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Short title: Sex and blood pressure variability

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WHAT'S NEW?

Women exhibit higher blood pressure variability despite having lower blood pressure values. Among the most significant independent factors influencing blood pressure variability are gender, age, and blood pressure values themselves. In particular, within the female group a stronger association was observed between increased blood pressure variability and hypertension-mediated organ damage. The measurement of blood pressure variability may hold significant implications for clinical practice.

ABSTRACT

Background: Hypertension is widely recognized as a significant risk factor for cardiovascular diseases. Beyond merely focusing on blood pressure levels, there is growing recognition of the importance of considering blood pressure variability (BPV).

Aims: The aim of this study was to compare factors influencing BPV in men and women in office and out-of-office measurements.

Methods: The study enrolled 120 women and 99 men recruited from an outpatient clinic between 2021 and 2022. All participants underwent a medical interview. Subsequently, office blood pressure measurements were conducted using two methods - unattended and attended measurements Following this, 24-hour ambulatory blood pressure monitoring was performed, and participants were instructed to conduct home blood pressure measurements for 7 days. Laboratory tests, echocardiography and carotid artery ultrasound were performed thereafter.

Results: BPV was higher in women, in older patients, those with diabetes and smokers. Female sex remained significant determinant of higher BPV in multiple regression analysis (b = –0.299; P = 0.002) after adjustment for age (b = 0.247; P = 0.01), body mass index (b = 0.012; P = 0.89), diabetes (b = –0.155; P = 0.08), smoking (b = 0.063; P = 0.48) and blood pressure values (b = 0.478; P <0.001). BPV is associated with parameters of subclinical organ-damage and this relationship is stronger for women than men.

Conclusions: Women exhibit higher BPV despite having lower blood pressure. Reducing the BPV, a multifaceted phenomenon related to organ damage, necessitates integrated intervention focused on optimizing blood pressure values on one hand, and managing metabolic risk factors and lifestyle modifications, notably including tobacco cessation, on the other.

Key words: arterial hypertension, blood pressure measurement, blood pressure variability, sex differences

INTRODUCTION

Hypertension stands as a crucial risk factor for cardiovascular diseases, accounting for a significant proportion of severe complications, including myocardial infarctions, strokes, and heart failure [1]. The epidemiology and clinical characteristics of hypertension differ significantly between men and women. Women develop more hypertension complications at the same blood pressure (BP) levels as men and benefit more from the same BP reduction [2]. Despite similar cardiovascular disease prevalence in both sexes, women often experience

delayed diagnosis and suboptimal treatment, emphasizing the need for sex-specific research to identify underlying physiological differences [3]. Moreover, due to the higher incidence of drug-induced side effects in women compared to men, antihypertensive pharmacotherapy may present a greater challenge in women [4]. The appropriateness of employing sex-specific thresholds for diagnosing and managing hypertension is currently a topic of debate [5].

However, it is crucial to acknowledge that blood pressure variability (BPV) is another critical independent risk factor that may influence the differences in the relationship between hypertension course and prognosis between the sexes [6, 7]. BPV relates to fluctuations in BP across varying time spans. BPV can be characterized depending on the time interval considered, ranging from very short- (intra-beat and beat-to-beat) to short-term (within 24 hours), mid-term (days), and long-term (weeks, months, encompassing variability during clinical visits), as outlined in the position paper by the European Society of Hypertension [8]. Various measures for BPV exist, with commonly used ones including standard deviation, coefficient of variation, weighted 24-hour standard deviation, and average real variability, all of which are precisely described in the aforementioned position paper [8].

In research on BPV, the influence of several factors has been demonstrated, such as age, lipid profile and elevated BP [9]. There is also growing body of evidence indicating that BPV differs between men and women. Nonetheless, numerous potential determinants remain unexplored.

The objective of this study was to investigate the impact of various factors on BPV, encompassing very short-term (office blood pressure measurement), short-term (ambulatory blood pressure monitoring), and mid-term BPV (home blood pressure monitoring), comparing men and women. Clinical parameters and adipocytokines were considered in the analysis to elucidate their potential roles in shaping BPV. Moreover, we compared the associations of BPV with subclinical hypertension-mediated organ damage in men and women.

METHODS

A total of 219 consecutive patients, comprising 120 women and 99 men, from the outpatient clinic who met the inclusion criteria and provided informed consent were recruited for the study. This is a cross-sectional study, and all included participants underwent a full set of examinations. Thus, the number of individuals included is equivalent to the number of study participants. All procedures were performed at the Outpatient Clinic of the First Department of Cardiology, Interventional Electrocardiology, and Hypertension. The time from recruitment to completion of examinations ranged from 1 to 4 weeks. The number of patients included in the

study was calculated for the original project, which aimed to compare attended and unattended blood pressure measurements, as noted in the publication [10].

The recruitment process began in January 2021 and concluded at the end of 2022. Eligible participants, aged 18 years or older and without clinically evident cardiovascular disease or chronic kidney disease, underwent a comprehensive subjective examination using a standardized questionnaire. Additionally, a thorough physical examination and precise anthropometric measurements, were performed as part of the study protocol, previously described [10].

Subsequently, BP measurements were conducted. Prior to their scheduled visit, participants were instructed to refrain from alcohol consumption and cigarette smoking for at least 3 hours. The BP measurements occurred in the afternoon in a quiet examination room at the outpatient clinic. All measurements were conducted in adherence to the European Society of Hypertension guidelines [11], utilizing the OMRON HEM 907 device (Omron Corporation, Kyoto, Japan). The measurements followed a standardized protocol:

1. Unattended automated office blood pressure measurements (UAOBP): After ensuring proper equipment setup the attending physician initiated the device and left the examination room. Following 5 minutes of seated rest, three consecutive BP measurements were automatically obtained at 1-minute intervals.

2. Office blood pressure measurements: After a 5-minute period of seated rest, the physician performed three consecutive conventional BP measurements at 1-minute intervals without engaging in conversation with the participant.

The sequence of these methods for each participant was randomized.

On the following day, after an overnight fast, each participant underwent laboratory assessments, including lipid profiling, HbA1C, as well as measurements of selected adipocytokines: adiponectin, leptin, and chemerin.

The concentration of adiponectin, chemerin and leptin were measured using the quantitative sandwich enzyme immunoassay technique (Human Adiponectin Quantikine ELISA Kit, Human Chemerin Quantikine ELISA Kit and Human Leptin Quantikine ELISA Kit; R&D Systems, Minneapolis, MN, US), according to the instructions provided by the manufacturers. Assay sensitivity was 0.246 ng/ml for adiponectin, 4.13 pg/ml for chemerin, and 7.8 pg/ml for leptin. The intra-assay and the inter-assay coefficients of variation was as follows: 3.5% and 6.5% for adiponectin; 3.9% and 7.3% for chemerin; 3.2% and 4.4% for leptin. Optical density was measured on a plate reader EL×808™ (Bio-Tek Instruments, Winooski, VT, US)

at the wavelength 450 nm and data were collected using Gen 5 (Bio-Tek, US) software. A four parametric logistic (4-PL) curve fit was used to generate the standard curve.

On the same day, 24-hour ambulatory blood pressure monitoring (ABPM) was carried out using SpaceLabs 90207 devices (Spacelabs Healthcare, Snoqualmie, WA, US). The ABPM measurements were taken at 15-minute intervals during the daytime (06:00–22:00 hours) and at 20-minute intervals during the nighttime (22:00–06:00 hours).

Participants were instructed to perform home blood pressure measurements (HBPM). They were advised to measure their BP while seated after a 5-minute rest, with one-minute intervals between readings. Specifically, they were asked to take two measurements in the morning upon waking and two measurements before bedtime for a continuous period of 7 days.

As a measure of BPV standard deviation was chosen. Variability was assessed separately for UAOBP and OBP. For HBPM, variability was assessed based on the range of 7-day measurements, considering values from morning and evening. Similarly, for ABPM, variability was separately assessed during the daytime, at nighttime, and over the entire day.

Additionally, for daytime and nighttime ABPM, correction was applied to account for the number of hours included in each of these subperiods. For the 24-hour ABPM, a weighted 24-hour standard deviation was used.

In addition, to evaluate BPV in ABPM, the average real variability (ARV) method was employed [12]. Following the BP measurements, transthoracic echocardiography, carotid artery ultrasound and carotid-femoral pulse wave velocity measurement were performed. The ultrasound examination was performed using the Vivid E95 device (GE Ultrasound, Horten, Norway), operated by an skilled practitioner, pulse wave velocity was measured with Sphygmocor device (AtCor Medical Pty Ltd, West Ryde, New South Wales, Australia) with details described elsewhere [10]. Hypertension-mediated organ damage was defined identically as in our previous publication [10].

The study protocol adhered to the ethical principles outlined in the 1975 Declaration of Helsinki and received approval from the Bioethics Committee of Jagiellonian University in Krakow (1072.6120.39.2020). Written informed consent was obtained from all participants.

Statistical analysis

The normality of the variables was assessed using the Shapiro–Wilk test. To assess homogeneity of variance, Levene's test was used. Data were analyzed using descriptive statistics, with continuous variables presented as mean (standard deviation) or median (interquartile range) when appropriate and categorical variables as percentages. To assess the correlation between

continuous variables and BPV, correlation analysis was employed. For variables with a normal distribution, Pearson correlation coefficients were calculated. For variables that did not follow a normal distribution, Spearman correlation was applied. To evaluate differences in BPV between groups, the Student's t-test was used for normally distributed variables, and the Mann–Whitney test for variables without a normal distribution. Subsequently, a multivariable linear regression was conducted, incorporating variables that significantly influenced BPV in the univariate analysis. Lastly, age and BMI-adjusted multivariable regression analysis of 24 hour systolic blood pressure and average blood pressure variability with parameters of subclinical hypertension-mediated organ damage was performed. All statistical tests conducted were two-tailed, and a *P*-value of less than 0.05 was deemed statistically significant. SPSS version 28.0 (IBM Corporation, Armonk, NY, US) was used for all statistical analyses.

RESULTS

In this study, 219 participants were included, with 55% of them being females. The mean age of the participants was 55.3 (13.5). Women were significantly older than men. The prevalence of comorbidities — diabetes mellitus and hypercholesterolemia — was similar in both sexes. There were no differences in antihypertensive drug usage, except for less frequent use of calcium channel blockers in women. Table 1 presents the characteristics of the study population.

Women did not differ from men with respect to office BP values. In both sexes, unattended BP was significantly lower than attended BP. For SBP this difference was more pronounced in women than in men (4.5 [10.2] vs. 2.4 [4.7] mm Hg; $P = 0.04$ for SBP, and 2.2 [3.6] vs. 1.5 [3.6] mm Hg; $P = 0.11$ for DBP). Home blood pressure in the evening was lower in women at a borderline significance level, while significant differences between sexes were revealed in 24-hour monitoring (Figures 1 and 2).

Parameters of BPV in ABPM were higher in women for both systolic and diastolic blood pressure standard deviation (SD) in day-time interval, as well as for ARV (Figures 1 and 2).

With regard to office BPV women did not differ from men in any of parameters analyzed both for systolic and diastolic BP.

Home BPV was significantly higher in women for morning systolic and diastolic BP, and for evening systolic BP, with no differences between sexes for evening diastolic BPV measurements.

Figures 1 and 2 present the blood pressure values and BPV for each measurement method.

In the univariate analysis, parameters of BPV correlated significantly with BP values, most significantly for 24-hour measurements (correlation coefficient for SBP and SBP ARV: r = 0.364; *P* <0.001; DBP and DBP ARV: r = 0.186; *P* = 0.03). Significant correlations were also observed for office measurements (attended DBP and DBP SD: r = 0.332; *P* <0.001) and HBPM (morning SBP and SBP SD: r = 0.169; *P* = 0.02). For attended SBP and DBP in home measurements, there was a trend towards correlations (attended SBP and SBP SD: r = 0.129; *P* = 0.056; HBPM DBP and DBP SD: r = 0.120; *P* = 0.09).

Age correlated positively with BPV only for systolic BP in all of the following: 24-hour monitoring (SBP ARV: r = 0.284; *P* = 0.002; DBP ARV: r = –0.039; *P* = 0.68), UAOBP (SBP SD: r = 0.215; *P* = 0.02; DBP SD: r = –0.172; *P* = 0.06), and HBPM (SBP SD for morning measurements: r = 0.237; *P* = 0.01; DBP SD: r = 0.113; *P* = 0.22).

Body mass index was significantly correlated with BPV parameters (SBP ARV: r = 0.243; *P* = 0.007; DBP ARV: r = 0.200; *P* = 0.04), and there was a trend toward a correlation with 24-hour blood pressure values (SBP: r = 0.166; *P* = 0.07).

Patients with diabetes exhibited higher BPV compared to non-diabetics (office unattended DBP SD 2.98 [1.68] vs. 2.08 [1.48]; *P* <0.001). Moreover the concentration of HbA1c correlated with systolic BPV in 24 hourr monitoring (SBP ARV r = 0.234; *P* = 0.01) and HBPM (morning SBP SD r = 0.350; *P* <0.001). For DBP ARV there was a trend towards significant correlation (r = 0.170; r = 0.06).

Smokers presented higher BPV in the night-time measurements for both systolic (night-time SBP SD 12.42 [5.12] vs. 11.43 [3.95]; *P* = 0.04) and diastolic (night-time DBP SD 9.39 [3.86] vs. 8.26 [2.89]; *P* = 0.02) parameters.

Morning HBPM as well as 24 hour measurements showed also significant correlation with chemerin concentrations (home morning SBP SD r = 0.400; *P* <0.001; home morning DBP r = 0.419; *P* <0.001; 24 hour SBP ARV r = 0.304; *P* = 0.005; 24 hour DBP ARV r = 0.240; *P* = 0.03).

No statistically significant associations were observed between the levels of leptin and adiponectin and BPV.

Age, body composition parameters, glucose metabolism, lipid levels, and serum adipocytokine concentrations were not related to in-office BPV.

In the multivariable analysis, female sex remained significantly related to higher BPV, similarly like age and BP values (Table 2).

Blood pressure variability and subclinical organ damages

Analysis of the relationship of BPV with parameters of cardiac performance and arterial structure and function revealed that in women, BPV was significantly related to left ventricular mass and concentric geometry, left atrial function, and arterial stiffness. In men, BPV was associated only with parameters of diastolic function (Table 3).

DISCUSSION

The main finding of this study is that women despite lower BP values have higher BPV. Female sex, similarly like age and BP values are the main determinants of BPV, however numerous metabolic and life-style parameters also play a role.

The existence of a positive correlation between the value of BP values and BPV has been extensively documented in the scientific literature [9, 13]. In a similar fashion in our study, elevated BP values, regardless of the measurement methodology, were associated with higher BPV.

Many studies have investigated the impact of age on BPV [14, 15]. Consistent with our investigation, advanced age has been repeatedly associated with an augmentation of BPV. This can be partially accounted for by the diminished sensitivity of the baroreflex, a phenomenon associated with heightened arterial stiffness stemming from age-related alterations in the structural composition of the arterial vessel wall [16].

Women in our study population were older, a difference attributable to the recruitment of consecutive hypertensive outpatients. This reflects epidemiological data showing a lower prevalence of hypertension in premenopausal women compared to age-matched men. Further analyses were adjusted for age.

The observed in our study gender-based difference in BPV aligns with existing literature, suggesting potential physiological and hormonal influences [14, 17]. The activity of autonomic and endocrine BP regulating factors differs between sexes and may impact BP fluctuations and its association with target organ damage and cardiovascular morbidity.

It is well known that women in the course of hypertension develop more concentric left ventricular remodeling and have higher prevalence of left ventricular hypertrophy [18]. Similarly, enlargement of the left atrium, an early sign of hypertensive heart disease has also been reported to be more prevalent in women than in men [5]. Moreover, postmenopausal women present higher arterial stiffness than male counterparts [19]. These differences significantly contribute to the their higher risk for atrial fibrillation, heart failure with preserved left ventricular ejection fraction and stroke.

Our study found that in women BPV was associated with left ventricular mass, left atrial volume and function, and arterial stiffness. This suggests that BPV should be regarded as a significant target for managing BP and reducing cardiovascular risk, particularly in women. While optimizing BP with long-acting drugs remains crucial in hypertension management, it's worth noting that target BP levels for women in this regard might appear lower than those for men, given the strong dependency of BPV on BP. This is supported by studies showing women have more complications and higher cardiovascular risk at lower SBP levels than men [20].

Consistent with previous observations in the literature, 24-hour blood pressure was lower in women, while differences in office BP (both attended and unattended measurements) between sexes were not significant [21, 22]. This reflects a more pronounced white coat effect in women [23]. Blood pressure variability in office measurements also did not differ between sexes, while significant differences were observed for 24-hour, daytime, and nighttime BP in ABPM, as well as for HBPM. In the study by Omboni et al. [17], higher BPV in ABPM was also observed in the female group, but only in the 24-hour and daytime measurements.

Based on our results, home blood pressure measurements and day-to-day BP variability may have important value for additional cardiovascular risk assessment.

The other factors influencing BPV, concurrently serving as cardiovascular risk factors, encompass cigarette smoking, lipid profile parameters — total cholesterol and low-density lipoprotein, the presence of diabetes, and glycemic control (HbA1C). A significant impact of these factors on BPV has been documented by other investigators. In the study by Johannson et al., higher BPV was found in diabetic patients compared to non-diabetics [9]. Boubouchairopoulou et. al. [13], reported that cigarette smoking was associated with heightened BPV. Finally, in the research by Shin et al. [24], elevated lipid profile values were positively correlated with BPV. These factors collectively influence on arterial stiffness, potentially explaining the higher BPV in individuals with these risk factors [6, 25].

Our study shows that chemerin modulates BPV parameters, but its role lost statistical significance in multivariable analyses. Mechanisms linking chemerin to BPV include decreased nitric oxide, increased reactive oxygen species in endothelial cells, enhanced sympathetic nerve function, and induced vascular contraction [26]. These pathways likely explain chemerin's observed influence on BPV.

Considering the results of our study, interventions targeting modifiable risk factors can be proposed to reduce BPV. It appears that diminishing BPV may be achievable through lowering BP, smoking cessation, as well as achieving better glycemic control assessed by HbA1c.

A limitation of this study was the small number of in-office measurements and the absence of subsequent medical visits, preventing the comparison of BPV between visits. A notable strength of the research was the relatively large number of participants and the analysis of various BPV modalities using BP measurement methods in the same group of patients.

In conclusion, numerous pertinent factors, including metabolic ones, impact BPV across both short- and long-term durations. Despite lower BP values, women present higher BPV in HBPM and ABPM.

Blood pressure variability has a stronger relationship with parameters of target organ complications in women compared to men. Therefore, BPV should be assessed on par with BP values.

Including BPV in health monitoring can help better prevent organ damage and more accurately estimate cardiovascular risk, especially in women.

Reducing the BPV, as a multifaceted phenomenon, necessitates an integrated intervention focused on optimizing target BP values on one hand, and managing metabolic risk factors and lifestyle modifications, notably including tobacco cessation, on the other.

In addition, further research is needed to explore sex differences in BPV

Article information

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Table 1. Characteristics of the study population by sex

Parameters	Study group n = 219	Female, n = 120 (55%)	Male, n = 99 (45%)	*P*-value
Age, years, mean (SD)	55.3 (13.5)	59.6 (10.8)	52.2 (15.2)	<0.001[a]
Height, cm, mean (SD)	168 (10)	162.2 (6.5)	177.1 (7.1)	<0.001[a]
Weight, kg, mean (SD)	83 (15)	76.4 (13.6)	90.8 (13.6)	<0.001[a]
BMI, kg/m^2, mean (SD)	29 (4.7)	29.1 (5.2)	28.9 (3.9)	0.42[a]
Dyslipidemia, n (%)	119 (54.3)	62 (52)	57 (58)	0.41[b]
Diabetes mellitus, n (%)	17 (7.8)	9 (8)	8 (8)	1[b]
Smoking, n (%)	99 (45)	47 (39)	52 (53)	0.6[b]
Antihypertensive treatment, n (%)	201 (91.8)	112 (92)	89 (87)	0.52[b]
Number of antihypertensive drugs:				0.34[a]
0, n (%)	18 (8.2)	9 (7.5)	9 (9)	
1, n (%)	48 (22.4)	25 (21)	24 (24)	
2, n (%)	65 (29.7)	42 (35)	23 (23)	
3, n (%)	54 (24.7)	30 (25)	24 (24)	
4, n (%)	33 (15.1)	14 (12)	18 (19)	

5, n (%) 6, n (%)	10 (4.6) 1 (0.5)	7 (6) 0 (0)	3 (3) 1 (1)	
ACEi or ARB, n (%)	171 (78)	93 (77)	78 (79)	0.62
Diuretics, n (%)	93 (42)	52 (44)	41 (41)	0.91[b]
Calcium channel blockers, n (%)	85 (35)	38 (32)	47 (47)	0.03[b]
β-blockers, n (%)	110 (50)	66 (55)	44 (44)	0.10[b]
Potassium-sparing diuretics, n (%)	14 (6.4)	10 (8)	4 (4)	0.32[b]
Other antyhypertensive drugs, n (%)	12 (5.5)	4 (3)	8 (8)	0.22[b]
Statins, n (%)	92 (42)	46 (39)	46 (46)	0.30[b]
Glucose, mmol/l, median (IQR)	5.15 (4.75–5.6)	5.1 (4.7–5.4)	5.2 (4.8–5.6)	0.09
HbA1c, %, median (IQR)	5.7 (5.5–6)	5.8 (5.5–6)	5.6 (5.4–5.9)	0.10
eGFR, ml/min/ 1.73 m^2 [CKD-EPI], median (IQR)	79.5 (65.3–93.3)	86.0 (72.8–98.3)	69.2 (61.0–82.7)	<0.001[c]
Total cholesterol, mmol/l, median (IQR)	4.7 (4.1–5.4)	4.9 (4.4–5.6)	4.3 (3.6–5)	0.001 [c]
LDL-C, mmol/l, median (IQR)	2.5 (1.9–3.2)	2.7 (2.2–3.3)	2.2 (1.8–3.1)	0.09 [c]
HDL-C, mmol/l, median (IQR)	1.5 (1.2–1.8)	1.6 (1.4–1.8)	1.3 (1.1–1.6)	<0.001[c]
Non-HDL-C, mmol/l, median (IQR)	3.1 (2.5–3.9)	3.3 (2.8–3.9)	3.1 (2.4–3.9)	0.27 [c]
TG, mmol/l, median (IQR)	1.2 (0.9–1.7)	1.2 (0.9–1.6)	1.4 (0.9–1.8)	0.18 [c]
Adiponectin, µg/ml, median (IQR)	6.8 (4.6–9.7)	8.4 (6.8–11.1)	4.7 (3.1–6.6)	<0.001[c]
Leptin, ng/ml, median (IQR)	14.3 (8.3–26.4)	23.3 (14.3–37.3)	8.6 (5.5–12.8)	<0.001 [c]
Chemerin, ng/ml, median (IQR)	78.2 (68.3–87.3)	83.2 (73.8–91.4)	74.3 (64.5–79.9)	<0.001[c]

[a]*P* for T-test. [b]*P* for χ^2 test. [c]*P* for Mann–Whitney test

Abbreviations: ACEi, angiotensin-converting enzyme inhibitors; ARBs, angiotensin receptor blockers; BMI, body mass index; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non high-density lipoprotein cholesterol; TG, triglycerides

Table 2. Multivariable regression analysis of factors influencing 24 hour systolic blood pressure variability (SBP AVR)

Factor	Coefficient	SE	*P*-value
Female Sex	0.299	0.095	0.002
Age	0.247	0.096	0.01
Diabetes	–0.155	0.087	0.08
Smoking	0.063	0.089	0.48
BMI	0.012	0.090	0.89
BP	0.478	0.095	<0.001
Chemerin	0.115	0.090	0.21

Abbreviations: BP, blood pressure; SBP ARV, systolic blood pressure average real variability; other — see Table 1

Table 3. Age and BMI-adjusted multivariable regression analysis of 24 hour systolic blood pressure and average blood pressure variability with parameters of subclinical hypertension-mediated organ damage

	LVMi	**RTW**	**LAVI**	**LAS**	**E/e'**	**IMT**	**PWV**
24 h SBP							
b (SE)	0.25 (0.07)	0.13 (0.07)	0.11 (0.06)	-0.19 (0.06)	0.14 (0.06)	0.27 (0.05)	0.38 (0.06)
P-value	<0.001	0.05	0.10	0.003	0.04	<0.001	<0.001
24 h SBP WOMEN							
b (SE)	0.40 (0.08)	0.23 (0.08)	0.20 (0.09	-0.18 (0.08)	0.09 (0.08)	0.29 (0.07)	0.34 (0.08)
P-value	<0.001	0.01	0.03	0.02	0.28	<0.001	<0.001
24 h SBP MEN							
b (SE)	0.06 (0.1)	0.01 (0.1)	0.04 (0.1)	-0.12 (0.11)	0.24 (0.1)	0.14 (0.09)	0.36 (0.08)
P-value	0.55	0.91	0.71	0.27	0.02	0.15	<0.001
24 h SBP ARV							

b (SE)	0.14 (0.09)	0.16 (0.08)	0.10 (0.008)	-0.22 (0.08)	0.12 (0.08)	0.08 (0.07)	0.17 (0.07)
P-value	0.09	0.052	0.22	0.005	0.13	0.28	0.03
24 h SBP ARV WOMEN							
b (SE)	0.29 (0.11)	0.27 (0.10)	0.05 (0.12)	-0.23 (0.01)	0.01 (0.11)	0.11 (0.10)	0.35 (0.10)
P-value	0.009	0.01	0.63	0.02	0.95	0.26	0.001
24 h SBP ARV MEN							
b (SE)	0.03 (0.12)	0.13 (0.12)	0.13 (0.12)	-0.14 (0.12)	0.35 (0.11)	-0.04 (0.09)	-0.04 (0.03)
P-value	0.85	0.29	0.29	0.25	0.01	0.68	0.18

Abbreviations: E/e', early mitral inflow velocity to mitral annulus velocity ratio; IMT, intima media thickness; LAS, left atrial strain; LAVI, left atrial volume index; LVMi, left ventricular mass index; PWV, pulse wave velocity; SBP, systolic blood pressure; RTW, relative wall thickness; other — see Table 2

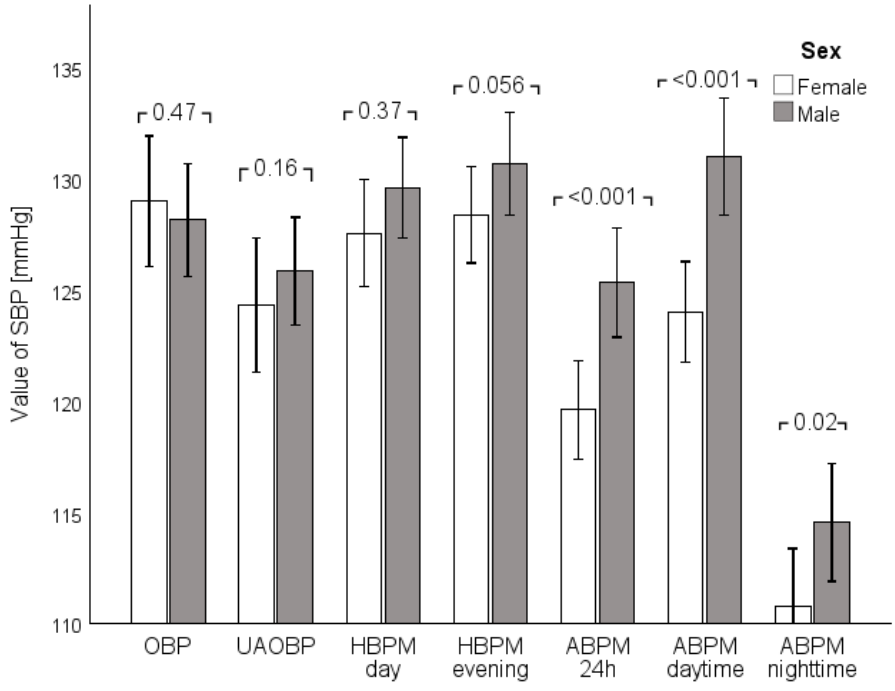

Figure 1. Values of systolic blood pressure and its variability depending on the measurement method — comparison between sexes

Abbreviations: ABPM, ambulatory blood pressure monitoring; HBPM, mean of 7 days of home blood pressure monitoring; OBP, office blood pressure measurement; SBP, systolic blood pressure; UAOBP, unattended office blood pressure measurement

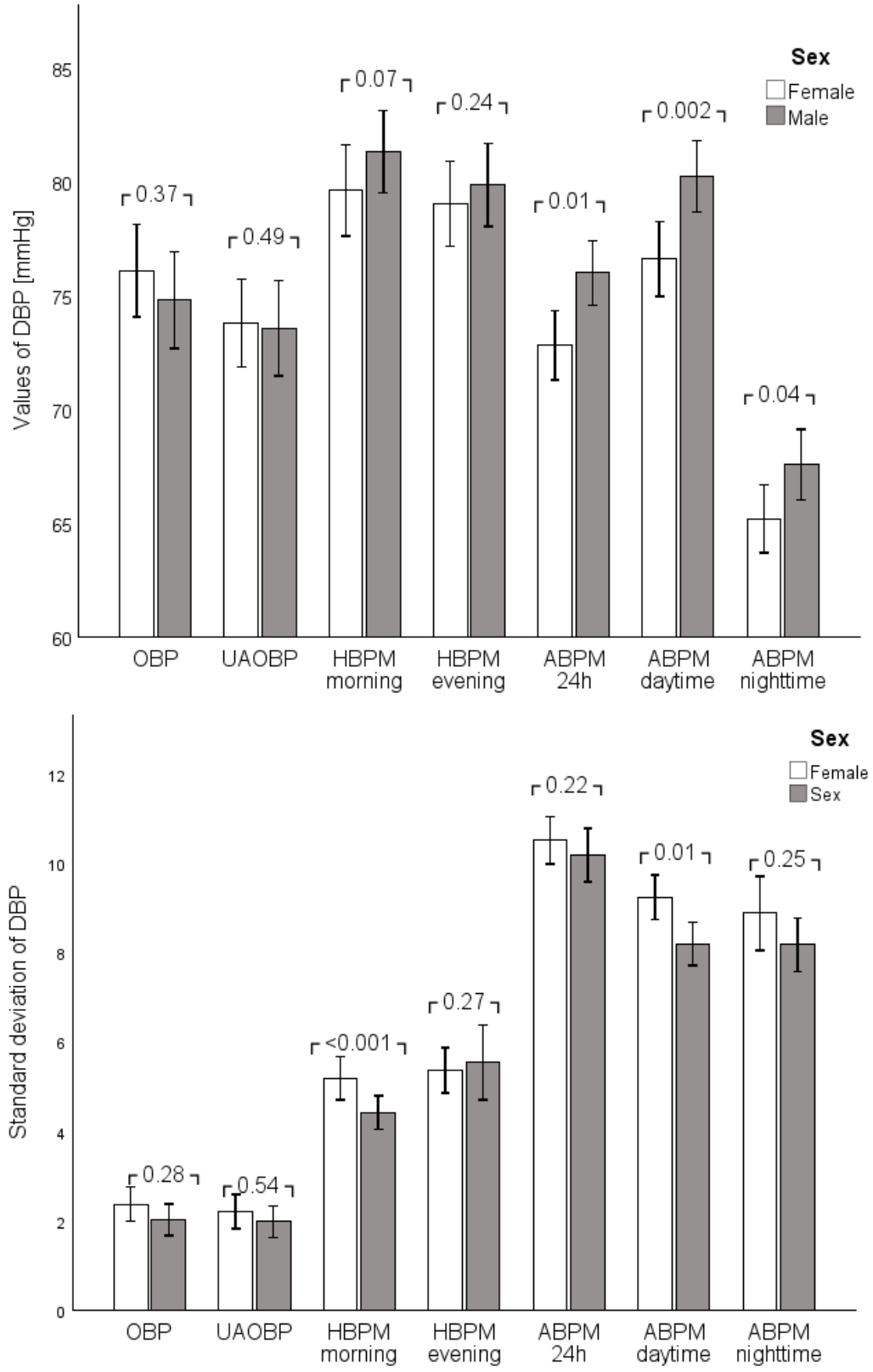

Figure 2. Values of diastolic blood pressure and its variability depending on the measurement method — comparison between sexes

Abbreviations: see Figure 1