Comparison of blood pressure and blood pressure changes in patients with positional and non-positional mild obstructive sleep apnea

Running Title: Positional mild OSA and blood pressure

Yi-Chih Huang, MD¹, Chun-Yao Lin, MD², Chou-Chin Lan, MD, PhD¹,³ Yao-Kuang Wu, MD¹,³, Chor-Shen Lim, MD¹, Chun-Yao Huang, MD¹, Hsuan-Li Huang, MD³,⁴, Kuan-Hung Yeh, MD³,⁴, Yu-Chih Liu, MD, PhD², Mei-Chen Yang, MD¹,³,*

¹ Division of Pulmonary Medicine, Department of Internal Medicine, Taipei Tzuchi Hospital, The Buddhist Tzuchi Medical Foundation.

² Divisions of Pulmonary, Critical Care and Sleep Medicine, Chang Gung Memorial Hospital at Keelung, Keelung City, Taiwan.

³ The School of Medicine, Tzu-Chi University, Hualien, Taiwan.

⁴ Division of Cardiology, Department of Internal Medicine, Taipei Tzuchi Hospital, The Buddhist Tzuchi Medical Foundation.

*Corresponding Author: Mei-Chen Yang, MD

Division of Pulmonary Medicine, Department of Internal Medicine, Taipei Tzuchi Hospital,
The Buddhist Tzuchi Medical Foundation, New Taipei City, Taiwan, and with the School of Medicine, Tzu-Chi University, Hualien, Taiwan.

No.289, Jianguo Rd., Xindian Dist., New Taipei City 23142, Taiwan

Tel: 886-2-66289779 Ext. 5709

Fax: 886-2-66289009

Email: mcyang37@yeah.net
ABSTRACT

**Background:** This retrospective study aimed to determine if there are differences in blood pressure (BP) between patients with positional-dependent and non-positional-dependent mild obstructive sleep apnea (OSA).

**Methods:** Patients who were referred for overnight polysomnography for suspected OSA between 2007 and 2011 were screened. A total of 70 patients with mild OSA were included and divided into two groups according to positional-dependency status of OSA: positional (n = 52) and non-positional (n = 18). The groups were compared for anthropometric and polysomnographic variables, presence of cardiovascular co-morbidities, morning and evening BP and the changes between evening and morning BP, and continuous positive airway pressure (CPAP) device usage.

**Results:** Demographic and anthropometric variables were similar; however, the prevalence of hypertension was significantly higher in the non-positional group (61.1%) than in the positional group (34.6%; P = 0.049). There were no between group differences in any of the BP measures. CPAP acceptance rates were very low in both groups (5.6% in non-positional and 11.5% in positional), although adherence was comparable.
**Conclusions:** The results of this study suggest that patients with positional-dependent mild OSA exhibit have similar BP (and evening to morning changes in BP) to patients with non-positional mild OSA.

**Key words:** Obstructive sleep apnea, positional sleep apnea, cardiovascular co-morbidities, hypertension, continuous positive airway pressure.
BACKGROUND

Severe obstructive sleep apnea (OSA) is associated with an increased risk of cardiovascular (CV) disease, including coronary artery disease, heart failure, and stroke [1]. Although often overlooked, even mild OSA has been associated with a higher prevalence of CV disease and significant CV co-morbidities, including hypertension and carotid artery atherosclerosis [2-5]. As the severity of OSA tends to worsen over time, active and effective treatment for mild OSA is essential [6].

The vast majority of patients with mild OSA exhibit position-dependent apnea, in which the presence and severity of symptoms are related to body position, and the associated gravitational changes, during sleep [7,8]. Specifically, gravity pulls the jaw and the tongue downwards while in the supine position, partially or fully obstructing the airway. Positional OSA is generally indicated by a total apnea-hypopnea index (AHI) $\geq 5$, with a $>50\%$ reduction in the AHI between the supine and lateral positions, and an AHI that normalizes (AHI $<5$) in the lateral position [7].

Various treatment modalities are available for mild OSA. Positional therapy, aimed at maintaining a non-supine sleep position, is often used as first-line treatment for patients with positional mild OSA, but has only moderate efficacy and poor adherence [9]. Continuous
positive airway pressure (CPAP) treatment significantly reduces the AHI and improves sleep efficiency in patients with OSA; however both patients with mild OSA and their clinicians are significantly less inclined to accept CPAP treatment [10]. Mandibular advancement devices, while generally preferred by patients over CPAP, are not as effective in reducing sleep apnea symptoms as CPAP [9]. In addition to its proven efficacy for improving apnea symptoms, effective CPAP treatment has also been shown be associated with significantly decreased risks for cardiovascular disorders, including reduced arterial stiffness and decreased blood pressure (BP) in patients with OSA [11-13].

Few studies have examined BP and CPAP use in patients with positional-dependent mild OSA. Therefore, the aim of this study was to determine if there are differences in BP (including morning BP, evening BP, and the different in BP between evening and morning) between patients who have positional and non-positional mild OSA. CPAP usage patterns in these patients were also evaluated.
METHODS

Study Subjects

A total of 874 consecutive adult patients (aged ≥ 18 years) were screened in this retrospective cohort study. All were referred to the Chest and Sleep Clinic of Taipei Tzuchi Hospital, The Buddhist Tzuchi Medical Foundation (New Taipei City, Taiwan) for overnight polysomnography (PSG) for suspected OSA between January 2007 and December 2011. Patients who did not have a previous diagnosis of OSA and who underwent full-night diagnostic PSG were included. Patients who underwent split-night sleep studies were excluded due to the inherent difficulty in separating the diagnostic and therapeutic portions of the study for postural effects [14]. Patients with PSG results that did not include at least 15 minutes of data obtained in both supine and non-supine positions were also excluded. This time period was chosen with reference to a previous study [14] and did not have to be consecutive. Patients with PSG results indicating non-apnea (AHI < 5/h), moderate OSA (AHI = 15-30/h) or severe OSA (AHI > 30/h) were also excluded. Only patients with mild OSA (AHI ≥ 5 to < 15) were included in the analyses. These patients were divided by positional-dependency of OSA. Positional OSA was defined as a > 50% reduction in the AHI between the supine and lateral positions and an AHI that normalized (AHI < 5) in a non-supine sleep position. This study was approved by the Institutional Review Board of the
Taipei Tzuchi Hospital, The Buddhist Tzuchi Medical Foundation. The informed patient consent was waived since this was retrospective study.

**Measurements**

*Anthropometric measurements and demographic data*

Medical history and anthropometric data were recorded prior to PSG study, and included body weight, height, body mass index, neck, waist, and hip circumference, waist-to-hip ratio, smoking status, hypertension, and presence of CV disease (defined as previous diagnosis of coronary artery disease or a history of cerebrovascular accident).

*Sleep parameters*

Excessive daytime sleepiness was evaluated using the Chinese version of the Epworth Sleepiness Scale (ESS) before overnight PSG study [15]. An attended, standard overnight PSG study was performed by trained sleep technicians at the sleep center. During PSG, electroencephalography (EEG), electrooculography, chin and bilateral anterior tibialis surface electromyography, electrocardiography, airflow through the nose and mouth (registered by thermistor), thoracoabdominal movements (registered by respiratory inductive plethysmography), position (by a sensor on the respiratory inductive plethysmograph), snoring, and oxygen saturation (by pulse oximetry) were recorded. The PSG study lasted for
at least 6 hours. PSG data were analyzed by manual scoring for every 30-second epoch by trained sleep technicians and were reviewed by sleep specialists.

Sleep stage was scored by trained sleep technicians using the standard criteria of Rechtschaffen and Kales [16]. An apnea event was defined as an 80-100% reduction in airflow lasting for at least 10 seconds. A hypopnea event was defined as a reduction in airflow of at least 50% for at least 10 seconds or at least a 30% reduction in airflow for at least 10 seconds as compared with baseline and associated with at least 3% oxygen desaturation or with an EEG arousal. AHI was calculated from the total number of apnea and hypopnea events per hour of sleep. The desaturation index was defined as > 3% oxygen desaturation per hour of sleep. The arousal index (AI) was defined as arousal episodes per hour of sleep [17]. Sleep efficiency was defined as the fraction of total sleep time to total recording time. Sleep latency was defined as the time from lights off to the first identifiable sleep stage. Rapid eye movement (REM) latency was defined as the time from the first identifiable sleep stage to the first REM sleep.

Blood pressure

BP was measured by trained technicians using an automated sphygmomanometer with an optimal cuff. Evening BP was measured after 15 minutes of rest and before sleep onset.
Morning BP was measured immediately upon awakening with the patient still in a supine position and attached to all PSG equipment. Two consecutive determinations were made on each occasion, separated by 5 minutes, and the results were averaged for both the evening and morning BP readings. Mean arterial blood pressure (MABP) was calculated using the systolic BP (SBP) and diastolic BP (DBP): MABP = 1/3 SBP + 2/3 DBP. Evening-to-morning SBP difference was determined by: morning BP – evening BP.

**CPAP acceptance and adherence**

All patients were offered treatment with CPAP. CPAP acceptance was defined as an expressed willingness to use CPAP, followed by use of a CPAP device at home for at least two weeks [18]. Patients who accepted CPAP were routinely followed up every 3 months at the outpatient clinic. At each visit, objective CPAP usage data recorded by the device software, including percentage of days used, percentage of nights during which CPAP was used for ≥ 4 hours, and the overall mean hours used per night.

High CPAP adherence was defined as ≥ 4 hours of CPAP per night for ≥ 70% of the nights [19]. Patients not meeting these levels of CPAP usage were recorded as having low CPAP adherence. Continued use of CPAP was defined as use of CPAP during the period up until the latest follow-up visit, as indicated by CPAP device data.
Statistical Analysis

Results for continuous variables are presented as mean ± standard deviation, whereas results for categorical variables are presented as number (percentage). A Kolmogorov-Smirnov test was used to test for normality. Baseline characteristics between the positional OSA and non-positional OSA groups were compared by two sample independent t-tests (continuous variables) or chi-square / Fisher’s exact test (categorical variables). Daytime sleepiness measurements and overnight polysomnography results between positional and non-positional groups were estimated by linear regression models. All statistical assessments were evaluated at a two-sided alpha level of 0.05 using SAS software, version 9.2 (SAS Institute, Inc., Cary, NC).
RESULTS

Baseline Characteristics and Anthropometric Measurements

The screening results are summarized in Figure 1. A total of 874 adult patients met the
inclusion criteria for screening. Of these, 46 were excluded for undergoing split-night studies,
352 for not having sufficient data in both supine and non-supine sleep positions, 103 for
having normal AHI, 100 for having moderate OSA, and 203 for having severe OSA.

The 70 patients who were included in the analysis were divided into two groups: positional
mild OSA (n = 52) and non-positional mild OSA (n = 18). The baseline characteristics of
these two groups are summarized in Table 1. All demographic and anthropometric variables
were similar between these two groups. Percentages of patients previously diagnosed
hypertension were significantly different between the two groups (P = 0.049); 61.1% in the
non-positional mild OSA group and 34.6% in the positional mild OSA group. All patients
with hypertension were receiving antihypertensive treatment.

Sleep Parameters

Table 2 summarizes the daytime sleepiness measurements and the overnight PSG results for
the non-positional and positional mild OSA groups. ESS scores were not significantly
different between these groups. After adjusting for hypertension, significant associations
were found for lateral AHI, supine posture percent, lateral posture percent, AI and sleep efficiency ($P < 0.001$, $P = 0.021$, $P = 0.021$, $P = 0.001$, $P = 0.013$, respectively). The positional group had a lower mean lateral AHI (1.9 ± 1.8 episodes/hours), lateral posture percent (37.9% ± 20.2%), AI (17.7 ± 9.6 episodes/hours) and higher supine posture percent (62.1% ± 20.2), sleep efficiency (78.2% ± 13.3%) than the non-positional group (lateral AHI: 8.6 ± 1.8 episodes/hours; lateral posture percent: 53.2% ± 24.1%; AI: 29.6 ± 19.5 episodes/hours; supine posture percent: 46.8% ± 24.1%; sleep efficiency: 74.9% ± 16.9%). All other factors were not significantly different between these groups.

**Blood Pressure**

As shown in Table 2, there were no significant differences between the non-positional and positional mild OSA groups in evening BP, morning BP or the evening-to-morning difference in BP. In addition, there were no significant changes in BP from morning to evening within each group.

**CPAP Acceptance and Adherence**

Table 3 shows descriptive statistics for CPAP acceptance and adherence. CPAP acceptance rates were low in both the non-positional (5.6%) and positional mild OSA (11.5%) groups. However, among those who accepted CPAP, the adherence rates were high (100% in the non-
positional group, and 80% in the positional group).
DISCUSSION

The prevalence of positional OSA in our study among patients with mild OSA was 74.3% (52 of 70 mild OSA patients). This percentage is similar to that reported in a previous study using the same definitions for positional-dependency [20]. Results of overnight PSG studies showed that patients in the positional group had lower AI and better sleep efficiency than the non-positional group, which also supports the results of a previous study [21]. AI was within normal limits for our positional group, but was higher than normal in the non-positional group. Sleep efficiency was lower than normal in both groups. While associations between OSA and hypertension have been generally accepted, studies have also shown that sleep-disordered breathing, short sleep, and poor sleep have been associated with hypertension in the general population [22-24]. Therefore, patients experiencing poor sleep quality should receive optimal treatment, regardless of OSA severity or positional dependency in order to reduce the risk of developing hypertension.

The prevalence of hypertension in both groups (61.1% of non-positional and 34.6% of positional patients) was similar to that reported in a previous study [25] and was higher in both groups than the prevalence in general population [24]. Much evidence supports elevated BP as a predictor of CV disease. Both increased SBP and increased DBP have been reported to be significant predictors of stroke and coronary artery disease [26]. On average, SBP and
DBP readings in the morning are 10%-20% lower than BP readings in the evening [27]. A recent study showed that when this morning dip is replaced by a morning surge in patients with hypertension, the risk of developing a major CV event rises significantly [28]. Ting et al reported that elevated morning SBP was associated with significantly greater respiratory disturbances, blood glucose, and metabolic syndrome score [29]. Many patients with OSA exhibit loss of the overnight dip in BP and exhibit morning BP elevations, possibly due to sympathetic nervous system overactivity [30,31]. In this study, patients in both the positional and non-positional groups showed a loss of evening to morning variation in BP, which supports the aforementioned findings. Our finding suggests that patients with positional mild OSA may have the same level of risk of developing CV disease as patients with non-positional mild OSA. Therefore, patients with mild OSA should receive active and effective treatment regardless of positional dependency.

Positional therapy is often relied upon as the primary treatment modality for positional mild OSA. A variety of strategies have been proposed for keeping patients in a non-supine position, including the use of tennis balls, vibrating positional alarms, and wearable devices [32,33]. However, while positional therapy can moderately reduce AHI in some patients, long-term compliance with positional therapy is poor as outlined in recent reviews [9,34]. In addition, since CPAP clearly provides superior improvement over positional therapy,
positional therapy is not recommended for first-line treatment of OSA [9].

The efficacy of CPAP in reducing apnea symptoms and the risk of CV disorders has been clearly demonstrated, making it the treatment of choice for patients with OSA; however, patients are often reluctant to accept CPAP treatment due to perceived inconvenience and discomfort [34,35]. Unsurprisingly, few patients in our study were willing to start CPAP treatment. Nonetheless, the majority of patients who accepted CPAP treatment showed a high level of adherence and continued use. Thus, educational support and physician involvement to increase patient awareness of the increased CV risks, even with mild OSA, and the benefits of CPAP treatment for CV health may improve patient acceptance and adherence [36].

This study is limited by the retrospective design and the small number of patients included. Larger controlled studies are warranted to further study the effects of positional dependency on BP, and CPAP efficacy and usage patterns in patients with mild OSA.

In conclusion, this study suggests that patients with positional mild OSA have similar BP and changes in BP to patients with non-positional OSA, and as such are likely to have a similar risk for developing CV disease. CPAP treatment should still be the first treatment choice for mild OSA, regardless of positional dependency. Increased efforts are warranted to improve
patient acceptance of CPAP.

A list of used Abbreviations

OSA: obstructive sleep apnea

CPAP: continuous positive airway pressure

CV: cardiovascular

PSG: polysomnography

AHI: apnea-hypopnea index

BP: blood pressure

ESS: Epworth Sleepiness Scale

EEG: electroencephalography

SBP: systolic BP

DBP: diastolic BP

MABP: man arterial blood pressure

BMI: body mass index

HTN: hypertension

CAD: coronary artery disease

CVA: cerebrovascular accident

DI: desaturation index

AI: arousal index

REM: rapid eye movement
SaO2: oxygen saturation

Competing interests: The author(s) declare that they have no competing interests.

Authors' contributions:
Yi-Chih Huang: Conception and design; Analysis and interpretation of data; Drafting of the manuscript; Final approval of the manuscript; statistical analysis; Obtaining funding
Chun-Yao Lin: Conception and design; Analysis and interpretation of data; Drafting of the manuscript; statistical analysis; Obtaining funding
Chou-Chin Lan: Acquisition of data; Analysis and interpretation of data; Critical revision of the manuscript; statistical analysis;
Yao-Kuang Wu: Acquisition of data, literature research
Chor-Shen Lim: Acquisition of data, literature research
Chun-Yao Huang: Acquisition of data, literature research
Hsuan-Li Huang: Acquisition of data, literature research
Kuan-Hung Yeh: Acquisition of data, literature research
Yu-Chih Liu: Conception and design; Analysis and interpretation of data; Drafting of the manuscript; Administrative, technical or material support
Mei-Chen Yang: Conception and design; Analysis and interpretation of data; Critical revision of the manuscript; Final approval of the manuscript; guarantor of integrity of the entire study;
definition of intellectual content; Supervision

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FIGURE LEGENDS

Figure 1. Flowchart of patient enrollment. PSG: polysomnography; OSA: obstructive sleep apnea; AHI: apnea-hypopnea index.
Table 1. Baseline characteristics of patients with non-positional and positional obstructive sleep apnea

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group</th>
<th>Non-positional (n = 18)</th>
<th>Positional (n = 52)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td></td>
<td>61.4 ± 15.3</td>
<td>56.5 ± 13.2</td>
<td>0.202</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td>0.921</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>6 (33.3)</td>
<td>18 (34.6)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>12 (67.7)</td>
<td>34 (65.4)</td>
<td></td>
</tr>
<tr>
<td>Neck circumference, cm</td>
<td></td>
<td>37.8 ± 3.6</td>
<td>37.3 ± 3.1</td>
<td>0.529</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td></td>
<td>92.6 ± 10.7</td>
<td>92.0 ± 10.6</td>
<td>0.824</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td></td>
<td>99.4 ± 10.0</td>
<td>100.7 ± 7.5</td>
<td>0.543</td>
</tr>
<tr>
<td>Waist-to-hip ratio, %</td>
<td></td>
<td>0.9 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>0.260</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td></td>
<td>26.3 ± 4.8</td>
<td>26.3 ± 3.4</td>
<td>0.985</td>
</tr>
<tr>
<td>Smoking history</td>
<td></td>
<td></td>
<td></td>
<td>0.252</td>
</tr>
<tr>
<td>Past-smoker</td>
<td></td>
<td>4 (22.2)</td>
<td>9 (17.3)</td>
<td></td>
</tr>
<tr>
<td>Never smoked</td>
<td></td>
<td>8 (44.4)</td>
<td>34 (65.4)</td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td></td>
<td>6 (33.4)</td>
<td>9 (17.3)</td>
<td></td>
</tr>
<tr>
<td>CV co-morbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any CV co-morbidity</td>
<td></td>
<td>11 (61.1)</td>
<td>19 (36.5)</td>
<td>0.069</td>
</tr>
<tr>
<td>HTN</td>
<td></td>
<td>11 (61.1)</td>
<td>18 (34.6)</td>
<td>0.049*</td>
</tr>
<tr>
<td>Anti-HTN drugs at bedtime</td>
<td></td>
<td>1 (9.1)</td>
<td>1 (5.6)</td>
<td>1.000</td>
</tr>
<tr>
<td>Dosage of anti-HTN drugs (number of tablets/day)</td>
<td></td>
<td>2.3 ± 1.3</td>
<td>1.8 ± 1.4</td>
<td>0.354</td>
</tr>
<tr>
<td>CAD</td>
<td></td>
<td>2 (11.1)</td>
<td>4 (7.7)</td>
<td>0.643</td>
</tr>
<tr>
<td>CVA</td>
<td></td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td></td>
</tr>
</tbody>
</table>

BMI: body mass index; CV: cardiovascular; HTN: hypertension; CAD: coronary artery disease; CVA: cerebrovascular accident.

Results are mean ± standard deviation for continuous variables and number (percentage) for categorical variables.

Between group comparisons were made by t-test (continuous variables) or Chi-square / Fisher’s exact tests (categorical variables).

* Indicates a significant between group difference, $P < 0.05$. 
**Table 2.** Comparison of daytime sleepiness measurements and overnight polysomnography results for patients with non-positional and positional obstructive sleep apnea

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Non-positional (n = 18)</th>
<th>Positional (n = 52)</th>
<th>Adjusted P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Daytime sleepiness measurements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESS score</td>
<td>10.6 ± 5.5</td>
<td>9.2 ± 5.0</td>
<td>0.184</td>
</tr>
<tr>
<td><strong>Overnight polysomnography</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AHI, episodes/hour</td>
<td>10.9 ± 2.5</td>
<td>10.1 ± 2.8</td>
<td>0.287</td>
</tr>
<tr>
<td>Supine AHI, episodes/hour</td>
<td>15.1 ± 5.7</td>
<td>16.5 ± 8.0</td>
<td>0.558</td>
</tr>
<tr>
<td>Lateral AHI, episodes/hour</td>
<td>8.6 ± 1.8</td>
<td>1.9 ± 1.8</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Supine posture percent, %</td>
<td>46.8 ± 24.1</td>
<td>62.1 ± 20.2</td>
<td>0.021*</td>
</tr>
<tr>
<td>Lateral posture percent, %</td>
<td>53.2 ± 24.1</td>
<td>37.9 ± 20.2</td>
<td>0.021*</td>
</tr>
<tr>
<td>DI, episodes/hour</td>
<td>4.7 ± 4.2</td>
<td>5.6 ± 3.4</td>
<td>0.229</td>
</tr>
<tr>
<td>AI, episodes/hour</td>
<td>29.6 ± 19.5</td>
<td>17.7 ± 9.6</td>
<td>0.001*</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>74.9 ± 16.9</td>
<td>78.2 ± 13.3</td>
<td>0.013*</td>
</tr>
<tr>
<td>Sleep latency, minute</td>
<td>27.6 ± 37.7</td>
<td>24.7 ± 21.2</td>
<td>0.409</td>
</tr>
<tr>
<td>REM latency, minute</td>
<td>113.0 ± 86.5</td>
<td>107.4 ± 49.4</td>
<td>0.154</td>
</tr>
<tr>
<td>S1, %</td>
<td>22.2 ± 18.8</td>
<td>18.5 ± 9.2</td>
<td>0.280</td>
</tr>
<tr>
<td>S2, %</td>
<td>61.5 ± 17.0</td>
<td>60.7 ± 9.1</td>
<td>0.880</td>
</tr>
<tr>
<td>S34, %</td>
<td>3.5 ± 5.4</td>
<td>5.1 ± 7.3</td>
<td>0.483</td>
</tr>
<tr>
<td>REM, %</td>
<td>12.8 ± 7.5</td>
<td>15.6 ± 7.1</td>
<td>0.901</td>
</tr>
<tr>
<td>SaO2 mean, %</td>
<td>93.1 ± 3.2</td>
<td>94.4 ± 1.7</td>
<td>0.074</td>
</tr>
<tr>
<td>SaO2 minimal, %</td>
<td>87.1 ± 4.2</td>
<td>85.2 ± 4.7</td>
<td>0.072</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evening SBP, mmHg</td>
<td>124.4 ±14.9</td>
<td>121.2 ±13.5</td>
<td>0.551</td>
</tr>
<tr>
<td>Evening DBP, mmHg</td>
<td>75.4 ± 10.6</td>
<td>74.8 ± 9.7</td>
<td>0.978</td>
</tr>
<tr>
<td>Evening MABP, mmHg</td>
<td>91.7 ± 11.7</td>
<td>90.2 ± 10.1</td>
<td>0.780</td>
</tr>
<tr>
<td>Morning SBP, mmHg</td>
<td>125.4 ±16.1</td>
<td>122.4 ±12.0</td>
<td>0.543</td>
</tr>
<tr>
<td>Morning DBP, mmHg</td>
<td>77.3 ±11.0</td>
<td>77.0 ± 11.1</td>
<td>0.840</td>
</tr>
<tr>
<td>Morning MABP, mmHg</td>
<td>93.3 ± 12.0</td>
<td>92.1 ± 10.7</td>
<td>0.914</td>
</tr>
<tr>
<td>Evening-to-morning SBP difference, mmHg</td>
<td>1.0 ± 10.5</td>
<td>1.2 ± 10.1</td>
<td>0.980</td>
</tr>
<tr>
<td>Evening-to-morning DBP difference, mmHg</td>
<td>1.9 ± 7.1</td>
<td>2.3 ± 10.9</td>
<td>0.805</td>
</tr>
<tr>
<td>Evening-to-morning MABP difference, mmHg</td>
<td>1.6 ± 7.4</td>
<td>1.9 ± 9.4</td>
<td>0.846</td>
</tr>
</tbody>
</table>

ESS: Epworth Sleepiness Scale; AHI: apnea-hypopnea index; DI: desaturation index; AI: arousal index; REM: rapid eye movement; SaO2: oxygen saturation; SBP: systolic blood pressure; DBP: diastolic blood pressure; MABP: mean arterial blood
pressure.

Results are mean ± standard deviation.

After adjusting for hypertension, associations between factors and groups were assessed by linear regression and $P$-values were estimated using Wald’s test.

* Indicates a significant association, $P < 0.05$. 
Table 3. Continuous positive airway pressure acceptance and adherence among patients with non-positional and positional obstructive sleep apnea

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-positional (n = 18)</td>
</tr>
<tr>
<td>CPAP acceptance, n (%)</td>
<td>1 (5.6%)</td>
</tr>
<tr>
<td>CPAP adherence</td>
<td></td>
</tr>
<tr>
<td>Level of adherence</td>
<td></td>
</tr>
<tr>
<td>High, n (%)</td>
<td>1 (100.0%)</td>
</tr>
<tr>
<td>Low, n (%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Continued CPAP use</td>
<td></td>
</tr>
<tr>
<td>Continued use, n (%)</td>
<td>1 (100.0%)</td>
</tr>
<tr>
<td>Discontinued use, n (%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Follow-up duration, months</td>
<td>47.0</td>
</tr>
</tbody>
</table>

CPAP: continuous positive airway pressure.

Results are number (percentage) for categorical variables and mean ± standard deviation for continuous variables.
Patients referred for PSG to rule out OSA (n = 874)

Patients received full-night PSG study (n = 828)

Patients with sufficiency sleep time (≥ 15 mins) in both sleep postures (n = 476)

Patients with OSA (AHI ≥ 5) (n = 373)

Patients with mild OSA (AHI ≥ 5 to < 15) included in analysis (n = 70)

Non-positional mild OSA (n = 18)

Positional mild OSA (n = 52)

Excluded split-night sleep study (n = 46)

Excluded insufficient sleep time (< 15 min) in both sleep postures (n = 352)

Excluded patients without OSA (AHI < 5) (n = 103)

Excluded patients with moderate (AHI 15 to 30) or severe OSA (AHI > 30)

Moderate OSA (n = 100)

(70 non-positional/30 positional)

Severe OSA (n = 203)

(179 non-positional/24 positional)