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DAILY BLOOD PRESSURE PARAMETERS AND STRUCTURAL AND FUNCTIONAL CHANGES IN THE MYOCARDIUM OF PATIENTS WITH HYPERTENSION AND FREQUENT VENTRICULAR EXTRASYSTOLE

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We examined 50 patients with stage II hypertension and frequent symptomatic ventricular extrasystoles and 32 patients with stage II hypertension without any cardiac arrhythmias. All patients underwent a comprehensive clinical and instrumental examination, daily blood pressure monitoring, and echocardiographic examination. It was determined that patients with ventricular extrasystoles had significantly ($p<0.04$) higher systolic blood pressure values during the day and at night. Systolic blood pressure variability at night showed the highest values in patients with frequent extrasystoles, which may indicate a complicated course of hypertension in this category of patients and increase the risk of target organ damage. In addition, frequent ventricular extrasystole in patients with stage II hypertension was associated with right heart overload, with an increase in the frequency of mitral valve fibrosis/calcification and tricuspid regurgitation, a decrease in ejection fraction and a deterioration in myocardial relaxation capacity.

Key words: essential arterial hypertension, ventricular extrasystole, echocardiography, daily blood pressure monitoring.

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ПОКАЗНИКИ ДОБОВОГО МОНІТОРУВАННЯ АРТЕРІАЛЬНОГО ТИСКУ ТА СТРУКТУРНО-ФУНКЦІОНАЛЬНІ ЗМІНИ МІОКАРДА У ХВОРИХ НА ГІПЕРТОНІЧНУ ХВОРОБУ І ЧАСТУ ШЛУНОЧКОВУ ЕКСТРАСИСТОЛІЮ

Було обстежено 50 пацієнтів з гіпертонічною хворобою II стадії та частою симптомною шлуночковою екстрасистолією та 32 пацієнти із гіпертонічною хворобою II стадії без будь-яких порушень серцевого ритму. Всім хворим проводилось комплексне клініко-інструментальне обстеження, добове моніторування артеріального тиску та ехокардіографічне дослідження. Визначено, що у пацієнтів з шлуночковими екстрасистолами реєструвались достовірно ($p<0.04$) вищі показники систолічного артеріального тиску в денний та нічний час. Показники варіабельності систолічного артеріального тиску в нічний час показали найбільші значення в пацієнтів з частою екстрасистолією, що може свідчити про ускладнений перебіг гіпертонічної хвороби у даної категорії хворих і підвищує ризик ураження органів-мішеней. Окрім того, часта шлуночкова екстрасистолія у хворих на гіпертонічну хворобу II стадії асоціювалась з перевантаженням правих відділів серця, зі збільшенням частоти реєстрації фіброзу/кальцинозу мітрального клапана та трикуспідальної регургітації, зменшенням фракції викиду і погіршенням релаксаційних можливостей міокарда.

Ключові слова: есенціальна артеріальна гіпертензія, шлуночкова екстрасистолія, ехокардіографія, добове моніторування артеріального тиску.

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Arterial hypertension (AH) is an urgent problem of our time, with an overall prevalence of about 30 %–45 % in adults, which is the same worldwide and leads to premature disability and death. With the progression of the disease, patients with hypertension have prerequisites for cardiac arrhythmias at all stages of the cardiovascular continuum, the most common of which are atrial fibrillation and extrasystole. Some of these patients have certain subjective signs (feeling of heart failure and/or palpitations), while arrhythmias in others are asymptomatic and therefore do not have appropriate monitoring and treatment [5, 11, 13].

Many studies have shown that the presence of ventricular extrasystole (VE) in patients with hypertension increases the risk of cardiac death, even in the absence of concomitant coronary heart disease [2]. In addition, systolic blood pressure (BP) was associated with the prevalence of VE, and frequent or complex VE was also associated with hypertension, and the prevalence of VE increased with increasing left ventricular (LV) myocardial mass [8]. To date, the question remains controversial: should we consider VE as a specific marker of malignant arrhythmias or as a marker of disease severity? Let's look at it from the point of view that LV hypertrophy in hypertension is the leading factor linking high blood pressure and ventricular arrhythmias. It is logical to consider it as a precursor to the development of VE [4, 6, 8].

Other studies have demonstrated the effect of nocturnal BP elevation (non-dipper) on the frequency of registration and severity of ventricular arrhythmias in patients who did not receive treatment for this condition [9]. A close pathophysiologic relationship between the dispersion of the Q-T interval and left ventricular myocardial mass in hypertension has also been shown. The relationship between the duration of the Q-T interval and the magnitude of its dispersion with circadian changes in blood pressure is now known. Some studies have demonstrated the relationship between the index of LV myocardial mass, the value of the Q-T interval dispersion, and the circadian profile of blood pressure. The most significant changes in these parameters were found in patients with a non-dipper circadian profile. The relationship between the duration of the corrected Q-T interval and the severity of structural and geometric LV remodeling has also been proven [4, 10]. This may explain the high risk of developing severe ventricular arrhythmias in patients with hypertension and LV hypertrophy.

Late ventricular potentials are another noninvasive method that can detect areas of fibrosis in the myocardium even in the absence of signs of coronary artery disease. It is known that areas of fibrosis surrounded by normal healthy tissue play a leading role in the development of the re-entry mechanism. In hypertension, due to microangiopathy and LV hypertrophy, subendocardial fibrous formations occur, which contribute to the circulation of the excitation wave and the development of SE even without concomitant coronary heart disease [1, 2, 3]**.** The autonomic nervous system plays an important role in the regulation of blood pressure and the development of hypertension. The Framingham study showed that individuals with high blood pressure had a decrease in heart rate variability. Also, low heart rate variability in people with normal blood pressure indicated a high risk of hypertension, which confirms the hypothesis of autonomic dysregulation. Some scientists have drawn attention to the pathogenetic relationship between the severity of hypertension and low heart rate variability. These disorders were circadian in nature and were more pronounced in patients with insufficient nocturnal BP lowering (non-dippers) [12]**.**

To date, a certain relationship between various arrhythmias and hypertension has been proven. In the presence of high blood pressure and LV hypertrophy, the risk of VE, paroxysmal arrhythmias, and sudden arrhythmic death increases significantly. Humoral and structural-functional factors also play an important role in the development and further progression of arrhythmias. There are a large number of noninvasive techniques that have different diagnostic significance and availability for studying arrhythmia predictors. However, this issue is still open today and continues to be studied.

The purpose of the study was to evaluate the parameters of daily blood pressure monitoring and structural and functional changes in the myocardium of patients with hypertension and frequent ventricular extrasystoles.

Materials and methods. The study included 50 patients with stage II hypertension (HTN) and frequent symptomatic ventricular extrasystole aged 27 to 75 (mean 58.2±0.9) years, who formed the main clinical group of the study. In addition, we examined 32 patients with stage II HTN without any cardiac arrhythmias (excluded by Holter electrocardiogram monitoring (HM ECG)) aged 32 to 72 (mean 55.9±1.7) years, who formed the comparison group in relation to the main clinical group. All patients underwent a comprehensive clinical, instrumental, and laboratory examination, which included: 1) general clinical and anthropometric examination, blood pressure measurement; 2) ECG in 12 standard leads; 3) daily blood pressure monitoring (DBPM); 3) HM ECG; 4) echocardiography (EchoCG).

BP monitoring was performed using the hardware and software complex of the DiaCard system (“Solvaig” JSC, Ukraine) according to a standard protocol. The following parameters were evaluated according to the data of DBPM: mean daily, daytime, and nighttime systolic (SBP, SBPd, and SBPn, respectively), diastolic (DBP, DBPd and DBPn, respectively) and pulse BP (PBP, PBPd and PBPn, respectively) in mm Hg; early rise of SBP and DBP (ER SBP and ER DBP, respectively) in mm Hg./h; hypertension index of time of SBP and DBP per day (SBP_HI and DBP_HI, respectively) in %; diurnary index of SBP and DBP (DI SBP and DI DBP, respectively) in %; variability of day and night SBP (Var SBPd and Var SBPn, respectively) and DBP (Var DBPd and Var DBPn, respectively) in mm Hg. The analysis of the nature of the daily profile of SBP and DBP was performed using standard criteria for the value of the DI for SBP and DBP separately: “dipper” – DI from 10 to 20 %, “non-dipper” – DI within 0 and 10 %, “night- peaker” – DI < 0 % and “over-dipper” – DI > 20 %.

Echocardiography studies were performed in one-dimensional and two- dimensional modes with color, pulse, and continuous wave Doppler using “My Lab 25” equipment (Italy) according to the recommendations for cardiac ultrasound. The following echocardiographic parameters were determined: 1) aortic diameter (dAo) in mm; 2) anteroposterior size of the left atrium (LA) in mm; 3) end-systolic and diastolic size of the left ventricle (ESD and EDD) in mm; 4) end-systolic and diastolic volume of the left ventricle (ESV and EDV) in ml; 5) left ventricular posterior wall thickness in diastole (LVPWT) in mm; 6)

interventricular septal thickness in diastole (IVST) in mm; 7) anteroposterior size of the right ventricle (RV) in mm; 8) relative wall thickness of the left ventricular myocardium (RWT) according to the formula RWM = (LVPWT + IVST) / EDD; 9) left ventricular myocardial mass (LVM) in g according to the Pen Convention formula 10) left ventricular myocardial mass index (LVMI) in g/m^2 as LVMI = LVM / S, where S is the patient's body area calculated by Dubois nomogram; 11) left ventricular ejection fraction (LVEF) in %; 12) ratio of early to late left ventricular filling velocity (Ve/Va).

According to the recommendations, the statistical processing of the study results was carried out using the methods of variation statistics using the StatSoft "Statistic" v. 12.0 program. The results were presented as median and interquartile range with the indication of the 25th and 75th percentiles or as percentages (%), which reflected the frequency of the trait in the sample. A comparison of relative values (%) was conducted using the criterion χ^2. Quantitative values of independent samples were compared using the Mann-Whitney and Kruskal-Wallis criteria.

Results of the study and their discussion. The analysis of the results of daily BP monitoring in patients with hypertension and ventricular extrasystole showed that patients with hypertension and frequent VE had significantly higher values of mean daily systolic blood pressure (SBP) and mean night systolic blood pressure (SBPn) compared with patients without extrasystoles (155 mm Hg vs. 147 mm Hg, p=0.01; 147 mm Hg vs. 140 mm Hg, p=0.04) (Table 1).

Table 1

Indices of daily blood pressure monitoring and circadian blood pressure profile in patients with hypertension and ventricular extrasystole

ABPM parameters	Patients without extrasystoles	Patients with VE	P1-2
	Group 1 (n=32)	Group 2 (n=50)	
SBP, mm Hg	148 (137; 158)	150 (139; 159)	ns
DBP, mmHg	86 (78; 92)	87 (78; 97)	ns
SBPd, mmHg	147 (134; 161)	155 (142; 168)	0.01
DBPd, mmHg	88 (80; 97)	89 (80; 100)	ns
SBPn, mm Hg	140 (127; 151)	147 (137; 154)	0.04
DBPn, mmHg	78 (71; 86)	77 (70; 89)	ns
PBP, mm Hg	59 (55; 68)	61 (55; 70)	ns
PBPd, mm Hg	56 (54; 70)	60 (55; 71)	ns
PBPn, mm Hg	60 (54; 66)	60 (54; 66)	ns
DI SBP, %.	7 (3; 9)	10 (6; 14)	0.01
DI DBP, %.	10 (4; 15)	12 (9; 16)	ns
SBP HI, %.	80 (57; 92)	90 (64; 97)	0.04
DBP_HI, %.	82 (56; 96)	81 (56; 96)	ns
Var SBP, mmHg	18 (15; 22)	18 (16; 25)	ns
Var SBPn, mmHg	11 (7; 15)	16 (12; 21)	0.04
Var DBPd, mm Hg	12 (9; 18)	14 (9; 19)	ns
Var DBPn, mmHg	9 (7; 14)	12 (8; 17)	ns
Dipper by SBP	12 (37.5 %)	15 (30.0 %)	ns
Non-dipper by SBP	15 (46.9 %)	25 (50.0 %)	ns
Night-peaker by SBP	4 (12.5 %)	8 (16.0 %)	ns
Over-dipper by SBP	1 (3. 1 %)	2 (4.0 %)	ns
Dipper by DBP	14 (43.8 %)	9 (18.0 %)	0.01
Non-dipper by DBP	11 (34.4 %)	38 (76.0 %)	0.0002
Night-peaker by DBP	3 (9.4 %)	0 (0)	0.03
Over-dipper by DBP	4 (12.5 %)	3 (6.0 %)	ns

Notes**:** 1 **–** ns – not significant, p>0.05; 2– VE – ventricular extrasystole; 3 – Intergroup differences significance of precents calculated using χ^2, of absolute values calculated by Kruskal-Wallis ANOVA test & Median test.

The mean value of the daily index (DI) for SBP was 7 % in the group of patients without arrhythmias, indicating insufficient nighttime BP reduction in these patients. In comparison, in patients with HTN, this percentage was significantly higher (10 %, p=0.01). The index of the time when SBP exceeded the permissible norm (IR SBP) was highest in the group of patients with HF, which revealed significant differences compared with patients without extrasystoles (90 % vs. 80 %, p=0.04). The variability of systolic blood pressure at night (SBPn) in patients with HTN was 16 mm Hg, significantly different from patients without arrhythmias, where SBPn was 11 mm Hg (p=0.04). Such variability in

SBP in patients with extrasystole and HF is critical and may indicate a complicated course of HF, as well as target organ damage.

The analysis of the circadian profile of SBP in patients with hypertension and VE showed that approximately half of the subjects in each group (50.0 % of patients with VE and 46.9 % of patients without arrhythmias) had a non-dipper daily profile, i.e., insufficient reduction of BP at night.

At the same time, the daily over-dipper profile, indicating an excessive decrease in blood pressure at night, was recorded in the smallest number of patients (4.0 % of patients with HF and 3.1 % of patients without extrasystoles). The analysis of circadian regulation of SBP in the examined groups of patients showed the smallest number of patients with a daily dipper profile and the largest number with a daily non-dipper profile in patients with HF, which was statistically significant compared with patients in the comparison group (18.0 % vs. 43.8 %, p=0.01; 76.0 % vs. 34.4 %, p=0.0002). Only three (9.4 %) patients with night hypertension (night-peaker) were detected in the group of patients without arrhythmias, which was significantly more than in the groups with VE, where we did not reveal such a daily profile in any case (p=0.03).

The results of the assessment of the structural and functional state of the heart according to the Echo-CG (Table 2) showed that in patients with HTN and frequent ventricular extrasystole, compared with patients without heart rhythm disorders, a significant increase in the size of the right ventricle (RV index, p=0.03), the ratio of the right ventricle to the end-diastolic size (RV/EDD index, p=0.01) was observed.

Table 2

Structural and functional state of the myocardium in patients with hypertension and ventricular extrasystole

Indices of echocardiography	Patients with HTN without extrasystoles	Patients with HTN and frequent VE	P1-2
	Group 1 (n=32)	Group 2 (n=50)	
ESD, mm	33.8 (32.5; 36.9)	33. 1 (30.9; 36.0)	ns
EDD, mm	51.0 (47.4; 54.0)	50.0 (47.2; 53.5)	ns
LA, mm	40.4 (38.0; 42.0)	40.5 (38.0; 42.0)	ns
LA/EDD	0.79 (0.76; 0.82)	0.79 (0.74; 0.85)	ns
RV, mm	24.9 (24.0; 28.4)	27.9 (24.8; 30.9)	0.03
RV/EDD	0.49 (0.46; 0.53)	0.55 (0.48; 0.61)	0.01
dAo, mm	32.8 (31.6; 34.3)	33.2 (28.5; 37.0)	ns
PWDT, mm	12.0 (11.3; 12.3)	12.0 (10.0; 13.0)	ns
IVSDT, mm	11.5 (10. 1; 12. 1)	12.0 (9.8; 14.4)	ns
RWT	0.46 (0.40; 0.50)	0.48 (0.42; 0.53)	ns
LVMI, g/m^2	146.0(135.3; 159.0)	152.6 (109. 1; 198.4)	ns
EF, %	60.3 (57.5; 63.5)	54.3 (50.2; 61. 1)	0.01
Ve/Va	1.45 (1.04; 1.59)	1. 18 (0.75; 1.34)	0.001
AC fibrosis/calcification	8 (25.0 %)	8 (16.0 %)	ns
Fibrosis/calcification of the MC	3 (9.4 %)	22 (44.0 %)	0.0009
Mitral regurgitation	27 (84.4 %)	39 (78.0 %)	ns
Aortic regurgitation	2 (6.3 %)	4 (8.0 %)	ns
Tricuspid regurgitation	13 (40.6 %)	33 (66.0 %)	0.02

Notes: 1. ns – not significant, p>0.05; 2. VE – ventricular extrasystole; 3. Intergroup differences significance calculated by the Kruskal-Wallis ANOVA test & Median test and percentages by the χ^2 criterion.

In addition, we evaluated the structural and functional state of the heart valves, although only patients with mild regurgitation and/or valve fibrosis/calcification were allowed to participate in our study. The incidence of mitral valve fibrosis/calcification was higher in patients with frequent extrasystole (44.0 %), which was significantly (p=0.0009) different from the comparison group (9.4 %). At the same time, the frequency of tricuspid regurgitation was highest in patients with VE. It was significantly different from the comparison group (66.0 % vs. 40.6 %, p=0.02).

Referring that the study included only patients with CHF stage I and NYHA class I-II and no echocardiographic signs of systolic LV myocardial dysfunction (EF > 40 %), only the diastolic variant of LV myocardial dysfunction was registered among the patients. At the same time, according to our data, the average value of ejection fraction (EF) in the examined groups of patients was lower in patients with frequent extrasystoles (54.3 % in patients with VE), which was significantly (p=0.01) different from patients without arrhythmias, in whom EF was 60.3 %.

The presence of frequent extrasystoles is associated with greater variability of SBP at night, which may indicate the possibility of a complicated course of HTN and an increased risk of target organ damage in these patients. There is literature data that levels of SBPd and SBPn are higher in patients with cardiac

arrhythmias. In the ARIC study (2002), frequent or complex VE was associated with elevated SBP, but the frequency and severity of VE increased with increasing LV myocardial mass [5]. The role of circadian changes in BP and the non-dipper profile on ventricular arrhythmogenesis has been shown now [7, 13]. It has been proven that a constant prolonged increase in blood pressure at night (non-dipper pattern) is an independent predictor of frequent and severe ventricular arrhythmias. The development of myocardial electrical instability against the background of changes in the circadian BP profile is explained by the direct relationship of BP changes with the duration of the Q-T interval and the magnitude of its dispersion, as well as with the severity of structural changes in the atria and ventricles, which leads to the electrical instability of the atrial and ventricular myocardium and contribute to the development of VE in these patients [6, 8]. Perhaps this is the explanation of the role of arrhythmias in HTN complications.

It should be noted that the overload of the right and left hearts in patients with hypertension is the pathomorphologic basis for the development of myocardial electrical instability and cardiac arrhythmias. Increased myocardial stress in the setting of hypertension along with excessive afterload stimulates myocardial hypertrophy, its structural remodeling with a disproportionate increase in fibrous tissue, a decrease in coronary blood flow, and the development of myocardial diastolic dysfunction [2, 4, 7]. In addition to increased blood pressure, other factors, such as angiotensin, demographic determinants, and genetic polymorphism, play an important role in the development and progression of hypertrophy, as evidenced by the weak correlation between blood pressure and LV myocardial mass. Myocardial hypertrophy leads to impaired myocardial kinetics of Ca^{2+}, Mg^{2+}, Na^{+}, and K^{+} ions, which contributes to the prolongation of the action potential and is a trigger in the mechanism of re-entry, early postdepolarization, and trigger activity. Along with LV hypertrophy, endothelial vascular dysfunction plays a role in the occurrence of arrhythmias [1, 2, 9, 12]. The presence of both of these factors significantly increases the risk of future cardiac events, including arrhythmias.

Conclusions

1. The presence of frequent ventricular extrasystoles in patients with stage II HF is accompanied by higher SBP during the day and night ($p<0.04$) and greater variability of SBP at night ($p=0.04$).

2. The presence of frequent VE in patients with stage II HTN was associated with right heart overload, with an increase in the frequency of fibrosis/calcification of the mitral valve ($p=0.0001$), and tricuspid regurgitation ($p=0.03$), a decrease in EF ($p=0.02$) and a deterioration in myocardial relaxation ($p=0.03$).

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