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ORIGINAL ARTICLE

Postprandial hypotension detected through home blood pressure monitoring: a frequent phenomenon in elderly hypertensive patients

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Postprandial hypotension (PPH) is a frequently under-recognized entity associated with increased morbidity and mortality. The prevalence of PPH detected through home blood pressure monitoring (HBPM) is unknown. To determine the prevalence and clinical predictors of PPH in hypertensive patients assessed through HBPM. Hypertensive patients of 18 years or older underwent home blood pressure (BP) measurements (duplicate measurements for 4 days: in the morning, 1 h before and 1 h after their usual lunch, and in the evening; OMRON 705 CP). PPH was defined as a meal-induced systolic BP decrease of $\geqslant$20 mm Hg. Variables identified as relevant predictors of PPH were entered into a multivariate logistic regression analysis. In total, 230 patients were included in the analysis, with a median age of 73.6 (interquartile range 16.9) years, and 65.2% were female. The prevalence of PPH (at least one episode) was 27.4%. Four variables were independently associated with PPH: age of 80 years or older (odds ratio (OR) 3.45, 95% confidence interval (CI) 1.35–8.82), body mass index (BMI) (OR 0.88, 95%CI 0.81–0.96), office systolic BP (OR 1.03, 95%CI 1.01–1.05) and a history of cerebrovascular disease (OR 3.29, 95%CI 1.03–10.53). PPH after a typical meal is a frequent phenomenon that can be detected through HBPM. Easily measurable parameters in the office such as older age, higher systolic BP, lower BMI and a history of cerebrovascular disease may help to detect patients at risk of PPH who would benefit from HBPM.
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INTRODUCTION

Postprandial hypotension (PPH) is a frequently under-recognized phenomenon associated with increased morbidity, such as syncope, falls, stroke and coronary events, and all-cause mortality.[1,2] Certain populations, namely the elderly, patients diagnosed with hypertension or diabetes, and patients with different causes of autonomic dysfunction, have been identified as particularly vulnerable.[3] Although the mechanism involved in PPH is not clearly understood, it appears to be secondary to a blunted sympathetic response to a meal.[4]

Most studies assessing PPH have been conducted in institutionalized geriatric subjects who consume standardized meals.[5,6] Data from ambulatory patients in their usual environment are scarce, and studies including such patients have used ambulatory blood pressure monitoring as a method to evaluate PPH.[7,8]

Home blood pressure monitoring (HBPM) is an increasingly used BP measurement strategy that offers some advantages over ambulatory blood pressure monitoring,[9,10] including better patient tolerance, lower cost, and greater suitability for the long-term follow-up of patients under treatment. However, this method has not previously been used to detect PPH in hypertensive patients. The purpose of our study was to determine the prevalence of PPH detected through HBPM in hypertensive subjects and to establish clinical predictors that are easily detectable in a doctor's office.

METHODS

Study population

This was a cross-sectional study that consecutively included hypertensive patients 18 years or older treated in the Hypertension Section of the Hospital Italiano de Buenos Aires. The patients performed HBPM as prescribed by their treating physician to assess hypertension control. The study protocol was approved by the local ethics committee, and all patients who agreed to participate provided informed consent. The medical records of all patients were reviewed to gather data regarding risk factors (diabetes, smoking status and dyslipidemia), a history of cardiovascular disease (coronary heart disease, cerebrovascular disease and peripheral artery disease) and the use of antihypertensive drugs. Laboratory data (fasting plasma glucose

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(FPG), total cholesterol, high-density lipoprotein, low-density lipoprotein, triglyceride levels and serum creatinine) from 6 months before HBPM were also gathered from the medical records.

Patients were asked to complete a diary, recording lunch time and postprandial-related symptoms (dizziness, fatigue and somnolence) during home blood pressure (BP) assessment. The recruitment period lasted from January 2013 to May 2013.

Anthropometric and BP measurements

Weight and height were assessed in all patients, and body mass index (BMI) was calculated as weight/height^2 (kg m^{-2}). BP was subsequently measured by a trained technician twice in the non-dominant arm, 2 min apart (the average of the two readings was used for analysis), after a 5-min rest with the patient in a sitting position; the arm was always supported at the heart level, and an appropriate cuff size was used according to the arm circumference: patients with an arm circumference of <26 cm used a small adult cuff (12×22 cm); patients with an arm circumference between 27 and 34 cm used a standard adult cuff (16×30 cm); and patients with an arm circumference of $\geqslant 35$ cm used a large adult cuff (16×36 cm). For this purpose, an automatic oscillometric device Omron 705 CP (Omron, Tokyo, Japan) was used; this device has previously been validated[11] against a mercury sphygmomanometer according to the revised protocol of the British Hypertension Society.[12]

After receiving appropriate training on its use, the patients returned home with the same oscillometric device used in the office and registered duplicate sitting BP readings (2 min apart) in the non-dominant arm for 4 days: in the morning (before breakfast and medication for those under treatment), 1 h before and 1 h after their usual lunch, and in the evening. Patients were instructed to measure home BP after a 5-min rest, with the legs uncrossed, the back supported and not talking. Additionally, morning readings were taken before breakfast and drug intake. First-day measurements were discarded from the analysis. Patients with <2 days of pre- and postprandial home BP readings were excluded from the analysis.

Definitions of clinical parameters

Hypertension was defined as antihypertensive drug use or a mean (average of morning and evening readings) home systolic or diastolic BP (discarding first-day measurements) of $\geqslant 135$ or 85 mm Hg, respectively.

Regarding cardiovascular risk factors, current smoking was defined as the daily use of tobacco products; diabetes was defined as an FPG of $\geqslant 126$ mg dl^{-1} on at least two occasions or the use of antidiabetic drugs; and dyslipidemia was defined according to the ATP III criteria[13] or the use of lipid-lowering drugs.

In relation to the history of cardiovascular disease, coronary heart disease was defined as a history of myocardial infarction, unstable angina, chronic stable angina or coronary bypass surgery; cerebrovascular disease was defined as a history of stroke or transient ischemic attack; and peripheral artery disease was defined as intermittent claudication, abnormal arterial Doppler examination, or peripheral revascularization in the lower limbs.

FPG, serum creatinine, total cholesterol, high-density lipoprotein, low-density lipoprotein and triglyceride levels were all measured in mg dl^{-1}.

Meal-induced BP variation was calculated as the difference between mean BP 1 h before and 1 h following lunchtime, as recorded in the patient's diary. A meal-induced systolic BP decrease of $\geqslant 20$ mm Hg was used to define PPH.[14]

Statistical analysis

Results are reported as the percentage, mean ± standard deviation, or median and interquartile range, according to the data distribution. The characteristics of patients with and without PPH were compared using the *t*-test or Mann–Whitney *U*-test for continuous variables and the chi-square test for categorical variables. A two-sided *P*-value of <0.05 was considered as statistically significant.

Variables associated with PPH in the univariate analyses were entered into a multiple logistic regression model to detect independent predictors of PPH. The model's calibration and discrimination were tested using the Hosmer–Lemeshow goodness-of-fit test and the area under the receiver operating characteristic curve, respectively.

RESULTS

The study included 255 hypertensive patients; 25 (9.8%) had <2 days of pre- and postprandial HBPM readings. Therefore, 230 patients were included in the analysis. The median age was 73.6 (interquartile range 16.9) years, and 150 (65.2%) patients were female (Table 1). Additionally, 91.3% were receiving antihypertensive drugs, with a mean of 2 (± 1.1) drugs per patient, and the mean office BP was 139 (± 16.2)/78.2 (± 9.9) mm Hg.

Overall, meal-induced BP decreases in 5 (± 9.8) and 4.4 (± 5.5) mm Hg were observed for systolic and diastolic BP, respectively. The home BP and heart rate profiles are depicted in Table 2 and Figures 1 and 2. Interestingly, when the mean home BP was calculated using the morning, evening, and pre- and postprandial readings, the mean was significantly lower than when it was calculated based on the morning and evening BP measurements only, following the current recommendation:[15] 136.1 (± 16.6)/74.6 (± 9.6) *vs.* 131.8 (± 14.5)/72.3 (± 9.1) mm Hg ($P<0.001/0.001$).

The prevalence of PPH (at least one episode) was 27.4% (63/230), and 8.7% (20/230) of patients had two or more episodes. Among the 28 subjects who collected only 2 days of pre- and postprandial measurements, 20 had no PPH episodes, 7 had 1 episode and 1 had 2 episodes. The prevalence of the white coat effect among subjects with PPH was not significantly different from those without PPH (19 *vs.* 24%, $P=0.43$).

Patients with PPH were older (Figure 3), had a lower BMI, a higher office systolic BP and higher prevalences of beta-blocker use and a history of cerebrovascular disease (Table 1). Univariate logistic regression analyses demonstrated that the following variables were associated with PPH, and they were entered into a multivariate regression model (*P*-value for goodness-of-fit = 0.5; area under the receiver operating characteristic curve = 0.75, 95% confidence interval (CI) 0.68–0.82): age, BMI, use of beta-blockers, a history of cerebrovascular disease and office systolic BP. Four variables were independently associated with PPH: age of 80 years or older, BMI, office systolic BP and a history of cerebrovascular disease (Table 3).

DISCUSSION

In our study, PPH detected using HBPM was a prevalent phenomenon among hypertensive patients and was associated with older age, lower BMI, a history of cerebrovascular disease and a higher office systolic BP. The reported prevalence of PPH is very heterogenous among studies, ranging from 9 to 70%, depending on the population age, the presence of co-morbidities and the method of BP measurement used.[8,16] The prevalence found in our study was consistent with some studies that also evaluated hypertensive patients and was lower compared with others, perhaps because of the younger population included in our study.[7,8,17]

Table 1 Characteristics of the study population

	Total	*With PPH*	*Without PPH*	*P-value*
n (%)	230 (100)	63 (27.4)	167 (72.6)	
Demographic characteristics				
Women (%)	65.2	68.3	64.1	n.s.
Age (IQR)	73.6 (16.9)	78.3 (12.2)	71 (18.9)	<0.001
BMI (IQR)	27.3 (5.3)	26.1 (3.5)	27.4 (5.4)	<0.001
Office-sitting SBP (mm Hg) (±s.d.)	139 (±16.2)	143.5 (±17.6)	137.4 (±15.4)	0.01
Office-sitting DBP (mm Hg) (±s.d.)	78.2 (±9.9)	77.8 (±11.4)	78.4 (±9.3)	n.s.
Antihypertensive therapy (%)	91.3	93.7	90.4	n.s.
Number of antihypertensive drugs (±s.d.)	2 (±1.1)	2.06 (±0.9)	1.9 (±1.1)	n.s.
Diuretics (%)	32.2	30.2	32.9	n.s.
Beta-blockers (%)	39.1	50.8	34.7	0.03
ACE-Is (%)	30	27	31.1	n.s.
ARBs (%)	40.9	47.6	38.3	n.s.
CCBs (%)	47.4	41.3	49.7	n.s.
Alpha-blockers (%)	4.8	6.3	4.2	n.s.
Other (%)	1.7	1.6	1.8	n.s.
Postprandial-related symptoms (dizziness, somnolence, fatigue)	30.3	27.8	31.3	n.s.
Risk factors, lab data and a history of CV disease				
Current smokers (%)	10.9	14.3	9.6	n.s.
Past smokers (%)	12.2	14.3	11.4	
Dyslipidemia (%)	82.6	81	83.2	n.s.
Diabetes (%)	7	7.9	6.6	n.s.
FPG ($mg\,dl^{-1}$) (IQR)	97 (12.2)	96 (13)	97 (13)	n.s.
SCr ($mg\,dl^{-1}$) (IQR)	0.88 (0.3)	0.9 (0.3)	0.87 (0.3)	n.s.
Total cholesterol ($mg\,dl^{-1}$) (±s.d.)	192.4 (±39.6)	188.1 (±34.6)	194 (±41.2)	n.s.
LDL cholesterol ($mg\,dl^{-1}$) (±s.d.)	113.6 (±35.4)	109.1 (±33.7)	115.2 (±36)	n.s.
HDL cholesterol ($mg\,dl^{-1}$) (±s.d.)	55.9 (±13.7)	56.9 (±13.9)	55.5 (±13.6)	n.s.
Triglyceridemia ($mg\,dl^{-1}$) (IQR)	98.5 (50.2)	93 (55)	98.5 (49)	n.s.
History of IHD (%)	7.4	12.7	5.4	n.s.
History of cerebrovascular disease (%)	6.5	14.3	3.6	0.003
History of heart failure (%)	1.7	3.3	1.2	n.s.
History of PAD (%)	6.1	6.3	6	n.s.

Abbreviations: ACE-Is, angiotensin converting enzyme inhibitors; ARBs, angiotensin receptor blockers; BMI, body mass index; CCBs, calcium channel blockers; CV, cardiovascular; DBP, diastolic blood pressure; IQR, interquartile range; FPG, fasting plasma glucose; HDL, high-density lipoprotein; IHD, ischemic heart disease; LDL, low-density lipoprotein; n.s. = not significant; PAD, peripheral artery disease; PPH, postprandial hypotension; SBP, systolic blood pressure; SCr, serum creatinine; s.d., standard deviation.

Table 2 Home blood pressure and heart rate profiles

	Total	*With PPH*	*Without PPH*	*P-value*
Number of BP measurements (±s.d.)	26.2 (±2.7)	26.2 (±2.7)	26.2 (±2.7)	n.s.
Mean SBP (mm Hg) (±s.d.)[a]	136.1 (±16.6)	142.4 (±17.5)	133.8 (±15.6)	<0.001
Mean DBP (mm Hg) (±s.d.)[a]	74.6 (±9.6)	74.8 (±9.6)	74.6 (±9.6)	n.s.
Mean HR (b.p.m.) (±s.d.)	68.3 (±9.6)	67 (±9.7)	68.8 (±9.5)	n.s.
Morning SBP (mm Hg) (±s.d.)	137.1 (±18.9)	145 (±19.2)	134.1 (±18)	<0.001
Morning DBP (mm Hg) (±s.d.)	75.4 (±10)	76.2 (±10.3)	75.1 (±9.9)	n.s.
Preprandial SBP (mm Hg) (±s.d.)	130 (±15.5)	139.3 (±16.5)	126.5 (±13.5)	<0.001
Preprandial DBP (mm Hg) (±s.d.)	72.2 (±10)	73.8 (±11.1)	71.6 (±9.5)	n.s.
Postprandial SBP (mm Hg) (±s.d.)	125 (±14.6)	124.3 (±15.4)	125.3 (±14.2)	n.s.
Postprandial DBP (mm Hg) (±s.d.)	67.8 (±9.7)	65.4 (±10)	68.7 (±9.5)	0.02
Preprandial HR (b.p.m.) (±s.d.)	68.2 (±10.6)	65.9 (±9.8)	69.1 (±10.8)	0.04
Postprandial HR (b.p.m.) (±s.d.)	71.8 (±10.6)	69.6 (±10.8)	72.6 (±10.5)	n.s.
Evening SBP (mm Hg) (±s.d.)	135.2 (±16.4)	139.7 (±18.1)	133.5 (±15.4)	0.01
Evening DBP (mm Hg) (±s.d.)	73.8 (±10.2)	73.5 (±10.3)	74 (±10.2)	n.s.
Meal-induced SBP variation (mm Hg) (±s.d.)	5 (±9.8)	15 (±9)	1.2 (±7.1)	<0.001
Meal-induced DBP variation (mm Hg) (±s.d.)	4.4 (±5.5)	8.4 (±5.6)	2.9 (±4.7)	<0.001
Meal-induced HR variation (b.p.m.) (±s.d.)	−3.6 (±4.9)	−3.8 (±5.1)	−3.5 (±4.8)	n.s.

Abbreviations: BP, blood pressure; b.p.m., beats per minute; DBP, diastolic blood pressure; HR, heart rate; n.s., not significant; PPH, postprandial hypotension; SBP, systolic blood pressure; s.d., standard deviation.
[a]Average of morning and evening measurements, discarding first-day readings.

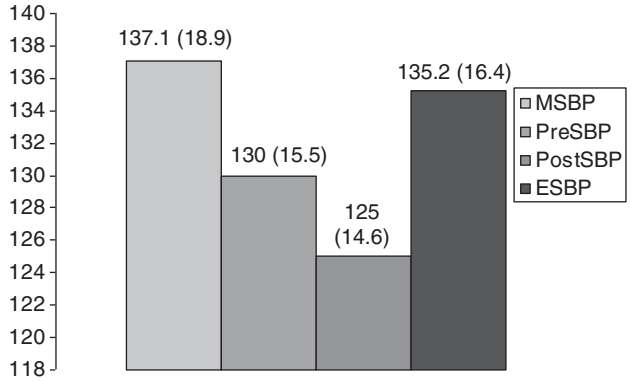

Figure 1 Comparison of morning, preprandial, postprandial and evening systolic blood pressure in the study population.

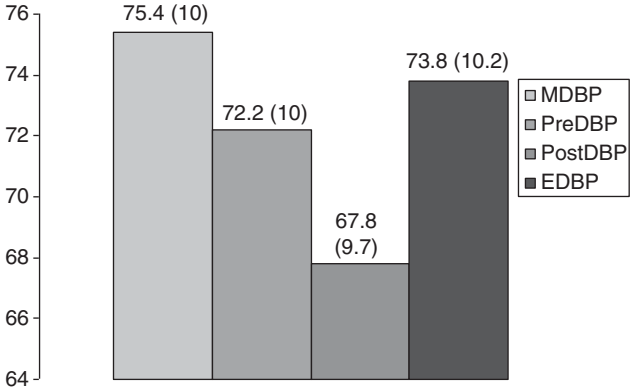

Figure 2 Comparison of morning, preprandial, postprandial and evening diastolic blood pressure in the study population.

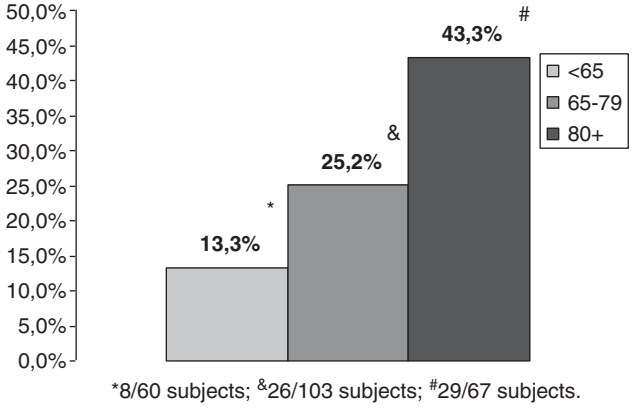

Figure 3 Prevalence of postprandial hypotension according to age group.

Age has been evaluated in several studies, including ours, as a PPH predictor, consistently showing a positive association.[18,19] Therefore, elderly people are an already fragile population at risk of suffering from PPH more frequently than younger people, increasing the complexity of hypertension management in such patients. Although our study included subjects 18 years or older, the median age of the population was 73.6 years. Indeed, although we see adult patients in general, most of them are elderly. The average age of patients attending the Hypertension Section of our Hospital is 66 years. In fact, only 26.1% of the patients included in the study were younger than 65 years.

Table 3 Multivariate analysis: predictors of postprandial hypotension

	OR (CI 95%)	*P-value*
Age group		
65–79 *vs.* <65 years	1.69 (0.68–4.2)	n.s.
80 or more *vs.* <65 years	3.45 (1.35–8.82)	0.01
History of cerebrovascular disease	3.29 (1.03–10.53)	0.045
BMI	0.88 (0.81–0.96)	0.002
Office SBP	1.03 (1.01–1.05)	0.01
BB therapy	1.86 (0.97–3.57)	ns

Abbreviations: BB, beta-blocker; BMI, body mass index; n.s., not significant; SBP, systolic blood pressure.

Hypertension is a co-morbidity typically associated with PPH.[14] In our study, all subjects had been diagnosed with hypertension, and interestingly, higher office systolic BP was an independent predictor of PPH. This is of particular interest, as the decision to intensify antihypertensive treatment is typically based on office BP measurements only. In that sense, we believe that elderly patients with uncontrolled hypertension may benefit from HBPM before any change in antihypertensive strategy. Future research should aim to establish whether hypertension management considering PPH episodes at home could help to reduce morbidity and mortality.

Decreases in postprandial BP have shown a high correlation with magnetic resonance imaging findings indicative of cerebrovascular damage.[2] Moreover, in some prospective studies, PPH has been associated with a higher incidence of transient ischemic attacks[20] and stroke,[1] which may explain the association between a history of cerebrovascular disease and PPH found in our study.

Low BMI has been associated with orthostatic hypotension,[21] but to the best of our knowledge, this is the first study to describe such an association with PPH. This could be related to the increased fragility observed in thinner elderly patients.

Other PPH predictors found in previous studies were not statistically significant in our study. Consistent with other studies,[8,22] we failed to find an association between PPH and diabetes, an entity in which autonomic dysfunction has been well documented. A tentative explanation may be that diabetes had recently been diagnosed or was well controlled among our patients. We do not have information on the possible presence of diabetic neuropathy in our population.

In accordance with previous studies,[8] PPH was not correlated with the presence of postprandial symptoms. In fact, no differences were found in our study regarding meal-induced BP variation among subjects with and without symptoms. It must be emphasized, however, that asymptomatic PPH is not a benign condition, given its association with asymptomatic cerebrovascular damage.[2] Therefore, clinical predictors that may raise the suspicion of PPH in the absence of symptoms may help to identify the patients who would benefit the most from PPH assessment by HBPM or another method. Such screening of asymptomatic hypertensive patients, particularly the elderly, for PPH should be encouraged.

Regarding antihypertensive drugs, some studies have found an association between PPH and diuretics,[17,19] which raises concern about whether antihypertensive drugs should be reduced or withdrawn in such cases. Other studies, including ours, have found

no association between PPH and treatment for hypertension.[8] Although the univariate analysis in our study indicated an association with beta-blocker use, statistical significance was lost in the multivariate analysis. One possible explanation is that beta-blocker use is related to uncontrolled office hypertension, which persisted as an independent predictor of PPH in the multivariate analysis, given that beta-blockers are not as useful as other drug classes for hypertension therapy in elderly subjects.[23,24] Although discrepancies exist regarding antihypertensive treatment and PPH, it would seem advisable to prescribe antihypertensive drugs away from meal times to avoid exaggerated postprandial BP declines rather than to reduce or discontinue antihypertensive treatment.

To the best of our knowledge, this is the first study to use HBPM as an out-of-office measurement method to assess PPH. HBPM provides extensive BP information over a long period of time with high reproducibility, and it has been shown to be better than office BP in the prediction of hypertensive target organ damage and cardiovascular prognosis.[25] In our study, we used a 4-day measurement protocol,[26,27] complying with the recommendations of the updated European guidelines on the use of HBPM regarding a monitoring schedule of at least 3 days of measurements.[9] In the particular case of PPH assessment, HBPM offers some advantages over ambulatory blood pressure monitoring: it provides pre- and postprandial measurements over several days *vs.* only 24-h measurements, and given that the patients must be awake to record BP, it avoids a possible 'napping effect' that could be observed on ambulatory blood pressure monitoring and could act as a confounder for postprandial BP decline. As a result, we believe that HBPM may be a suitable strategy for PPH screening.

One interesting finding in our study was that the average home BP was significantly reduced when considering morning, pre- and postprandial, and evening readings when compared with the currently recommended average of morning and evening measurements only.[15,28] The protocol that should be used to guide treatment in patients at risk of PPH constitutes a matter for future research that may widen HBPM indications.

Finally, our results should be interpreted within the context of the study limitations. First, PPH detected through HBPM has not previously been defined. Although the variability inherent to BP might have influenced the BP decrease after lunch that was observed in our study, we feel that using a decrease of at least 20 mm Hg might have helped to offset this possible confounding factor. Second, patients consumed their usual meals; there was no standardization. However, the lack of standardized meals could be regarded as one study strength because it allows a more 'real-life' approach, which may be more useful for physicians making treatment decisions based on this type of information. Third, only lunch-related PPH could be evaluated, as the device used has a limited memory capacity. As a consequence, PPH related to breakfast and dinner could not be assessed. However, even one single episode of PPH has been shown to increase mortality,[29] and its detection should not be disregarded. Moreover, lunch has been identified as one of the meals in which greater postprandial BP decreases occur.[30] Fourth, subjects receiving antihypertensive drugs took their medication at different times of the day, as they were used to doing before their inclusion in the study. For patients taking their medication near lunchtime, the postprandial BP decrease could have been exaggerated as a result of the drug effect.

In conclusion, PPH was a frequent phenomenon after a typical meal in our study population, particularly in the elderly. HBPM was useful for the detection of PPH and feasible in elderly patients. Easily measurable parameters such as older age, higher office systolic BP, lower BMI, and a history of cerebrovascular disease may help to detect patients at risk of PPH who would benefit from HBPM.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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