in patients with OSA.

Commentary: A commentary on this article appears in this issue on page 301.

Keywords: apnea-hypopnea index, arterial stiffness, obstructive sleep apnea, pulse wave velocity, WatchPAT

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Although the current gold standard for diagnosing obstructive sleep apnea (OSA) is overnight polysomnography, the procedure is expensive and time consuming to administer. The WatchPAT is a portable device for which multiple studies have demonstrated good correlation with full polysomnography; however, the effects of arterial stiffness on WatchPAT variables still need to be elucidated.

Study Impact: This study demonstrates that arterial stiffness may affect the respiratory index measured by the WatchPAT in patients with OSA. Therefore, we believe that greater care should be taken when using the WatchPAT to diagnose OSA, especially in patients with severe arterial stiffness.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by repetitive complete or partial obstruction of the upper airway during sleep. The diagnosis and treatment of OSA are critical to avoid the potential risks of fatal cerebrovascular or cardiovascular events, and death from causes mainly related to the atherosclerotic process.[1,2] However, at least 80% of moderate-to-severe OSA remains undiagnosed in middle-aged adults.[3] The current gold standard for diagnosing OSA is in-laboratory overnight polysomnography (PSG),[4] but despite providing the most accurate description of sleep disorders, it is limited by high costs, long waiting lists in some areas, and the requirement for overnight assessment. Therefore, alternative approaches are required.

The WatchPAT is a wrist-worn ambulatory device that is used to diagnose or screen for OSA, and is categorized as a type 3 monitoring device by the American Academy of Sleep Medicine (AASM).[5] Multiple studies have demonstrated that the respiratory parameters calculated using the WatchPAT correlates well with that calculated by full PSG.[6–8] In Japan, the WatchPAT has mainly been used to screen for OSA. The variables calculated by the WatchPAT algorithm are based on peripheral arterial tone (PAT), heart rate, oxyhemoglobin saturation, and actigraphy,[9] allowing indirect detection of apnea and hypopnea events through PAT signal attenuation, heart rate increase, and desaturation.[6,10,11] Given that the PAT measurement is based on a physiological signal reflecting changes in vascular volume in the digit using the finger-mounted plethysmograph,[9] an impaired peripheral vascular response might

affect the respiratory variables measured by the WatchPAT. Indeed, patients using α-adrenergic receptor-blocking agents cannot be accurately evaluated by WatchPAT, because such agents are known to affect the PAT signal.[12,13] The role of more common conditions, such as arterial stiffness or atherosclerosis, has not been adequately considered in the literature.

Brachial-ankle pulse wave velocity (baPWV) is a widely used modality for evaluating arterial stiffness.[14] The baPWV can be noninvasively and indirectly assessed by calculating the time that it takes a pulse wave to travel along the vasculature between the brachium and ankle. Previous research has shown that baPWV is an independent predictor of the presence of coronary artery disease, myocardial injury, and future cardiovascular events.[15,16] Therefore, we hypothesized that arterial stiffness (ie, a high pulse wave velocity [PWV]) would attenuate the accuracy of the respiratory variables measured by WatchPAT. In the current study, we investigated the effect of arterial stiffness, as measured by baPWV, on WatchPAT variables in patients with OSA.

METHODS

Study Design

We enrolled adult patients with suspected OSA in whom the diagnosis was made at Chiba University Hospital between April 2015 and March 2016. Registrations were consecutively conducted at each patients' first visit at the Sleep Disordered Breathing Clinics in our hospital (if consent to participating in our study were obtained). Patients with suspected other sleep conditions (ie, nongeneral OSA such as heart failure with central sleep apnea, narcolepsy, or obvious neuromuscular disease) were not registered. Exclusion criteria for the study were history of permanent pacemaker, nonsinus cardiac arrhythmias, and use of α-adrenergic receptor-blocking agents or short-acting nitrates (24-hour washout period required). Before the study, patients completed a questionnaire concerning daytime sleepiness (Epworth Sleepiness Scale), medical history, and physical data (eg, weight and height). Patients first underwent home-based portable monitoring using a WatchPAT 200 (Itamar Medical Ltd., Caesarea, Israel) before visiting the hospital for standard in-laboratory full overnight PSG (mean duration between WatchPAT and PSG was 39.1 ± 26.5 days). On the same day as the patient underwent PSG, the baPWV was measured. Informed consent was obtained from all participants after the protocol was approved by the Institutional Review Board of Chiba University Hospital.

Home-Based Testing by WatchPAT

For home study, patients received instructions on how to use the WatchPAT device. These instructions addressed proper application of the device and were accompanied by a demonstration of correct use, and lasted 5 to 10 minutes. The WatchPAT is a four-channel, unattended, ambulatory monitoring device that records PAT signals, heart rate, oxygen saturation, and actigraphy. The WatchPAT calculates clinical parameters such as respiratory event index and 4% oxygen desaturation index (ODI) by its automated and proprietary algorithm (analyzed data reports are automatically made in the default setting). This device is worn around the wrist and has one finger probe that contains a PAT sensor for measuring PAT changes in peripheral arterial beds, a heart rate sensor, and an oximetry sensor for measuring arterial oxygen saturation. The wrist unit contains the actigraphy sensor for measuring wrist movements through a three-dimensional accelerometer. Using the WatchPAT software (ZZZ PAT version 4.4.64.p, Itamar Medical Ltd., Caesarea, Israel), the resulting data were automatically analyzed to identify respiratory events and sleep states. The automated analysis used actigraphy to identify wake-sleep states. Respiratory events were detected as follows: (1) termination of respiratory disturbances led to a surge of sympathetic activity that influenced peripheral arterial vasoconstriction; (2) vasoconstriction of the peripheral arterial bed by alpha receptors resulted in an attenuation of PAT signal; and (3) PAT signal attenuation, increased heart rate, and oxygen desaturation were analyzed by the automatic computerized algorithm of the WatchPAT system.[5] Manual editing of automated scoring is possible but was avoided in the current study to allow the assessment of only algorithm performance rather than that of the algorithm plus human operator. In addition, a previous study demonstrated the validation of automated scoring.[5,7]

In-Laboratory Testing by Polysomnography

All patients underwent standard, in-laboratory, full overnight PSG, using an E Series monitor (Compumedics, Victoria, Australia). The following were recorded: electroencephalography (EEG; C4-M1, O2-M1, C3-M2, O1-M2), electrooculography, submental and bilateral anterior tibial electromyography, electrocardiography, respiratory effort by thoracoabdominal piezoelectric belts, nasal airflow by nasal pressure cannula, nasal and oral flow by a thermistor, finger pulse oximetry, snoring recording by a neck microphone, and assessment of body posture by a thoracic belt sensor. The 2007 American Academy of Sleep Medicine alternative criteria was used to score the PSG.[8] Apnea was defined as a reduction in nasal airflow to < 10% of the baseline for ≥ 10 seconds. Hypopnea was defined as a reduction in nasal airflow signal amplitude of ≥ 50% for ≥ 10 seconds in association with either an oxygen desaturation of ≥ 3% or arousal on EEG. OSA was defined as an apnea-hypopnea index (AHI) of ≥ 5 events/h, combined with predominantly obstructive respiratory events. OSA severity was classified according to the AHI, as follows: mild, 5–15; moderate, 15–30; and severe, ≥ 30 events/h. All PSG tests were scored by a single technician.

Brachial–Ankle Pulse Wave Velocity

The baPWV was measured in the supine position, using an automated device (Colin form; OMRON COLIN Co., Ltd. Tokyo, Japan) that measures arterial pulse waves in both the brachial and posterior tibial arteries by the oscillometric method.[14] The pressure waveforms of the brachial and tibial arteries were obtained from occlusion and monitoring cuffs around the upper arm and the ankle. The transmission distance between the brachium and ankle was calculated automatically based on the height of the patient.[17] The average of the left and right values was used for analysis. Several studies have reported the cutoff value of baPWV for cardiovascular disease. Maeda et al.

compared 3,628 outpatients with diabetes and showed baPWV of 1400 cm/s as statistically adequate cutoff points for cardiovascular events and mortality.[18] Kim et al. reported baPWV > 1700 cm/s was strongly associated with the presence and severity of cardiovascular disease.[15] Based on these studies, we decided upon a baPWV threshold from 1300 cm/s to 1800 cm/s.

Data and Statistical Analysis

The following data were derived from in-laboratory PSG testing: total sleep time, rapid eye movement (REM) sleep, deep non-REM (NREM) sleep, 4% ODI, and AHI (PSG-AHI). The following data were derived from the home WatchPAT testing: total sleep time, REM sleep, deep NREM sleep, 4% ODI, and respiratory event index (WP-AHI). Demographic variables are presented as mean ± standard deviation, unless otherwise stated. Differences between the PSG and WatchPAT measurements were compared by paired *t* tests. Bland-Altman plots were used to determine the agreement between PSG and WatchPAT measurements in the study groups. Receiver operator characteristic (ROC) curves were constructed to illustrate the diagnostic performance of the WP-AHI, which was compared to the reference standard (ie, PSG-AHI ≥ 30 events/h for a positive result, and PSG-AHI < 30 events/h for a negative result). The area under the ROC curves, and the sensitivity and specificity for WP-AHI cutoff values were calculated. Pearson correlation coefficients between the PSG-AHI and WP-AHI were calculated; subgroup analyses according to different thresholds of the PWV values (ie, 1300, 1350, …, 1750, and 1800 cm/s) were also performed to explore the effect of arterial stiffness. Values of $P < .05$ by two-tailed tests were considered to denote statistical significance. All statistical analyses were done using JMP Pro Version 12.0 (SAS Institute, Cary, North Carolina, United States).

RESULTS

Participant Characteristics

Characteristics of the study population are shown in **Table 1**. A total of 61 patients (48 males, 13 females) were studied, for whom the average age was 57.1 ± 13.5 years and the body mass index was 26.5 ± 4.4 kg/m^2. Fifty-four patients (88.5%) had at least one comorbidity other than obesity, with the greatest percentages having hypertension (49.2%), diabetes mellitus (24.6%), or dyslipidemia (19.7%). All the patients recruited had OSA, with mild OSA in 6.6%, moderate OSA in 16.2%, and severe OSA in 77.2%.

Comparison of Sleep and Breathing Variables Between PSG and WatchPAT

The comparison of sleep and respiratory variables between PSG and WatchPAT are shown in **Table 2**. The WP-AHI was significantly lower than the PSG-AHI. Total sleep time, deep NREM sleep, and REM sleep were significantly higher in WatchPAT than in PSG. However, there was no significant difference in the 4% ODI between the WatchPAT and PSG.

The Bland-Altman plot illustrated that as PSG-AHI increased, the WatchPAT tended to underestimate the respiratory event index, indicating that proportional bias existed in respiratory indices between WatchPAT and PSG, as shown in **Figure 1**. ROC curves for respiratory event indices are shown in **Figure 2**. The area under the curve for PSG-AHI ≥ 30 events/h was 0.84 (95% confidence interval [CI]: 0.73–0.95, $P = .0008$). When diagnosis of OSA was defined to WP-AHI cutoff values of 18.6 events/h, the sensitivity and specificity for the WatchPAT to identify PSG-AHI ≥ 30 events/h were 0.79 (95% CI: 0.64–0.89) and 0.80 (95% CI: 0.44–0.97), respectively. As shown in **Figure 3**, there was a moderate correlation for the respiratory event indices between WatchPAT and PSG ($r = .69$, $P < .0001$).

Table 1—Characteristics of study population (n = 61).

Age (years)	57.1 ± 13.5
Sex (M/F)	48/13
BMI (kg/m^2)	26.5 ± 4.4
Height (cm)	166.5 ± 8.6
Weight (kg)	72.5 ± 16.1
ESS score	6.2 ± 4.6
PSQI score	11 ± 2.9
Hypertension, n (%)	30 (49.2)
Arrhythmia, n (%)	2 (3.7)
Ischemic heart disease, n (%)	2 (3.7)
Diabetes mellitus, n (%)	12 (19.7)
Dyslipidemia, n (%)	49 (80.3)
Obesity, n (%)	39 (63.9)

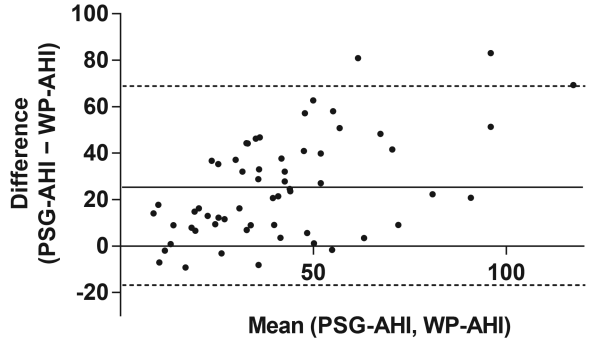
Data are presented as the mean ± standard deviation for numerical data and n (%) for categorical data. BMI = body mass index, ESS = Epworth Sleepiness Scale, PSQI = Pittsburgh Sleep Quality Index.

Table 2—Sleep and breathing characteristics comparing polysomnography and WatchPAT (n = 61).

	WatchPAT	PSG	*P*
TST (minutes)	403 ± 87 (175–572)	338 ± 91 (134–502)	< .0001
Deep NREM sleep (% TST)	3.9 ± 6.5 (0.9–28.6)	6.4 ± 6.8 (0.0–29.3)	< .0001
REM sleep (% TST)	24.2 ± 8.9 (4.0–41.9)	16.2 ± 7.9 (0–36.7)	< .0001
4% ODI (events/h)	20.9 ± 17.7 (0–70.1)	23.8 ± 20.7 (0.1–90.9)	.13
Respiratory indices (events/h)	WP-AHI: 28.4 ± 19.2 (1.0–82.7)	PSG-AHI: 53.6 ± 30.2 (6.5–152.0)	< .0001

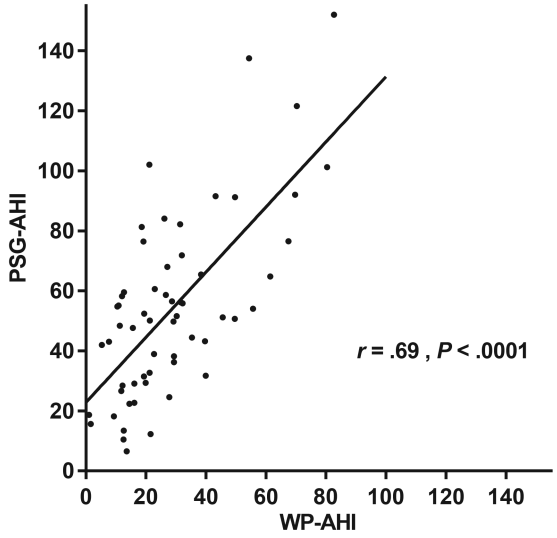
Data are presented as the mean ± standard deviation (minimum–maximum). 4% ODI = 4% oxygen desaturation index, AHI = apnea-hypopnea index, NREM = non-rapid eye movement, PSG = polysomnography, PSG-AHI = apnea-hypopnea index as measured by PSG, REM = rapid eye movement, TST = total sleep time, WP-AHI = respiratory event index as measured by WatchPAT.

Figure 1—Bland-Altman plot of respiratory event indices.



Differences were calculated as the PSG-AHI minus WP-AHI. Means for the PSG and WatchPAT findings are on the x-axis, and differences are on the y-axis. Solid lines on the y-axis are drawn from 0, mean difference. Dotted lines on the y-axis are drawn from mean ± 1.96 × standard deviation of the differences. PSG = polysomnography, PSG-AHI = apnea-hypopnea index as measured by PSG, WP-AHI = respiratory event index as measured by WatchPAT.

Figure 3—Scatterplot of PSG-AHI by WP-AHI.

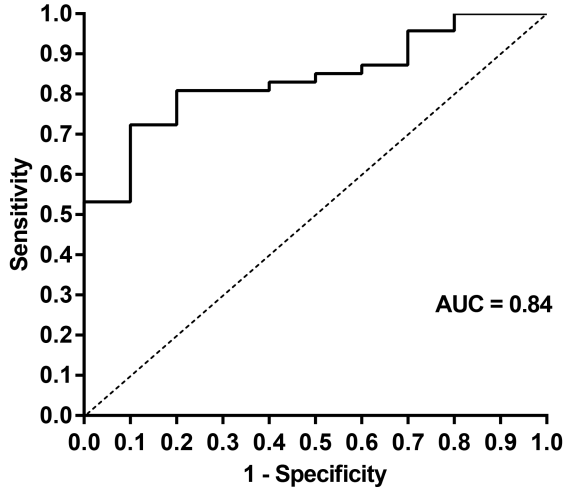


A moderate correlation was found (r = .69, P < .0001, n = 61) between PSG-AHI and WP-AHI. PSG-AHI = apnea-hypopnea index as measured by polysomnography, WP-AHI = respiratory event index as measured by WatchPAT.

The Effect of Arterial Stiffness on the Relationship Between WatchPAT and PSG

To evaluate the effect of arterial stiffness, we compared the correlations between WP-AHI and PSG-AHI groups with PWV results lower and higher than the selected PWV (range 1300–1800 cm/s), as shown in **Table 3**. In patients with lower PWV values than each cutoff value (from 1300 to 1800 cm/s), there were very high correlations for the respiratory event indices between the WatchPAT and the PSG groups. Among them, patients with a PWV under 1300 cm/s has the highest correlation between PSG-AHI and WP-AHI (r = .811, P = .0001). As the PWV cutoff value increased, the correlation became lower. Conversely, patients with a PWV over 1550 cm/s have no statistical correlation for the respiratory event indices between the WatchPAT and the PSG. **Figure 4** shows the representative difference classified by a PWV of 1500 cm/s (lower; n = 33, r = .78, P < .0001 higher; n = 27, r = .40, P = .04).

Figure 2—ROC curve identifying pathologic apnea and hypopnea events from sleep for the WatchPAT and PSG.



The comparison threshold was set at PSG-AHI ≥ 30 events/h on PSG. PSG = polysomnography, PSG-AHI = apnea-hypopnea index as measured by PSG, ROC = receiver operating characteristic.

Table 3—Correlation coefficients between PSG-AHI and WP-AHI according to different thresholds of the PWV values.

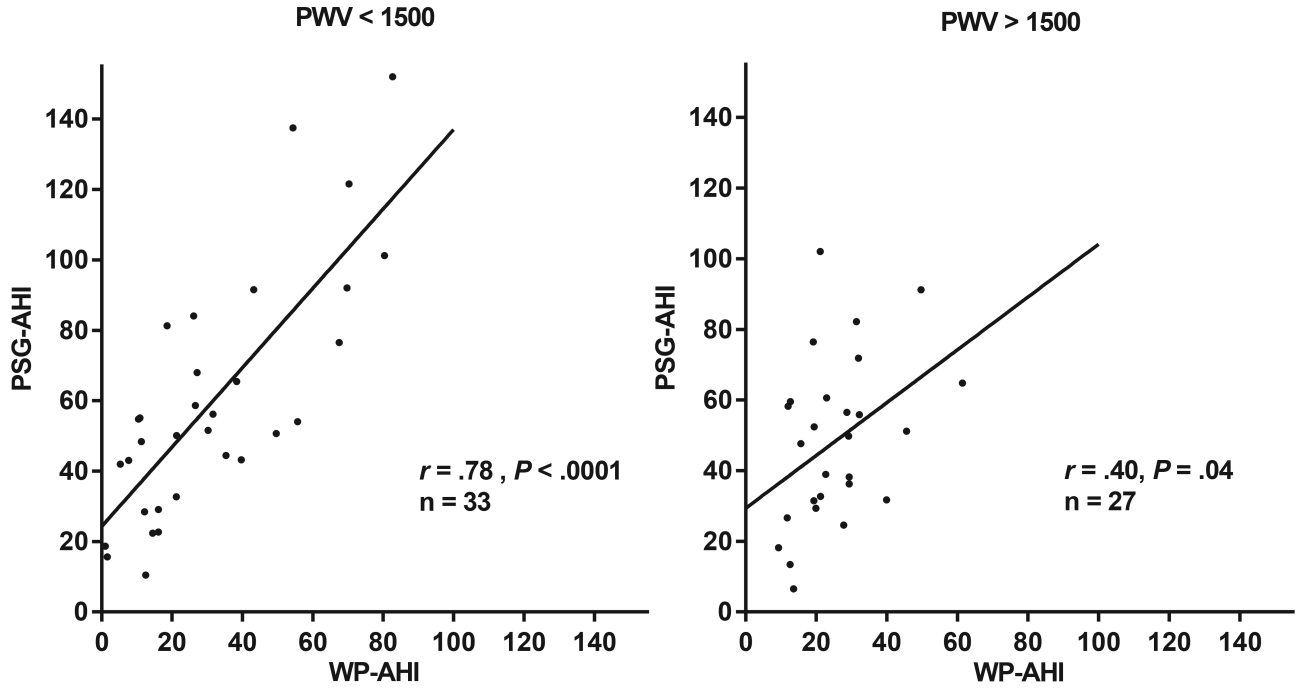
PWV Threshold (cm/s)	Lower Than Threshold		Higher Than Threshold	
	r	*P*	*r*	*P*
1300	.811	.0001	.652	< .0001
1350	.759	.0002	.675	< .0001
1400	.710	.0002	.697	< .0001
1450	.780	< .0001	.523	.0022
1500	.782	< .0001	.397	.040
1550	.769	< .0001	.392	.053
1600	.745	< .0001	.496	.026
1650	.745	< .0001	.460	.055
1700	.749	< .0001	.368	.18
1750	.733	< .0001	.431	.21
1800	.719	< .0001	.545	.21

PSG-AHI = apnea-hypopnea index as measured by polysomnography, PWV = pulse wave velocity, WP-AHI = respiratory event index as measured by WatchPAT.

DISCUSSION

This is the first study, to our knowledge, to have used baPWV to evaluate how arterial stiffness affects the respiratory variables measured by WatchPAT in patients with OSA. We showed that

Figure 4—Scatterplots of PSG-AHI by WP-AHI based on the PWV threshold of 1500 cm/s.



Data are shown for patients with a PWV under 1500 cm/s **(A)** and a PWV over 1500cm/s **(B)**. The Pearson correlation coefficients were .78 ($P < .0001$) and .40 ($P < .04$), respectively. PSG-AHI = apnea-hypopnea index as measured by polysomnography, PWV = pulse wave velocity, WP-AHI = respiratory event index as measured by WatchPAT.

overall correlation and agreement were good between WP-AHI and PSG-AHI, and that this correlation was strongest among patients with low arterial stiffness. By contrast, there was low correlation between WP-AHI and PSG-AHI among patients with high arterial stiffness. These results indicate that arterial stiffness should be considered, especially in patients suspected of arterial stiffness, when evaluating by WatchPAT. The mean WP-AHI was also lower than the mean PSG-AHI in this study, suggesting that the WatchPAT may underestimate the respiratory event index, partially because of the effect of arterial stiffness.

The Effect of Arterial Stiffness Measured by PWV on WatchPAT Variables

In the current study, the WP-AHI in the high PWV group had worse correlation with PSG-AHI than that in the lower group. This indicated that reduction in the extensibility of the peripheral arteries due to increased stiffness was related to the estimated number of apnea and hypopnea events analyzed by the WatchPAT. Arterial stiffness reflects physiological changes in the mechanical and structural properties of the vascular wall that lead to reduced arterial compliance. In turn, variation in arterial stiffness has been correlated with cardiovascular outcomes, and is an emerging prognostic factor with hypertension, diabetes, obesity, and dyslipidemia.[19] Given that the PAT is a major component variable in assessments using the WatchPAT, it was anticipated that arterial stiffness would affect the related respiratory index, especially for patients in clinical settings (eg, diversity of medications, complications, age, and OSA severity). Most previous studies of the WatchPAT have excluded patients with diabetes mellitus, peripheral neuropathy, vasculopathy, or cardiac disease because these diseases may theoretically affect peripheral blood flow.[7] Few studies have focused on the effect of arterial impairment on results from the WatchPAT. Aging is also associated with impaired peripheral vascular tone and can affect the WatchPAT data, but Onder et al. demonstrated that aging did not negatively affect the PAT-recorded respiratory index.[20] However, they did show that there was a significant difference between young and old groups in PAT- and PSG-recorded stage N3 sleep.[20] This finding may be attributed to aging and impaired vascular tone.

WP-AHI Compared With PSG-AHI

We also showed that the mean WP-AHI was lower than the mean PSG-AHI, even though most studies that validated the WatchPAT demonstrated good agreement with PSG for the respiratory event index. This discordance can be explained by the differences in study populations. The Brand-Altman plot illustrated that as PSG-AHI increased, the WatchPAT tended to underestimate the respiratory event index. Thus, consistent with research by other authors,[6,21] we observed proportional bias when using the WatchPAT in this study. This may be explained, in part, by the fact that the WatchPAT had more difficulty detecting individual events when multiple events occurred over a brief period.[21] It was also notable that our research included patients with severe OSA (mean AHI 53.6 events/h,

and severe OSA in 77%), whereas most previous studies have tended to include patients with mild-to-moderate OSA. This important difference in the study population may also have caused the results to be underestimated. Given that most symptomatic patients who consult their doctor have severe OSA (eg, daytime sleepiness or severe snoring), it is important to know that the WatchPAT might underestimate the respiratory variables in these patients.

Clinical Implications

The WatchPAT is a simple and patient-friendly portable device for assessing suspected OSA, providing suitably accurate assessment of respiratory disturbance during sleep with minimal technical effort and low cost. The device has been widely used in clinical settings and trials already (eg, screening in certain diseases and occupations, determination of treatment effect).[10,22–24] However, our results demonstrated a note of caution that arterial stiffness may affect the respiratory index measured by the WatchPAT, and that it might be necessary to consider the grade of arterial stiffness when assessing test results using this device. This is especially important because, compared with earlier studies, our population was closer to that typically seen in clinical settings. Indeed, we included patients with more severe OSA and with several comorbidities, including hypertension, diabetes mellitus, and dyslipidemia, whereas previous studies of the WatchPAT have mainly included patients with mild-to-moderate OSA and no comorbidities.[7] In real-word clinical situations, we therefore think that the WatchPAT might underestimate the respiratory event index when compared with PSG. Given this, clinicians might need to assess the merits of the WatchPAT device over full PSG before performing an assessment for patients with OSA using WatchPAT.

Limitations

There are several limitations to our study. First, this study included a small number of patients with OSA and it was conducted at a single hospital. Second, there is a possibility of night-to-night variation because the WatchPAT and PSG assessments were not performed on the same nights (the duration between WatchPAT and PSG was 39.1 ± 26.5 days). This may affect our results to some extent, whereas our study originally intended to evaluate the efficacy and accuracy of home-based WatchPAT in practical use in actual clinical situations (ie, a portable modality for use in screening or for the diagnosis of in-home situations) compared to that of in-laboratory PSG. However, other studies have demonstrated good correlation for respiratory disturbance between the WatchPAT and PSG when recorded on different nights, so we do not think that this will have had a major influence on the relationship between the WP-AHI and PSG-AHI.[5,25] Third, in our study, we found that total sleep time was significantly longer in WatchPAT than in PSG. Possible reasons include the following: (1) because WatchPAT does not monitor EEG, total sleep time may have been underestimated in our subjects, and (2) the first-night effect during in-hospital PSG testing may decrease the total sleep time (versus home testing). Additionally, regarding longer deep sleep time in WatchPAT than that in PSG, O'Brien et al. showed poor relationships in sleep stage characteristics between WatchPAT and PSG; in particular, agreement for deep sleep was not well achieved.[22] In the current study, it remains uncertain whether WatchPAT underestimated deep sleep time. Fourth, it should be noted that the baPWV mainly represents large arteries, whereas WatchPAT represents peripheral arteries. Nevertheless, baPWV is known to be partially influenced by peripheral arteries.[26] Activation of α-sympathetic nerves, which is essential for detecting the respiratory index when using the WatchPAT, has been demonstrated to reduce forearm vascular and fingertip vascular resistance when using α-adrenergic agonist.[13] Therefore, we consider it reasonable to use baPWV to assess arterial stiffness as a factor that can change fingertip blood flow. Finally, regarding body mass index and Epworth Sleepiness Scale score, the averages of our study were quite low (26.5 ± 4.4 kg/m^2 and 6.2 ± 4.6, respectively) compared to other general previous reports for OSA. We believe that this disagreement may be because of population difference (eg, East Asian versus Western countries) regardless of the presence of severe OSA. Therefore, some differences may exist between our study population and those of others.

CONCLUSIONS

In conclusion, we have shown that there was a strong correlation between WP-AHI and PSG-AHI as PWV values decreased, but that there was low or no correlation as PWV increased, indicating that arterial stiffness may affect the respiratory event index measured by the WatchPAT. The results of the current study suggest exercising caution when using the WatchPAT to diagnose OSA in patients with severe arterial stiffness.

ABBREVIATIONS

AASM, American Academy of Sleep Medicine
AHI, apnea-hypopnea index
baPWV, brachial-ankle pulse wave velocity
EEG, electroencephalography
ODI, oxygen desaturation index
OSA, obstructive sleep apnea
PAT, peripheral arterial tone
PSG, polysomnography
PSG-AHI, apnea-hypopnea index as measured by polysomnography
REM, rapid eye movement
WP-AHI, respiratory event index as measured by WatchPAT

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Address correspondence to: Corresponding author: Jiro Terada, MD, PhD, Department of Respirology, Graduate School of Medicine, Chiba University, 1-8-1 Inohana, chuo-ku, Chiba City, Chiba, Japan; Tel: +81-43-222-7171; Fax: +81-43-226-2176; Email: jirotera@chiba-u.jp

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