

UDC 618.3-06-008.6:616.12-008.331.1-07"34"

DOI: 10.15587/2519-4798.2026.362854

BLOOD PRESSURE MONITORING IN PREGNANT WOMEN AT RISK OF DEVELOPING PREECLAMPSIA

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The aim of research – to determine the hemodynamic patterns of preeclampsia development in pregnant women based on ambulatory blood pressure monitoring.

Material and methods. A prospective cohort study included 161 women in the second half of pregnancy. The main group consisted of 77 pregnant women at risk for preeclampsia; the comparison group included 50 women with gestational hypertension; the control group consisted of 34 healthy pregnant women. Blood pressure was sampled by using a non-invasive automatic ambulatory blood pressure monitoring machine for 24h.

Results. Although the average 24-hour BP is lower than the established level of 140/90 mmHg, a more detailed evaluation during ambulatory blood pressure monitoring (ABPM) in pregnant women at high risk of developing gestational hypertension and preeclampsia at the preclinical stage allows to identify following predictors: an increase systolic (SBP), diastolic (DBP), and mean arterial pressure and their amplitude; an increase the rate of the morning surge of SBP to 16.4 ± 2 and DBP to 14.5 ± 2.4 mmHg/hour; a decrease circadian index of SBP to $9.8 \pm 0.64\%$ and DBP to $7.7 \pm 1\%$; an increase SBP variability (day/night) = $12.2 \pm 0.6 / 9.77 \pm 0.4$ and DBP variability (day/night) = $9.1 \pm 0.44 / 8.41 \pm 0.45$; the occurrence of elevated blood pressure episodes during the daytime (SBP up to $2.0 \pm 0.53\%$, DBP up to $2.15 \pm 0.36\%$) and during the nighttime period (SBP up to $5.6 \pm 1.4\%$, DBP up to $7.85 \pm 0.36\%$).

Conclusions. ABPM allows to detect pathological hemodynamic changes (specifically isolated nocturnal hypertension and increased BP variability) at the preclinical stage. This makes the method indispensable for predicting preeclampsia and the timely adjustment of clinical management strategies for high-risk patients

Keywords: pregnancy, preeclampsia, ambulatory blood pressure monitoring, hemodynamics, nocturnal hypertension, blood pressure variability, endothelial dysfunction

How to cite:

Loskutova, T., Petulko, A. (2026). Blood pressure monitoring in pregnant women at risk of developing preeclampsia. ScienceRise: Medical Science, 1 (66), 29–34. <http://doi.org/10.15587/2519-4798.2026.362854>

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1. Introduction

Hypertensive disorders in pregnancy (HDP) remain among the most challenging problems in modern obstetrics, complicating 5% to 10% of all pregnancies worldwide, with a steadily increasing trend observed over recent decades [1]. This rise is associated with the increasing average maternal age, the obesity epidemic, the widespread use of assisted reproductive technologies, and the growing prevalence of concomitant comorbidities [2]. Traditional office blood pressure (BP) measurement records only point-in-time hemodynamic parameters, failing to reflect 24-hour circadian fluctuations, identify "white coat" phenomena or masked hypertension, and – most importantly – assess nocturnal blood pressure levels [3, 4]. It is the disruption of the nocturnal hemodynamic profile that serves as a critically important predictor of adverse obstetric and cardiovascular events. In this regard, ambulatory blood pressure monitoring (ABPM) acts as an indispensable tool for assessing the vascular status of pregnant women, particularly in high-risk

groups, allowing to detect subclinical predictors of preeclampsia (PE) long before clinical manifestation.

Endothelial dysfunction in PE does not develop instantaneously. For an extended period, the maternal organism attempts to compensate these alterations by activating the sympathoadrenal system and the renin-angiotensin-aldosterone system. At this stage, while office blood pressure may remain within normal limits due to daytime adaptive mechanisms, ambulatory blood pressure monitoring (ABPM) enables the detection of early warning signs, such as circadian rhythm disruption, elevated nocturnal pressure, and increased variability of hemodynamic parameters. Consequently, isolated office blood pressure measurement during a physician visit is insufficient for an adequate assessment of the status of high-risk pregnant women [5].

Over the last decade, the evidence base supporting the superiority of ambulatory blood pressure monitoring over routine clinical (office) measurement has steadily

expanded. The use of ABPM allows to identify specific clinical phenotypes of arterial hypertension. Identifying these phenotypes is of fundamental importance for the stratification of obstetric risks and the selection of clinical management strategies for pregnancy [3]. White-coat hypertension (WCH) is distinguished as a condition where office blood pressure consistently exceeds threshold values (140/90 mmHg), whereas mean 24-hour ABPM values remain within normal reference ranges. In pregnant women, the presence of WCH is associated with an increased risk (compared to normotensive women) of progression to true gestational hypertension or preeclampsia; thus, these patients require close monitoring. However, identifying WCH helps avoid the unwarranted prescription of antihypertensive medications, which could potentially lead to iatrogenic arterial hypertension and dangerous placental hypoperfusion [3].

Secondly, masked hypertension (MH) is characterized normal office blood pressure but consistently elevated values according to ABPM [3]. In obstetric practice, masked hypertension has the greatest danger as it remains undiagnosed during routine antenatal surveillance. Patients with MH carry a similar, or even higher, risk of developing severe PE, fetal growth restriction, and placental abruption as women with sustained hypertension [5, 6].

Thirdly, isolated nocturnal hypertension. Physiological pregnancy is characterized by a nocturnal reduction in blood pressure (the "dipping" phenomenon), which provides necessary recovery for the cardiovascular system and optimizes uteroplacental blood flow during sleep [7]. An increase blood pressure exclusively at night (or the absence of its physiological decline) is one of the earliest markers of endothelial dysfunction, increased vascular resistance, and impaired autonomic regulation [8]. The presence of nocturnal hypertension is regarded as a potent independent predictor for the development of preeclampsia [5].

The aim of the study was to identify the hemodynamic patterns of preeclampsia development in high-risk pregnant women based on 24-hour ambulatory blood pressure monitoring.

2. Materials and methods

The study was conducted at Obstetrics and gynecology department of Dnipro State Medical University, Dnipro, Ukraine, in 2020–2024 which based in Municipal non-commercial enterprise "City multi-profile clinical hospital for mother and children named after prof. M. F. Rudniev" of the Dnipro city council. A prospective cohort study covered 161 women in the second half of pregnancy. The diagnosis of Pregnancy induced Hypertension and estimation pregnancy of high-risk preeclampsia development was based on Order No. 151 of the Ministry of Health of Ukraine [9]. The exclusion criteria for the study were the presence of Anti-Phospholipid Syndrome, Chronic Hypertension, Diabetes Mellitus, Glomerulonephritis.

The study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki, following Good Clinical Practice guidelines and applicable legal regulations, and was approved by the Ethics Committee of the Dnipro State Medical University (protocol No. 5 dated September 21, 2020). Informed consent to participate in the study was obtained from all participants.

The main group (high-risk group) consisted of 77 pregnant women whose individual risk of developing preeclampsia, as calculated by first-trimester combined screening, exceeded the 1:100 threshold, or who had high-level clinical predictors of PE development in accordance with Order No. 151 of the Ministry of Health of Ukraine. Comparison group – 50 pregnant women with gestational hypertension or mild PE which was diagnosed based on an Order No. 151 of the Ministry of Health of Ukraine. The control group (C) was formed by 34 conditionally healthy pregnant women with a non-complicated obstetrical anamnesis and without risk factors for preeclampsia. All women were undergoing a clinical and laboratory examination. Data recorded included maternal age, gestation week at diagnosis, gravidity, parity, previous preeclampsia, blood pressure, proteinuria, gestation weeks at delivery, birth weight, stillbirth. Laboratory data at diagnosis included liver and renal function, platelet count, hematocrit, and hemoglobin.

Central hemodynamics were assessed using 24-hour ambulatory blood pressure monitoring systems, "Solveig – ABP-01" (Ukraine) and ABPM-02 ("Meditech", Hungary), with measurements taken every 15 minutes during the day and every 30 minutes at night. The following parameters were determined automatically: systolic (SBP), diastolic (DBP), mean arterial pressure (MAP), and heart rate (HR), as well as their minimum, maximum, and average values for the daytime, nighttime, and 24-hour periods. Additionally, the pressure load index (PLI) and blood pressure variability (BPV) were recorded. Based on the average daytime and average nighttime SBP and DBP, the daily index (DI), the amplitude of SBP, DBP, MAP, and HR and the morning surge rate (MSR) of SBP and DBP were calculated.

Statistical processing of the study results was performed by using licensed computer programs Microsoft Excel 2010 and Graph Pad Prism 5 using methods of parametric and nonparametric statistics. The normality of the distribution of quantitative traits was assessed using Shapiro-Wilk and Kolmogorov-Smirnov criteria, analysis of variance, odd t-test, Mann-Whitney test, χ^2 test with conjugation of conjugation tables and Yates correction, Fisher's exact test was used. The difference between the values was considered significant by $p < 0.05$.

3. Results

Clinical characteristics of the study groups demonstrated that they were representative in terms, age and parity. The mean age of women in the primary group was 25.4 ± 0.55 years, in the control group – 25.13 ± 0.68 years, and in the comparison group – 26.16 ± 0.8 years. The number of primiparous women in the primary group was 62 (80.5%), in the control group – 26 (76.5%), and in the comparison group – 37 (74%) ($p > 0.05$).

The primary ABPM indicators, specifically: SBP (systolic blood pressure), ASBP (average systolic blood pressure), DBP (diastolic blood pressure), ADP (average diastolic blood pressure), MAP (mean arterial pressure), AMAP (average mean arterial pressure), HR (heart rate), AHR (average heart rate), BPV (blood pressure variability), and PLI (pressure load index) for 24-hour, daytime, and nocturnal periods, along with their comparative characteristics across the study groups, are presented in Table 1.

Table 1

Comparative characteristics of ABPM indicators across clinical groups (M ± m)

ABPM Parameters		Clinical groups				
		Control, n=34	Comparison, n=50	Δ %	Main, n=77	Δ %
SBP (mmHg)	24-hour	102.3 ± 2	122.5 ± 2.47*	+19.7	109.5 ± 0.95*.#	+ 7
	Daytime	105.3 ± 2.4	124.3 ± 2.4*	+18	112.0 ± 1.1*.#	+ 6.4
	Nighttime	91.9 ± 3.2	115.2 ± 2.9*	+25.3	101.0 ± 1*.#	+ 9.9
ASBP (mmHg)	24-hour	47.2 ± 1.9	61.0 ± 3.4*	+29.2	51.1 ± 2.5#	+ 8.3
	Daytime	39.0 ± 2.5	55.0 ± 3.8*	+41	49.8 ± 2.7*	+ 27.6
	Nighttime	26.3 ± 2.7	34.2 ± 2.4*	+30	36.2 ± 1.4*	+ 37.6
DBP (mmHg)	24-hour	60.5 ± 1.5	80.5 ± 1.7*	+33.3	65.8 ± 0.9*.#	+ 8.8
	Daytime	64.0 ± 1.6	81.8 ± 1.78*	+27.8	68.3 ± 0.87*.#	+ 6.7
	Nighttime	57.0 ± 1.56	78.8 ± 1.7*	+38.2	62.9 ± 1.29*.#	+ 10.4
ADBP (mmHg)	24-hour	37.0 ± 2.4	48.9 ± 2.85*	+16.1	42.9 ± 2.7	+ 15.9
	Daytime	34.2 ± 2.1	46.3 ± 3.1*	+35.4	39.1 ± 2.58	+ 14.3
	Nighttime	19.3 ± 2.8	30.3 ± 1.4*	+57	33.7 ± 1.25*.#	+ 74.6
MAP (mmHg)	24-hour	74.4 ± 1.47	94.4 ± 1.84*	+26.9	80.4 ± 0.7*.#	+ 8
	Daytime	77.2 ± 1.25	95.6 ± 1.93*	+23.8	82.8 ± 0.82*.#	+ 7.3
	Nighttime	68.6 ± 1.8	90.8 ± 2*	+32.5	75.6 ± 1*.#	+ 10.2
AMAP (mmHg)	24-hour	35.6 ± 1.7	44.2 ± 2.3*	+24	43.4 ± 2.6*	+ 21.9
	Daytime	31.5 ± 1.7	41.2 ± 2.5*	+30.8	38.5 ± 1.2*	+ 22.2
	Nighttime	17.3 ± 2.8	29.8 ± 1.2*	+72.3	28.1 ± 1.3*	+ 62.4
HR (bpm)	24-hour	88.7 ± 1	87.0 ± 1.4	-2	91.0 ± 1.1	+ 2.6
	Daytime	90.9 ± 1.1	89.0 ± 1.5	-2.1	95.0 ± 1.1	+ 4.5
	Nighttime	81.0 ± 1.5	78.6 ± 1.4	-3	84.4 ± 1.6	+ 4.2
AHR (bpm)	24-hour	38.3 ± 3.8	52 ± 1.9*	+35.7	56.1 ± 2.6*	+ 46.5
	Daytime	36.5 ± 3.9	50.2 ± 2*	+37.5	51.4 ± 3.2*	+ 40.8
	Nighttime	15.5 ± 1.6	21.7 ± 2*	+40	26.8 ± 1.6*	+ 72.9
BPV 24-hour, (mmHg)	SBP	8.6 ± 0.53	13.5 ± 0.7*	+57	11.9 ± 0.4*	+ 38.4
	DBP	7.32 ± 0.76	10.0 ± 0.4*	+36.6	9.9 ± 0.45*	+ 35.2
	HR	7.6 ± 1.25	10.5 ± 0.36*	+34.2	13.0 ± 0.53*.#	+ 71
BPV daytime, (mmHg)	SBP	9.83 ± 0.4	13.8 ± 0.87*	+40.8	12.2 ± 0.6*	+ 24
	DBP	7.47 ± 0.62	9.3 ± 0.5*	+25.7	9.1 ± 0.44*	+ 21.8
	HR	6.78 ± 0.84	10.4 ± 0.35*	+52.9	12.3 ± 0.62*.#	+ 81.4
BPV nighttime, (mmHg)	SBP	6.78 ± 0.35	12.8 ± 0.8*	+76.5	9.77 ± 0.4*	+ 44
	DBP	5.76 ± 0.66	8.8 ± 0.65	+52.8	8.41 ± 0.45*	+ 46
	HR	4.8 ± 0.59	6.4 ± 0.64	+33.3	7.8 ± 0.3*	+ 62.5
PLI 24-hour, %	SBP	-	24.2 ± 4.5	+100	3.74 ± 0.7#	+ 100
	DBP	-	27.0 ± 5.3	+100	6.0 ± 0.27#	+ 100
	MAP	-	37.1 ± 5.85	+100	2.8 ± 0.27#	+ 100
PLI daytime, %	SBP	-	21.7 ± 4.2	+100	2.0 ± 0.53#	+ 100
	DBP	-	27.6 ± 5.3	+100	2.15 ± 0.36#	+ 100
	MAP	-	35.5 ± 5.76	+100	1.9 ± 0.47#	+ 100
PLI nighttime, %	SBP	-	34.6 ± 6.4	+100	5.6 ± 1.4#	+ 100
	DBP	-	31.2 ± 5.8	+100	7.85 ± 0.36#	+ 100
	MAP	-	38.4 ± 6.2	+100	3.3 ± 0.58#	+ 100

Note: * – statistically significant differences compared to the control group ($p < 0,05$); # – Statistically significant differences compared to the comparison group ($p < 0,05$), Δ – percentage difference relative to the control group, %

Blood pressure amplitude (the difference between maximum and minimum values) serves as a marker of vascular wall elasticity and the autonomic nervous system's capacity to adequately regulate vascular tone throughout the 24-hour period. Physiological pregnancy is characterized by a specific circadian pattern with a relatively wide amplitude, reflecting the high adaptability of the cardiovascular system to changes in postural status and physical activity.

In the study of daily systolic blood pressure amplitude (ASBP), it was established that during both day-

time and nighttime periods, ASBP in the study group was higher than in the control group ($p < 0.05$) and did not statistically differ from the parameters of the preeclampsia group. The ASBP in the comparison group was 61.0 mmHg versus 47.2 mmHg in the control group. Nighttime diastolic blood pressure amplitude (ADBP) was significantly higher in high-risk pregnant women (33.7 mmHg) than in controls (19.3 mmHg) and those with preeclampsia (30.3 mmHg), all differences significant ($p < 0.05$). A similar trend was observed for the

mean arterial pressure amplitude (AMAP), which in the study group significantly exceeded control values, approaching the levels of the comparison group, and was 62.4% higher at night ($p < 0.05$).

Data analysis showed a significantly higher level of 24-hour, daytime, and nighttime SBP and DBP variability in pregnant women of the study group. Over the 24-hour period, SBP variability in the study group (11.9 mmHg) exceeded the values of healthy pregnant women by 38.4%, and DBP variability (9.9 mmHg) was 35.2% higher ($p < 0.05$). At night, SBP variability in the study group was 44% higher, and DBP variability was 46% higher compared to the control group ($p < 0.05$), indicating an impairment of baroreflex regulation.

The Pressure Load Index (PLI) is defined as the percentage of measurements exceeding threshold values during a specified period (daytime $>140/90$ mmHg, nighttime $>120/80$ mmHg). Higher PLI values indicate a longer duration during which the vessels remain in a vasospastic state. The study of the pressure load index

revealed that no episodes of SBP or DBP elevation above threshold values were recorded in the control group, resulting in a PLI of 0. However, in the study group, episodes of blood pressure elevation were observed even prior to the detection of stable hypertension by conventional methods. The highest frequency of these episodes occurred during the nighttime hours (nighttime PLI SBP was 5.6%, and PLI DBP was 7.85%).

ABPM results demonstrate a significantly higher morning blood pressure surge (MBPS) rate (Table 2) in pregnant women of the study group, which is essentially comparable to the values observed in women with manifested preeclampsia. The morning surge rate of systolic blood pressure (MSR SBP) in the study group was found to be 2.1 times higher than in the control group, while the MSR DBP was 2.6 times higher than in the control group and even 1.3 times higher than in the preeclampsia group ($p < 0.05$). Such morning pressure rise constitutes immense stress for the uterine and placental vessels, potentially provoking microthrombosis and placental abruption.

Table 2

Analysis of morning blood pressure surge rate in pregnant women ($M \pm m$)

ABPM parameters	Main group, n=77	Comparison group, n=50	Control group, n=34
MRSR SBP (mmHg/h)	$16.4 \pm 2.00^*$	$18.8 \pm 2.76^*$	7.7 ± 0.94
MRSR DBP (mmHg/h)	$14.5 \pm 2.40^*$	$11.0 \pm 1.47^*$	5.5 ± 0.84
DI SBP (%)	$9.8 \pm 0.64^{*,\#}$	$7.3 \pm 0.78^*$	12.7 ± 0.53
DI DBP (%)	$7.7 \pm 1.00^{*,\#}$	$3.6 \pm 1.00^*$	10.9 ± 0.63
DI MAP (%)	$8.6 \pm 0.85^{*,\#}$	$4.9 \pm 0.93^*$	11.7 ± 0.56

Note: * Significant difference ($p < 0.05$) compared to the control group; # Significant difference ($p < 0.05$) compared to the comparison group

Another equally important parameter is the dipping index (DI) or dipper status. In healthy individuals, nocturnal BP typically falls by $\geq 10\%$, a profile termed the “dipper” pattern. When this decline is blunted ($<10\%$), a non-dipping blood pressure pattern (NDBP) manifests, reflecting sympathetic overactivity, endothelial dysfunction, arterial stiffness, and low-grade vascular inflammation. Analysis of the dipping index indicates that pregnant women in Main and comparison groups are characterized by an insufficient nocturnal blood pressure drop. The DI SBP in the study group was 9.8%, which is 1.3 times lower than in the control group (12.7%). The DI DBP in the study group was 7.7%, which is 1.4 times lower than normal values ($p < 0.05$).

4. Discussion

For a long time, hypertensive disorders of pregnancy (HDP) were considered exclusively as transient conditions that resolve completely after delivery. However, contemporary research recognizes that a history of preeclampsia and gestational hypertension is a potent, sex-specific risk factor for cardiovascular diseases [10, 11]. Statistics indicate that women with gestational hypertensive disorders have a 4-fold higher risk of developing chronic hypertension, a 2-fold higher risk of ischemic heart disease and stroke, as well as an increased risk of developing end-stage renal disease and dementia later in life [1, 12].

Based on our findings, the analysis of ABPM parameters after the 20th week of pregnancy allows to

identify predictors for the development of gestational hypertension even during the pre-clinical stage.

Although mean 24-hour office blood pressure (BP) may remain below the established threshold of 140/90 mmHg, the presence of preclinical changes – such as increased nocturnal SBP and DBP amplitude, an increased morning surge rate, a reduced dipping index (non-dipping pattern), increased variability, and the presence of a pressure load index – clearly distinguishes these women from healthy pregnant individuals. Our findings are consistent with previous studies [13, 14] and indicate that ABPM (Ambulatory Blood Pressure Monitoring) allows to identify four key BP phenotypes: normotension, white-coat hypertension, masked hypertension, and sustained hypertension. The detection of masked and nocturnal hypertension is critical for the early prediction of preeclampsia.

It was established that nocturnal diastolic blood pressure and mean arterial pressure in the main group significantly exceed the values of healthy pregnant women, indicating that pathological changes are already occurring in the microcirculatory bed even when average office BP values remain within the generally accepted normal range [14]. An increase in nocturnal peripheral resistance indicates the inability of the vascular system to relax, which is a direct consequence of reduced nitric oxide production and the dominance of vasoconstrictor factors, such as endothelin-1.

Research [8] shows that the association between preeclampsia (PE) and systolic, diastolic, and nocturnal systolic hypertension manifests during the second half of

pregnancy and indicates abnormal placentation. The presence of a hidden "hypertensive load" is critical. The transformation of the BP profile from "dipper" to "non-dipper" in pregnant women is critically dangerous: it hinders physiological nocturnal hypervolemia and reduces uteroplacental blood flow during the period when the fetus requires maximum plastic supply for growth. It has been proven that the "reverse dipper" pattern, according to 24-hour ABPM data prior to delivery, is a significant independent predictor of preterm labor and the necessity of delivery via Cesarean section [13–16].

The morning blood pressure surge is a physiological phenomenon characterized by a sharp increase blood pressure during the first hours after awakening. This phenomenon is associated with the morning activation of the sympathoadrenal system, increased vascular resistance, and peak morning cortisol release. An excessive morning surge (pathological MBPS) is a well-recognized predictor of stroke, myocardial infarction, and left ventricular hypertrophy in the general population of cardiology patients. In the context of obstetrics, pathological morning surge is considered an independent risk factor for the development of eclampsia and placental abruption [14].

Large-scale cohort studies, supported by the National Institutes of Health and published in 2024–2025, have demonstrated that even isolated pathological blood pressure signs (a rapid increase of BP or the absence of physiological dipping) during the first half of pregnancy (up to 20 weeks) – which did not result in the development of classical preeclampsia – are significantly associated with an increased risk of developing hypertension over the subsequent 14 years postpartum [12]. Understanding chronobiological patterns allows for the optimization of preventive doses of acetylsalicylic acid (strictly before bedtime) and antihypertensive drugs (labetalol, nifedipine) by aligning the pharmacokinetics of medications with the individual patient's daily peak hemodynamic loads. This approach may not only help reduce the incidence of severe preeclampsia, preterm birth, and perinatal losses but also play a critical, life-saving role in the primary prevention of long-term cardiovascular catastrophes, preserving a woman's quality of life and health for decades to come.

Study limitations:

1. Small sample size.
2. There are no established reference ranges for ambulatory blood pressure monitoring parameters based on gestational age.

Prospects for further research. The results of the study can be used to develop a model for predicting preeclampsia and creation a personalized management algorithm to prevent pregnancy complications.

5. Conclusions

1. Ambulatory Blood Pressure Monitoring (ABPM) is a method that enables the detection of early signs of endothelial dysfunction, autonomic imbalance, and vascular stiffness, facilitating a transition to a per-

sonalized pregnancy management strategy.

2. For the high-risk group for preeclampsia, a specific pathological hemodynamic profile is characteristic as early as the beginning of the second trimester.

3. Pathological hemodynamic profile for high-risk group for preeclampsia includes ($p < 0.05$):

– increased amplitude of systolic and diastolic blood pressure ($61,0 \pm 3,4$ and $48,9 \pm 2,85$ respectively vs $47,2 \pm 1,9$ and $48,9 \pm 1,9$ in C group), particularly a nocturnal increase, which indicates a loss of vascular elasticity;

– an abnormally high rate of morning blood pressure surge (SBP $16,4 \pm 2,0$ mmHg/h vs $7,7 \pm 0,94$ mmHg/h and DBP $14,5 \pm 2,4$ mmHg/h vs $5,5 \pm 0,84$ mmHg/h in C group), reflecting hypersympathicotonia;

– the blunting of physiological nocturnal blood pressure dipping (the development of "non-dipper" or "reverse dipper" status) (DI SBP $9,8 \pm 0,64$ % vs $12,7 \pm 0,53$ % in C group; DI DBP $7,7 \pm 1,00$ % vs $10,9 \pm 0,63$ % in C group);

– an increase SBP variability (day/night) = $12.2 \pm 0.6 / 9.77 \pm 0.4$ and DBP variability (day/night) = $9.1 \pm 0.44 / 8.41 \pm 0.45$; the occurrence of elevated blood pressure episodes during the daytime (SBP up to $2.0 \pm 0.53\%$, DBP up to $2.15 \pm 0.36\%$) and during the nighttime period (SBP up to $5.6 \pm 1.4\%$, DBP up to $7.85 \pm 0.36\%$) which initiates shear stress on the endothelium and microvascular target organ damage.

Conflict of Interest

The authors declare that they have no conflicts of interest regarding this study, including financial, personal, authorship, or any other factors that could have influenced the research and the results presented in this article.

Funding

This research was conducted without external financial support.

Data availability

The data will be made available upon reasonable request.

Use of artificial intelligence

The authors confirm that no artificial intelligence (AI) technologies were used in the preparation of this work.

Author Contributions

Tetiana Loskutova: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Visualization, Project administration, Funding acquisition; **Albina Petulko:** Conceptualization, Methodology, Software, Formal analysis, Investigation, Resources, Data curation, Writing – original draft, Writing – review & editing, Visualization, Project administration, Funding acquisition.

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Received 13.04.2026

Received in revised form 06.05.2026

Accepted 25.05.2026

Published 29.05.2026

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