Hypertension Research (2022) 45:1468–1475
https://doi.org/10.1038/s41440-022-00950-z

ARTICLE

Orthostatic hypotension and night-time dipper patterns in geriatric outpatients

Lavinia M. A. Patetta[1] · Alessandro Reffo[1] · Caterina Trevisan[1,2] · Chiara Curreri[1] · Valter Giantin[1] · Alessandro Franchin[1] · Giuseppe Sergi[1]

Received: 10 November 2021 / Revised: 22 April 2022 / Accepted: 26 April 2022 / Published online: 9 June 2022

Abstract

Orthostatic hypotension (OH) and blood pressure circadian dysfunctions are common in older adults and may be related to aging-related autonomic nervous system deficits. This study aimed to evaluate the relationship between orthostatic and nocturnal blood pressure changes in geriatric outpatients. This cross-sectional study was carried out with 425 Italian individuals aged ≥65 years (mean age 75.8 ± 7.1 years) who attended a hypertension outpatient clinic from January 2013 to January 2020. Each patient underwent orthostatic testing and noninvasive 24-h blood pressure monitoring (ABPM). OH was detected in 38.1% of patients, and these individuals were more likely to have abnormal circadian blood pressure patterns (reverse and nondipper) than those without OH (61.7% vs. 51.7%; $p = 0.045$). In linear regression, after adjusting for potential confounders, orthostatic and nocturnal changes in systolic blood pressure were inversely associated ($\beta = -0.63$, 95% CI [−0.95; −0.32]; $p < 0.001$). This association was stronger in patients ≥80 years. OH is highly prevalent in older patients and is associated with altered nocturnal blood pressure profiles, especially in the oldest old. Because both OH and altered blood pressure patterns are associated with elevated cardiovascular risk and mortality, our study suggests that elderly patients with OH should undergo noninvasive 24-h blood pressure monitoring.

Keywords Ambulatory blood pressure monitoring · Aged · Orthostatic hypotension · Prevalence

Introduction

Orthostatic hypotension (OH) is a common health problem in the elderly population, and its prevalence increases with advancing age [1–3]. The current literature suggests that OH has both clinical and prognostic importance. Despite some conflicting results [4], many studies have shown OH to be associated with an increased incidence of cardiocerebrovascular events and mortality [5, 6].

Hypertension is frequent in patients with OH [7], and OH may be associated with alterations in blood pressure (BP) control. In this study, we focused not only on OH but also on circadian BP profiles, i.e., extreme dipper, dipper, reverse dipper or nondipper patterns, which can be easily evaluated through noninvasive 24-h blood pressure monitoring (ABPM). Like OH, some nocturnal profiles, such as reverse dipper or nondipper patterns, tend to present more frequently with advancing age [8] and have been associated with higher cardiovascular risk [9].

The literature contains reports of a relationship between orthostatic BP and nocturnal changes in systolic BP (SBP) [10–12]. In particular, it has been shown that lower orthostatic BP may contribute to diurnal and nocturnal BP profiles [11, 12]. Since both OH and altered nocturnal BP profiles are related to autonomic system deficits and since such dysfunctions are more common in older people [13–16], we hypothesized that the association between OH and abnormal dipping BP patterns may be stronger in individuals with advanced age. The aim of this study was therefore to

These authors contributed equally: Lavinia M. A. Patetta, Alessandro Reffo.

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41440-022-00950-z.

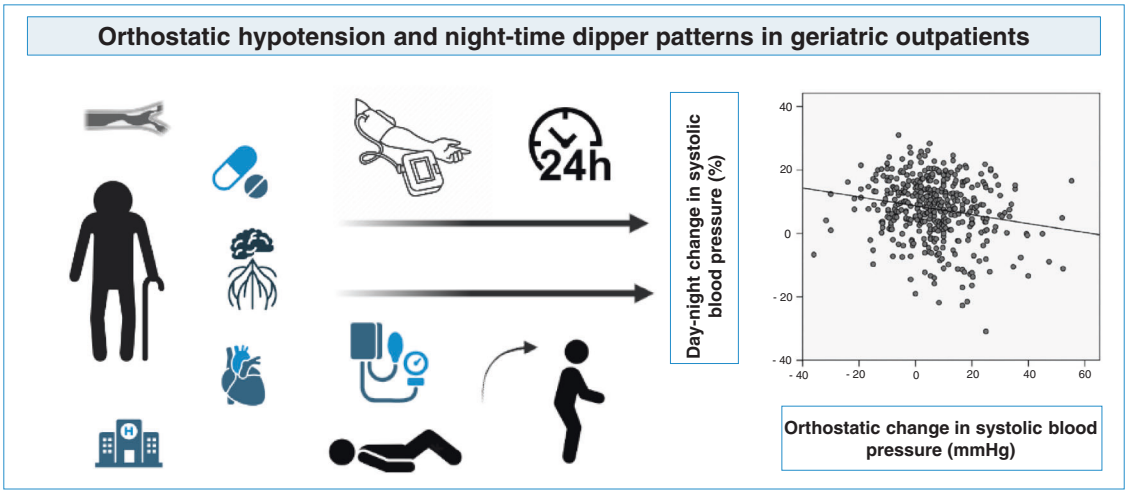
✉ Lavinia M. A. Patetta
lavinia.patetta@gmail.com

[1] Department of Medicine (DIMED), Geriatrics Division, University of Padua, Padua, Italy

[2] Department of Medical Sciences, University of Ferrara, Ferrara, Italy

Graphical Abstract

Orthostatic and nocturnal changes in systolic blood pressure are inversely associated in older adults, probably due to aging-related autonomic nervous system deficits.



investigate the relationship between OH and day-night blood pressure changes in a sample of older outpatients.

Methods

Study design

This was a cross-sectional study. The flow chart of the sample selection is shown in Fig. 1. Of the 510 individuals aged ≥65 years who visited the Hypertension Outpatient Clinic of the Geriatric Division at the University Hospital of Padua from January 2013 to January 2020, we selected 440 who satisfied the following criteria: (1) had stable health status in the last 6 months (i.e., absence of hospitalizations and acute clinical conditions); (2) underwent orthostatic pressure tests; and (3) underwent ABPM. Those with severe obesity (BMI > 40 kg/m²), underweight (BMI < 18 kg/m²), a history of collagenopathies, ongoing cancer therapy (e.g., chemotherapy, radiation therapy or immunotherapy), and any type of arrhythmia (e.g., extra beats, tachy-brady syndrome, atrioventricular blocks, ventricular arrhythmia) in their medical history except for atrial fibrillation [17, 18] (confirmed by medical records or electrocardiogram) were excluded. From the initial sample of 440 individuals, 10 outpatients were excluded because of incomplete ABPM data, and 5 were excluded because of incomplete orthostatic test data, resulting in a final analytical sample of 425 patients.

Sample size

The current literature suggests that the prevalence of OH in the 1st and 3rd minutes in older outpatients is approximately

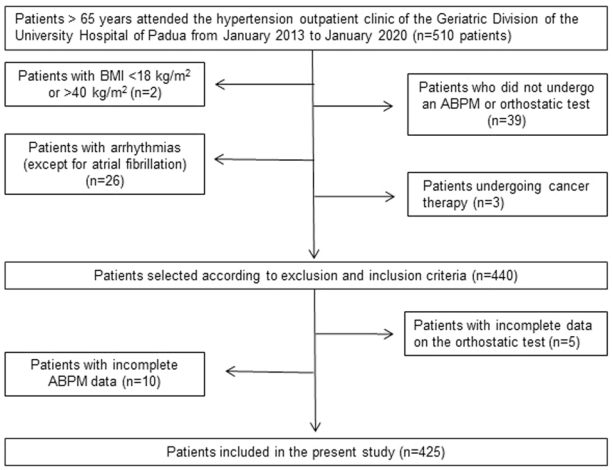


Fig. 1 Flow chart. BMI body mass index, ABPM ambulatory blood pressure monitoring

30% [1–3]. Since we had data to detect OH at the 5th minute as well, we expected to find an increased prevalence of OH of approximately 10%, reaching 40%. Assuming an $\alpha = 0.05$ and a power of 80%, we estimated that a sample of at least 178 individuals would be necessary to detect such OH prevalence.

Patient characteristics

Physicians collected anthropometric data, medical history, and information on ongoing treatments and lifestyle (smoking and alcohol consumption habits) for each patient. Among the chronic diseases, we considered arterial hypertension, history of cardiovascular diseases, hyperlipidemia, stroke, chronic pneumopathies, diabetes mellitus, liver

disease, and previous or current cancer with no ongoing targeted therapy (either for no medical indication or patient refusal). Concerning cardiovascular diseases, we considered chronic congestive heart failure, angina pectoris, history of myocardial infarction, atrial fibrillation, and peripheral artery disease. Hypertension was defined as a systolic BP ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg [19] or ongoing use of antihypertensive medication. The diagnosis of diabetes mellitus or lipid disorders was derived from the patients' medical records.

The study was carried out in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments. The study protocol was approved by the local ethics committee, and all participants (or their next of kin) were fully informed about the nature, purpose, and procedures of the study and gave their written informed consent.

Orthostatic test

After 5 min of rest, BP was measured three times in the sitting position on both arms using a gallium auscultatory sphygmomanometer with an appropriately sized cuff. Following this, heart rate and BP were measured 3 times with the participant in the supine position and then 3 times after 1, 3, and 5 min of standing. Any symptoms occurring while the measurements were being taken were also recorded. The presence of OH was defined as a decrease of at least 20 mmHg in systolic BP and/or 10 mmHg in diastolic BP at each time interval after assuming a standing position [20]. We defined supine BP as the average of the 3 supine BP measurements and supine hypertension (SH) as the presence of supine SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg [21]. As an additional measure, we considered orthostatic changes in SBP, calculated as the difference between the averages of the 3 supine and the 3 standing BP measures. The measurements were taken for all patients in the morning.

24-hour ABPM

Twenty-four-hour ABPM was performed with a Takeda TM-2430 (A&D Company, Tokyo, Japan). This device employs the oscillometric method and has been validated [22]. An appropriately sized cuff was placed on the arm on which the highest blood pressure had been registered. The following BP parameters were recorded over at least 24 h every 15 min during the day (7:00 AM to 10:00 PM) and every 20 min during the night (10:00 PM to 7:00 AM): mean blood pressure (MBP), systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR). Over the same 24-h period, patients recorded their activities, particularly mealtimes and sleep times. We considered an ABPM to be valid if the duration was 24 h and at least 70% of measurements were valid. Diurnal and nocturnal blood pressure (BP) were defined as the mean BP values during the waking time and the sleeping time, respectively. BP variability was evaluated by standard deviation (SD). Patients were classified into four groups based on the percentage variation in nocturnal BP compared with diurnal BP (night-time BP change): reverse dipper, if it was <0%; nondipper if it was 0–10%; dipper if it was 10–20%; and extreme dipper, if it was >20% [17]. Nocturnal dipper profiles were grouped into "nondip" (ND), which included reverse dippers and nondippers, or "dip" (D), which included dippers and extreme dippers.

Statistical analysis

All statistical analyses were performed in SPSS version 25 (IBM Corp., Armonk, NY). Data are expressed as the means ± SDs for continuous variables or as counts and percentages for categorical variables. Patients were divided into groups according to the presence/absence of OH and their age (65–79 years vs. over 80 years). The outpatients' characteristics were compared with Student's t test, ANOVA, Mann–Whitney or Kruskal–Wallis tests, as appropriate, depending on the normal distribution of the continuous variables. The χ^2 test was instead used to compare the frequencies of the categorical variables between groups. Pearson's correlation was used to investigate the association between orthostatic change in SBP (mmHg) and day-night changes in SBP (%). This relationship was also analyzed by linear regression, considering orthostatic changes in SBP expressed as 5 mmHg increases as the exposure. The relationship between OH and nondipper profiles was studied by binary (considering nondipper vs. dipper profile, as an outcome) and multinomial (considering dipper vs. reverse, nondipper, or extreme dipper, as an outcome) logistic regressions. Linear and logistic regressions were adjusted for factors that could be potential confounders in the studied association. These factors were selected based on the results of the univariate analysis and on the scientific rationale that could link them to both the exposure and the outcome with a confounding role (for a causal diagram, please see Supplementary Fig. 1). The final set of confounders included age, sex, alcohol intake, cardiovascular disease, antihypertensive polytherapy (≥3 drugs), 24-h BP and supine BP. Statistical significance was set at $p < 0.05$.

Results

The study was carried out on 425 patients with a mean age of 75.8 ± 7.1 years (range 65–97 years), 128 (30.1%) of whom were over 80 years of age. Most of the participants

Table 1 Characteristics of the total sample and by presence/absence of orthostatic hypotension

	All	OH	No OH	*p* value
N (%)	425	162 (38.1%)	263 (61.9%)	
Sex (female)	274 (64.5%)	89 (54.9%)	185 (70.3%)	0.002
Age (years)	75.8 ± 7.1	76.6 ± 6.9	75.4 ± 7.2	0.06
BMI (kg/m^2)	27.3 ± 4.0	27.1 ± 4.0	27.5 ± 4.0	0.96
Alcohol (≥3 AU/day)	134 (31.5%)	56 (34.6%)	78 (29.7%)	0.33
N° chronic diseases	1.5 ± 1.5	1.7 ± 1.6	1.4 ± 1.4	0.06
Arterial Hypertension	330 (77.6%)	140 (86.4%)	190 (72.2%)	0.001
Cardiovascular diseases	77 (18.1%)	36 (22.2%)	41 (15.6%)	0.05
Chronic congestive heart failure	24 (5.6%)	12 (7.4%)	12 (4.6%)	0.28
Myocardial infarction	20 (4.7%)	10 (6.2%)	10 (3.8%)	0.35
Angina pectoris	15 (3.5%)	8 (4.9%)	7 (2.7%)	0.28
Peripheral artery disease	37 (8.7%)	18 (11.1%)	19 (7.2%)	0.21
Atrial Fibrillation	106 (24.9%)	56 (34.6%)	50 (19%)	<0.001
Stroke	34 (8.0%)	10 (6.2%)	24 (9.1%)	0.36
Hyperlipidemia	137 (32.2%)	42 (25.9%)	95 (36.1%)	0.03
Diabetes Mellitus	62 (14.6%)	30 (18.5%)	32 (12.2%)	0.08
Fall history	76 (17.9%)	38 (23.5%)	38 (14.4%)	0.02
Antihypertensive drugs	307 (72.2%)	130 (80.2%)	177 (67.3%)	0.004
N° antihypertensive drugs	1.6 ± 1.4	1.9 ± 1.3	1.5 ± 1.4	<0.001
Thiazide diuretics	107 (34.8%)	43 (26.5%)	64 (24.3%)	0.64
Loop diuretics	58 (18.8%)	37 (22.8%)	21 (7.9%)	<0.001
Potassium-sparing diuretics	37 (12.1%)	12 (7.4%)	25 (9.5%)	0.48
β-blockers	115 (37.4%)	55 (33.9%)	60 (22.8%)	0.01
Calcium channel blockers	100 (32.5%)	48 (29.6%)	52 (19.8%)	0.02
α-blockers	25 (8.1%)	14 (8.6%)	11 (4.2%)	0.08
alpha-2- agonists	11 (3.5%)	5 (3.1%)	6 (2.3%)	0.75
Ace-inhibitors	134 (43.6%)	57 (35.2%)	77 (29.3%)	0.23
ARBs	106 (34.5%)	37 (22.8%)	69 (26.2%)	0.48

BMI body mass index, *AU* alcohol unit, *OH* orthostatic hypotension, *ACE-inhibitors* angiotensin-converting enzyme – inhibitors, *ARBs* angiotensin receptor blockers

were women ($n = 274$, 64.5%), 16.5% had a BMI > 30 kg/m^2, and only two patients had autonomic dysfunction related to Parkinson's disease. Prior to ABPM, 77.6% of subjects reported having already had a diagnosis of arterial hypertension, and 72.2% were on antihypertensive therapy.

The prevalence of OH was 38.1%. As shown in Table 1, patients with OH had a higher frequency of arterial hypertension, atrial fibrillation, dyslipidemia, cardiovascular disease, and previous falls. The OH group also took more antihypertensive drugs and were more likely to be undergoing treatment with β-blockers, calcium channel blockers, and loop diuretics.

Supine BP and orthostatic SBP and DBP changes up to the 5th minute in OH and non-OH patients are shown in Table 2. The decrease in pressure was more pronounced at 1 min after standing and was greater for systolic BP. During orthostatic testing, 28.2% of patients presented with symptoms (e.g., feeling of dizziness, instability or lightness headed), and the majority were patients with OH (36.4% vs. 23.2%, $p = 0.002$).

The 24-h ambulatory blood pressure measures are shown in Table 3. ABPM yielded an average of 93.8 ± 6.8% valid measurements. Patients with OH had higher standard deviation (SD) values in the 24-h SBP, day-time SBP, and night-time SBP measures. The frequencies of the various nocturnal blood pressure profiles in the total sample were 17.6% reverse dippers, 37.9% nondippers, 36.2% dippers, and 8.2% extreme dippers. The reverse dipper profile was more frequent in patients over 80 years of age than in those under 80 years (26.6% vs. 13.8%, $p = 0.002$).

The OH group was more likely than the non-OH group to present ND profiles (61.7% vs. 51.7%; $p = 0.045$) and had a higher frequency of reverse dippers (22.2% vs. 14.8%; $p = 0.036$). In logistic regression, after adjusting for confounders, we found a significant association between OH and ND profiles (OR = 1.67 [95% CI: 1.07–2.59],

Table 2 Blood pressure measures during orthostatic test

	All ($n = 425$)	OH ($n = 162$)	No OH ($n = 263$)	*P* value
Orthostatic BP changes				
Supine SBP (mmHg)	144 ± 20	148 ± 21	141 ± 19	<0.001
Supine DBP (mmHg)	82 ± 11	84 ± 11	80 ± 11	<0.001
Supine Hypertension	275 (64.9%)	119 (73.5%)	156 (59.3%)	0.02
Orthostatic SBP 1 min (mmHg)	136 ± 21	129 ± 21	140 ± 20	0.24
Orthostatic SBP 3 min (mmHg)	136 ± 21	130 ± 20	140 ± 20	<0.001
Orthostatic SBP 5 min (mmHg)	138 ± 21	132 ± 20	140 ± 20	<0.001
Orthostatic DBP 1 min (mmHg)	81 ± 12	78 ± 12	84 ± 12	<0.001
Orthostatic DBP 3 min (mmHg)	82 ± 12	79 ± 12	84 ± 11	<0.001
Orthostatic DBP 5 min (mmHg)	83 ± 12	80 ± 12	84 ± 11	<0.001
Orthostatic changes in SBP (mmHg)	7 ± 14	17 ± 14	0 ± 9	0.001
Orthostatic changes in DBP (mmHg)	0 ± 8	6 ± 7	−3 ± 5	0.004

OH orthostatic hypotension, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

Table 3 24-hour ambulatory blood pressure measures

	All ($n = 425$)	OH ($n = 162$)	No OH ($n = 263$)	*p* value
24-hour BP				
24-hour SBP (mmHg)	135 ± 14	134 ± 15	135 ± 14	0.77
24-hour DBP (mmHg)	74 ± 7	74 ± 7	74 ± 7	0.86
SD of 24-hour SBP (mmHg)	20 ± 5	21 ± 5	19 ± 5	0.03
SD of 24-hour DBP (mmHg)	14 ± 3	14 ± 4	14 ± 4	0.19
24-hours HR (bpm)	70 ± 9	70 ± 9	69 ± 9	0.07
Day time BP				
Day time SBP (mmHg)	138 ± 14	137 ± 14	139 ± 14	0.30
Day time DBP (mmHg)	77 ± 7	76 ± 7	76 ± 7	0.55
SD of day time SBP (mmHg)	19 ± 5	20 ± 5	19 ± 5	0.01
SD of day time DBP (mmHg)	14 ± 4	15 ± 4	13 ± 4	0.05
Day time HR (bpm)	72 ± 9	73 ± 9	72 ± 9	0.30
Night time BP				
Night time SBP (mmHg)	128 ± 19	129 ± 19	127 ± 18	0.12
Night time DBP (mmHg)	69 ± 9	69 ± 9	68 ± 8	0.25
SD of night time SBP (mmHg)	15 ± 5	16 ± 5	15 ± 5	0.05
SD of night time DBP (mmHg)	10 ± 3	10 ± 3	10 ± 4	0.83
Night time HR (bpm)	64 ± 9	66 ± 9	62 ± 9	0.001
Day-night changes in BP				
Day-night changes in SBP (mmHg)	10 ± 13	8 ± 14	12 ± 13	0.01
Day-night changes in DBP (mmHg)	8 ± 7	7 ± 8	8 ± 7	0.01
Dipper profiles				0.045
Non-Dipper	236 (55.5%)	100 (61.7%)	136 (51.7%)	
Dipper	189 (44.5%)	62 (32.8%)	127 (48.3%)	

OH orthostatic hypotension, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *SD* standard deviation

$p = 0.02$), particularly reverse dipper (OR = 2.19 [95% CI: 1.16–4.18], $p = 0.016$) (Supplementary Table 1). Considering only patients with OH, we found that ND profiles were more frequent in those aged ≥80 years ($n = 41$) than in those aged 65–79 years ($n = 59$) (ND frequency: 78.8% vs. 53.6%; $p = 0.001$).

In simple linear regression, we observed an inverse relationship between orthostatic SBP changes and nighttime

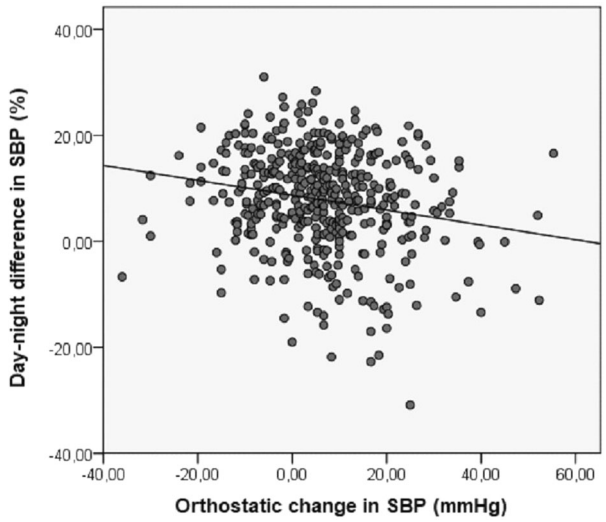


Fig. 2 Regression line for the relationship between orthostatic and day-night changes (%) in systolic blood pressure. SBP systolic blood pressure

SBP changes ($\beta = -0.63$ [95% CI: −0.95, −0.32], $p < 0.001$) (Fig. 2). In the multivariable analysis, we confirmed that each 5 mmHg increase in orthostatic SBP change was associated with an average reduction of 0.80% in day-night SBP change (Table 4).

Although the interaction between age group and orthostatic SBP changes was not significant ($p = 0.27$), the association between night-time SBP change (%) and orthostatic SBP change was stronger in patients ≥80 years. Furthermore, for patients >80 years of age, this association was stronger after considering all confounders (Table 4). Similar results were also observed for DBP.

Table 4 Linear regression between orthostatic and day-night changes in systolic blood pressure

	Model 1[a]		Model 2[b]	
	β coefficients (95% CI)	*p* values	β coefficients (95% CI)	*p* values
All ($n = 425$)				
Per 5 mmHg increase in orthostatic SBP change	−0.61 (−0.92; −0.29)	<0.001	−0.80 (−1.12; −0.48)	<0.001
Age < 80 years ($n = 128$)				
Per 5 mmHg increase in orthostatic SBP change	−0.47 (−0.84; −0.11)	0.01	−0.70 (−1.09; −0.30)	0.001
Age ≥ 80 years ($n = 297$)				
Per 5 mmHg increase in orthostatic SBP change	−0.89 (−1.49; −0.29)	0.004	−1.00 (−1.58; −0.42)	0.001

CI confidence interval, *SBP* systolic blood pressure

[a]Model 1 is adjusted for age and sex

[b]Model 2 is adjusted for age, sex, alcohol consumption (≥3 UA/day), cardiovascular disease, antihypertensive drugs, 24-h SBP and supine SBP

Discussion

This study confirms the relationship between postural changes and nocturnal BP in elderly patients attending a hypertension outpatient clinic. Orthostatic BP changes were associated with more marked alterations in the nocturnal BP profile in patients over 80 years of age.

The prevalence of OH in our sample was higher than that reported in patients in similar care settings [1–3, 7], probably because of the older age of our population (mean age 75.8 years). Several studies have shown a higher frequency of OH in elderly individuals [1–3], which may be related to the physiological aging process. The stiffer myocardium and the concomitant diastolic heart dysfunction of older people precipitate stroke volume reduction as a result of orthostatic-induced diminished preload, while increased arterial stiffness contributes to the inadequate vasoconstrictive response [23]. Aging predisposes patients to a decline in autonomic nervous system function, and older patients have a high prevalence of conditions that can cause OH, such as a high number of comorbidities and medications. In fact, the majority of our sample (72.2%) was under antihypertensive therapy, which may exacerbate age-related variations in postural BP [24, 25]. In addition, frailty status is a risk factor for OH, which has been shown to be very common among frail older individuals, especially when measured within the first minute [26, 27]. Indeed, frailty exacerbates age-related physiological changes by altering compensatory responses to orthostatic changes and increasing the risk of adverse outcomes, such as accidental falls.

Another factor that could have been responsible for the high prevalence of OH was how it was defined: we measured orthostatic BP not only in the early upright period (1–3 min) but also after 5 min of standing, which allowed us to identify patients with not only classic OH (at the first and 3rd minute) but also with delayed OH (DOH). Several studies consider DOH to be an early form of OH and is associated with higher mortality [28, 29].

In our study, orthostatic BP changes exhibited a significant inverse relationship with the day-night BP difference, and accordingly, most patients with OH presented nondipping or reverse-dipping patterns. This finding confirms the data reported by Voichanski et al. [10], although their study participants were much younger (mean age 58 ± 18) and had a lower prevalence of OH (10%). In addition,

the study excluded individuals who could potentially have undergone ABPM monitoring but presented with specific clinical conditions (e.g., AF, OSAS, or sleep disorders), and the cuff was always placed on the left arm rather than on the arm where the highest blood pressure had been recorded. Fagard et al. [11] found a two- to threefold-fold greater prevalence of OH in reverse dippers than in dippers, although patients with major cardiovascular diseases or severe comorbidities were excluded from this study, and the sample population was slightly younger and had a lower prevalence of OH than our sample. Similar findings were obtained by Kario et al. [12], who corroborated the relationship between OH and reverse dipping. On the other hand, Narkiewicz et al. [30], in a study on men aged between 18 and 45 years with mild essential hypertension, found that postural BP changes did not predict nocturnal BP reduction.

In summary, these results suggest that postural and nocturnal BP changes may be related and that the association may be more marked in older individuals. Supporting this hypothesis, we found the relationship between postural and nocturnal BP changes to be significantly stronger in patients over 80 years of age. As has been amply demonstrated, aging is characterized by a physiological decline in autonomic nervous system function. This means that older people often present with impairment of the sensitivity of alpha-1 adrenergic receptors and reduced baroreceptor sensitivity with a resulting high risk of OH [13]. Day-night blood pressure gradients have also been associated with autonomic dysfunctions, such as reduced parasympathetic activity and abnormal sympathetic activation at night [14, 15], which were also confirmed in older hypertensive patients [16]. Therefore, age-related autonomic changes can predispose individuals to both OH and a nondipper profile, consistent with the stronger relationship observed between orthostatic and nocturnal BP changes in the oldest old patients.

Pressure variability may play a relevant role in this relationship, and it is influenced by baroreflex dysfunction and vascular aging, two components of the mechanism underlying orthostatic hypotension. Due to postural changes, OH influences BP variability, measured through ABPM as the standard deviation (SD). In our sample, patients with OH presented a higher BP variability than those without OH. Therefore, one could argue that autonomic system dysfunction can result in OH, as well as in a wide BP variability in daytime, nighttime, and 24-h periods, also influencing nighttime circadian patterns.

The strengths of this study lie in the inclusion of patients in the older age groups, measurement of orthostatic BP at 5 min of standing, and the overall good quality of the ABPM records that include a high percentage of valid measurements. Among the study limitations are the cross-sectional design and a lack of data on drug therapy. We did not have detailed information regarding the timing and dosage of antihypertensive therapy, which could have influenced the nocturnal blood pressure profile, or the use of statins or hypoglycemic agents. In addition, the small number of patients with orthostatic hypertension did not allow us to explore the association between this condition and nighttime BP changes. Further studies are needed to better investigate this aspect.

In conclusion, this study confirms OH to be highly prevalent in geriatric patients attending the hypertension outpatient clinic. Patients with OH, especially the oldest old, are more likely to also present changes in their nocturnal BP profiles. Future prospective studies on older people in other settings would be useful to clarify the pathophysiological basis of the relationship between OH and ABPM parameters and its prognostic risk factors.

Compliance with ethical standards

Conflict of interest The authors declare no competing interests.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

1. Saedon NI, Pin Tan M, Frith J. The prevalence of orthostatic hypotension: a systematic review and meta-analysis. J Gerontol A Biol Sci Med Sci. 2020;75:117–22.
2. Rutan GH, Hermanson B, Bild DE, Kittner SJ, LaBaw F, Tell GS. Orthostatic hypotension in older adults. The Cardiovascular Health Study. CHS Collaborative Research Group. Hypertension 1992;19:508–19.
3. Freud T, Punchik B, Kagan E, Barzak A, Press Y. Orthostatic hypotension and overall mortality in 1050 older patients of the outpatient comprehensive geriatric assessment unit. Geriatr Gerontol Int. 2018;18:1009–17.
4. Masaki KH, Schatz IJ, Burchfiel CM, Sharp DS, Chiu D, Foley D, et al. Orthostatic hypotension predicts mortality in elderly men: the Honolulu Heart Program. Circulation. 1998;98:2290–5.
5. Luukinen H, Koski K, Laippala P, Kivelä SL. Prognosis of diastolic and systolic orthostatic hypotension in older persons. Arch Intern Med. 1999;159:273–80.
6. Verwoert GC, Mattace-Raso FU, Hofman A, Heeringa J, Stricker BH, Breteler MM, et al. Orthostatic hypotension and risk of cardiovascular disease in elderly people: the Rotterdam study. J Am Geriatr Soc. 2008;56:1816–20.
7. Di Stefano C, Milazzo V, Totaro S, Sobrero G, Ravera A, Milan A, et al. Orthostatic hypotension in a cohort of hypertensive patients referring to a hypertension clinic. J Hum Hypertens. 2015;29:599–603.
8. Kaul U, Omboni S, Arambam P, Rao S, Kapoor S, Swahney JPS, et al. Blood pressure related to age: The India ABPM study. J Clin Hypertens (Greenwich). 2019;21:1784–94.
9. Salles GF, Reboldi G, Fagard RH, Cardoso CR, Pierdomenico SD, Verdecchia P, et al. Prognostic effect of the nocturnal blood pressure fall in hypertensive patients: the ambulatory blood pressure collaboration in patients with hypertension (ABC-H) meta-analysis. Hypertension. 2016;67:693–700.

10. Voichanski S, Grossman C, Leibowitz A, Peleg E, Koren-Morag N, Sharabi Y, et al. Orthostatic hypotension is associated with nocturnal change in systolic blood pressure. Am J Hypertens. 2012;25:159–64.
11. Fagard RH, De Cort P. Orthostatic hypotension is a more robust predictor of cardiovascular events than nighttime reverse dipping in elderly. Hypertension. 2010;56:56–61.
12. Kario K, Eguchi K, Nakagawa Y, Motai K, Shimada K. Relationship between extreme dippers and orthostatic hypertension in elderly hypertensive patients. Hypertension. 1998;31:77–82.
13. Barantke M, Krauss T, Ortak J, Lieb W, Reppel M, Burgdorf C, et al. Effects of gender and aging on differential autonomic responses to orthostatic maneuvers. J Cardiovasc Electrophysiol. 2008;19:1296–303.
14. Costa A, Bosone D, Ramusino MC, Ghiotto N, Guaschino E, Zoppi A, et al. Twenty-four-hour blood pressure profile, orthostatic hypotension, and cardiac dysautonomia in elderly type 2 diabetic hypertensive patients. Clin Auton Res. 2016;26:433–9.
15. Zhang Y, Agnoletti D, Blacher J, Safar ME. Blood pressure variability in relation to autonomic nervous system dysregulation: the X-CELLENT study. Hypertens Res. 2012;35:399–403.
16. Abate G, D'Andrea L, Battestini M, Zito M, Di, Iorio A. Autonomic nervous activity in elderly dipper and non-dipper patients with essential hypertension. Aging (Milano). 1997;9:408–14.
17. Parati G, Stergiou G, O'Brien E, Asmar R, Beilin L, Bilo G, et al. 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;32:1359–66.
18. Giantin V, Perissinotto E, Franchin A, Baccaglini K, Attanasio F, Maselli M, et al. Ambulatory blood pressure monitoring in elderly patients with chronic atrial fibrillation: is it absolutely contraindicated or a useful tool in clinical practice and research? Hypertens Res. 2013;36:889–94.
19. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. ESC Scientific Document Group. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39:3021–104.
20. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, Biaggioni I, et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Auton Neurosci. 2011;161:46–8.
21. Jordan J, Tank J, Heusser K, Reuter H, Biaggioni I. What do we really know about supine hypertension in patients with orthostatic hypotension. Curr Opin Cardiol. 2019;34:384–9.
22. Palatini P, Frigo G, Bertolo O, Roman E, Da Cortà R, Winnicki M. Validation of the A&D TM-2430 device for ambulatory blood pressure monitoring and evaluation of performance according to subjects' characteristics. Blood Press Monit. 1998;3:255–60.
23. Toba A, Ishikawa J, Harada K. Orthostatic hypotension and association of arterial compliance in elderly patients with hypertension: a pilot study. Blood Press Monit. 2017;22:274–8.
24. Poon IO, Braun U. High prevalence of orthostatic hypotension and its correlation with potentially causative medications among elderly veterans. J Clin Pharm Ther. 2005;30:173–8.
25. Hajjar I. Postural blood pressure changes and orthostatic hypotension in the elderly patient: impact of antihypertensive medications. Drugs Aging. 2005;22:55–68.
26. Romero-Ortuno R, Cogan L, Foran T, Kenny RA, Fan CW. Continuous noninvasive orthostatic blood pressure measurements and their relationship with orthostatic intolerance, falls, and frailty in older people. J Am Geriatr Soc. 2011;59:655–65.
27. Kocyigit SE, Soysal P, Bulut EA, Aydin AE, Dokuzlar O, Isik AT. What is the relationship between frailty and orthostatic hypotension in older adults? J Geriatr Cardiol. 2019;16:272–9.
28. Gibbons CH, Freeman R. Clinical implications of delayed orthostatic hypotension: a 10-year follow-up study. Neurology. 2015;85:1362–7.
29. Gibbons CH, Freeman R. Delayed orthostatic hypotension: a frequent cause of orthostatic intolerance. Neurology. 2006;67:28–32.
30. Narkiewicz K, Piccolo D, Borella P, Businaro R, Zonzin P, Palatini P. Response to orthostatic stress predicts office-daytime blood pressure difference, but not nocturnal blood pressure fall in mild essential hypertensives: results of the harvest trial. Clin Exp Pharm Physiol. 1995;22:743–7.