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Blood Pressure Variability: Marker or Predictor of Cardiovascular Risk?

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Abstract

***Background*:** Regardless of the mean blood pressure (BP) value, short-term and long-term BP variability (BPV) are associated with the development and progression of target organ damage and predictors of cardiovascular complications and mortality. The purpose of the present study was to evaluate the prognostic significance of increased BPV in patients with arterial hypertension (AH).

Methods and Results: The study consisted of two stages. In the first stage, a retrospective analysis of 365 ABPM (24-hour ambulatory blood pressure monitoring) results was carried out. As a result of the analysis, 271 patients aged 56.1±10.0 years with uncontrolled AH Grades 1-3 (ESC/ESH, 2018) were included in this study. Depending on the values of BPV, AH patients were divided into two groups: Group 1 consisted of patients with normal BPV (n=145), and Group 2 consisted of patients with increased BPV (n=126). The second stage included 91 patients with uncontrolled hypertension without permanent antihypertensive therapy who had increased SBPV.

We found statistically significant differences in BP between the AH patients with normal BPV and increased BPV. Thus, in the group with normal BPV, compared with increased BPV, the parameters of the average 24-h systolic BP (SBP), daytime SBP, and nighttime SBP were statistically lower (141±14.6 vs. 147.2±20.2 mmHg, $P<0.004$; 142.8±15.1 vs. 148.4±20.7 mmHg, $P<0.01$; and 136.2±15.5 vs. 143.8±21.4 mmHg, $P<0.001$; respectively).

A statistically significant moderate direct correlation was found between the average 24-h SBP and the average 24-h and daytime SBP variability (SBPV) ($r_s=0.49$ and $r_s=0.40$ respectively, $P<0.001$ in all cases). A statistically significant moderate to weak direct correlation also was found between the average daytime SBP, and the average 24-h and daytime SBPV ($r_s=0.45$ and $r_s=0.37$, respectively, $P<0.001$ in all cases). A moderate direct correlation was found between nighttime SBP and 24-hour SBPV ($r_s=0.52$, $P<0.001$) and between nighttime SBP and daytime SBPV ($r_s=0.42$, $P<0.001$). Weak direct correlations were found between the average 24-h SBPV and central SBP (SBPc) ($r_s=0.34$, $P<0.001$), as well as between the average 24-h and daytime SBPV and central pulse pressure (PPc) ($r_s=0.33$ and $r_s=0.32$, respectively, $P<0.001$ in all cases). A weak direct correlation was found between carotid intima-media thickness (CIMT) and the average 24-h and daytime SBPV ($r_s=0.37$ [$P<0.001$] and $r_s=0.3$ [$P=0.04$]).

Conclusion: The increased SBPV is associated with impaired diurnal blood pressure profile (DBPP) and structural and functional changes in blood vessels, in particular, an increase in SBPc and PP in the aorta, and CIMT thickening, which characterizes increased BPV as a predictor of vascular remodeling in patients with uncontrolled AH.(**International Journal of Biomedicine. 2023;13(3):66-71.**)

Keywords: arterial hypertension • blood pressure variability • 24-hour ambulatory blood pressure monitoring • target organ damage

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Abbreviations

ABPM, 24-hour ambulatory blood pressure monitoring; **AH**, arterial hypertension; **ARV**, average real variability; **BP**, blood pressure; **BPV**, BP variability; **BMI**, body mass index; **CIMT**, carotid intima-media thickness; **DBP**, diastolic BP; **DBPP**, diurnal blood pressure profile; **DBPV**, DBP variability; **GFR,** glomerular filtration rate; **IVST**, interventricular septal thickness; **LVDD**, LV diastolic dysfunction; **LVH**, left ventricular hypertrophy; **MAU**, microalbuminuria; **PP**, pulse pressure; **PPc**, central pulse pressure; **PWV**, pulse wave velocity; **PWT**, posterior wall thickness; **SBPV**, SBP variability; **SBP**, systolic BP; **uACR**, urine albumin-creatinine ratio.

Introduction

High blood pressure (BP) is a leading risk factor for cardiovascular disease. BP shows marked fluctuations in the short and long term.[1] Such fluctuations over time, expressed in appropriate terms of descriptive statistics (standard deviation, coefficient of variation), are called BP variability (BPV). Regardless of the mean BP value, short-term and long-term BPV are associated with the development and progression of target organ damage and predictors of cardiovascular complications and mortality.[2,3]

For a long time, BPV was considered as a random variable that does not deserve attention when working with a patient. The impetus for the beginning of BPV study was the introduction into clinical practice of the technique of daily monitoring of blood pressure (ABPM) in the 1970s. One of the first works concerning the study of BP variability dates back to the early 1990s when Italian scientist Frattola and colleagues,[4] using invasive 24-hour BP monitoring, demonstrated the relationship between increased BPV and the severity of target organ damage. All markers of target organ damage (microalbuminuria, increased PWV, left ventricular hypertrophy, and atherosclerotic plaques in the carotid arteries) are independent predictors of death from cardiovascular disease.

In 2000, *Circulation* published an article by Dirk Sander and co-authors[5] devoted to the study of the association of BPV with the risk of early progression of atherosclerosis in patients with hypertension. In 2010, a number of publications appeared with the results of the ASCOT BPLA study on BPV, in which, for the first time, BP was assessed not by the results of ABPM but within and between visits. Thus, in *The Lancet*, the results of an analysis of BPV, conducted by Professor Peter Rothwell,[6,7] were published, according to which high long-term variability in systolic blood pressure (SBP) has a much more pronounced direct relationship with the frequency of cerebrovascular and coronary events than the average level of SBP in the brachial artery.

The purpose of the present study was to evaluate the prognostic significance of increased BPV in patients with arterial hypertension (AH).

Materials and Methods

The study consisted of two stages. In the first stage, a retrospective analysis of 365 ABPM results was carried out. As a result of the analysis, 271 patients aged 56.1±10.0 years with uncontrolled AH Grades 1-3 (ESC/ESH, 2018) were included in this study.

Exclusion criteria were symptomatic hypertension, acute coronary syndrome, chronic heart failure (NYHA FC>III), cardiac arrhythmia, history of stroke and myocardial infarction, diabetes, renal impairment, severe co-morbidities.

Office BP was measured using a mercury sphygmomanometer, according to Korotkov's method. BP was measured 3 times, and the means of these measurements were used in the analyses. The 24-hour ABPM was performed using a BR-102 plus (SCHILLER, Switzerland). BP was measured during the daytime (07:00–23:00) every 30 min and at night (23:00–07:00) every 60 min. The interpretation of the results was based on generally accepted recommendations for ABPM quality criteria: monitoring duration of at least 23 hours, 50 successful measurements, and no "gaps" in the record lasting more than 1 hour.

Depending on the values of BPV, AH patients were divided into two groups: Group 1 consisted of patients with normal BPV (n=145), and Group 2 consisted of patients with increased BPV (n=126). When creating groups with increased and normal variability in SBP and DBP, we conditionally set the threshold levels of daytime SBP variability (SBPV) – 15 mmHg, daytime DBP variability (DBPV) – 14 mmHg, and nighttime SBPV and DBPV – 15 mmHg and 12 mmHg, respectively.

The second stage included 91 patients with uncontrolled hypertension without permanent antihypertensive therapy who had increased SBPV, according to ABPM. The mean age of the patients was 52.8±11.9 years.

The pulse contour analysis was carried out using the SphygmoCor device (AtCor Medical, Australia), which obtains peripheral arterial pressure waveforms by applying an arterial applanation tonometer to the wrist. Such indicators as the central SBP (SBPc), central DBP (DBPc), central PP (PPc), and PWV were analyzed.

All patients underwent echocardiography with the determination of the left ventricular mass index (LVMI), ultrasound examination of the carotid intima-media thickness (CIMT), as well as the determination of the level of microalbuminuria (MAU), blood creatinine, and glomerular filtration rate (GFR) calculation according to the CKD-EPI equation.

Statistical analysis was performed using the statistical software package SPSS version 26.0 (SPSS Inc, Armonk, NY: IBM Corp). The normality of distribution of continuous variables was tested by the Kolmogorov-Smirnov test with the Lilliefors correction and Shapiro-Wilk test. Baseline characteristics were summarized as frequencies and percentages for categorical variables and as mean± standard deviation (SD) for continuous variables. For data with normal distribution, inter-group comparisons were performed using Student's t-test. Spearman's rank correlation coefficient (r_s) was calculated to measure the strength and direction of the relationship between two variables. A probability value of $P<0.05$ was considered statistically significant.

The study protocol was reviewed and approved by the Ethics Committee of the Republican Specialized Centre of Cardiology. All participants provided written informed consent.

Results

At the first stage, after analyzing the data of 271 ABPM in patients with uncontrolled hypertension, we obtained a wide range of characteristics of SBPV and DBPV. In 153(56.4%) patients, an increased average 24-h SBPV of 16.1±4.4mm was noted. Increased average 24-h DBPV (12.6±3.4 mmHg) was noted in 84(31%) patients.

In the group with increased BPV, the average daytime SBPV and DBPV were 19.1±4.2 and 13.8±3.6 mmHg, respectively. The average nighttime SBPV and DBPV were 15.5±6.1 and 12.4±7.7 mmHg. We found statistically significant differences between the AH patients with normal BPV and increased BPV. Thus, in the group with normal BPV, compared with increased BPV, the parameters of the average 24-h SBP, daytime SBP, and nighttime SBP were lower by 6mmHg (141±14.6 vs. 147.2±20.2 mmHg; $P<0.004$), 6 mmHg (142.8±15.1 vs. 148.4±20.7 mmHg; $P<0.01$), and 7 mmHg (136.2±15.5 vs. 143.8±21.4 mmHg; $P<0.001$), respectively.

In addition, between the groups with normal BPV and increased BPV, there were statistically significant differences in the degree of nocturnal fall in SBP: 5.4±10.7 mmHg and 2.9±8.3 mmHg, respectively ($P<0.03$) (Table 1). There was a greater tendency for higher values of the daytime and nighttime load of SBP and DBP in the group with increased BPV than in the group with normal BPV. The value of pulse pressure (PP) also prevailed in the group with increased BPV, compared with normal BPV (62±14.3 mmHg versus 59.6±11.7 mmHg, but without statistically significant differences (Table 1).

When assessing nocturnal BP decrease (dipping) in AH patients with normal and increased BPV, it turned out that most patients in both groups had an unfavorable daily BP index. In all patients, the pathological variants of DBPP were found, among which a "non-dipper" variant prevailed (51% of cases in the group with normal BPV and 42.8% in the group with increased BPV). In the group with normal BPV, 25.5% of cases had the optimal degree of nocturnal SBP reduction (dipper), while in the group with increased BPV it was 19.8%. In the group with normal BPV, there were 1.6 times fewer people with a steady increase in night SBP (night-peaker) than in the group with increased BPV (22.7% and 36.5%, respectively), which has an important prognostic value. Patients with an increased degree of nocturnal SBP reduction (over dipper) were 0.60% and 0.79%, respectively. No statistically significant differences were found when comparing the indicators of nocturnal BP-dipping.

The second stage analysis, where 91 patients with uncontrolled hypertension and increased SBPV were examined, showed that a high cardiovascular risk characterized the patients. Thus, more than 80% of patients had increased body weight and obesity, half were diagnosed with LV hypertrophy and LV diastolic dysfunction, and 60% were diagnosed with increased PWV and thickening of CIMT (Table 2).

Evaluation of BPV during the office BP measurement showed that the number of patients with a difference in SBP of more than 5 mmHg between the first and second measurements amounted to 15(16.4%) patients. Between the second and third measurements, the difference was also

Table 1.
ABPM indicators considering BPV.

Parameter	Normal BPV		Increased BPV		*P*-value
	M±SD	95% CI	M±SD	95% CI	
Average 24-h SBP, mmHg	141±14.6	139-143	147.2±20.2	143.7-150.7	0.004
Average 24-h DBP, mmHg	83.3±12.07	81-85	85.6±13.4	83.2-87.9	0.138
Average daytime SBP, mmHg	142.8±15.1	140-145	148.4±20.7	144.7-152	0.01
Average daytime DBP, mmHg	84.9±12.6	82.9-86.9	86.9±13.7	84.5-89.3	0.211
Average nighttime SBP, mmHg	136.2±15.5	133.7-138.72	143.8±21.4	140.1-147.6	0.0001
Average nighttime DBP, mmHg	78.7±11.8	76.8-80.6	81.6±14.4	79.1-84.1	0.069
Nocturnal SBP fall,%	5.4±10.7	3.7-7.2	2.9±8.3	1.4-4.4	0.03
Nocturnal DBP fall,%	7.4±8.9	6-9	6.3±9.6	4.6-8.05	0.328
Daytime SBP load, %	57.6±25.3	53.5-61.7	62.3±22.2	58.4-66.2	0.107
Daytime DBP load, %	39.1±30.7	34.1-44.1	43.1±29.1	38-48.2	0.273
Nighttime SBP load, %	83.4±19.7	80.2-86.6	86.2±18.8	82.9-89.5	0.234
Nighttime DBP load, %	48.8±32	43.6-53.9	51.5±32.7	45.8-57.2	0.493
PP, mmHg	59.6±11.7	57.7-61.53	62±14.3	59.5-64.5	0.129

observed in 16(17.5%) patients, and between the first and third measurements - in 50(55%) patients. In 10(11%) patients, we did not find a difference >5 mmHg between measurements.

Table 2.

Clinical characteristics of AH patients (n=91) with increased SBPV

Parameter		
SBP, mmHg (M±SD / 95% CI)	161.3±14.5	157.3-165.3
DBP, mmHg (M±SD / 95% CI)	93.1±10.6	90.1-96.04
BMI >30 kg/m^2, (n / %)	48	52.7%
BMI >25<30 kg/m^2, (n / %)	25	27.4%
LVH, (n / %)	31	34%
LVDD, (n / %)	16	17.5%
PWV >10 m/sec, (n / %)	31	34%
CIMT ≥0.9 mm, (n / %)	28	30.7%

In AH patients with increased BPV, the assessment of the relationship between BP and ABPM data on the Chaddock scale revealed a statistically significant moderate direct correlation between the average 24-h SBP and the average 24-h and daytime SBPV (r_s=0.49 and r_s=0.40 respectively, *P*<0.001 in all cases). A statistically significant moderate to weak direct correlation also was found between the average daytime SBP, and the average 24-h and daytime SBPV (r_s=0.45 and r_s=0.37, respectively, *P*<0.001 in all cases). A moderate direct correlation was found between nighttime SBP and 24-hour SBPV (r_s=0.52, *P*<0.001) and between nighttime SBP and daytime SBPV (r_s=0.42, *P*<0.001). Weak direct correlations were also found between the average 24-h SBPV and SBPc (r_s=0.34, *P*<0.001), as well as between the average 24-h and daytime SBPV and PPc (r_s=0.33 and r_s=0.32, respectively, *P*<0.001 in all cases). A weak direct correlation was found between CIMT and the average 24-h and daytime SBPV (r_s=0.37 [*P*<0.001] and r_s=0.3 [*P*=0.04]). We did not find statistically significant correlations between the increased SBPV, parameters of the functional state of the kidneys, and indicators of the structural state of the left ventricle. It should also be emphasized that increased nighttime SBPV was also not significantly associated with the studied parameters (Table 3).

Table 3.

Correlation analysis between increased SBPV, ABPM data, and target organ damage parameters.

Parameter	Average 24-h SBPV		Average daytime SBPV		Average nighttime SBPV	
	r_s	*P*	r_s	*P*	r_s	*P*
Average 24-h SBP	0.49	<0.001	0.40	<0.001	0.037	0.724
Average daytime SBP	0.45	<0.001	0.37	<0.001	0.015	0.884
Average nighttime SBP	0.52	<0.001	0.42	<0.001	0.11	0.289
SBPc	0.34	0.001	0.33	0.001	0.08	0.443
DBPc	0.16	0.118	0.17	0.1	-0.1	0.36
PPc	0.33	0.002	0.32	0.001	0.14	0.131
PWV	-0.193	0.06	-0.2	0.06	-0.07	0.5
CIMT	0.37	<0.001	0.3	0.04	0.2	0.06
IVST	0.22	0.03	0.18	0.07	-0.33	0.755
PWT	0.071	0.5	0.04	0.65	-0.026	0.8
LVMI	0.071	0.5	0.03	0.75	0.055	0.6
uACR	0.02	0.8	0.05	0.63	0.003	0.974
GFR	-0.12	0.2	-0.14	0.16	-0.045	0.67
Creatinine	0.14	0.17	0.244	0.18	0.15	0.14
MAU	-0.21	0.845	-0.46	0.65	-0.023	0.83

Discussion

The relationship between increased BPV and cardiovascular diseases has been shown in previous studies.[8-11] A number of studies reported significant associations between high average real variability (ARV) and the presence and progression of subclinical organ damage.[12-16]

In our patients with uncontrolled AH and increased SBPV, the average 24-h, daytime and nighttime SBP values were higher than in AH patients with normal SBPV. It can be assumed that impaired BPV is associated with an additional increase in SBP and significantly increases the risk of damage to target organs. Analysis of DBPP showed that in the AH patients with increased SBPV, there was a significantly low rate of nighttime SBP reduction, thereby placing an additional load on the target organs.

In addition, data from a number of studies indicate that increased SBP is associated with the progression of atherosclerosis. Thus, in a well-known meta-analysis,[17] after adjusting for demographic indicators, a correlation was found between SBP and the progression of atherosclerosis. After adjusting for age, patients' PWV was shown to increase by 1.14 m/s for every 20 mmHg increase in SBP. However, the correlation between mean BP or DBP and PWV was weak, while it was negative in our study. Many investigators have studied potential mechanisms for the relationship between BPV and poor cardiovascular outcomes. First, BPV may be a marker of arterial stiffness associated with reduced compliance of large elastic arteries. In a study by Tursunova et al.,[18] increased daytime SBPV and PP variability in patients with isolated systolic AH correlated with an increase in arterial stiffness, compared with patients with systolic-diastolic AH, and an increased vascular stiffness (carotid-femoral PWV >10 m/s) increased the risk of developing LV concentric hypertrophy and concentric remodeling, which adversely affected the prognosis.[19]

Our data on the relationship between BPV and kidney damage are consistent with a prospective study by Hung et al.,[20] which included 300 Han Chinese participants with hypertension (mean age of 63.5 years) and investigated whether short-term BPV is correlated with hypertensive nephropathy. Five different BPV parameters were derived from ambulatory BP monitoring (ABPM), including standard deviation (SD), weighted SD (wSD), coefficient of variation (CoV), successive variation (SV), and ARV. The renal event was defined as >50% reduction in baseline eGFR. The Cox proportional hazard regression (HR) model to assess the independent effects of BPV showed that 24-h SBP (HR=1.105; 95% CI=1.020-1.197, *P*=0.015) and 24-h DBP (HR=1.162; 95% CI=1.004-1.344, *P*=0.044) were independently associated with renal events. However, BPV parameters were only associated with renal events univariately, but not after adjusting for baseline characteristics, 24-h mean BP, and office BP.

In this study, we found a direct correlation between CIMT and the average 24-h and daytime SBPV. This is consistent with a study by Xiong et al.[15] who provided the evidence that, for the subjects from the southern area of China, all of the indices of SBPV for daytime and 24 h had significant correlation with CIMT.

Short-term variability of 24-hour SBP showed an independent, although moderate, relation to aortic stiffness in hypertension in a study by Schillaci et al.[16] These results were consistent with our findings.

Conclusion

Our data showed that increased BPV is associated with impaired diurnal blood pressure profile and structural and functional changes in blood vessels, in particular, an increase in SBPc and PP in the aorta, and CIMT thickening, which characterizes increased BPV as a predictor of vascular remodeling in patients with uncontrolled AH. A well-controlled 24-h and daytime SBPV should be prioritized in managing AH.

Competing Interests

The authors declare that they have no competing interests.

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