

Nocturnal Blood Pressure Patterns in Hypertensive Patients with Obstructive Sleep Apnea Syndrome in Angola

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Abstract: The main cardiovascular consequence of Obstructive Sleep Apnea Syndrome (OSAS) is arterial hypertension (AH), considered since 2003 a second cause of AH. This study aimed to evaluate nocturnal blood pressure patterns (BPP) in hypertensive patients with OSAS in Angola. A descriptive, cross-sectional observational study was carried out in a private clinic in Angola. Demographic and clinical variables, polysomnography, and ABPM parameters were included. Patients were classified as a dippers, non-dippers, and reverse dippers. One-way ANOVA test, Kruskal-Wallis's test, and chi-square test were used as appropriate. The sample consisted of 70 individuals: 39 (55.7%) were male. The mean age was 53.63 ± 9.14 years and the BMI was 35.82 ± 6.57 kg/m². An association between patients with a past medical history of hypertension and BPP was found ($p < 0.05$). Mean O₂ saturation was significantly lower in the reverse dippers ($p = 0.25$). Although insignificantly, the dippers were younger and less obese. In contrast, the proportion of smokers and diabetics was higher in the reverse dippers. No correlation was found between the apnea-hypopnea index, the mean blood pressure, and the Epworth sleepiness scale. In the present study, the prevalence of non-dipping and reverse dipping patterns was high. It needs to be confirmed with future prospective studies.

Keywords: Arterial hypertension; Non-dipper; Reverse dipper; Obstructive sleep apnea syndrome.

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1. Introduction

Sleep has a modeling function mediated by humoral, autonomic, and hemodynamic mechanisms. In sleep disorders, mainly in Obstructive Sleep Apnea Syndrome (OSAS), are altered due to constant and intermittent breathing pauses and consequent hypoxia that activates the sympathetic nervous system. The increase in sympathetic activity resulting from these intermittent breathing events during sleep is considered the most precise mechanism in developing cardiovascular disorders in individuals with OSAS [1].

The main cardiovascular consequence of OSAS is arterial hypertension (AH), considered since 2003 a second cause of AH, according to the seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure [2]. Nocturnal systolic and diastolic blood pressure tend to fall between 10 and 20%, to daytime values, which define the physiological dipping pattern. When this reduction is less than 10%, it is known as a non-dippers. On the other hand, the increase in nocturnal systolic or diastolic blood pressure is known as reverse dippers or

risers [3]. Ambulatory blood pressure measurement (ABPM) is a diagnostic tool increasingly used in clinical practice in the evaluation of hypertensive patients [4, 5]. The non-dipping pattern, as well as the inverted dipping pattern, have been associated with echocardiographic changes in left ventricular geometry [6, 7], left atrial function [8], increased cardiovascular and cerebrovascular risk, and increased morbidity and mortality with high prognostic value [9]. Absence or decrease in physiological reduction and increase in blood pressure during sleep are characteristics of AH associated with OSAS [10-12]. However, data on the prevalence of the reverse dipping pattern in patients with obstructive sleep apnea syndrome are scarce [13, 14].

The present study aims to evaluate 24-hours blood pressure patterns in hypertensive patients with OSAS in Angola.

2. Methodology

2.1 Study design

This observational, retrospective, and cross-sectional study included patients with OSAS followed up at the sleep consultation at Clínica Girassol in Luanda from April 2014 to December 2016 who also underwent ambulatory blood pressure measurement (ABPM). The information from database included: demographic and clinical variables, level 3 polysomnography, and ABPM parameters. Demographic and clinical variables were collected from a data collection instrument developed from the Berlin Questionnaire [16] and the STOP BANG Questionnaire [17] used in the Sleep Respiratory Pathology Outpatient Clinic of our institution. The degree of daytime sleepiness was assessed by ESS (possible score ranged from 0 to 24 points) [15]. All participants were informed and consent to participate for conduct this study. The confidentiality of the information extracted from the participants' records was maintained and the data extracted were secured and made available only to the study's principal investigators.

2.2 Study population

The study population included all patients monitored at the sleep consultation at Clínica Girassol who underwent both the OSAS study and the ABPM study in the referred period. Patients were classified as a dippers when there was a drop in systolic or diastolic blood pressure (BP) at night to daytime BP greater than 10%; as a non-dippers if this drop is not observed in systolic or diastolic BP, and as an reverse dippers when an increase in nocturnal systolic or diastolic BP is observed [3]. Patients were classified according to the severity of OSAS as mild if the apnea-hypopnea index (AHI) is from 5 to 14.9 events/hour, moderate if the AHI is from 15 to 29.9 events/hour, and severe when the AHI is equal to or greater than 30 events/hour, according to the guidelines of the American Academy Sleep Medicine 2007 [1]. Patients were further classified as having excessive daytime sleepiness (EDS) if they scored 10 or greater on the Epworth Sleepiness Scale (ESS), [15].

We included hypertensive patients aged 32 to 77 years who underwent the OSAS study, and the ABPM study. Patients who did not undergo the ABPM study and who had incomplete surveys were excluded.

2.3 Definition of Risk Factors and Comorbidities

Obstructive sleep apnea syndrome was defined as a clinical situation characterized by excessive daytime sleepiness, cardiorespiratory and neurocognitive disorders secondary to repeated episodes of total or partial obstruction of the upper airway that causes oxyhemoglobin desaturation and AHI > 5 events/hour [18]. Hypertension was defined based on a previous diagnosis of hypertension or treatment with antihypertensive therapy or hypertension diagnosed during ABPM. Diabetes mellitus was defined as a previous diagnosis of diabetes mellitus or treatment with antidiabetic drugs. Dyslipidemia

was defined as a previous diagnosis of dyslipidemia or treatment with any lipid-lowering medication. Obesity was defined as BMI>30kg/m². Smoking was defined based on current smoking (Yes/No). Alcoholic habits were defined based on the current status (Yes/No). Coffee consumption habits were defined based on the current status (Yes/No). Daytime sleepiness was assessed using the Epworth Sleepiness Scale [15].

2.4 Level 3 Polysomnography

Level 3 polysomnography (PSG3), also known as a cardiorespiratory sleep study, was performed on a modified portable system using Embletta MPR equipment for home diagnosis of OSAS. The PSG3 evaluates cardiorespiratory parameters by recording five parameters: oronasal breathing, respiratory noise, thoracoabdominal movements, body position, electrocardiographic tracing, and pulse oximetry [19]. The following variables were evaluated: apnea-hypopnea index (AHI), oxyhemoglobin desaturation index (ODI), minimum O₂ saturation and mean O₂ saturation. Data analysis was performed by visual inspection of the PSG3 record, as well as based on automatic calculations performed in the program in accordance with the Academy of American Medicine Sleep AASM 2007 standard. The Apnea-Hypopnea Index (AHI) is calculated by summing the number of respiratory events (apneas + hypopneas) per hour. The oxyhemoglobin desaturation index (ODI) is the result of the ratio between the number of desaturations resulting from the total collapse of the airway (apnea) lasting 10s or more, in association with hypopnea (partial collapse) with a 30% reduction of airway and oxyhemoglobin desaturation of 4% with the number of apnoeas and hypopneas recorded in the PSG3. The ODI was considered normal when equal or less than 5 events/hour and pathological if greater than 5 events/hour. [20].

2.5 Ambulatory Blood Pressure Measurement

The 24-hour ambulatory blood pressure measurement (ABPM) was performed using the IEM Model Mobil-O-Graph equipment (New Generation 24h ABPM Classic); the dataset was transferred and analyzed at the workstation with the HMC (Hypertension Management Software) program. Germany). The cuff was placed on the non-dominant arm and the patients were instructed to carry out their usual day-to-day activities without any restriction. Patients were instructed to record in the diary the time they went to sleep and when they woke up. The device was set to take BP readings at 15-minute intervals during the day (7:00–23:00 hours) and at 20-minute intervals during the night (23:00–7:00 hours). Each ABPM dataset was first automatically scanned to remove artifact readings according to preselected editing criteria. Systolic readings >290 or <60 mm/Hg and diastolic readings >195 or <30 mm/Hg were automatically removed. The recording was analyzed to obtain SBP and DBP averages over 24 hours during the day and night. The night pressure drop was calculated according to the following formula:

$$(\%) 100 \times [1 - (\text{night blood pressure}/\text{daytime blood pressure})]$$

2.6 Statistical analysis

The normality of the distribution was analyzed using the Shapiro-Wilks test. Categorical variables are described using absolute and relative frequencies. Continuous variables are expressed by the mean and standard deviation (SD) or by median and percentiles. One-way ANOVA test, Kruskal-Wallis test and chi-square test were used. Fisher's exact test was also used when chi-square assumptions were not met. No sample size was calculated. A significance level of $\alpha = 5\%$ was considered in all hypothesis tests. The analysis was performed using the Statistical Package for the Social Sciences program (SPSS, version 20.0).

3. Results

Of the 200 individuals diagnosed with OSAS and followed up at the sleep consultation at our institution, 70 patients who underwent both polysomnography, and ABPM and had the questionnaires duly completed were included in this study. Of these, 55.7% were male, with a mean age of 53.63 ± 9.14 years, and BMI was 35.82 ± 6.57 kg/m². The demographic and clinical characteristics of the study population and distributed by nocturnal pressure patterns (dipper vs non-dipper versus reverse dipper) are shown in Table 1, according to the total distribution.

Table 1. Distribution of demographic and clinical characteristics in the total population and according to nocturnal blood pressure characteristics.

	Total N= 70	Dipper N= 21	Non-Dipper N= 38	Reverse Dipper N=11	P-value
Age, mean+sd	53.63±9.14	51.1±10.52	53.95±8.68	57.36±6.86	.175 &
BMI	35 (32-40)	32 (31-39)	36 (32-41)	36 (31-41)	.350 Ø
Gender					.571 ¥
Male n (%)	39 (55.7)	13 (61.9)	19 (50.0)	7 (63.6)	
Female n (%)	31 (44.3)	8 (33.1)	19 (50.0)	4 (36.4)	
RF and Co-morbidities					
Diabetes Mellitus n (%)	17 (24.5)	5 (23.8)	9 (23.7)	3 (27.3)	.969 ¥
Hypercholesterolemia n (%)	24 (35.8)	8 (38.1)	13 (37.1)	3 (27.3)	.809 ¥
Obesity n (%)	61 (87.1)	16 (76.2)	36 (94.7)	9 (81.8)	.100 §
Smoking Habits n (%)	33 (47.1)	11 (52.4)	14 (36.8)	8 (72.7)	.094 ¥
Alcoholic Habits n (%)	27 (38.6)	10 (47.6)	15 (39.5)	2 (18.2)	.263 ¥
Coffee intake n (%)	27 (38.6)	10 (47.6)	15 (39.5)	2 (18.2)	.263 ¥
Hypertension					
Referred HT n (%)	63 (90.0)	17 (81.7)	37 (97.4)	9 (81.8)	.041§
Medicated HT n (%)	57 (83.4)	16 (76.2)	33 (86.8)	8 (72.8)	.403§
Symptomatology					
Snoring n (%)	54 (77.1)	15 (71.4)	30 (78.9)	9 (81.8)	.733§
Epworth SE , mean+sd	9.79±4.9	11.38±5.22	8.66±4.76	10.64±4.59	.108&
Epworth's SE					.269§
ESE < 10 n (%)	33 (47.1)	7 (33.3)	21 (55.3)	5 (45.5)	
ESE > 10 n (%)	37/52.9)	14(66.7)	17(44.7)	6(54.5)	
OSAS					.150§
Mild n (%)	28 (40.0)	9 (42.9)	15 (39.5)	4 (36.4)	
Moderate n (%)	24 (34.3)	7 (33.3)	16 (42.1)	1 (9.1)	
Severe n (%)	18 (25.7)	5 (23,8)	7 (18.4)	6 (54.5)	

Legend. FR - Risk factors, SE - Sleepiness scale, OSAS - Obstructive sleep apnea syndrome. &- One way ANOVA, ¥- Chi-square test; § - Fisher's exact test; Ø - Kruskal-Wallis test.

Obesity (87.1%) and smoking (47.1%) were the two main cardiovascular risk factors, followed by hypercholesterolemia and diabetes mellitus in 35.8 and 24.5%, respectively. In turn, alcoholic habits and coffee intake were reported in 38.6% of cases. Of the total sample, 90.0% of patients knew that they were hypertensive and 83.4% were undergoing therapy regularly. In 77.1% of patients, snoring was the complaint that most brought pa-

tients to the consultation. The registered levels of OSAS were mild, moderate, and severe in 40.0, 34.8, and 25.7%, respectively. The mean Epworth Sleepiness Scale (ESS) was 9.79 ± 4.9 , with excessive daytime sleepiness in 52.9% of cases. Nocturnal pressure patterns were classified as dippers in 30.0%, non-dippers in 54.3% and reverse dippers in 15.7%.

Table 2 shows the parameters of PSG3 and ABPM in the total population and distributed by nocturnal pressure patterns. In the total sample, the median AHI, ODI, and percentage minimum O₂ saturation and mean O₂ saturation were 18 events/hour, 18 events/hour, 77 and 94%, respectively.

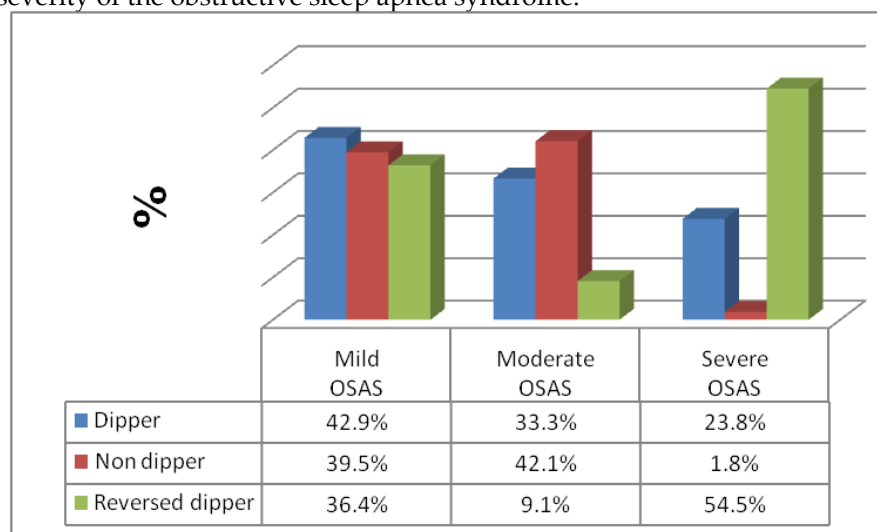
Table 2. Polysomnography and ABPM results in the total population and according to nocturnal blood pressure characteristic.

	Total N=70	Dipper N= 21	Non-dipper N= 38	Reverse Dipper N = 11	P-value
Polysomnography					
AHI (/h) Median (IQR)	18 (12-31)	17 (12-35)	17 (11-26)	33 (13-64)	.196 Ø
ODI Median (IQR)	18 (12-33)	18 (12-34)	15 (9-26)	33 (13-64)	.165 Ø
Percentage of O ₂ ST <90% Median (IQR)	2 (1-15)	2 (0.6-15)	2 (1-9)	14 (5-41)	.146 Ø
Minimal O ₂ Sat (%) Median (IQR)	77(69-84)	82 (69-86)	76 (66-84)	73 (70-83)	.469 Ø
Average O ₂ Sat (%) Median (IQR)	94 (93-95)	94 (93-95)	95 (93-96)	91 (91-93)	.025 Ø
ABPM					
24-hour SBP [mmHg] Median (IQR)	129 (118-140)	128 (116-141)	128 (118-139)	131 (125-145)	.320 Ø
24-hour DBP [mmHg] Median (IQR)	78 (72.84)	77 (73-82)	79 (72-85)	78 (69-90)	.974Ø
Daytime SBP [mmHg] Median (IQR)	132 (122-145)	136 (121-146)	131 (122-145)	138 (135-148)	.272Ø
Daytime DBP [mmHg] Median (IQR)	80 (76-86)	81 (79-88)	81 (76-86)	78 (72-86)	.299Ø
Nighttime SBP [mmHg] Median (IQR)	123 (116-135)	116 (104-130)	123 (116-135)	143 ((130-143)	<.0001Ø
Nighttime DBP [mmHg] Median (IQR)	72 (65-80)	65 (61-79)	74 (68-80)	82 (76-91)	<.0001Ø
Nocturnal SBP descent [mmHg] Mean± SD	6.87±7.74	14.45±4.48	5.34±3.73	-(8.59±7.88)	<.0001Ø
Nocturnal DBP descent [mmHg] Mean± SD	10.29±10.29	20.57±5.93	8.97±6.16	-(4.82±6.35)	<.0001Ø

Legend. ABPM - Ambulatory blood pressure measurement, AHI - Apnea hypopnea index, DBP - Diastolic blood pressure IQR - Interquartile range; ODI Oxyhemoglobin desaturation index, SBP - Systolic blood pressure, ST Saturation. Ø - Kruskal-Wallis test. *p<.050 **p<.010.

In the present study, except for AH in the past medical history, where a higher proportion of non-dippers was recorded when compared to the other two groups, we did not find significant differences in the other clinical and demographic variables. However, we can see that the dippers are younger and less obese and have more hypercholesterolemia than the reverse dippers. Regarding social habits, dippers had more smoking habits than non-dippers, but less than reverse dippers. As for alcoholic habits and coffee consumption, the dippers had a higher frequency of alcohol consumption when compared to the other two groups.

Snoring was more frequent in the inverted dipper, as well as the severity of the obstructive sleep apnea syndrome - 54.5% in the reverse dippers had severe OSAS (Figure 1). One aspect that seemed surprising to us was that the ESS value, which reflects the severity of daytime sleepiness, was higher in dippers than in non-dippers and similar in reverse dippers. Finally, except for the mean O₂ saturation, which was significantly lower in the reverse dippers (91%); we found no differences between the groups in the other polysomnographic variables. The oxyhemoglobin desaturation index and the percentage of saturation below 90% of total sleep time were higher in the reverse dippers.

Figure 1. Prevalence of the dipper, non-dipper and inverted-dipper pattern in relation to the severity of the obstructive sleep apnea syndrome.

Legend. OSAS - Obstructive sleep apnea syndrome.

As expected, mean nocturnal systolic and diastolic pressures were significantly higher on the reverse dippers than on the non-dippers and on the dippers (median and IQR) [143 (140-150) mmHg versus 123 (116-135) mmHg] versus [116 (104-130) mmHg; $p < 0.0001$] and 82 (76-91) mmHg 74 (68-86) mmHg versus 66 (61-79) mmHg, $p < 0.0001$, respectively], the same was observed in the means of nocturnal blood pressure drop. The other ABPM parameters were similar between the three groups. We found a correlation between the AHI and the other polysomnographic variables, but not with the mean daytime, nighttime, or 24-hour systolic or diastolic BP, nor with the ESS (see Table 3).

Table 3. Partial correlation between apnea-hypopnea index, Epworth sleepiness scale, nocturnal saturation of oxygen, and blood pressure (adjusted for age and body mass index).

		ESE	O ₂ Sat Nadir	O ₂ Sat Mean	24-hours SBP	Daytime SBP	Nighttime SBP	24-hours DBP	Daytime DBP	Nighttime DBP
AHI	R	.213	-.425	-.451	.197	.194	.123	-.078	.033	.021
	P Value	.088	.000**	.000**	.115	.112	.330	.539	.796	.866

Legend. AHI - Apnea Hypopnea Index. DBP - Diastolic Blood Pressure. ESS - Epworth Sleepiness Scale. SBP - Systolic Blood Pressure. O₂ Sat - Oxygen Saturation. ** $p < .010$.

4. Discussion

In Angola, there are no data on the prevalence of OSAS. On the other hand, it is known that in Angola, arterial hypertension has become an increasingly important public health problem. The prevalence of arterial hypertension in Angola ranged 23% (95% CI: 21% to 25.2%) to 38% (95% CI: 32.83-44.90%) [21, 22]. Studies indicate that among patients with hypertension, the frequency of OSAS is around 50% [10]. Therefore, it is expected that OSAS is also a highly prevalent syndrome in our country. To the best of our knowledge, this is the first study carried out in Angola that evaluated nocturnal BP patterns in an adult population of hypertensive patients with OSAS.

In this study, male, middle-aged, and obese patients were predominant, results that are in line with those reported in the literature. The meta-analysis performed by Cuspidi et al., showed that the percentage of men was 84%, the mean age range was 44 to 77 years and the mean BMI ranged from 24.0 ± 3.0 kg/m² to 34.0 ± 6.0 kg/m² [12]. Excessive daytime sleepiness (EDS) is a very common symptom reported by patients with OSAS [23].

In the present study, most patients (52.9%) had EDS, which is in line with what was found in other studies that report a prevalence of EDS between 50 and 80% [23]. The mean ESS score in our population was 9.79 ± 4.9 ; hypertensives with a dipping pattern had a higher mean ESS score than non-dippers and it was similar to reverse dippers. Conflicting data are reported in the literature regarding ESS in hypertensive vs non-hypertensive patients, most studies have shown that the mean ESS score in non-hypertensive patients was higher than in hypertensive patients [24, 25].

In contrast, Kapur et al., reported that the association of sleep-disordered breathing with hypertension is stronger in individuals who report daytime sleepiness than in those who do not [26], is justified in the excessive stimulation of the sympathetic nervous system observed in hypertensive patients and patients with OSAS [24, 25]. In our study, we found that this may also explain the findings since hypertensive patients with OSAS, the activation of the sympathetic system is even more exacerbated. On the other hand, OSAS can cause cognitive impairment that decreases the perception of excessive sleepiness, often confused with tiredness, fatigue, or lethargy, which could be another explanation to justify our results [20].

Obesity, type 2 diabetes mellitus, and hypercholesterolemia are the most frequent risk factors for both arterial hypertension and OSAS. Several studies explained that this is followed by other non-genetic factors linked to lifestyle (diet, physical activity, consumption of alcohol, tobacco) [27]. In our study, we found that the obesity and smoking were the two main risk factors for both arterial hypertension and OSAS, followed by hypercholesterolemia and diabetes mellitus. Hypoxia for intermittent periods due to the collapse of the upper airways in OSAS, lasting more than 5 hours, can reduce insulin sensitivity without adequate return of serum levels and, in addition, causes the death of beta cells, altering glycemic parameters. In 31 studies, changes in glycated hemoglobin were observed in cases of severe AHI [27]. In turn, chronic smoking promotes increased resistance of the upper airways, hypoxia, hypercapnia, and snoring due to inflammatory factors and changes in mucociliary consequently reduce the caliber and promote the collapse of the airways.

The stimulus of nicotine can cause important changes in sleep phases. The effects of smoking and OSAS on the development of arterial hypertension are well known. These two conditions have synergistic effects on increasing blood pressure, the risk of developing atherosclerosis, and increasing cardiovascular morbidity [28]. All this together may explain the fact that we found a higher proportion of diabetics and smokers in the reverse dippers when compared to the other two groups. Snoring is a frequent symptom in patients with OSAS reported in up to 92.4% of cases [29]. In our study, 77.1% of the patients had snoring as their main complaint and the frequency of snoring was higher in the reverse dippers (81.9%). The high prevalence of reverse dippers and non-dippers found in the present study is in line with what has been described by other authors. A meta-analysis carried out by Cuspidi et al., which included 1,562 patients with obstructive sleep apnea syndrome (OSAS), in 14 studies, revealed a prevalence of the non-dipper pattern between 30 and 90% [12]. However, data on the prevalence of inverted dipper patterns in patients with obstructive sleep apnea syndrome are scarce and revealed a prevalence of inverted dipper of 13 to 19% [13, 14].

While in the general population, this pattern is found in less than 5% [30]. In 56 individuals with OSAS (mean age 48.59 ± 13.27 years and BMI 27.53 ± 3.23 kg/m²) with and without arterial hypertension, Ma et al., found a prevalence of 19.6% of reverse dippers. In turn, in 307 normotensive individuals with OSAS, Lee et al., report a prevalence of reverse dippers (12.7%), very similar to ours. In both studies, the authors also found that dippers were relatively younger than non-dippers and reverse dippers [13, 14]. Ma et al. demonstrated that the non-dippers had a higher BMI than the dippers and reverse dippers and Lee et al., found no significant differences in BMI between the three groups. This contrasts with our study, where BMI, although not statistically significant, was higher in the reverse dippers when compared to the other two groups [13, 14].

Our results showed that there is a tendency for patients with severe OSAS to present a reverse dipping pattern. Patients with reverse dipping pattern had higher AHI, lower minimum saturation of O₂ and significantly lower mean saturation of O₂, which is consistent with the literature. The increase in BP in patients with OSAS is multifactorial and not fully understood. OSAS promotes an increase in BP due to sympathetic hyperactivity, systemic inflammation, oxidative stress, endogenous vasoactive factors, and endothelial dysfunction. The main feature of OSAS is persistent sympathetic hyperactivity, and sympathetic activity is exacerbated in patients with OSAS, resulting in peripheral vasoconstriction, BP elevation, even during wakefulness [10]. For individuals with OSAS, some authors believe that reverse dipping pattern is worse than a non-dipping due to higher nocturnal blood pressure combined with hypoxia and hypercapnia, causing a series of abnormal reactions and mechanisms. Others believe that the reverse dipping pattern is a transitional phase due to the increase in BP in OSAS, characterized by a sympathetic activation of greater magnitude than that observed in other patterns of nocturnal BP [13].

ABPM can be considered in the initial evaluation of patients once PSG has confirmed the diagnosis of OSAS. The prevalence of hypertension is underdiagnosed in patients with OSAS if BP is assessed only in the office [31, 32]. In addition, ABPM is a predictor of cardiovascular risk [31] and may be of particular importance in the assessment of hypertension in patients with OSAS, and should be suspected in all hypertensive patients, especially if the AH is predominantly diastolic, nocturnal, and refractory to treatment [11, 32, 33]. Finally, masked hypertension, defined as normal office BP with elevated ambulatory or home BP, is very common in patients with OSAS. The regular use of ABPM will “unmask” the masked hypertension in these patients [32, 33]. If ABPM is combined with polysomnography, which is also a non-invasive, simple, and inexpensive test, the sleep-wake period can be better defined to improve the accuracy of calculating the dipper, non-dipper and inverted dipper patterns [13].

However, we would like to emphasize that, according to Cuspidi et al., the available evidence on the association between OSAS and the inverted dipper pattern is still scarce. In addition, given the potential negative synergistic effect of these two conditions, studies focused on this topic must be made available, hence the importance of the work presented here [34]. This study has limitations, of which we highlight the fact that the sample size is small, which may limit the power of statistics to show differences between groups. Level 3 polysomnography was used, an exam that may have underestimated the AHI by recording the total time of the exam and not the sleep time with its efficiency, not knowing whether the respiratory events occurred during wakefulness or sleep. The strength of the study is that it was the first study of nocturnal BP patterns in hypertensive patients with OSAS in Angola.

5. Conclusion

In Angola, we found a high prevalence of inverted dipper and non-dipper patterns in hypertensive patients with OSAS. It is essential to carry out prospective studies to assess the prevalence of OSAS in hypertensive patients, especially in those with nocturnal or therapy-resistant hypertension.

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Supplementary Materials: None.

References

1. Flemons WW, Buysse D et al. The Report of American Academy of Sleep Medicine TaskForce. Sleep Related Breathing Disorders in Adults: Recommendations for Syndrome. Definition and Measurement Techniques in Clinical Research. *Sleep* 1999;22:667-89.
2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. *JAMA* 2003; 289: 2560–2572. doi:10.1001/jama.289.19.2560.
3. Parati G, Stergiou G, O'Brien E, Asmar R, Beilin L, Bilo G, Clement D, de la Sierra A, de Leeuw P, Dolan E, Fagard R, Graves J, Head GA, Imai Y, Kario K, Lurbe E, Mallion JM, Mancia G, Mengden T, Myers M, Ogedegbe G, Ohkubo T, Omboni S, Palatini P, Redon J, Ruilope LM, Shennan A, Staessen JA, van Montfrans G, Verdecchia P, Waeber B, Wang J, Zanchetti A, Zhang Y. European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular variability. European Society of Hypertension practice guidelines for ambulatory blood pressure monitoring. *J Hypertens*. 2014 Jul;32(7):1359-66. doi: 10.1097/HJH.0000000000000221.
4. Madin K, Iqbal P. Twenty four hour ambulatory blood pressure monitoring: a new tool for determining cardiovascular prognosis. *Postgrad Med J*. 2006; 82(971): 548–551, doi:10.1136/pgmj.2006.046409.
5. O'Brien E, Kario K, Staessen JA, de la Sierra A, Ohkubo T. Patterns of ambulatory blood pressure: clinical relevance and application. *J Clin Hypertens (Greenwich)*. 2018 Jul;20(7):1112-1115. doi: 10.1111/jch.13277.
6. Feijão A, Victória Pereira S, Cardona L, Morais H. The influence of dipper vs non-dipper pattern on left ventricular function and ascending aorta dimensions in hypertensive patients in Angola. *Arterial Hypertension* 2019;23(3):197-202. doi:10.5603/AH.a2019.0014
7. Cuspidi C, Tadic M, Sala C, Carugo S, Mancia G, Grassi G. Reverse dipping and subclinical cardiac organ damage: a meta-analysis of echocardiographic studies. *J Hypertens*. 2021 Aug 1;39(8):1505-1512. doi: 10.1097/HJH.0000000000002836
8. Tadic M, Cuspidi C, Pencic B, Mancia G, Grassi G, Kocijancic V, Quarti-Trevano F, Celic V. Impact of different dipping patterns on left atrial function in hypertension. *J Hypertens*. 2020 Nov;38(11):2245-2251. doi: 10.1097/HJH.0000000000002542.
9. Cuspidi C, Sala C, Tadic M, Gherbesi E, De Giorgi A, Grassi G, Mancia G. *J Clin Hypertens (Greenwich)*. Clinical and prognostic significance of a reverse dipping pattern on ambulatory monitoring: An updated review. 2017 Jul;19(7):713-721. doi: 10.1111/jch.13023.
10. Furlan SF, Braz CV, Lorenzi-Filho G, Drager LF. Management of Hypertension in Obstructive Sleep Apnea. *Curr Cardiol Rep*. 2015 Dec;17(12):108. doi: 10.1007/s11886-015-0663-z.
11. Parati G, Ochoa JE, Bilo G, Mattaliano P, Salvi P, Kario K, Lombardi C. Obstructive sleep apnea syndrome as a cause of resistant hypertension. *Hypertens Res*. 2014 Jul;37(7):601-13. doi: 10.1038/hr.2014.80.
12. Cuspidi C, Tadic M, Sala C, Gherbesi E, Grassi G, Mancia G. Blood Pressure Non-Dipping and Obstructive Sleep Apnea Syndrome: A Meta-Analysis. *J Clin Med*. 2019 Sep 2;8(9):1367. doi:10.3390/jcm8091367.
13. Ma Y, Sun S, Peng CK, Fang Y, Thomas RJ. Ambulatory Blood Pressure Monitoring in Chinese Patients with Obstructive Sleep Apnea. *J Clin Sleep Med*. 2017 Mar 15;13(3):433-439. doi:10.5664/jcsm.6498.
14. Lee S, Thomas RJ, Kim H, Seo HS, Baik I, Yoon DW, Kim SJ, Lee SK, Shin C.. Association between high nocturnal blood pressure and white matter change and its interaction by obstructive sleep apnoea among normotensive adults. *J Hypertens*. 2014;32:2005-2012. doi:10.1097/HJH.0000000000000290.
15. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*. 1991 Dec;14(6):540-5. doi:10.1093/sleep/14.6.540.
16. Senaratna CV, Perret JL, Matheson MC, Lodge CJ, Lowe AJ, Cassim R, Russell MA, Burgess JA, Hamilton GS, Dharmage SC. Validity of the Berlin questionnaire in detecting obstructive sleep apnea: A systematic review and meta-analysis. *Sleep Med Rev*. 2017 Dec;36:116-124. doi:10.1016/j.smrv.2017.04.001.
17. Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire: A Practical Approach to Screen for Obstructive Sleep Apnea. *Chest*. 2016 Mar;149(3):631-8. doi:10.1378/chest.15-0903.
18. Geer JH, Hilbert J. Gender Issues in Obstructive Sleep Apnea. *Yale J Biol Med* 2021 Sep 30;94(3):48
19. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman J Jr, Friedman L, Hirshkowitz M, Kapen S, Kramer M, Lee-Chiong T, Loubé DL, Owens J, Pancer JP, Wise M. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep*. 2005;28:499–521. doi:10.1093/sleep/28.4.499.
20. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*. 2007 Jul;132(1):325-37. doi: 10.1378/chest.07-0040
21. Pires JE, Sebastião YV, Langa AJ, Nery SV. Hypertension in Northern Angola: prevalence, associated factors, awareness, treatment and control. *BMC Public Health*. 2013; 13: 90, doi: 10.1186/1471-2458-13-90.
22. Paquissi, F.C., et al., Hypertension among Outpatients at a General Hospital in South Angola: Prevalence, Awareness, Treatment, and Control. *Clin Med Insights Cardiol*, 2016. 10: p. 111-6.
23. Rosenberg R, Schweitzer PK, Steier J, Pepin JL. Residual excessive daytime sleepiness in patients treated for obstructive sleep apnea: guidance for assessment, diagnosis, and management. *Postgrad Med*. 2021 Sep;133(7):772-783. doi: 10.1080/00325481.2021.1948305. Epub 2021 Jul 22.

24. Tam W, Ng SS, To KW, Ko FW, Hui DS. The interaction between hypertension and obstructive sleep apnea on subjective daytime sleepiness. *Clin Hypertens (Greenwich)*. 2019 Mar;21(3):390-396. doi: 10.1111/jch.13485.
25. Ulander M, Hedner J, Stillberg G, Sunnergren O, Grote L. Correlates of excessive daytime sleepiness in obstructive sleep apnea: Results from the nationwide SESAR cohort including 34,684 patients. *J Sleep Res*. 2022 Dec;31(6):e13690. doi: 10.1111/jsr.13690.
26. Kapur VK, Resnick HE, Gottlieb DJ, Sleep Heart Health Study Group. Sleep disordered breathing and hypertension: does self-reported sleepiness modify the association? *Sleep*. 2008 Aug;31(8):1127-32.
27. Tahrani AA, Ali A. Obstructive sleep apnoea and type 2 diabetes. *Eur Endocrinol*. 2014;10(1):43-50. doi:10.17925/EE.2014.10.01.43
28. Zhu H, Xu H, Chen R, Liu S, Xia Y, Fu Y, et al. Smoking, obstructive sleep apnea syndrome and their combined effects on metabolic parameters: evidence from a large cross-sectional study. *Sci Rep*. 2017;7:8851. doi:10.1038/s41598-017-08930
29. Bailly S, Destors M, Grillet Y, Richard P, Stach B, Vivodtzev I, Timsit JF, Lévy P, Tamisier R, Pépin JL; scientific council and investigators of the French national sleep apnea registry (OSFP). Obstructive Sleep Apnea: A Cluster Analysis at Time of Diagnosis. *PLoS One*. 2016 Jun 17;11(6):e0157318. doi: 10.1371/journal.pone.0157318.
30. Ohkubo T, Imai Y, Tsuji I, Nagai K, Watanabe N, Minami N, Kato J, Kikuchi N, Nishiyama A, Aihara A, Sekino M, Satoh H, Hisamichi S.. Relation between nocturnal decline in blood pressure and mortality. *Am J Hypertens*. 1997;10:1201-1207. doi:10.1016/s0895-7061(97)00274-4.
31. Wolf J, Hering D, Narkiewicz K. Non-dipping pattern of hypertension and obstructive sleep apnea syndrome. *Hypertens Res*. 2010;33(9):867–871. doi: 10.1038/hr.2010.153.
32. Baguet JP, Boutin I, Barone-Rochette G, Levy P, Tamisier R, Pierre H, Boggetto-Graham L, Pépin JL. Hypertension diagnosis in obstructive sleep apnea: self or 24-hour ambulatory blood pressure monitoring? *Int J Cardiol*. 2013 Sep 1;167(5):2346-7. doi: 10.1016/j.ijcard.2012.11.037.
33. Baguet JP, Hammer L, Lévy P, Pierre H, Rossini E, Mouret S, Ormezzano O, Mallion JM, Pépin JL. Night-time and diastolic hypertension are common and underestimated conditions in newly diagnosed apnoeic patients. *J Hypertens*. 2005;23(3):521–527. doi:10.1097/01.hjh.0000160207.58781.4e.
34. Cuspidi C, Gherbesi E, Tadic M. Is obstructive sleep apnoea the most important determinant of reverse dipping? Hypothesis and evidence. *J Clin Hypertens (Greenwich)*. 2019 Oct;21(10):1594-1595. doi:10.1111/jch.13682.