




RESEARCH LETTER**Hypotensive episodes revealed by ambulatory blood pressure monitoring in nursing home residents**

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Funding information

The study was supported by Cassa di Risparmio di Pistoia e Pescia Foundation.

INTRODUCTION

Susceptibility to hypotension increases at advanced age,^{1,2} particularly in nursing home (NH) residents, a vulnerable population with multiple coexisting risk factors for low blood pressure (BP).

Hypotension may be responsible for symptoms such as dizziness, confusion, and drowsiness, which impair autonomy and quality of life. Moreover, hypotension in older people is a frequent cause of syncope and falls, which are more likely to occur in NH residents due to concomitant conditions such as dementia, gait, and balance disorders, which magnify the risk of falls and related complications.^{3,4}

Ambulatory blood pressure monitoring (ABPM) is a valuable diagnostic tool to investigate low BP⁵ and may be useful to detect hypotension in NH residents. However, ABPM is rarely used in this setting and data regarding ambulatory BP in NH residents are scarce.

This study aimed to assess the prevalence of hypotensive episodes in NH residents undergoing ABPM and to analyze the associated clinical features and ambulatory BP profile.

METHODS

We retrospectively analyzed data from 100 subjects aged 60 or older consecutively admitted to the NH of the “Filippo Turati” Foundation in Gavinana, Pistoia, Italy, who underwent comprehensive geriatric assessment including ABPM (Spacelabs Healthcare, model 90207).

Hypotensive episodes on ABPM were defined as systolic BP (SBP) drops ≥ 20 mmHg between two consecutive measurements, reaching a minimum SBP of < 100 mmHg. This cutoff is consistent with previous studies dealing with ambulatory hypotension.^{1,2} We adopted a more conservative approach excluding individuals with only night-time hypotension, as very low BP values frequently occur during sleep and their prognostic value is questionable. Based on night-time BP dipping (i.e., difference between mean daytime and night-time SBP, expressed as percentage of daytime SBP), participants were classified as “dippers” (dipping $> 10\%$), “non-dippers” (dipping $0\%–9\%$), and “reverse dippers” (nocturnal rise in BP). White coat effect was also assessed, defined as a difference of > 20 mmHg between office SBP and mean daytime SBP on ABPM.

Multivariable logistic regression was used to identify clinical variables associated with hypotensive episodes

TABLE 1 Clinical characteristics of the study sample according to the presence of hypotensive episodes on ABPM

	Overall sample (n = 91)	Hypotensive episodes on ABPM (n = 50)	No hypotensive episodes on ABPM (n = 41)	p
Age (years), mean ± SD	82.7 ± 9.6	83.9 ± 8.7	81.3 ± 10.4	0.198
Female, n (%)	56 (61.5)	32 (64.0)	24 (58.5)	0.594
BMI (kg/m ²), mean ± SD	24.3 ± 3.3	23.8 ± 3.0	24.8 ± 3.5	0.133
Barthel Index, median (IQR)	30 (10–75)	40.0 (5.0–81.25)	30.0 (10.0–67.5)	0.888
Length of NH stay (days), median (IQR)	346 (22.0–1357.0)	518.5 (19.8–1648.3)	315 (28.5–1086.5)	0.696
Syncope, n (%)	6 (6.6)	4 (7.4)	3 (6.5)	0.863
Falls history, n (%)	44 (48.4)	28 (51.9)	21 (45.7)	0.537
Comorbidities				
Charlson Comorbidity Index	5 (5–6)	5 (5–6)	5 (4.5–6)	0.581
Hypertension, n (%)	46 (50.5)	25 (50.0)	21 (51.2)	0.908
Diabetes, n (%)	9 (9.9)	6 (12.0)	3 (7.3)	0.457
Stroke/TIA, n (%)	26 (28.6)	12 (24.0)	14 (34.1)	0.286
Coronary artery disease, n (%)	14 (15.4)	7 (14.0)	7 (17.1)	0.686
Heart failure, n (%)	16 (17.6)	13 (26.0)	3 (7.3)	0.020
Atrial fibrillation, n (%)	15 (16.5)	13 (26.0)	2 (4.9)	0.007
Chronic kidney disease, n (%)	4 (4.4)	2 (4.0)	2 (4.9)	0.839
Creatinine, mean ± SD	0.93 ± 0.31	0.9 ± 0.3	1.0 ± 0.3	0.073
Creatinine <1 mg/dl, n (%)	61 (67.0)	38 (76)	23 (57.5)	0.062
Dementia, n (%)	59 (64.8)	35 (70.0)	24 (58.5)	0.254
MMSE score, median (IQR)	17 (3–25)	15 (0–23.25)	18 (9.5–25.5)	0.188
NPI, median (IQR)	6 (0–16)	10 (0–16)	6 (0–12)	0.098
Parkinson, n (%)	1 (1.9)	1 (1.9)	0 (0.0)	0.354
Hemoglobin, mean ± SD	12.7 ± 1.5	12.5 ± 1.6	12.9 ± 1.5	0.287
Serum protein, mean ± SD	6.7 ± 0.8	6.6 ± 0.8	6.8 ± 0.8	0.280
Albumin, mean ± SD	3.8 ± 1.6	3.6 ± 0.5	4.1 ± 2.3	0.268
Medications				
Number of medications, median (IQR)	3 (2–4)	4 (2–4)	3 (2–4)	0.560
Number of antihypertensives, median (IQR)	1 (0–2)	1 (0–2)	1 (1–2)	0.726
ACE, n (%)	36 (39.6)	19 (38.0)	17 (41.5)	0.737
ARB, n (%)	2 (2.2)	1 (2.0)	1 (2.4)	0.887
Calcium antagonists, n (%)	14 (15.4)	9 (18.0)	5 (12.2)	0.445
Diuretics, n (%)	36 (39.6)	21 (42.0)	15 (36.6)	0.599
Beta-blockers, n (%)	4 (4.4)	2 (4.0)	2 (4.9)	0.839
Alpha-blockers, n (%)	4 (4.4)	1 (2.0)	3 (7.3)	0.218
Nitrates, n (%)	9 (9.9)	4 (8.0)	5 (12.2)	0.505
Digoxin, n (%)	15 (16.5)	14 (28.0)	1 (2.4)	0.001
Antiarrhythmics, n (%)	3 (3.3)	3 (5.6)	0 (0.0)	0.105
Antidepressants, n (%)	28 (30.8)	13 (26.0)	15 (36.6)	0.276
Benzodiazepines, n (%)	37 (40.7)	26 (52.0)	11 (26.8)	0.015
Antipsychotics, n (%)	19 (20.9)	9 (18.0)	10 (24.4)	0.456
Office blood pressure				
Office SBP, mean ± SD	134.1 ± 14.7	133.0 ± 14.0	135.5 ± 15.5	0.433
Office DBP, mean ± SD	80.0 ± 10.2	79.5 ± 10.2	80.6 ± 10.2	0.599
Supine SBP, mean ± SD	134.0 ± 16.4	131.3 ± 14.4	137.2 ± 18.1	0.088
Supine DBP, mean ± SD	77.3 ± 10.4	77.4 ± 10.7	77.3 ± 10.3	0.988
Orthostatic hypotension ^a , n (%)	8/49 (16.3)	4/28 (14.3)	4/21 (19.0)	0.655
White coat effect, n (%)	33 (36.3)	27 (54.0)	6 (14.6)	<0.001

TABLE 1 (Continued)

	Overall sample (n = 91)	Hypotensive episodes on ABPM (n = 50)	No hypotensive episodes on ABPM (n = 41)	p
ABPM				
ABPM: 24 h SBP, mean ± SD	121.5 ± 14.9	113.2 ± 10.7	131.5 ± 13.2	<0.001
ABPM: 24 h DBP, mean ± SD	68.0 ± 10.9	62.6 ± 8.9	74.5 ± 9.7	<0.001
ABPM: daytime SBP, mean ± SD	123.0 ± 15.9	114.1 ± 12.1	134.0 ± 12.7	<0.001
ABPM: daytime DBP, mean ± SD	70.3 ± 11.8	64.5 ± 10.2	77.3 ± 9.7	<0.001
ABPM: night-time SBP, mean ± SD	119.8 ± 16.1	112.2 ± 12.5	129.2 ± 15.1	<0.001
ABPM: night-time DBP, mean ± SD	65.7 ± 11.6	60.6 ± 9.3	72.0 ± 11.1	<0.001
ABPM: daytime SD, mean ± SD	13.4 ± 3.6	13.8 ± 3.5	12.8 ± 3.8	0.186
Nocturnal dipping, n (%)				
Dippers	26 (28.6)	14 (28.0)	12 (29.3)	0.894
Non-dippers	35 (38.5)	14 (28.0)	21 (51.2)	0.023
Reverse dippers	30 (33.0)	22 (44.0)	8 (19.5)	0.013

Abbreviations: ABPM, ambulatory blood pressure monitoring; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; BMI, body mass index; DBP, diastolic blood pressure; IQR, interquartile range; MMSE, Mini-Mental State Examination; NH, nursing home; NPI, Neuropsychiatric Inventory; SBP, systolic blood pressure; SD, standard deviation; TIA, transient ischemic attack.

^aOrthostatic hypotension was assessed in 49 of 91 subjects who were able to stand.

among those showing an association with the outcome in univariate analysis ($p < 0.1$). To avoid multicollinearity in the multivariable regression model, the intercorrelations among variables were checked using a correlation matrix of two-sided Spearman rho correlation coefficients. Correlations of ≥ 0.50 were considered to be significant. Statistical significance was set at $p < 0.05$. Statistical analyses were performed using SPSS Statistics, version 26 (IBM Corp, Armonk, NY). The study was approved by the local institutional review board.

RESULTS

The study sample included 91 residents (mean age 83, 56% female). Hypotensive episodes were observed in 50 residents (55%), with 35 (38%) reaching a minimum SBP of <90 mmHg. No clinical correlate of hypotensive episodes was recorded. Residents with hypotensive episodes had significantly lower ambulatory BP and more frequently showed a reverse dipping profile and a white coat effect. Office BP and prevalence of orthostatic hypotension were similar between the two groups (Table 1).

Residents with hypotensive episodes showed a higher prevalence of heart failure and atrial fibrillation and more commonly received digoxin and benzodiazepines, while antihypertensive prescriptions were similar between the two groups. No significant difference was observed in cognitive performance, functional status, and comorbidity burden (Table 1). At multivariable analysis, hypotensive episodes were independently associated with digoxin (OR 20.0, 95% CI 2.21–181.19,

$p = 0.008$), benzodiazepines (OR 4.0, 95% CI 1.45–11.22, $p = 0.007$), and serum creatinine <1 mg/dl (OR 3.9, 95% CI 1.28–12.17, $p = 0.017$).

DISCUSSION

In this sample of NH residents undergoing ABPM, hypotensive episodes were highly prevalent (55%) and related with a specific ABPM profile including lower mean BP values, reverse dipping, and white coat effect. This profile suggests that a certain degree of autonomic dysfunction may be present as a consequence of cardiovascular aging and comorbidities, leading to higher BP during the night-time and orthostatic/postprandial hypotension during the daytime. Second, overtreatment may contribute to hypotensive episodes, as white coat effect may determine excessive BP lowering in residents presenting with high office BP.

Hypotensive episodes were independently associated with benzodiazepines, consistently with previous studies describing a hypotensive effect of this class.⁶ An independent association with digoxin was also observed, which may be related to comorbidities such as heart failure and atrial fibrillation. Moreover, chronic treatment with digoxin was found to lower BP in previous studies. Hypothesized mechanisms include increased baroreflex sensitivity, enhanced parasympathetic activity, and withdrawal of elevated sympathetic tone due to improved cardiac inotropism.^{7,8} Finally, our study revealed an independent association between hypotensive episodes and low serum creatinine, which is common in older adults with sarcopenia.⁹ This implies a

link between low muscle mass and hypotensive risk, possibly deriving from the crucial role of muscle mass in the BP response to gravity stress.¹⁰

No clinical correlate of hypotensive episodes was recorded in our sample. Yet, symptoms of hypotension are frequently atypical in older individuals and subjects with cognitive impairment are less likely to report them.³ Therefore, ABPM may reveal hypotensive episodes that would otherwise be unrecognized.

The present study confirms that NH residents have a significant predisposition for hypotensive episodes. ABPM can significantly contribute to hypotension diagnosis of subjects at risk in this vulnerable, older population.

ACKNOWLEDGMENT

Open Access Funding provided by Università degli Studi di Firenze within the CRUI-CARE Agreement.

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

AUTHOR CONTRIBUTIONS

Conception and design: Giulia Rivasi, Enrico Mossello, and Andrea Ungar. Acquisition, analysis, and interpretation of data: all authors. Drafting the article: Giulia Rivasi, Enrico Mossello, Michele Brignole, Andrea Ungar, and Ludovica Ceolin. Revising of the article for important intellectual content: all authors. Final approval of the version to be published: all authors.

SPONSOR'S ROLE

None.

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How to cite this article: Rivasi G, Mossello E, Turrin G, et al. Hypotensive episodes revealed by ambulatory blood pressure monitoring in nursing home residents. *J Am Geriatr Soc*. 2021;1-4. doi:10.1111/jgs.17570