White Coat Hypertension in Patients With Obstructive Sleep Apnea-Hypopnea Syndrome*

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Background: The strength of the association between obstructive sleep apnea-hypopnea syndrome (OSAHS) and systemic hypertension could be affected by methodologic problems in the definition of hypertension.

Study objectives: To determine the frequency of white coat hypertension (WCH) in patients with OSAHS, and to analyze the characteristics of patients with OSAHS and WCH.

Patients and interventions: Ninety-nine consecutive patients with OSAHS and 20 healthy control subjects were included into the study. Twenty-four-hour ambulatory BP monitoring (ABPM) and urinary catecholamines were determined simultaneously with the polysomnographic study. Arterial blood gases and lung volumes were also measured.

Results: Office hypertension was diagnosed in 45 patients, while the control group included 54 normotensive patients with OSAHS. After ABPM, hypertension was confirmed in 30 patients with OSAHS and office hypertension. WCH was diagnosed in the remaining 15 patients (33%). Patients with WCH presented higher values of sleep onset latency and wake after sleep onset than normotensive and sustained hypertensive patients. No other differences in sleep parameters, function tests, or urinary catecholamines were found between the OSAHS groups.

Conclusion: The results indicate that WCH is a frequent phenomenon in patients with OSAHS, and that it is not predictable by clinical variables. (CHEST 2004; 125:817–822)

Key words: ambulatory BP monitoring; hypertension; sleep apnea; white coat hypertension

Abbreviations: ABPM = ambulatory BP monitoring; AHI = apnea-hypopnea index; BMI = body mass index; OSAHS = obstructive sleep apnea-hypopnea syndrome; \( \text{SaO}_2 \) = oxyhemoglobin saturation; WASO = wake after sleep onset; WCH = white coat hypertension

The strong association between obstructive sleep apnea-hypopnea syndrome (OSAHS) and systemic hypertension is well recognized.\(^1,2\) Forty to 60% of patients with OSAHS have arterial hypertension.\(^3\) Although this association is complicated by confounding factors, such as obesity, age, and sex, previous studies\(^4-7\) have identified OSAHS as an independent predictor for sustained hypertension.

However, the strength of this association could be affected by some methodologic problems in the definition of hypertension. Accuracy in the diagnosis of hypertension is fundamental to identify potential risk factors in epidemiologic studies as well as to indicate hypotensive treatment.

BP measurements obtained in the clinic have long served as the basis for determining the risk of hypertension.\(^8\)
hypertension in patients with OSAHS. Nevertheless, many patients with high BP in the physician's office are normotensive elsewhere. White coat hypertension (WCH), the pressor response triggered by BP measured by the doctor in the office, can be estimated by measuring the difference between the office BP and average daytime ambulatory BP monitoring (ABPM). Besides its clinical importance, an adequate control of white coat effect is essential in epidemiologic studies.

While WCH seems to occur in 24 to 39% of the general hypertensive population, this clinical condition has rarely been described in patients with OSAHS. Moreover, the frequency of WCH in patients with OSAHS could be affected by the changes in the autonomic nervous function of these patients. Although individuals with WCH do not necessarily report anxiety surrounding clinic visits or during normal activities, such situational BP elevations are generally understood to represent classic stress responses. In this sense, it has been suggested that patients with sustained hypertension and subjects with WCH could have a different pattern of urinary catecholamine excretion. The effect of sympathetic overactivity of many patients with OSAHS on white coat response has not been previously evaluated. Therefore, the aim of this study was to determine the frequency of WCH in patients with OSAHS. We have also analyzed the characteristics of patients with OSAHS and WCH.

**Patients and Methods**

**Subjects**

Ninety-six consecutive patients with OSAHS were selected for study. Patients were excluded from the study for the following reasons: (1) unwillingness or inability to perform the testing procedure; (2) obstructive or restrictive lung disease demonstrated on pulmonary function testing; (3) congestive heart failure, valvular heart disease, or atrial fibrillation; (4) suspected secondary hypertension; (5) current drug or mechanical treatment for sleep apnea; (6) therapy with steroids, nonsteroidal anti-inflammatory drugs, contraceptive, tricyclic antidepressants, or postmenopausal substitutive estrogen; (7) abnormal thyroid function; (8) morbid obesity (body weight > 150% ideal); and (9) stroke, angina pectoris, myocardial infarction, or angio-plasty in the last 12 months. Twenty healthy subjects with normal office BP were studied as control group. Control subjects were judged healthy by history, physical examination, ECG, basal spirometry, and chest radiography.

Antihypertensive treatment (diuretics, beta-blocking agents, angiotensin-converting enzyme inhibitors, or calcium-channel blockers) was discontinued at least 3 weeks before the examination. Subjects were asked not to eat for 4 h before the study, and they were also asked to refrain from using coffee, tea, and alcohol for ≥ 12 h before each study, and tobacco for ≥ 2 h before each study. The study was approved by the Institutional Ethics Committee at the hospital. All subjects gave their written informed consent prior to enrollment.

**ABPM**

Twenty-four-hour ABPM was performed on each patient (model 90207; Spacelabs; Redmond, WA), using an oscillometric method. BP was measured every 30 min during the day (8 AM to 11 PM) and every 60 min during the night (11 PM to 8 AM) on a workday. An appropriate cuff was used and placed on the nondominant arm. Patients were instructed not to move their arm during the ongoing measurement, and during the recordings the subjects were asked to carry out their ordinary daily activities, and to go to bed no later than 11 PM.

Office BP was measured by a random zero sphygmomanometer with the subject sitting for at least 5 min. The diagnosis of office hypertension was based on the mean of three readings obtained on two separate visits (between 1 week and 2 weeks apart) ≥ 140/90 mm Hg. WCH was defined as office hypertension associated with a mean daytime BP ≤ 135/85 mm Hg. Therefore, sustained hypertension was defined as a mean daytime BP ≥ 135/85 mm Hg. The existence of a nocturnal decrease in BP of ≥ 10% defined a “dipper” status.

**Blood and Urine Analysis**

Determinations of serum levels of cholesterol, triglycerides, and glucose were performed using an autoanalyzer. The day of ABPM determination, subjects were requested to collect separate urine samples from 8 AM to 11 PM (diurnal) and from 11 PM to 7 AM (night). Urine specimens for each sample were collected in polyethylene containers, acidified with HCl 6 mol/L as preservative, and stored at −40°C before analysis.

Urinary excretion of norepinephrine and epinephrine were determined as previously described. A 5-mL aliquot of a urine sample was filtered; 3.4 dihydroxybenzylamine (internal standard) and 0.1% ethylenediamine tetra-acetic acid were added to the filtrate, adjusted to pH 6.5, and subsequently placed on a Biorex 70 cation exchange column (Bio-Rad; Munich, Germany). After the sample entered completely into the resin, the column was washed with distilled water and the catecholamines were eluted with 10 mL of 0.65 mol/L boric acid. After this procedure, 20 µL of the effluent were injected into a high-performance liquid chromatography system composed of a high-performance liquid chromatography HPLC pump (model 510; Waters; Milford, MA), 20 µL injection valve (Rheodyne LLC; Rohnert Park, CA), a chromatographic column (HR-80, RP-C18; ESA; Chelmsford, MA), coulometric detector (model Coulochem II; ESA), high-sensitivity analytical cell model 5011 (ESA), and conditioning cell model 5021 (ESA). Concentrations of detected compounds were personal computer calculated using integration software 712 HPLC system controller (Version 1.2; Gilson; Madison, WI) that measures the heights of the peaks and relates them to external standards.

Intra-assay coefficients of variation were 3% for norepinephrine and 3% for epinephrine. Interassay coefficients of variation were 9% for norepinephrine and 10.5% for epinephrine. Results were expressed in terms of micrograms per gram of creatinine.

**Polysomnography**

During the same night when they were collecting urine specimens, healthy subjects and patients with OSAHS underwent polysomnography from 11 PM to 7 AM. EEG (C3-A2, C4-A1), electro-oculogram, chin electromyogram, electromyograms of the tibialis anterior of both legs, and ECG were continuously recorded. Breathing was monitored using nasal cannulae, oronasal thermistors, and thoracoabdominal strain gauges. Simultaneously, oxyhemoglobin saturation (SaO2) was monitored with a pulse oximeter (Pulsox DP-8; Minolta; Osaka, Japan). Sleep was...
analyzed using the standard criteria\textsuperscript{17} for epochs of 20 s, and the following sleep variables were calculated: total sleep time, wake time after sleep onset (WASO), and sleep efficiency, defined as the ratio of total sleep time to sleep episode duration. An obstructive apnea/hypopnea event was characterized by a > 50% decrease from baseline in the amplitude of breathing for > 10 s associated with either oxygen desaturation of > 3% or an arousal in the presence of continued respiratory efforts.\textsuperscript{18} The apnea-hypopnea index (AHI) was established as the number of apneas/hypopneas per hour of sleep. OSAHS was defined as excessive daytime sleepiness unexplained by other factors plus five or more obstructed breathing events per hour during sleep.\textsuperscript{18} As indexes of nocturnal oxygen saturation, the mean Sa\textsubscript{O\textregistered} throughout the night, the mean low Sa\textsubscript{O\textregistered} (mean of the minimum value for Sa\textsubscript{O\textregistered} in each 30-s epoch), and the minimum Sa\textsubscript{O\textregistered} (lowest values recorded during sleep) were computed.

Arterial blood gas values were measured with subjects in a seated position, while they breathed room air. Spirometry was performed by means of a pneumotachograph and static lung volumes were measured with a constant-volume body plethysmograph (MasterLab Body; Erich Jaeger GmbH, Würzburg, Germany) in accordance with European Respiratory Society standardization.\textsuperscript{19}

Statistical Analysis

The comparisons between the groups were performed by the one-way analysis of variance, including the effects of covariates (sex, age, body mass index [BMI], and smoking, categorized as yes/no). Post hoc analysis was performed using the Bonferroni test for multiple comparisons. The $\chi^2$ test was used for evaluating frequencies.\textsuperscript{20} A multiple logistic regression analysis was performed to identify the factors determining WCH. The independent variables included in the model were gender, age, BMI, smoking, diabetes (yes/no), AHI, and minimum Sa\textsubscript{O\textregistered}, as well as the variables that reached statistical significance in univariate analysis. These analyses were performed using the Statistical Package for the Social Sciences for Windows (Release 8.0; SPSS; Chicago, IL). In all cases, $p < 0.05$ was considered to be significant. Data are expressed as mean ± SD.

**RESULTS**

Office hypertension was diagnosed in 45 patients with OSAHS. Thirty-five of these subjects were in World Health Organization hypertension stage 1 (systolic BP < 159 mm Hg, diastolic BP < 99 mm Hg), 8 patients were in stage 2 (systolic BP, 160 to 179 mm Hg/diastolic BP, 100 to 109 mm Hg), and 2 patients were in stage 3 (systolic BP > 180 mm Hg/diastolic BP >110 mm Hg). The normotensive OSAHS group was composed of 54 patients. After ABPM determination, hypertension was confirmed in 30 patients with OSAHS and office hypertension, the frequency of WCH being 33% (95% confidence interval, 18 to 48%). In the normotensive OSAHS and control groups, all patients presented normal ABPM findings.

There was no significant difference in sex, age, BMI, or smoking habits between the OSAHS and control groups (Table 1). Metabolic parameters were

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control Subjects (n = 20)</th>
<th>Normotensive Group (n = 54)</th>
<th>WCH (n = 15)</th>
<th>Sustained Hypertension (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>55 ± 11</td>
<td>57 ± 9</td>
<td>58 ± 13</td>
<td>54 ± 8</td>
</tr>
<tr>
<td>Male gender, %</td>
<td>90</td>
<td>91</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>BMI</td>
<td>31.1 ± 3.8</td>
<td>28.5 ± 4.6</td>
<td>30.3 ± 4.9</td>
<td>31.1 ± 4.3</td>
</tr>
<tr>
<td>Smoker, %</td>
<td>20</td>
<td>16</td>
<td>21</td>
<td>18</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>0</td>
<td>3.7</td>
<td>6.7</td>
<td>3.3</td>
</tr>
<tr>
<td>Serum glucose, mmol/L</td>
<td>5.0 ± 0.5</td>
<td>5.1 ± 0.6</td>
<td>5.2 ± 0.9</td>
<td>5.3 ± 0.8</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.28 ± 0.81</td>
<td>5.21 ± 0.77</td>
<td>5.33 ± 0.91</td>
<td>5.61 ± 1.03</td>
</tr>
<tr>
<td>High-density lipoprotein cholestrol, mmol/L</td>
<td>1.27 ± 0.24</td>
<td>1.29 ± 0.22</td>
<td>1.31 ± 0.37</td>
<td>1.33 ± 0.51</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.32 ± 0.77</td>
<td>1.37 ± 0.84</td>
<td>1.41 ± 1.32</td>
<td>1.46 ± 1.41</td>
</tr>
<tr>
<td>Office BP, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>121.4 ± 7.2</td>
<td>122.4 ± 7.5</td>
<td>144.6 ± 6.3†</td>
<td>147.0 ± 10.7†</td>
</tr>
<tr>
<td>Diastolic</td>
<td>70.3 ± 6.9</td>
<td>70.5 ± 7.4</td>
<td>91.8 ± 4.6†</td>
<td>93.0 ± 10.2†</td>
</tr>
<tr>
<td>Daytime ambulatory BP, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>121.5 ± 7.0</td>
<td>122.6 ± 6.9</td>
<td>127.8 ± 7.6</td>
<td>140.1 ± 10.6†</td>
</tr>
<tr>
<td>Diastolic</td>
<td>72.3 ± 5.5</td>
<td>75.0 ± 5.2</td>
<td>75.2 ± 5.3</td>
<td>89.1 ± 9.7†</td>
</tr>
<tr>
<td>Nighttime ambulatory BP, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>106.1 ± 7.3</td>
<td>115.1 ± 11.2</td>
<td>127.2 ± 12.9†</td>
<td>131.7 ± 15.0†</td>
</tr>
<tr>
<td>Diastolic</td>
<td>67.2 ± 5.9</td>
<td>68.9 ± 6.4</td>
<td>71.8 ± 5.1</td>
<td>82.1 ± 11.5†</td>
</tr>
<tr>
<td>Nondipper, %</td>
<td>20</td>
<td>67†</td>
<td>73†</td>
<td>66†</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD unless otherwise indicated.
†p < 0.001 compared with normotensive OSAHS group.
‡p < 0.01 compared with normotensive OSAHS group.
§p < 0.001 compared with WCH.
‡p < 0.01 compared with control group.

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similar in normotensive, WCH, and sustained hypertension OSAHS groups and in control subjects. These parameters remained similar in study groups after controlling for gender, age, BMI, and smoking habits.

Table 1 shows office BP and ABPM measurements in the control group and in patients with OSAHS and normotension, WCH, or sustained hypertension. There were no differences in office BP between patients with WCH and patients with confirmed hypertension. Using ABPM, patients classified as WCH showed higher nighttime systolic BP in relation to the normotensive OSAHS and control groups. Prevalence of "nondipper" status was lower in control group (20%) than in normotensive patients (67%), WHC (73%), and sustained hypertension (66%) OSAHS groups (p < 0.01).

Patients with WCH have a higher sleep onset latency and WASO than normotensive and sustained hypertensive patients (Table 2). These parameters remained significantly higher in the WCH group after controlling for gender, age, and BMI. No other differences in sleep characteristics, lung function data, or urinary catecholamines were found between the OSAHS groups. In the logistic regression model, WCH was not predicted by any of the variables included in the model (gender, age, BMI, smoking status, diabetes, sleep onset latency, or WASO).

### Discussion

This study, the first to our knowledge to analyze the WCH phenomenon in patients with OSAHS, showed that one third of these patients, diagnosed hypertensives with office BP measurements, presented WCH. Although published estimates vary widely, WCH has been reported to occur in approximately 30% of hypertensive patients. The highest prevalence of WCH has been described in diabetic patients. In patients with type 2 diabetes mellitus, prevalence of WCH ranges from 23 to 62%, and reaches 74% in type 1 diabetic patients without nephropathy. However, WCH is less frequent in those patients with more severe hypertension. In a large study in a general hypertensive population, Verdecchia et al. reported a 19% prevalence of WCH, which increased to 33% when only patients with the mildest hypertension were considered.

The frequency of WCH in our patients with OSAHS is similar to that found in a population of mild-to-moderately hypertensive subjects treated at primary care centers in our city. In this group of subjects, the frequency of WCH was 39% (95% confidence interval, 33 to 45%), and it was also inversely proportional to severity of office BP values.

Although the association of hypertension and obe-

### Table 2—Sleep Parameters, Functional Data, and Urinary Catecholamines in the Control and OSAHS Groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control Subjects</th>
<th>Normotensive Group</th>
<th>WCH</th>
<th>Sustained Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time, min</td>
<td>379.2 ± 39.2</td>
<td>351.4 ± 71.9</td>
<td>345.7 ± 88.6</td>
<td>339.2 ± 71.9</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>84.2 ± 7.5</td>
<td>78.8 ± 12.2</td>
<td>78.9 ± 18.2</td>
<td>75.1 ± 14.9</td>
</tr>
<tr>
<td>Sleep onset latency, min</td>
<td>34.4 ± 10.6</td>
<td>12.3 ± 12.3</td>
<td>46.2 ± 55.5 §</td>
<td>13.5 ± 25.3§</td>
</tr>
<tr>
<td>WASO, min</td>
<td>33.8 ± 33.3</td>
<td>108.0 ± 38.1§</td>
<td>169.9 ± 46.9</td>
<td></td>
</tr>
<tr>
<td>AH1, h</td>
<td>2.4 ± 1.8</td>
<td>30.7 ± 18.3</td>
<td></td>
<td>36.3 ± 18.5</td>
</tr>
<tr>
<td>Mean nocturnal SaO₂, %</td>
<td>96 ± 1</td>
<td>94 ± 2</td>
<td>91 ± 7</td>
<td></td>
</tr>
<tr>
<td>Mean low nocturnal SaO₂, %</td>
<td>91 ± 2</td>
<td>73 ± 10</td>
<td></td>
<td>76 ± 9</td>
</tr>
<tr>
<td>Minimum SaO₂, %</td>
<td>89 ± 2</td>
<td>71 ± 8</td>
<td>72 ± 7</td>
<td></td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>80.3 ± 8.2</td>
<td>75.9 ± 10.5</td>
<td>77.1 ± 7.4</td>
<td>74.5 ± 8.5</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>37.9 ± 3.9</td>
<td>38.1 ± 3.8</td>
<td>38.4 ± 4.5</td>
<td>41.7 ± 7.2</td>
</tr>
<tr>
<td>FVC, L</td>
<td>3.46 ± 0.75</td>
<td>3.45 ± 0.67</td>
<td>3.44 ± 1.34</td>
<td>3.35 ± 0.92</td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>2.79 ± 0.66</td>
<td>2.74 ± 0.63</td>
<td>2.89 ± 1.09</td>
<td>2.74 ± 0.91</td>
</tr>
<tr>
<td>FEV₁/FVC, %</td>
<td>79.3 ± 5.2</td>
<td>79.2 ± 10.5</td>
<td>85.1 ± 6.5</td>
<td>80.8 ± 10.0</td>
</tr>
<tr>
<td>Diurnal norepinephrine, μg/g</td>
<td>63.2 ± 43.8</td>
<td>81.2 ± 78.0</td>
<td>105.1 ± 114.0</td>
<td>65.9 ± 41.9</td>
</tr>
<tr>
<td>Diurnal epinephrine, μg/g</td>
<td>9.4 ± 11.9</td>
<td>9.0 ± 7.5</td>
<td>15.1 ± 15.6</td>
<td>8.4 ± 3.9</td>
</tr>
<tr>
<td>Nocturnal norepinephrine, μg/g</td>
<td>28.9 ± 11.3</td>
<td>53.3 ± 40.5</td>
<td>44.7 ± 42.3</td>
<td>45.4 ± 19.0</td>
</tr>
<tr>
<td>Nocturnal epinephrine, μg/g</td>
<td>1.2 ± 1.2</td>
<td>3.1 ± 1.7</td>
<td>2.3 ± 0.9</td>
<td>3.6 ± 1.8</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD unless otherwise indicated.

1p < 0.05 compared with normotensive OSAHS group.
2p < 0.01 compared with normotensive OSAHS group.
3p < 0.01 compared with WHC.
4p < 0.01 compared with control group.

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osity is well recognized in patients with OSAHS, our patients with OSAHS and sustained hypertension had similar BMIs than normotensive OSAHS subjects. In this sense, epidemiologic studies7–26 have noted that hypertension is related to obesity and to other metabolic abnormalities (sometimes called “syndrome X”) consisting of insulin resistance, increased abdominal fat, and dyslipidemia. Moreover, it has been recently demonstrated that obesity and OSAHS are independently associated with insulin resistance, and that insulin resistance is a significant factor for hypertension in OSAHS patients.27 This last variable was not controlled in our study.

In contrast with other authors,10,22,23 we did not find an association between WCH and female gender. The reason why we could not find this association could be due to the scarce number of women in our study groups, secondary to low prevalence of OSAHS in women.

We failed to identify differential characteristics in sympathetic tone between normotensive and WCH patients with OSAHS. It is difficult to justify the higher sleep onset latency and WASO found in patients with WCH compared with sustained hypertension or normotensive groups. Although excessive daytime somnolence, tiredness, asphyxic episodes during the night or nonrefreshing sleep, among other symptoms, characterize patients with OSAHS, our results suggest that subjects with WCH and OSAHS have lower hypersomnolence than the other groups of patients with OSAHS.

The influence of psychological factors on daytime sleepiness could explain this finding. Several personality characteristics of subjects with WCH could attenuate the hypersomnolence due to obstructive sleep apneas. These patients display an increased emotional reactivity and higher levels of anger, anxiety, or depression, as compared with sustained hypertensive patients.28 As bedtime state anxiety has been significantly correlated with sleep latency,29 the higher values of sleep onset latency found in our patients with OSAHS and WCH could simply reflect the stress surrounding any clinical procedure.

Moreover, an anxious personality pattern could produce a state of constant emotional arousal. In fact, it has been also demonstrated that elevated symptoms of stress and anxiety are associated with subjective sleep complaints and EEG evidence of hyperarousal during sleep.30 Thus, the higher WASO found in the WCH group with respect to sustained hypertension or normotensive OSAHS groups could suggest that patients with OSAHS and WCH sleep differently.

As sleep architecture has not been previously explored in patients with WCH, whether or not they had OSAHS, we cannot determine if the finding of altered sleep onset latency and WASO might have interesting relevance to other populations of hypertension. The long-term prognostic significance and the exact repercussion of WCH remain unclear. Although longitudinal data on morbidity and mortality are scarce, they suggest a more benign outcome than in sustained hypertension.10,31–33 In accordance with this, we observed in our study that both systolic and diastolic BP levels in patients classified as WCH were similar to those observed in the normotensive OSAHS group. However, other studies21,34 show that WCH implies an increase in cardiovascular risk. In fact, in matched groups of normotensive subjects with WCH, it has been demonstrated that WCH is responsible for left ventricular dysfunction and abnormalities in carotid structure and function.35–37 This being so, Bidlingmeyer et al28 suggest that WCH might represent a transitional state toward the development of persistent ambulatory hypertension.

While the prevalence of WCH among patients with OSAHS is similar to the prevalence found in numerous studies of patients without OSAHS, the observation is still interesting in OSAHS. Although the relation between hypertension and OSAHS is well established,5,7 risks of a missed diagnosis of hypertension could occur in patients with OSAHS as in other populations.10,21–23,25. Our results suggest that the occurrence of WCH could lead to overdiagnosis of hypertension among patients with OSAHS; unless ABPM is not conducted, it can skew upwards the prevalence of hypertension in OSAHS, thereby leading to potentially flawed conclusions regarding the causative association of hypertension and OSAHS.

To summarize, we observed that WCH is a frequent phenomenon in patients with OSAHS, which could lead to overdiagnosis of hypertension among these subjects. Available evidence suggests that subjects with WCH are likely a group of intermediate risk between normotensives and sustained hypertensives. Thus, the mischaracterization of patients with WCH as hypertensive on the basis of office BP alone could underestimate cardiovascular complications in hypertensive patients with OSAHS.

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