ORIGINAL ARTICLE

Development and clinical application of a new technique for detecting ‘sleep blood pressure surges’ in sleep apnea patients based on a variable desaturation threshold

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Obstructive sleep apnea (OSA) places an enormous pressure load on the cardiovascular system by inducing a temporary blood pressure (BP) surge (sleep BP surge (SLBPS)), often resulting in target organ damage and cardiovascular events, such as left ventricular hypertrophy, sudden death, myocardial infarction and stroke. Accurate measurement of SLBPS would be valuable for the risk stratification of OSA patients. We developed a new oxygen-triggered BP monitoring system based on a variable SpO₂ threshold (VT algorithm) to selectively detect severe SLBPS, which are associated with morbidity, and evaluated its performance in comparison with a previous technique based on a fixed SpO₂ threshold (FT algorithm). In 23 OSA patients, the correlation between individual minimum SpO₂ values and SLBPS was not significant when the FT algorithm was used alone ($r=0.400$, $P=0.058$) but became significant ($r=0.725$, $P<0.0001$) when the VT algorithm was additionally used. In another 13 OSA patients, when the FT algorithm was eliminated from the FT+VT algorithm, the number of BP readings was drastically reduced (36 ± 22.7 vs. 61 ± 55.0 times, $P=0.004$) with a similar correlation between minimum SpO₂ and SLBPS. The correlation between the apnea hypopnea index and SLBPS was significant when measured with the present method, but not when assessed with ambulatory BP monitors (ABPM) simulation ($r=0.519$, $P=0.001$ vs. $r=0.149$, $P=0.385$). In conclusion, oxygen-triggered BP monitoring with a variable threshold is able to detect severe OSA-related BP surges more specifically and reduce the number of BP readings required during sleep compared with detection using a fixed threshold or the conventional ABPM method.

Hypertension Research (2011) 34, 922–928; doi:10.1038/hr.2011.52; published online 26 May 2011

Keywords: blood pressure; home blood pressure monitoring; nocturnal hypoxia; sleep apnea syndrome

INTRODUCTION

Obstructive sleep apnea (OSA) places an enormous pressure load on the cardiovascular system by inducing a temporary blood pressure (BP) surge (sleep BP surge (SLBPS)) because of the desaturation of blood oxygen (O₂ desaturation) during apnea episodes^{1,2} as well as negative intrathoracic pressure.^{3,4} This pressure load is regarded as a potential mechanism of left ventricular hypertrophy⁵ and to trigger cardiovascular diseases,^{6–8} such as sudden death,^{5,9} myocardial infarction¹⁰ and stroke.¹¹ As BP surge and BP variability are considerably associated with baroreceptor reflex function and sympathetic nervous activation,^{1,12} the degree of SLBPS may differ among patients, even when they show similar degrees of O₂ desaturation.¹³ In addition to the assessment of OSA severity using the apnea hypopnea index (AHI), the assessment of SLBPS is valuable for the risk stratification of OSA patients and predicting cardiovascular diseases.

Ambulatory BP monitors (ABPM), which are used to monitor nocturnal BP during sleep, measure BP at fixed intervals without synchronization with sleep apnea (SA) episodes. Consequently, they often fail to detect SA-related SLBPS and can not discriminate SA-related SLBPS from diurnal BP variations.

To resolve these problems of ABPM, we developed an oxygen-triggered BP monitor based on a combination of a home BP monitor and a pulse oximeter (Ox-triggered BP monitoring), which detects SA-related SLBPS using oxygen desaturation signals (an SpO₂ fall below a fixed threshold (FT) algorithm).¹⁴ However, we faced the following difficulties: first, in severe OSA patients, BP measurements were triggered so frequently that the sleep of the patient was considerably disturbed. Second, despite this high frequency of BP measurement, FT algorithm-based Ox-triggered BP monitoring sometimes failed to detect marked SLBPS during more severe SA episodes because of the introduction of a period in which BP measurement was

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Received 16 November 2010; revised 15 February 2011; accepted 17 February 2011; published online 26 May 2011

prohibited, which was enforced following previous minor SA episodes to reduce the number of BP measurements.

Thus, we developed a new Ox-triggered BP monitoring technique based on an algorithm with a variable SpO₂ threshold (VT algorithm), which detects marked SLBPS induced by severe SA episodes and reduces the incidence of successive BP measurements during sleep, and evaluated the performance of this new monitoring system in comparison with conventional FT algorithm-based BP monitoring in SA patients.

METHODS

Oxygen-triggered BP monitoring system

As shown in Figure 1, the oxygen-triggered BP system consists of an OLV-3100 pulse oximeter (Nihon Kohden, Tokyo, Japan), a HEM-780 validated cuff-oscillometric BP monitor (Omron Healthcare, Kyoto, Japan), a personal computer, and an interface circuit that allows communication between the personal computer and the pulse oximeter or BP monitor. The pulse oximeter was set to obtain optical pulse wave signals (wavelengths: 660 and 940 nm) in the right index finger and to generate pulse oximetry (SpO₂) readings every 5 s. The cuff of the BP monitor was attached to the left upper arm of the patient. A software program continuously monitored SpO₂ and triggered a BP reading when the SpO₂ value fell below the threshold level, which was set automatically as described below. Once triggered, the BP monitor took three measurements of systolic BP, diastolic BP and pulse rate (PR) based on the cuff-oscillometric principle at intervals of 15 s. The software program stored the SpO₂, BP and PR readings, and the time when the measurement took place.

Design of the BP-measurement-triggering algorithm

In the previous version of this technique,¹⁴ BP measurement was triggered when the SpO₂ value fell below a fixed threshold, which was set at the SpO₂ value observed immediately before bedtime (baseline level) minus 10% (FT algorithm, Figure 2a). However, the fixed threshold was found to be too sensitive for some severe OSA patients, resulting in BP measurements being triggered too frequently. Although a lower threshold would prevent such frequent BP measurement in severe patients, it would lead to BP measurements not being triggered in mild OSA patients, who do not present with severe episodes. Therefore, the fixed threshold was set at a sensitive level, despite this causing frequent BP measurement in severe patients. To alleviate this problem, a period in which BP measurement was prohibited for 10 min after a previous measurement was introduced. However, this was found to disrupt the detection of marked SLBPS because severe SA episodes tended to occur during the prohibition period.

In this version of our system, we have introduced a variable threshold, which was initially set to the baseline value (SpO₂ value immediately before bedtime) minus 10%, and thereafter, was continuously renewed according to the current minimum SpO₂ value (VT algorithm, Figure 2b). Therefore, BP measurement was triggered when the SpO₂ value fell below the previous minimum SpO₂ value. However, if the most severe SA episode in a night occurred early in the night, no further BP measurements would be triggered, resulting in the BP profile not being recorded. To avoid such problem, the new algorithm is

Figure 1 Scheme of the oxygen-triggered blood pressure (BP) monitoring system.¹⁴

designed to vary the threshold level in proportion to the time from the last BP measurement (see the slope of the threshold level in Figure 2b).

The rate of change of the threshold (RCOT) was set to 10% per hour according to the results of computer simulations as described below. The simulation program used RCOT of 6, 10 or 20% per hour, and the actual data from nine OSA patients, that is, three mild, three moderate and three severe patients. The performance of the algorithm was evaluated in two studies.

First, apnea episodes in which BP readings should be performed (requisite episodes) and those that should be overlooked (holding episodes) were marked on the SpO₂ records of each of the nine patients by multiple researchers of the study group. The criteria for the 'required' episodes included being accompanied with the minimum SpO₂ in the night, or displaying similarly severe desaturation after a long time, for example, at 3 h after the last requisite episode. The criterion for the 'holding' episodes was episodes occurring at a short interval after the last requisite episode regardless of the degree of desaturation. A simulation program with RCOT of 6, 10 and 20% per hour was run separately over the 114 designated episodes (82 requisite and 32 holding episodes) in the nine patients. The success rate, which was defined as the percentage of correct actions, that is, the identification of the requisite episodes and the overlooking of the holding episodes was evaluated for each RCOT. A success rate of 95% or more was considered acceptable. The success rate for requisite episodes was 86.6, 95.1 and 100.0% for RCOT of 6, 10 and 20% per hour, respectively, and that for holding episodes was 100.0% regardless of RCOT. Thus, the performance of RCOT of 10 and 20% per hour were considered acceptable.

Second, we evaluated the number of BP readings per night. The SpO₂ records of the nine patients included many episodes. However, from a practical point of view, we considered that the number of episodes for which BP should be measured should be limited to 15 per night. In computer simulations with RCOT of 6, 10 and 20% per hour, the number of patients in which 15 or more episodes were selected for BP measurement was 0, 0 and 5 (56%), respectively. Thus, RCOT of 6% and 10% per hour were considered appropriate.

Therefore, an RCOT of 10% was adopted as the best setting for detecting SA episodes while inducing an acceptable number of BP measurements in most patients.

Figure 2 Comparison of blood pressure (BP) monitoring methodologies between the fixed threshold algorithm (upper panel) and the variable threshold algorithm. The closed circles indicate the timing of sleep apnea (SA) episodes detected with the fixed threshold algorithm or the variable threshold algorithm. The striped arrows indicate the BP measurement prohibition period, which was enforced for 10 min after the last BP reading to avoid successive BP readings in severe SA patients. The stars indicate overlooked severe SA episodes, which took place during the BP measurement prohibition period. The variable threshold was increased depending on the current minimum SpO₂ value in order to detect severe sleep apnea episodes and avoid successive BP readings, and was also increased in proportion to the time from the previous BP reading in order to detect less severe episodes that appeared after a long interval.

Clinical evaluation

The performance of the VT algorithm was compared with the FT algorithm and computer simulation of conventional ABPM (ABPM simulation), which was performed at the same time as the Ox-triggered BP monitoring, with regard to the following three points. The ABPM simulation measured BP at fixed intervals of 30 min.

First, the ability of each method to detect severe SLBPS was evaluated. As SLBPS involve a temporary BP elevation induced by O₂ desaturation, it was assumed that the magnitude of O₂ desaturation is negatively correlated with the degree of SLBPS. Therefore, we hypothesized that, if the VT algorithm was better at detecting severe SLBPS than the FT algorithm, it would show a better correlation between the magnitude of O₂ desaturation and the degree of SLBPS. In this study, SLBPS was defined as the mean of the three highest systolic BP values minus the mean of the three lowest systolic BP values during sleep in each individual. We also defined the minimum SpO₂ as the mean of the three lowest SpO₂ values during sleep in a given individual.

Second, we evaluated the correlation between SLBPS and AHI. We hypothesized that a system that is more able to specifically detect SLBPS induced by severe SA episodes would show a closer correlation between SLBPS and AHI. AHI is known to reflect advanced target organ damage and risk of cardiovascular events, although AHI may not be direct cardiovascular risk factor.

Third, we compared the number of BP measurements among the different methods.

Study patients

The study patients were outpatients in whom polysomnography (PSG) was planned for the diagnosis of SA. The clinical characteristics of the study patients

Table 1 Clinical characteristics of the study patients

Variable	Study 1 (n=23)	Study 2 (n=13)
Age (years)	57.5 ± 13.4	52.0 ± 12.6
Females (%)	8.7	7.7
Body mass index (kg m ⁻²)	28.6 ± 3.0	30.2 ± 7.3
Hypertension (%)	78.3	84.6
Hyperlipidemia (%)	34.8	23.1
Diabetes (%)	8.7	0.0
<i>Antihypertensive drugs</i>		
ACEI/ARB use (%)	60.9	69.2
CCB use (%)	56.5	69.2
Diuretic use (%)	30.4	46.2

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker.
Data are given as the mean ± s.d. or percentages.

are shown in Table 1. We excluded patients with respiratory disease, malignancies or with a previous history of cardiovascular disease. The study subjects were obese and frequently accompanied with medicated hypertension and hyperlipidemia.

Study protocol

Study 1. We evaluated the effect of adding the VT algorithm to the previously used FT algorithm in 23 OSA patients (Table 1). Specifically, the results produced using both the FT and VT algorithms (FT+VT algorithm) were compared with those produced with the FT algorithm alone. The subjects were randomly assigned to one of the two algorithm conditions, that is, the FT algorithm alone or the VT+FT algorithm, in a crossover design for 2 consecutive nights. This allocation was conducted by an independent study coordinating center (Division of Cardiovascular Medicine, Jichi Medical University of School of Medicine).

Study 2. We also evaluated the effect of eliminating the FT algorithm from the VT+FT algorithm in 13 OSA patients (Table 1). The subjects were randomly assigned to either the VT+FT algorithm or the VT algorithm alone in a crossover design for 2 consecutive nights. This allocation was also conducted by the abovementioned independent study coordinating center.

In both Study 1 and Study 2, in addition to Ox-triggered BP monitoring, PSG was simultaneously conducted for 2 consecutive nights. PSG measures, including SpO₂, electroencephalogram, electrocardiology, electromyography, electrooculography (L (left), R (right)), airflow, and thoracic and abdominal movement, were assessed using the PS2 Plus Sleep Watcher system (Compumedics, Melbourne, VIC, Australia). We calculated AHI as the mean number of apnea and hypopnea events per hour of sleep. Apnea was defined as the complete or almost complete cessation of airflow, and hypopnea was defined as a decrease in airflow or thoracoabdominal excursion of at least 50% of the baseline value for 10 s or longer, accompanied by a 3% or higher decrease in SpO₂. In both study groups, PSG indices (AHI, arousal index, apnea index, lowest SpO₂ and SpO₂ < 90%), systolic BP, diastolic BP and PR were not significantly different between the 2 consecutive nights. Patients with central SA were not included in either study. The baseline values for BP, PR and the PSG indices in Study 1 and Study 2 are shown in Table 2.

Values of $P < 0.05$ (two-tailed) were considered statistically significant. All statistical analyses were performed with the software package SPSS version 11.0J (SPSS, Chicago, IL, USA).

These studies were approved by the institutional research board of Washiya Hospital, and written informed consent was obtained from each participant.

RESULTS

Figure 3 shows representative results of Ox-triggered BP monitoring with the VT algorithm. As seen in the figure, BP measurement was selectively initiated during severe SA episodes that induced swift BP surges, whereas the ABPM simulation did not detect the BP variation that occurred in most episodes.

Table 2 Clinic blood pressure, pulse rate and polysomnography indices of the study patients

Variables	Study 1 (n=23)			Study 2 (n=13)		
	FT algorithm	FT+VT algorithm	P-value	FT+VT algorithm	VT algorithm	P-value
AHI	35.7 ± 18.3	36.5 ± 22.2	0.69	33.3 ± 19.9	32.2 ± 16.0	0.75
Apnea index	14.7 ± 12.6	15.2 ± 16.8	0.91	12.3 ± 12.6	14.8 ± 11.9	0.60
SpO ₂ < 90% (%)	8.9 ± 10.1	9.2 ± 11.9	0.93	15.9 ± 13.2	15.7 ± 15.2	0.97
Lowest SpO ₂ (%)	76.7 ± 7.2	78.0 ± 6.2	0.51	74.2 ± 6.3	75.1 ± 8.8	0.78
Arousal index	29.5 ± 15.4	23.3 ± 19.7	0.14	35.8 ± 14.2	36.8 ± 15.6	0.83
Systolic BP (mm Hg)	120.9 ± 12.1	122.7 ± 15.3	0.37	116.4 ± 13.9	118.6 ± 13.7	0.14
Diastolic BP (mm Hg)	74.3 ± 10.4	75.2 ± 9.8	0.57	69.1 ± 7.3	71.9 ± 9.2	0.07
PR (b.p.m.)	58.4 ± 6.7	58.5 ± 7.7	0.76	61.1 ± 9.8	62.1 ± 9.5	0.41

Abbreviations: AHI, apnea hypopnea index; DBP, diastolic blood pressure; FT, fixed threshold; PR, pulse rate; SBP, systolic blood pressure; VT, variable threshold.
Data are given as the mean ± s.d.

Figure 3 Representative case who exhibited sleep blood pressure surges that were detected by oxygen-triggered blood pressure monitoring with the variable threshold algorithm and with fixed interval monitoring (ABPM simulation). The lines with closed circles indicate systolic and diastolic blood pressure values measured by oxygen-triggered monitoring, the 'x' symbols mark pulse rate measured by oxygen-triggered monitoring, the line with open boxes indicates systolic blood pressure measured by the ABPM simulation (see left scale), and the line without symbols indicates SpO₂ (blood oxygen saturation level; see right scale).

Effect of the VT algorithm (Study 1)

In Study 1, as shown in Figure 4, the correlation between the minimum SpO₂ and SLBPS, which was not significant when the two values were measured with the FT algorithm ($r=0.400$, $P=0.058$, Figure 4a), was found to be significant when the FT+VT algorithm was used ($r=0.725$, $P<0.001$, Figure 4b). In addition, the slope of the regression line for the minimum SpO₂ value vs. SLBPS rose three-fold when the FT+VT algorithm was applied (-3.250 vs. -1.149), suggesting that the VT algorithm considerably improved the ability of the systems to detect SLBPS caused by severe SA episodes.

Also in Study 1, the correlation between AHI and SLBPS, which was not significant when the FT algorithm was applied ($r=0.231$, $P=0.289$, Figure 4c), became significant when the FT+VT algorithm was used ($r=0.444$, $P=0.034$, Figure 4d).

The number of BP readings increased slightly when the FT+VT algorithm was used, but the difference was not significant (77 ± 78.6 vs. 71 ± 66.7 times, $P=0.78$).

Effect of eliminating the FT algorithm (Study 2)

In Study 2, the correlation coefficient (r) between SpO₂ and SLBPS was 0.844 ($P<0.001$) when the FT+VT algorithm was used and 0.679 ($P=0.011$) when the VT algorithm was used (graphic not shown). Although the correlation coefficient was reduced slightly by eliminating the FT algorithm, the slope of the regression line between SpO₂ and SLBPS remained unchanged (-1.701 vs. -1.636).

The number of BP readings was markedly reduced by eliminating the FT algorithm (36 ± 22.7 vs. 61 ± 55.0 times, $P=0.004$, Figure 5). A reduction in the number of BP readings was seen in all subjects except two, who showed slight increases.

Comparison with ABPM simulation

Finally, we compared the results produced by Ox-triggered BP monitoring and ABPM simulation. We combined the data obtained with the FT+VT algorithm from studies 1 and 2 ($n=36$). As shown in Figure 6, the correlation between the minimum SpO₂ and SLBPS was significant for both Ox-triggered BP monitoring (Figure 6a) and ABPM simulation (Figure 6b). However, the correlation coefficient produced by Ox-triggered BP monitoring ($r=0.700$, $P<0.0001$) was markedly higher than that produced by ABPM simulation ($r=0.420$, $P=0.010$). In addition, the number of SLBPS of 55 mm Hg or higher

(pathological BP surge¹⁵) was significantly higher for Ox-triggered BP monitoring than ABPM simulation (8 (34.8%) vs. 4 (17.4%)), and the correlation between SLBPS and AHI was significant for Ox-triggered BP monitoring ($r=0.519$, $P=0.001$, Figure 6c) but not for ABPM simulation ($r=0.149$, $P=0.385$, Figure 6d).

There were no significant differences in the association between minimum SpO₂ and SLBPS or that between AHI and SLBPS among the patients taking the different classes of antihypertensives listed in Table 1 (data not shown).

DISCUSSION

In this study, we developed a new BP monitoring system based on an improved BP-measurement triggering algorithm, and evaluated its ability to specifically detect SLBPS induced by severe O₂ desaturation and to reduce the number of BP readings required in OSA patients, who were obese and were frequently accompanied with medicated hypertension and hyperlipidemia.

SA-induced SLBPS is caused by sympathetic nervous activation because of the reduction in blood oxygen saturation and an increase in blood CO₂.¹⁶ Therefore, the magnitude of O₂ desaturation and the degree of subsequent SLBPS are closely correlated. Thus, we measured the performance of this new BP triggering algorithm by assessing the degree of correlation between the minimum SpO₂ value (a measure of SA-triggered O₂ desaturation) and SLBPS (a measure of the O₂ desaturation-induced BP surge).

In Study 1, by combining the VT algorithm with the FT algorithm (FT+VT algorithm), the correlation between the minimum SpO₂ and SLBPS was strengthened without a significant increase in the number of BP measurements. The improvement in the correlation can be explained as follows. The FT algorithm often overlooked severe SA episodes, most of which followed previous less severe episodes, that took place during the BP measurement prohibition period invoked by the previous episode. In contrast, the VT algorithm was programmed to initiate BP measurement during the prohibition period when greater O₂ desaturation occurred. As a result, the VT algorithm had an improved chance of responding to severe SA episodes, which characterize severe OSA. The closer correlation between the minimum SpO₂ and SLBPS found by the FT+VT algorithm indicates that, in comparison with the FT algorithm, the FT+VT algorithm is able to more specifically detect BP surges induced by severe SA episodes.

Figure 4 Correlations between the minimum SpO₂ values (blood oxygen saturation level) and sleep blood pressure surge (SLBPS) detected with the fixed threshold (FT) algorithm alone (a) and with a combination of the FT algorithm and the variable threshold (VT) algorithm (b), and the correlations between the apnea hypopnea index and SLBPS (lower) produced with the FT algorithm alone (c) and with a combination of the FT algorithm and VT algorithm (d).

Figure 5 Comparison of the numbers of BP readings between those induced by a combination of the fixed threshold (FT) algorithm and the variable threshold (VT) algorithm (left) and those produced with the VT algorithm alone. The closed circles and lines indicate the mean $\pm$ s.d., respectively.

Importantly, despite taking some BP readings during the prohibition period, the FT+VT algorithm did not significantly increase the number of BP readings in Study 1, probably because the initiation of BP reading was limited to severe SA episodes, and BP measurement during less severe episodes was well controlled. In addition, in Study 2, excluding the FT algorithm markedly reduced the number of BP readings without significantly reducing the correlation coefficient between the minimum SpO₂ value and SLBPS compared with the results produced with the FT+VT algorithm. This suggests that the VT algorithm is able to detect severe SLBPS more specifically. The most likely reason for the large reduction in the number of BP readings in Study 2 was the absence of BP measurement during mild SA episodes. This explanation is supported by the finding that the reduction in the number of BP readings caused by the VT algorithm was more extensive in patients who showed a high number of BP readings when measured with the FT+VT algorithm. The reduction in the correlation coefficient between SpO₂ and SLBPS remained minimal even when the FT algorithm was excluded.

Different from chronically impaired autonomic nerve activity and renal dysfunction, which often lead to a diurnal profile with a 'stably' high BP (non-dipping profile), OSA causes hypoxia-induced BP surges (SLBPS) resulting in augmented short-term BP variability. Most OSA patients show a non-dipping profile; however, the degree of SLBPS is independent of dipper/non-dipper status and might provide additional information about risks in OSA patients. According to our hypothesis, ABPM with the fixed interval measurement

Figure 6 Correlation between the minimum SpO₂ value (blood oxygen saturation level) and sleep blood pressure surges (SLBPS) detected with Ox-triggered blood pressure monitoring (a) and that produced with fixed-interval blood pressure monitoring (b), and the correlations between the apnea hypopnea index and SLBPS detected with Ox-triggered blood pressure monitoring (c) and fixed-interval blood pressure monitoring (d).

may fail to detect SLBPS because it does not continuously measure BP. Meanwhile, Ox-triggered BP monitoring measures BP at the onset of apnea episodes, and therefore, its records BP changes because of SLBPS more precisely than ABPM. Figure 3 supports this hypothesis by demonstrating SLBPS detected by the Ox-triggered monitoring, most of which were overlooked by the ABPM simulation. More importantly, as well as oxygen saturation, the AHI, a conventional indicator of OSA severity, was also significantly correlated with the SLBPS detected with Ox-triggered BP monitoring, but not with those detected with ABPM simulation (Figure 6). This indicates that Ox-triggered BP monitoring is superior to conventional ABPM with fixed intervals for detecting SA-specific exaggerated SLBPS in OSA patients. This interpretation is supported by the finding that the number of pathological BP surges of ≥ 55 mm Hg¹⁶ detected by Ox-triggered BP monitoring was significantly higher than that found by conventional ABPM simulation.

Ox-triggered monitoring could be clinically useful in terms of providing additional information for cardiovascular risk stratification in OSA patients. In general, the severity of OSA has been evaluated with AHI. However, cardiovascular risk in OSA patients can not be fully predicted using AHI, because AHI is only an index of the frequency of apnea and hypopnea episodes. Meanwhile, high SLBPS can be interpreted as a consequence of not only frequent apnea episodes but also other cardiovascular load factors, such as disordered baroreceptor sensitivity and chemoreceptor sensitivity during apnea episodes. Thus, we presume that SLBPS is a more direct index of cardiovascular risk than AHI.

As described earlier, we hypothesized that, in comparison with ABPM, Ox-triggered BP monitoring is more capable of detecting SA-specific exaggerated SLBPS. In future, the efficacy of detecting SLBPS with the

present method should be tested in terms of its association with target organ damage and prognostic power. However, in this study, which focused on the performance of the new system, we intended to confirm the utility of Ox-triggered BP monitoring by comparing its outcomes with those with the ABPM, which is widely used to assess nocturnal hypertension. Specifically, we compared, the correlations between SLBPS and minimum SpO₂ (Figures 6a and b) and the correlation between SLBPS and AHI (Figures 6c and d) obtained using the two methods. Although the minimum SpO₂ and AHI may not be perfect indices of cardiovascular risk, they do at least partially reflect cardiovascular risk. Therefore, comparisons of their correlations with SLBPS in a given population are able to suggest the clinical potential of the present method compared with the conventional method.

Study limitations

Ox-triggered BP monitoring with different algorithms was performed on different nights in each subject, and so altered physiological conditions, such as the degree of SA and arousal, sleep time, BP and the PR level may have affected the outcomes. The study focused on evaluating the performance of the system to detect SLBPS and did not discuss the associations between its outcomes with current target organ damage and cardiovascular prognosis. Finally, the study did not assess the tolerance or comfort of the subjects.

In conclusion, oxygen-triggered BP monitoring with a variable threshold is able to detect severe apnea episode-related sleep BP surges more specifically and reduce the number of BP readings required during sleep compared with BP monitoring with a fixed threshold and conventional ABPM.

This new home BP monitoring system is compact and simple and could be used to assess SA-related sleep BP surges reproducibly over multiple nights in clinical practice.

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