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Is it Mandatory to do a 24 hour ABPM in all Patients with Moderate to Severe Obstructive Sleep Apnoea?

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Abstract

Background Obstructive sleep apnoea (OSA) has been described as a risk factor for arterial hypertension (HT). One of the proposed mechanisms linking these conditions is non dipping (ND) pattern in nocturnal blood pressure, however evidence is variable and based on specific populations with underlying conditions. Data for OSA and ND in subjects residing at high altitude are currently unavailable.

Objective Identify the prevalence and association of moderate to severe OSA with HT and ND pattern in hypertensive and non-hypertensive otherwise healthy middle-aged individuals in residing at high altitude (Bogotá:2640 mt)

Methods Adult individuals with diagnosis of moderate to severe OSA underwent 24 hour- ambulatory blood pressure monitoring (ABPM) between 2015 and 2017. Univariable and multivariable logistic regression analysis were performed to identify predictors of HT and ND pattern.

Results Ninety-three (93) individuals (male 62.4% and median age 55) were included in the final analysis. Overall, 30.1% showed a ND pattern in ABPM and 14.9% had diurnal and nocturnal hypertension. Severe OSA (higher apnea-hiponea index [AHI]) was associated with HT ($p = 0.006$), but not with ND patterns ($p = 0.54$) in multivariable regression. Smoking status and lowest oxygen saturation during respiratory events where independently associated with ND pattern ($p = 0.04$), whereas age ($p = 0.001$) was associated with HT.

Conclusions In our sample, one in three individuals with moderate to severe OSA have non dipping patterns suggesting lack of straight association between OSA and ND. Older individuals who have higher AHI are more likely to have HT, and those who smoke have a higher risk of ND. These findings add aditional information to the multiple

Keywords
- obstructive sleep apnoea
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mechanisms involved in the relationship between OSA and ND pattern, and questions the routine use of 24-hour ABPM, particullary in our region, with limited resources and healthcare acces. However, further work with more robust methodology is needed to draw conclusions.

Introduction

Obstructive sleep apnoea syndrome (OSA) is a common disease with a prevalence of 9-38% in general population, and reaches 28% in Latin America.[1] It is considered an independent risk factor for cardiovascular disorders,[2] including arterial hypertension (HT), stroke, coronary syndrome, heart failure and chronic kidney disease.[3–5] HT is a common finding in individuals with newly diagnosed OSA,[6] as is OSA in individuals with chronic HT.[7] Older individuals with higher body mass index (BMI) are at higher risk of having OSA and HT.[7,8] Data in Latin America report an increase in AHI with age; however age is a confounding factor, and currently is not clear which is the normal Apnoea-hypopnea Index (AHI) in elderly individuals.[9] Individuals with dual diagnosis (HT and OSA) are at higher risk of adverse cardiovascular outcomes, highlighting the importance of adequate and prompt clinical assessment, diagnosis, and treatment.

The physiological mechanisms linking HT and OSA are multiple.[10] Recurrent airway collapse during apnoeic episodes lead to intermittent hypoxia resulting in transient elevated blood pressure (BP), particularly diastolic blood pressure.[10] These episodes have also been associated with microarousals and sleep fragmentation, which increase catecholamine levels and oxidative stress.[11] Crucially, over time, these brief surges in sympathetic activity during sleep in OSA could lead to nocturnal hypertension (night-time BP $>$ 120/70 mm Hg). These processes lead to endothelial dysfunction and systemic inflammation and constitute the pathophysiological bases of vascular damage[12] and resistant hypertension.[13] In normal conditions, blood pressure follows a circadian pattern in which daytime mean pressure is at least 10% higher than the night-time mean. Subjects with a normal nocturnal fall in systemic blood pressure on 24-hour Ambulatory Blood Pressure Monitoring (ABPM) are referred as dippers, while those that do not show this pattern are referred as non-dippers.[14] The prevalence of ND BP in individuals with OSA is 48 to 84%,[15–19] and it has been associated with an increased risk of HT-induced target organ damage and cardiovascular events.[19,20] The ABPM may be useful to assess the impact of OSA on HT, but this is not a paradigm in all patients with a recent diagnosis of sleep apnea. The ABPM role has been studied in specific populations abroad.[15,17,21,22] In Latin America only three Brazilian studies, which included individuals with specific characteristics, have evaluated the association between ND pattern and OSA.[23–25]

In this study, we aim to determine the prevalence of ND blood pressure pattern in patients with moderate to severe OSA, examine the associated risk factors for HT and ND, and discuss the clinical value of routine ABPM as a screening tool from ND pattern in this population.

Material and Methods

Study Participants

From a prospective cohort of subjects in a Sleep Disorders Centre in Bogota Colombia we selected and collected baseline and demographic data of 93 adults older than 18 years diagnosed with moderate to severe OSA between 2015 and 2017. The diagnosis of moderate to severe OSA was defined as the presence of an apnea hypopnea index $\geq$ 15 per hour, sleep efficiency $\geq$ 60% and at least one episode of REM sleep. We included both naïve and known hypertensive patients. We excluded individuals who refuse to participate in the study, those with heart failure, chronic kidney disease, Parkinson's disease, previous stroke (Rankin $\geq$4), lung disease with saturation $<$ 88%, positional apnoea, pregnancy and patients on CPAP. From a universe of 4320 subjects, 1209 met inclusion criteria but only 93 individuals agree to participate and were included in the analysis. 1116 candidates were excluded because of impossibility to attend or refuse ABPM, unfeasibility of follow up and incomplete PSG data.

Regulatory approval was granted from the local ethical committee. All subjects gave written informed consent during their first visit.

Evaluations

Polysomnography

All subjects underwent attended overnight polysomnography (Type I)[26] in our Sleep Lab. Electroencephalogram (EEG), electro-oculogram (EOG), electromyography of the chin and anterior tibialis, electrocardiogram (ECG), nasal airflow (nasal pressure transducer- Pro-Thec), thoracoabdominal movements (inductive respiratory bands), arterial oxygen saturation (pulse oximetry- Massimo), body position sensors, and video were registered in each study. All studies were read and classified by the attending sleep medicine expert (EO). based on Academy of Sleep Medicine Scoring Manual version 2.0.[27]

Ambulatory BP Monitoring

Subjects underwent 24-hour blood pressure monitoring within the first week after PSG. Using appropriate cuff sizes, blood pressure (BP) was measured every 15 minutes during the day (06:00-22:00), and every 30 minutes at night (22:00-06:00). Subjects were advised not to modify their ordinary

daily routine and not to move their arms during the ongoing measurement. They were also instructed not to do exercise or have sexual activity during the duration of the monitoring. Activity, bedtime, and time of awakening were recorded by participants in diaries (►**Supplement**). The criteria used to define the dipping or non-dipping pattern where based on the European Society of Hypertension position paper on ambulatory blood pressure monitoring.[26] All ABPM readings were reviewed by cardiologists.

Clinical Evaluation

All participants had a clinical evaluation and completed medical questionnaires regarding sleep quality, symptoms of OSA, and excessive daytime sleepiness (►**Supplement**). Anthropometric measurements, including neck and waist circumferences, body mass index, heart rate and oxygen saturation were gathered. Office BP was determined automatically and manually (Welch Allyn DS44-11CB and Littman Classic II) with the subject resting in a sitting position. Systolic values corresponded to Korotkoff Phase I (First tapping) and diastolic to Phase V (Silence). Two measurements were taken, and the mean value was recorded. Blood pressure measurements were taken in the morning. These procedures followed the American Heart Association Guidelines.[28]

Definitions

The respiratory events were classified according to the criteria of American Academy of Sleep Medicine: apnoea was defined as a $\geq$90% decreased in airflow compared to baseline, at least 90% of the event's duration meets the amplitude reduction criteria and the duration of the event lasts at least 10 seconds. Hypopnea was defined as a partial reduction in airflow (between 30-90% of baseline), occurring for at least 90% of the event's, the duration of this drop occurs for a period of at least 10 seconds, and is associated with arousals and/or oxygen desaturation $\geq$4%.[27]

The classification of OSA was as follows: No OSA (IAH $<$ 5/h), mild OSA (AHI: $\geq$5 and $<$15/h), moderate OSA (AHI: $\geq$ 15/h and $<$ 30/h), and severe OSA (AHI$\geq$ 30/h).[27]

Blood pressure values were classified as follows: normal when the diurnal systolic and diastolic BPs were $<$135 and $<$85 mm Hg, respectively, and nocturnal $<$120 and $<$70 mm Hg, respectively. Dipping patterns were defined as follows: A normal BP dip was based on a BP reduction $\geq$10% but $<$20% during sleep compared with the awake period. Extreme dippers were defined as a $\geq$20% reduction in BP during sleep compared with the awake period. A ND BP was defined as a $\geq$0% but $<$10% reduction in BP during sleep compared with wake period. Reverse dippers were defined as a $<$0% reduction (riser) in BP during sleep compared with during the awake period.[26]

Outcomes

The primary outcome was to obtain the prevalence of non-dipping pattern in our population with moderate to severe OSA. The secondary outcome was identifying factors associated with ND patterns and HT in individuals with moderate to severe OSA.

Statistical Analysis

Baseline characteristics and quantitate values were described using measures of central tendency. We compared the baseline characteristics between participants who had ND and those who did using Chi-square and Man Whitney's tests for normally distributed data and Wilcoxon rank sum tests for those that showed a non-normal distribution on the Shapiro-Wilk's test (►**Table 1**). The reported baseline characteristics included those that were gathered during clinical evaluation and that have been previously identified as independent predictors of ND (obesity, smoking status, snoring, waist and neck circumference, BMI, hypertension). We used univariable logistic regression to identify the predictors associated with hypertension and with ND. Variables that were significant ($p < 0.05$) in the univariable analyses or were consistently reported in previous literature were included in the multivariable analysis. Two multivariable models were developed: Model 1 included hypertension as dependent variable and all significant baseline and demographic variables were included as independent variables. Model 2 included ND as dependent variables and all the rest were included as independent variables. The rationale behind creating two separate models was to obtain predictors that would help clinicians identify individuals with OSA at high risk of hypertension (model 1), and those at high risk of ND (model 2) that would benefit from ABPM.

Martingale residuals plots were analysed to assess for nonlinearity. Multicollinearity was examined using the variance inflation factor (VIF). Analyses were performed using RStudio version 1.1.453m, using the packages "survival", "survminer," and "ggplot2".

Results

We included 93 adults (median age 55 years old, interquartile rage [IQR] 44-61; 62.4% females) with moderate to severe OSA (median AHI 40.3/h). 34.4% of the participants had a previous diagnosis of HT and 48.8% were hypertense during ABPM monitoring. 2.1% had exclusive nocturnal hypertension, and 30.1% individuals were classified as non-dippers, of whom 18% were hypertense (►**Table 1**).

Univariable regression analysis showed that older age (OR 1.01, 95% CI 1-1.02) and higher AHI index (OR 1.01 95% CI 1.0-1.01), were associated with hypertension (►**Table 2A**). These variables, together with the ones previously reported in the literature as significant predictors (BMI, smoking, snoring), were included in a multivariable model. The final model showed that age, smoking, and having a higher AHI were significantly associated with hypertension (►**Table 2B**).

Regarding ND patterns, univariable regression analysis showed that medical history of refractory HT (OR 1.59, 95% CI 1-2.53), smoking (OR 1.42, 95% CI 1.02-1.98), older age (OR 1.01, 95% CI 1-1.01), lower diurnal diastolic blood pressure (OR 0.98 95% CI 0.97-0.99), and reduced blood pressure variability (OR 0.96 95% CI 0.93-0.99) were associated with an increased risk of ND (►**Table 3A**). These variables, together with ones reported in the literature as significant predictors of ND (snoring, BMI) were included in the multivariable model.

Table 1 Baseline characteristics.

Variable	Overall	Non dipping BP		
		No	Yes	p value
Demographics	n = 93	n = 65	n = 28	
Age	55 [44, 61]	55 [43-58]	59.50 [46.75-64]	**0.036**
Sex (male)	58 (62.4)	43 (66.2)	15 (53.6)	0.351
Clinical evaluation				
Regular alcohol consumption	9 (9.7)	7 (10.8)	2 (7.1)	0.719
Regular smoking	8 (8.6)	3 (4.6)	5 (17.9)	**0.051**
Subjective snoring	89 (95.7)	62 (95.4)	27 (96.4)	1.000
Weight (kg)	73 [67- 84]	73 [68-85]	71 [63.50-81.12]	0.224
Height (cm)	168 [162-173]	168 [162-173]	167 [163.75-172.25]	0.775
BMI	26 [24- 28.5]	26.10 [24.60-29]	26.35 [23.38-28]	0.407
Neck	39 [36-42]	39 [36-41]	39 [34.50-42.38]	0.933
Abdomen	94 [88, 102]	94 [89-102.30]	92 [86-100.50]	0.210
Hypertension	32 (34.4)	23 (35.4)	9 (32.1)	0.816
Refractory hypertension	4 (4.3)	1 (1.5)	3 (10.7)	0.080
Systolic	120 [110-130]	113 [105-125]	118 [103.50-123.25]	0.789
Diastolic	75 [70-80]	73 [68-78]	73 [67.75-77.25]	0.930
Heart rate	72 [67- 78]	72 [68-80]	72 [66.75-78.25]	0.753
Polysomnography				
IAH	28.2 [19.8- 37.7]	26.20 [19.50-37.40]	29.80 [21.45-41.22]	0.563
Oxygen saturation	88 [86-91]	88 [87-91]	88 [85.75-90.25]	0.261
AMBP				
Hypertension AMBP	45 (48.4)	33 (50.8)	12 (42.9)	0.507
Diurnal systolic	123 [113-131]	125 [115-134]	121.50 [106.50-126]	0.079
Diurnal diastolic	78 [74-84]	80 [75-86]	75 [70-80.25]	**0.005**
Nocturnal systolic	105 [98-118]	103 [95-114]	114.50 [100.75-120]	**0.028**
Nocturnal diastolic	66 [61-72]	64 [61-69]	70.50 [64.50-75]	**0.016**
Nocturnal systolic decrease (%)	12 [7-18]	17 [12-21]	6 [4-7.25]	<**0.001**
Nocturnal diastolic decrease (%)	14 [9-22]	17 [14-25]	7 [4.38-9.32]	<**0.001**
Blood pressure variability %)	12 [10-14]	12 [11-14]	11 [9.75-13]	**0.018**
Pulse pressure	42 [37-49]	42 [37-49]	41.50 [35.75-46.75]	0.963
Heart rate (mean)	72 [67-79]	72 [68-80]	72 [66.75-78.25]	0.753
Nocturnal hypertension	16 (17.2)	10 (15.4)	6 (21.4)	0.552
Diurnal hypertension	23 (24.7)	18 (27.7)	5 (17.9)	0.434
Dipping pattern (%)				<**0.001**
Dipping	45 (48.4)	45 (69.2)	0 (0.0)	
Non dipping	28 (30.1)	0 (0.0)	28 (100.0)	
Extreme dipping	20 (21.5)	20 (30.8)	0 (0.0)	
Reverse dipping	0 (0.0)	0 (0.0)	0 (0.0)	

n [%] or Median [IQR]; BMI: body mass index; IAH: Index apnoea hypopnea.

Table 2 Univariable (A) and multivariable (B) logistic regression analysis of predictors of hypertension individuals with moderate to severe OSA.

Variable	Univariable log regression		Multivariable log regression	
	OR	p value	aOR	p value
Demographics				
Age	1.02 (1.01-1.03)	**0.000**	1.02 (1.01-1.03)	**0.001**
Sex (female)	0.83 (0.68-1.02)	0.076		
Clinical evaluation				
Alcohol comsumption	1.12 (0.80-1.56)	0.510		
Smoking	1.19 (0.83-1.68)	0.337	0.87 (0.61-1.24	0.454
Subjective snoring	0.65 (0.41-1.06)	0.082	0.69 (0.44-1.09)	0.113
Weight (kg)	1.00 (0.99-1.01)	0.670		
Height (cm)	1.00 (0.99-1.00)	0.751		
BMI	1.02 (0.99-1.05)	0.183	1.00 (0.97-1.03)	0.834
Neck	1.01 (0.98-1.03)	0.690		
Abdomen	1.00 (1.00-1.01)	0.366		
Heart rate	0.99 (0.98-1.01)	0.353		
Polysomnography				
AHI	1.01 (1.00-1.01)	**0.001**	1.01 (1.00-1.01)	**0.006**
Oxygen saturation	0.99 (0.96-1.02)	0.610		
AMBP				
Diurnal systolic	1.01 (1.01-1.02)	0.000		
Diurnal diastolic	1.01 (1.00-1.02)	0.226		
Nocturnal systolic	1.01 (1.01-1.02)	0.000		
Nocturnal diastolic	1.01 (1.00-1.02)	0.091		
Nocturnal systolic decrease (%)	1.00 (1.00-1.00)	0.082		
Nocturnal diastolic decrease (%)	1.00 (1.00-1.00)	0.082		
Blood preassure variability %)	1.02 (0.99-1.06)	0.167		
Pulse pressure	1.02 (1.01-1.03)	0.000		
Heart rate (mean)	0.99 (0.98-1.00)	0.124		

AHI: Index apnoea hypopnea; BMI: body mass index.
*Variables from AMBP were not included in the multivariable model to obtain predictors to select high risk individuals that would benefit from more extensive assessment of hypertension like AMBP monitoring.

Only smoking (aHR 1.46 95% CI 1.01-2.12) was independently associated with ND pattern after adjusting (►**Table 3B**).

Finally, indicators of hypoxemia where analyzed. Oximetry means values during respiratory events, during REM/NREM sleep and overall mean arterial oximetry values where compared between Dipping and Non Dipping subjects. ND pattern correlates with lower mean oxigen saturation during respiratory events (►**Table 4**). We did not include T < 90% data because of lack of these data in the PSG reports.

Discussion

Current clinical recommendations state that ABPM should be performed in the majority of subjects with OSA and/or HT given the high prevalence of co-diagnosis and its impact in cardiovascular outcomes. Using a prospective cohort of middle-aged individuals with moderate to severe OSA, we found that AHI is associated with HT, but not with ND pattern. This finding is congruent with previous literature suggesting a link between OSA and HT but opposes findings suggesting an unequivocal association between severe OSA and ND.

The prevalence of ND pattern that we found was 30.1%, which is lower than previously reported (48-84%).[15–19] A recent meta-analysis by Cuspidi et al, included 1562 patients with OSA from 14 studies and found a ND prevalence of 59.1%.[15] However, they included individuals with mild OSA (AHI > 5 and <15/h), who were evaluated at sleep or cardiology clinics. Given that Hispanic individuals or those in developing countries have a different incidences of

Table 3 Univariable (A) and multivariable (B) logistic regression analysis of predictors of non-dipping individuals with moderate to severe OSA.

Variable	Univariable log regression		Multivariable log regression	
	OR	p value	aOR	p value
Demographics				
Age	1 (1-1.01)	**0.035**	1 (1-1.01)	0.073
Sex (female)	0.89 (0.73-1.08)	0.255		
Clinical evaluation				
Alcohol comsumption	0.91 (0.66-1.26)	0.592		
Smoking	1.42 (1.02-1.98)	**0.037**	1.46 (1.01-2.12)	**0.043**
Subjective snoring	1.05 (0.65-1.68)	0.822	1.25 (0.78-2)	0.337
Weight (kg)	0.99 (0.98-1)	0.220		
Height (cm)	1 (0.99-1)	0.774		
BMI	0.98 (0.96-1.01)	0.444	0.97 (0.94-1)	0.097
Neck	0.99 (0.97-1.02)	0.760		
Abdomen	0.99 (0.98-1)	0.366		
Hypertension	0.97 (0.79-1.18)	0.765		
Refractory hypertension	1.59 (1-2.53)	**0.045**	1.37 (0.84-2.22)	0.193
Systolic	0.99 (0.99-1)	0.580		
Diastolic	0.99 (0.98-1)	0.222		
Heart rate	1 (0.99-1.02)	0.090		
Polysomnography				
AHI	1 (0.99-1)	0.695	0.99 (0.98-1)	0.54
Oxygen saturation	0.97 (0.95-1)	0.120	0.98 (0.95-1.02)	0.415
AMBP				
Hypertension AMBP	0.93 (0.77-1.13)	0.489		
Diurnal systolic	0.99 (0.98-1)	0.308		
Diurnal diastolic	0.98 (0.97-0.99)	0.001		
Nocturnal systolic	1 (1-1.01)	0.007		
Nocturnal diastolic	1.01 (1-1.02)	0.016		
Nocturnal systolic decrease (%)	0.99 (0.99-1)	0.926		
Nocturnal diastolic decrease (%)	1 (0.99-1)	0.382		
Blood preassure variability %)	0.96 (0.93-0.99)	0.017		
Pulse pressure	1 (0.99-1.01)	0.372		
Heart rate (mean)	0.99 (0.98-1)	0.818		

AHI, Index apnoea hypopnea; BMI, body mass index.
*Variables from AMBP were not included in the multivariable model to obtain predictors to select high risk individuals that would benefit from more extensive assessment of non-dipping like AMBP monitoring.

cardiovascular risk factors compared to other populations, their findings should be applied cautiously to populations like ours. To the best of our knowledge, only three studies, all conducted in Brazil, have examined this in the Latin population. Genta Pereira et al[23] included 153 individuals with hypertension with ND or reverse dipping and found a prevalence of OSA in 50% of them. Correa et al,[24] included 89 individuals with OSA and obesity who underwent ABPM. They found a 75% prevalence of ND pattern. Jenner et al[25] evaluated arterial stifness in patients with OSA and found a 54.7% prevalence of ND pattern. Given the characteristics of the individuals included in these studies (hypertense and obese) we hypothesised that their results might be overestimating the prevalence and predictive risk of OSA and ND for the general population.

Our findings suggest that daytime clinical assessment of HT it is still a useful tool, particularly in those individuals with moderate to severe OSA. Although daytime office BP does not replace the continuous measurement of blood pressure for 24 hours, it is a useful tool and can be done

Table 4 Oximetry values comparison between Dipping and Non-Dipping pattern.

OXYMETRY VALUES	AMBP PATTERN	n	media	Standar deviation	Normality Test p value	Difference of sample means (Shapiro Wilk)	Mann-whitney test for independent samples p value
Mean Oxygen saturation during respiratory events	Dipping	65	84,76	2,95	0,0010	1,6000	0,0150
	Non dipping	28	83,16	3,25	0,3190		
Mean Oxygen saturation in NREM	Dipping	65	89,69	2,36	0,0000	0,9400	0,2702
	Non dipping	28	88,75	4,28	0,0000		
Mean Oxygen saturation in REM	Dipping	65	89,09	3,55	0,0030	0,8400	0,3248
	Non dipping	28	88,25	4,81	0,0049		
Oxygen saturation overall mean value	Dipping	65	88,57	2,88	0,1345	1,1800	0,1312
	Non dipping	28	87,39	4,18	0,0039		

without the need of extensive and expensive ambulatory monitoring. We suggest that among OSA patients there is a particular population (older age men, smoking, and moderate to severe AHI) that is associated with ND pattern and nocturnal hypertension.

The variable most associated with cardiovascular impact of OSA (including HT) is hypoxemia. Recently hypoxemia is taking the lead from the AHI.[29,30] We hypothesize that the predictable lower oxygen saturation in high altitude of Bogota could be a differential factor and indeed we did find a lower mean oxigen saturation during respiratory events in patients with ND pattern. However, it was not the degree and hypoxemia burden that we would expect to Bogotás altutude; the altitude above sea level could lead to adaptive mechanisms to chonic hypoxia downplaying the value and impact of hypoxemia at high altitude. Data on healthy infants residing at Bogotás high altitude show higher apnea-hypopnea indexes and more prominent desaturation with respiratory events than do those living at low altitude.[31] At our best knowledge there are no current data of sleep hypoxemia threshold in adults at the altitude of Bogotá.

We found a relatively low number of individuals (14.9%) with nocturnal hypertension, which is markedly lower compared to the rates reported in other studies ((73.2%) (21), 50% (22), 61% (24)). Regarding this, it should be highlighted the caveat that our population was composed of hypertensive individuals without renal or cardiovascular complications that predispose to nocturnal hypertension and sympathetic hyperactivity. Also, point out that OSA is not the only cause of nocturnal hypertension. There are different conditions associated with nocturnal hypertension such as: advanced age, sedentariness, insomnia, diabetes mellitus, chronic kidney disease (CKD) and heart failure among others.[32,33] Aditionally, asians are likely to have nocturnal hypertension because of their higher salt intake and higher salt sensitivity.[33]

The clinical value of our findings is important in the context of settings where resources are scare and access to health care is limited; adequate risk assessment and adequate selection of individuals that would benefit from ABPM is highly needed. Our findings suggest that older individuals evaluated at the sleep clinic with severe OSA should be assessed for HT and those who smoke for ND. Cost-benefit studies needed to evaluate the need and utility of ABPM as a method to screen these individuals.

We think our results could be relevant to public health recommendations on the cardiovascular impact of smoking. Numerous studies have examined the association between smoking status and OSA,[34–36] however, few data exist on the risk difference for ND between smokers and non-smokers.[36,37] We found smoking to be an independent risk factor for ND in individuals with moderate to severe OSA.

Our study has several strengths. Firstly, we included a large population of middle-aged patients with diverse risk factors who were clinically assessed by trained specialists. Secondly, PSG and ABPM followed our institution protocol, reducing bias. The study has several limitations. Firstly, we

excluded individuals with mild or no OSA and this could account for selection bias. Secondly, low recruitment of participants could impact the true clinical value of our data. Third, recruitment from a single centre might not reflect real-life samples of outpatient patients. Fourth, as previously mentioned, our population was composed of hypertensive individuals without renal or cardiovascular complications that predispose to nocturnal hypertension and sympathetic hyperactivity. Finally, because of the feasibility of this study, the main limitation was the small sample size that limits the statistical analysis.

Conclusions

The prevalence of ND patterns in our group of middle-aged adults was lower compared to other populations. We found an association between moderate to severe OSA and HT, but not with ND pattern. Older individuals who have higher AHI are more likely to have HT, and those who smoke have a higher risk of ND. These findings add aditional information to the multiple mechanisms involved in the relationship between OSA and ND pattern and questions the routine use of 24-hour ABPM, particullary in our region, with limited resources and healthcare acces. However, further work with more robust methodology is needed to draw conclusions.

Conflict of Interest
None declared.

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