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SYSTOLIC, DIASTOLIC, AND PULSE BLOOD PRESSURE DURING PREMATURE VENTRICULAR CONTRACTIONS: RELATIONSHIP WITH CHARACTERISTICS OF ECTOPIC BEATS

Yu.V.Shubik[1], A.B.Korneev[1], M.A.Baturova[2], A.N.Morozov[3], V.V.Pivovarov[4], G.K.Zaytsev[4], V.M. Tikhonenko[1], A.Yu.Kormilytsin[4], M.V.Gordeeva[1], M.V.Berman[1]

[1]St. Petersburg State University, Russia, St. Petersburg, 7-9 Universitetskaya emb.; [2]OOO Northwest Center for Diagnosis and Treatment of Arrhythmias, Russia, St. Petersburg, 40-4 Lunacharsky ave.; [3]Pavlov First St. Petersburg State Medical University, Russia, St. Petersburg, 6-8 Lev Tolstoy str.; [4]NAO Incart, Russia, St. Petersburg. Russia, 12 Fermskoe road.

The aim is to assess the relationship between systolic, diastolic, and pulse blood pressure (SBP, DBP, PBP) during ventricular extrasystoles (VE) and the individual characteristics of ectopic beats.

Methods. The primary method of investigation was BP measurement for each heartbeat. Inclusion criteria were the presence of ≥10000 monomorphic VE per day. A total of 53 patients were included, either without structural heart changes or with minimal structural alterations. The mean of systolic, diastolic, and pulse BP (SBP, DBP, and PBP) during VE (SBP VE, DBP VE, PBP VE) and during post-extrasystolic sinus contraction (post VE SBP, post VE DBP, post VE PBP) were calculated for each patient as fractions of 1.0.

Results. The QRS complex width in VE originating from the right ventricular outflow tract is greater than from the left ventricular outflow tract; fragmentation of the QRS complex is more commonly observed in these VE. Significant correlations were observed between SBP VE and mean coupling interval (CI), PBP VE and CI, and SBP VE and PBP VE, though not between DBP VE and CI. DBP VE was significantly associated with VE count and daily VE percentage, while PBP VE was associated with left ventricular ejection fraction. It has been shown that post-VE SBP and post VE DBP are lower, while post VE PBP is higher compared to the corresponding parameters of sinus beats preceding the VE. Significant relationships were found between post VE SBP and post VE PBP, the duration of the post-extrasystolic pause, and the presence of paired VE; between post VE DBP and post VE PBP, DBP VE, CI VE, the presence of non-sustained ventricular tachycardia, and daily VE percentage; between post VE PBP and DBP VE, the presence of non-sustained ventricular tachycardia, daily VE percentage, and post-extrasystolic pause duration. Post VE PBP was equally determined by values of post VE SBP and DBP.

Conclusion. With the shortening of the VE coupling interval, its SBP decreases, while DBP increases slightly, which may determine its hemodynamic significance. In post-extrasystolic sinus beats, both SBP and DBP decrease.

Key words: ventricular extrasystole; premature ventricular contractions; arrhythmia-associated cardiomyopathy; “beat-to-beat” method; measurement of systolic, diastolic, pulse arterial pressure at each heartbeat; postextrasystolic potentiation; hemodynamic effectiveness; Holter electrocardiogram monitoring

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Corresponding author: Yuri Shubik, E-mail: yshubik@mail.ru

Yu.V.Shubik - ORCID ID 0000-0002-8736-1575, A.B.Korneev - ORCID ID 0000-0002-6796-8302, M.A.Baturova - ORCID ID 0000-0003-3811-6810, A.N.Morozov - ORCID ID 0000-0003-2598-7000, V.V.Pivovarov - ORCID ID 0000-0002-8426-215X, G.K.Zaytsev - ORCID ID 0000-0002-8822-9197, V.M.Tikhonenko - ORCID ID 0000-0002-6262-1326, A.Yu.Kormilytsin - ORCID ID 0000-0002-6373-4285, M.V.Gordeeva - ORCID ID 0000-0002-9437-8535, M.V.Berman - ORCID ID 0000-0001-9554-5553.

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When evaluating the clinical significance of any cardiac rhythm disturbances, three primary aspects are typically considered: symptomatology and its impact on the patient’s quality of life; increased risk of sudden cardiac death (SCD); and the development and progression of chronic heart failure (CHF). These aspects are relevant for both supraventricular and ventricular arrhythmias.

For example, the potential for psychogenic reactions in patients with supraventricular tachycardias (SVTs), various forms of atrial fibrillation (AF) and flutter, and ventricular extrasystole (VE) and tachycardia is well-established [1, 2]. All these arrhythmias are associated with increased mortality, including SCD [9-15]. VEs, in particular, are recognized for their potential to trigger life-threatening

ventricular arrhythmias, such as monomorphic and polymorphic ventricular tachycardias, ventricular flutter, and fibrillation, which may result in SCD [14-16]. The risk is especially high in patients with underlying structural or genetic heart diseases, including channelopathies and cardiomyopathies [17-29].

Another significant clinical consequence of rhythm disturbances is their potential to cause arrhythmia-associated cardiomyopathies (AACs). Contemporary guidelines on managing AF, SVTs, ventricular arrhythmias, and SCD prevention have dedicated sections discussing AACs [17-19, 30, 31]. AACs are linked to tachyarrhythmias due to a high heart rate and, in the case of VEs, to the frequency of ventricular ectopy. The greater the VE burden detected via 24-hour Holter electrocardiography (ECG), the higher the likelihood of AAC development.

The 2020 Russian Ministry of Health guidelines recommend addressing VEs either pharmacologically or via catheter ablation if they are associated with clinical symptoms or lead to ventricular dilation and reduced left ventricular (LV) myocardial contractility, provided they exceed 15% of total daily heartbeats [18]. The presence or absence of structural heart disease does not affect this recommendation. Meanwhile, the 2022 ESC guidelines set the "critical" VE burden at 10% [17]. For asymptomatic patients with "idiopathic" VEs exceeding 20% of daily beats, catheter ablation may be considered to prevent AAC. Similar criteria are being debated for inclusion in the forthcoming 2025 Russian Ministry of Health recommendations. Thus, VE burden is often viewed as the primary determinant of AAC risk.

However, VE burden alone may not fully capture the hemodynamic significance of VEs. Even a burden exceeding 20% does not always result in AAC over decades [32]. Factors such as QRS width and fragmentation (markers of ventricular dyssynchrony), morphology, coupling interval, polymorphism, and others may play a role [19, 33-35], potentially influenced by the arrhythmogenic substrate's location.

The hemodynamic insufficiency of premature beats can be assessed using parameters such as reduced systolic blood pressure (SBP), elevated diastolic blood pressure (DBP), and changes in pulse blood pressure (PBP) as an integral metric. The clinical significance of postextrasystolic potentiation (PESP)-the hemodynamic characteristics of sinus beats following compensatory pauses after VEs-is also discussed in the literature [36-39]. Studies suggest a prognostically adverse role for PESP in CHF patients, those with prior myocardial infarction, and individuals with AAC caused by frequent VEs. Investigating SBP, DBP, and PBP of postextrasystolic contractions may provide insights into this phenomenon. These metrics are likely individualized, and their accurate assessment has been methodologically challenging.

Currently, blood pressure for individual heartbeats can be measured invasively (using an arterial catheter connected to a transducer) or noninvasively through methods such as photoplethysmography, continuous tonometric monitoring (Volume Clamp Method), impedance cardiography, or applanation tonometry. Beat-to-beat BP measurement is particularly relevant for evaluating patients with syncope [40, 41]. In this study, a method based on the "unloaded artery" principle, capable of capturing the complete BP waveform, was utilized. The degree of BP alteration during VEs largely determines their hemodynamic significance.

This study aimed to assess the relationship between systolic, diastolic, and pulse blood pressure during ventricular extrasystoles and the individual characteristics of ectopic beats

METHODS

The clinical study adhered to the standards of Good Clinical Practice (GCP) and the principles of the Declaration of Helsinki. Approval was granted by the local ethics committee at the "Northwest Center for Diagnosis and Treatment of Arrhythmias" (St. Petersburg, Russia). Written informed consent was obtained from all participants.

The primary inclusion criterion was the presence of at least 10,000 monomorphic VEs per day, as determined by Holter ECG monitoring. Exclusion criteria included the presence of any cardiomyopathy (including arrhythmia-induced cardiomyopathy) or channelopathy, clinically significant chronic heart failure, reduced left ventricular ejection fraction (LVEF), and acute or exacerbated chronic conditions. Patients with polymorphic VEs were also excluded if VEs of other morphologies accounted for more than 1% of their total daily count.

A total of 53 patients (21 men), aged between 16 and 87 years (mean age 56.5 ± 2.4 years), were included in the study. In 21 cases, idiopathic VE was the sole manifestation of the condition, constituting Group 1. Hypertension was diagnosed in 20 patients, and an additional 9 patients presented with a combination of hypertension and ischemic heart disease (Group 2). Myocarditic cardiosclerosis, as confirmed by gadolinium-enhanced magnetic resonance imaging (MRI), was diagnosed in 3 patients (Group 3). Structural, functional, and electrocardiographic characteristics of the patients are summarised in Table 1.

Additionally, paired VEs were detected in 32 cases, nonsustained ventricular tachycardia (VT) in 14 cases, and QRS complex fragmentation in VEs in 19 cases. Based on the results of 12-lead electrocardiograms (ECG) [44-47], the approximate localization of the arrhythmogenic substrate was assessed as follows: in 23 patients, the right

Table 1.

Some structural-functional and electrocardiographic characteristics

Indicator	M±m
LV EF, %	61.7±1.35
LV thickness, mm	9.93±0.33
QRS complex width during VE, ms	140.38±2.19
Average CI during VE, ms	539.3±13.59
Post-extrasystolic pause, ms	1053.86±28.92
Average number of VEs per day	18611.0±1743.8
Average % VEs*	17.8±1.7

Note: hereinafter, LVEF - left ventricle ejection fraction; CI - coupling interval; VE - ventricular extrasystole; * - of the total number of heartbeats per day.

ventricular outflow tract (RVOT); in 12 patients, the left ventricular outflow tract (LVOT); and in 18 patients, other locations.

In addition to the aforementioned data, the following key parameters were assessed in patients:

- Mean SBP, DBP, and PBP of the sinus beat preceding the VE;
- Mean SBP, DBP, and PBP during the VE;
- Mean SBP, DBP, and PBP of the sinus beat following the VE (designated as post-VE SBP, post-VE DBP, and post-VE PBP, respectively).

The average SBP, DBP, and PBP during VE (SBP VE, DBP VE, and PBP VE) and the average post-VE SBP, post-VE DBP, and post-VE PBP for each patient were determined as fractions of 1.0. The value of 1.0 corresponded to the average SBP, DBP, and PBP of the sinus beats preceding the VE.

The primary method of investigation was beat-to-beat blood pressure measurement using the "Cardiotechnika-SAKR" device (NAO Incart, St. Petersburg, Russia, patents RU 2694737 C1 by V.V. Pivovarov et al. and RU 2698447 C1 by V.V. Pivovarov et al.). This method has been previously described in our publications on beat-to-beat blood pressure measurement during persistent atrial fibrillation and VE [42, 43].

The method involves continuous analysis of the finger vessel volume using a photoplethysmographic signal and a tracking electropneumatic system, which generates pressure to counteract changes in the arterial diameter of the finger under a cuff. The distal blood pressure measurement is calibrated against brachial blood pressure by adjusting the continuous pressure signal to align with the moments of Korotkoff sounds, simultaneously recorded during conventional blood pressure measurement on the contralateral arm.

This technique enables measurement of SBP and DBP and the calculation of PBP for each individual heartbeat, whether sinus or ectopic. The duration of each measurement session, determining SBP, DBP, and PBP for every beat, was 15 minutes.

TTable 2.

Indicators characterizing the hemodynamic significance of ventricular extrasystole

Indicator	Value
SBP VE across the entire group of patients	0.73±0.09. p<0.0001
DBP VE across the entire group of patients	1.11±0.10. p<0.0001
PP VE across the entire group of patients	0.12±0.19. p<0.0001
Correlation between SBP VE and CI	r=0.65. p<0.0001
Correlation between DBP VE and CI	r=0.04. p=0.76
Correlation between PP VE and CI	r=0.81. p<0.0001
Correlation between SBP VE and PP VE	r=0.75. p<0.0001
Correlation between DBP VE and % VE	r=0.51. p<0.008
Correlation between DBP VE and nVE	r=0.45. p<0.02
Correlation between PP VE and LVEF	r=-0.43. p<0.02

Note: hereinafter, DBP - Diastolic Blood Pressure; VE - Premature Ventricular Contraction; PP - Pulse Pressure; SBP - Systolic Blood Pressure; nVE - Number of VEs per day

Statistical analysis

Statistical analysis was performed using the SPSS software package. Student's t-test was applied to compare the means of two groups. One-way analysis of variance (ANOVA) with Tukey's post hoc test was used for comparisons involving more than two groups. The Chi-square test (χ^2 test) was employed for the analysis of categorical data. Pearson›s correlation coefficient was used to assess the linear relationship between two quantitative variables. Multiple linear regression was applied to evaluate the influence of several independent variables on a dependent variable. A p-value of <0.05 was considered the threshold for statistical significance.

RESULTS

The first stage of data analysis involved characterising patient groups and identifying the features of VE. As expected, a statistically significant correlation was observed between patient age and the presence or absence of heart disease. The mean age of patients with heart disease was 65.00±2.10 years, while for those without heart disease it was 42.85±3.82 years (p<0.0001).

Age was also correlated with the localisation of the arrhythmogenic substrate. The mean age of patients with VE originating from the right ventricular outflow tract (RVOT) was 46.61±3.19 years, compared to 59.42±4.73 years for VE from the left ventricular outflow tract (LVOT), and 67.61±3.45 years for other localisations. Differences between RVOT and LVOT were not statistically significant (p=0.059), whereas differences between RVOT and other localisations were highly significant (p=0.0002). Among the 23 patients with RVOT-originating VE, the arrhythmia was idiopathic in 17 cases. In contrast, only 2 of 12 patients with LVOT-originating VE and 1 of 18 with other localisations had idiopathic VE. The differences were statistically significant between RVOT and LVOT (p=0.003), and even more so between RVOT and other localisations (p=0.00002).

The left ventricular (LV) wall thickness in patients with idiopathic VE was 8.5±1.17 mm, compared to 11.42±1.02 mm in patients with hypertension (HTN) or a combination of HTN and coronary artery disease (CAD), and 10.83±1.22 mm in patients with post-myocarditis cardiosclerosis. Statistically significant differences were found between the first and second groups (p<0.05) and between the first and third groups (p<0.05), but not between the second and third groups (p>0.05). Similar differences were observed in LV wall thickness based on the localisation of the arrhythmogenic substrate: RVOT 8.50±0.34 mm, LVOT 11.42±0.42 mm, and other localisations 10.83±0.41 mm. Significant differences were found between RVOT and LVOT (p=0.0001) and between RVOT and other localisations (p=0.0004).

The LVEF did not differ significantly between groups with and without organic heart disease. No significant differences were found in the number of VE per day on Holter ECG monitoring or the percentage of VEs over 24 hours among

patients with different arrhythmogenic substrate localisations. The mean QRS duration of VEs was significantly longer for VEs originating from the right ventricular outflow tract (RVOT) (147.8±3.3 ms) compared to those from the left ventricular outflow tract (LVOT) (131.7±3.7 ms; p=0.049). These findings align with data on QRS fragmentation: fragmentation was not observed in any of the 12 patients with LVOT-originating VEs, but was present in 13 of 23 patients with RVOT-originating VEs and in 6 of 18 patients with other localisations. The differences in QRS fragmentation between RVOT and LVOT were also statistically significant (p=0.0008).

The mean coupling interval (CI) of VEs originating from the RVOT was 518.7±11.9 ms, compared to 525.0±29.4 ms for LVOT-originating VEs and 575.2±30.7 ms for other localisations. No statistically significant differences were observed between these groups. When combining RVOT and LVOT localisations and comparing them to all other localisations, the mean CI values (520.7±12.5 ms vs. 575.2±30.7 ms) also did not differ significantly. Excluding patients with interpolated VEs, allorhythmias, or obvious parasystole from the analysis did not significantly affect these findings.

The second stage of analysis focused on assessing SBP VE, DBP VE, and PBP VE as parameters largely defining the hemodynamic significance of VEs. These values were calculated as fractions of 1.0, where 1.0 represented the SBP, DBP, and PBP of sinus beats preceding the VEs. The results are presented in Table 2. SBP VE, DBP VE, and PBP VE did not significantly differ across patient groups with varying arrhythmogenic substrate localisations, nor were they influenced by QRS duration or fragmentation. Similarly, no differences in these parameters were observed between patients with idiopathic VEs and those with hypertension (HTN), combined HTN and coronary artery disease (CAD), or myocarditis-related cardiosclerosis.

However, SBP VE was closely associated with mean CI: as mean CI decreased, SBP VE also decreased. This relationship is illustrated graphically in Figure 1.

The correlation between the mean coupling interval (mean CI) and diastolic blood pressure during VEs (DBP VE) was not statistically significant. Pulse blood pressure during VEs (PBP VE) was more strongly associated with mean CI than systolic blood pressure during VEs (SBP VE): as mean CI decreased, PBP VE also decreased. Thus, PBP VE was determined by SBP VE but not by DBP VE. A close correlation was observed between SBP VE and PBP VE. Figure 2 graphically illustrates the relationships between SBP VE, DBP VE, and PBP VE. It is evident that the reduction in PBP VE was minimally influenced by an increase in DBP VE and was primarily determined by a decrease in SBP VE.

Apart from these relationships, DBP VE showed a statistically significant correlation (in decreasing order of strength) with the percentage of VEs over 24 hours and the total number of VEs per day on Holter ECG monitoring. The relationships of DBP VE with DBP post-VE (DBPpost VE), PBP post-VE (PBPpost VE), and the duration of the postextrasystolic pause are discussed in a subsequent section.

For PBP VE, a statistically significant correlation was found with LVEF. No correlations were found with other parameters. Excluding patients with interpolated VEs or apparent parasystole did not significantly alter these findings. However, the correlation between SBP VE and LVEF became statistically significant (r = -0.47, p=0.02).

The third stage of analysis focused on characterising the phenomenon of PESP. No significant differences were observed in the duration of the postextrasystolic pause between groups with and without organic heart disease or across different arrhythmogenic substrate localisations. The pause duration correlated with mean CI (r = 0.39, p=0.004) and DBP VE (r = 0.24, p=0.03).

Postextrasystolic SBP (SBPpost VE), DBPpost VE, and PBPpost VE were evaluated as indicators reflecting PESP characteristics. These values were calculated as fractions of 1.0, where 1.0 represented SBP, DBP, and PBP of sinus beats preceding the VEs. Unlike most published studies, which compared postextrasystolic SBP with SBP from 8–10 subsequent sinus beats, this study evaluated PESP based on single postextrasystolic beats due to the high VE burden (≥10,000 VEs/day, up to 45,000/day) per the inclusion criteria.

The results are presented in Table 3. Differences in SBPpost VE, DBPpost VE, and PBPpost VE compared to their respective pre-VE sinus values were relatively minor but highly statistically significant, with small standard deviations. These values were independent of the arrhythmogenic substrate localisation, QRS width, or QRS fragmentation. Similarly, no differences were found between patients with idiopathic VEs, those with hypertension (including combined hypertension and CAD), or myocarditis-related cardiosclerosis.

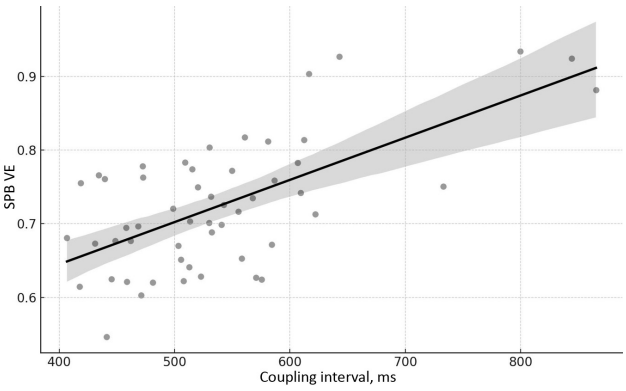

Figure 1. Relationship between CI and SBP during VE.

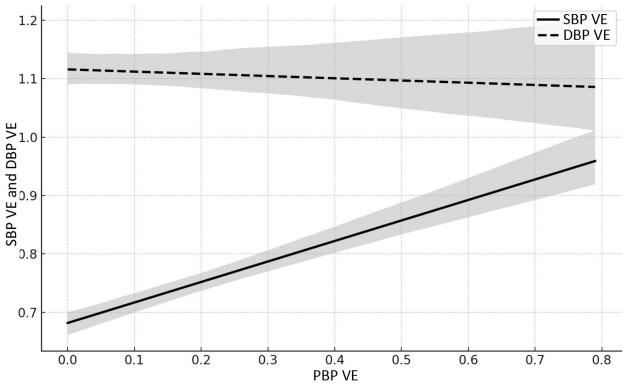

Figure 2. Relationship among SBP, DBP, and PBP during VE.

Excluding interpolated VEs and apparent parasystole from the analysis did not affect the results, except for the correlation between PBPpost VE and the number of VEs per day, which, along with the percentage of VEs, became statistically significant (r = -0.56, p=0.007).

A statistically significant correlation was observed between SBPpost VE and the duration of the compensatory pause (Figure 3), as well as between PBPpost VE and the duration of the compensatory pause. In contrast, the correlation between DBPpost VE and the postextrasystolic pause duration was not statistically significant.

The formation of the integral PBPpost VE parameter differed substantially from PBP VE. While the latter was primarily determined by SBP VE with minimal dependence on DBP VE, PBPpost VE was equally influenced by SBPpost VE and DBPpost VE, as shown in Figure 4.

DISCISSION

The determination of treatment strategies for VEs, as outlined in modern guidelines, is primarily based on the presence or absence of structural heart changes. Another crucial factor is symptomatology and the frequency of VEs. The number of ectopic beats directly correlates with the likelihood of developing AAC, regardless of the presence or absence of organic heart disease. However, in clinical practice, the progression to AAC varies among patients: some with a high VE burden develop AAC relatively quickly, while others remain unaffected over many years or even decades of observation[32].

In limited publications, several additional hemodynamically significant characteristics of VEs have been proposed as contributors to AAC formation. These include QRS width and fragmentation, morphology, prematurity index, and polymorphism. The *beat-to-beat* blood pressure measurement method allows the evaluation of these and other properties of VEs, enabling an understanding of their potential hemodynamic significance.

A key feature of VEs, as premature cardiac contractions, is their significant reduction in SBP. This reduction is attributed to insufficient ventricular filling, leading to decreased stroke volume and impaired cardiac pump function. Increased DBP may also play a role as a potential contributor to LV diastolic dysfunction. Additionally, the shortening of diastolic duration-the phase during which myocardial perfusion via coronary arteries occurs-can further compromise the blood supply to the myocardium. PBP, as a composite measure of SBP and DBP, emphasizes these changes when present.

The method also facilitates the evaluation of an intriguing, yet underexplored, property of VEs termed PESP. The existing literature on PESP presents conflicting findings. Physiological increases in SBP during sinus contractions following a compensatory pause have been attributed to enhanced contractility caused by elevated intracellular calcium levels in cardiomyocytes during the pause. Conversely, pathological conditions, such as chronic heart failure and myocardial infarction, have been associated with reduced SBP due to inadequate ventricular filling, which diminishes stroke volume. DBP typically remains unchanged but may increase in cases of LV diastolic dysfunction.

This study included patients with a high number of monomorphic VEs. Arrhythmogenic substrate localization was determined approximately using a standard 12-lead ECG. Following current guidelines, two groups were identified: those with RVOT arrhythmogenic substrate" and "all other localizations," which differ in treatment approaches. Additionally, a third group with "LVOT arrhythmogenic substrate" was analyzed based on ECG characteristics.

Table 3.

Indicators characterizing the hemodynamic significance of ventricular extrasystole

Indicator	Value
Post VE SBP across the entire patient group	0.98±0.05. p=0.02
Post VE DBP across the entire patient group	0.91±0.01. p<0.0001
Post VE PBP across the entire patient group	1.12±0.03. p=0.0001
Correlation between post VE SBP and post VE PBP	r=0.58. p=0.002
Correlation between post VE SBP and PEP	r=0.40. p=0.003
Correlation between post VE SBP and the presence of paired VEs	r=-0.40. p=0.04
Correlation between post VE DBP and post VE PBP	r=-0.61. p=0.001
Correlation between post VE DBP and VE DBP	r=0.51. p=0.008
Correlation between post VE DBP and CI of VE	r=0.48. p=0.01
Correlation between post VE DBP and the presence of NSVT	r=0.44. p=0.03
Correlation between post VE DBP and daily VE percentage	r=0.43. p=0.03
Correlation between post VE PBP and VE DBP	r=-0.52. p=0.01
Correlation between post VE PBP and the presence of NSVT	r=-0.48. p=0.03
Correlation between post VE PBP and daily VE percentage	r=-0.41. p=0.04
Correlation between post VE PBP and PEP	r=0.38. p=0.005

Note: hereinafter, PEP - Post-extrasystolic pause NSVT - Non-sustained ventricular tachycardia.

Inclusion criteria mandated either the absence of structural heart changes or minimal structural abnormalities. Patients with significant LV hypertrophy, previous myocardial infarction, clinically significant chronic heart failure, reduced pump function, or chamber enlargement-including those with a history of myocarditis-were excluded. Among hypertensive patients, the mean LV wall thickness was 11.4 mm, while in other groups, it remained within normal limits. LVEF was within normal ranges and did not differ significantly among the patient groups.

The present study included patients with a high number of monomorphic VEs. Arrhythmogenic substrate localization was determined approximately using a standard 12-lead ECG. Following current guidelines, patients were divided into two groups: "RVOT arrhythmogenic substrate localiza-

tion" and "all other localizations," which differed in treatment approaches. Additionally, a third group of patients with "LVOT arrhythmogenic substrate localization," easily identifiable by ECG, was included.

Inclusion criteria required the absence of structural heart changes or minimal structural abnormalities. Patients with significant left ventricular hypertrophy, prior myocardial infarction, clinically significant chronic heart failure, reduced pump function, or chamber enlargement-including those with a history of myocarditis-were excluded. Even among patients with hypertension, the mean LV wall thickness was only 11.4 mm, and for others, it did not exceed normal limits. LVEF was within normal ranges and did not differ significantly among the patient groups.

As expected, patients with "idiopathic" VEs were significantly younger. These patients were more likely to have arrhythmogenic substrates localized in the RVOT. Patients with LVOT VEs were older, while those with VEs from other sources were the oldest.

An interesting finding was the differences in the characteristics of the QRS complex during VEs based on arrhythmogenic substrate localization. VEs originating from the RVOT were wider than those from the LVOT. Half of the RVOT VEs showed fragmentation (similar to VEs from other localizations), whereas none of the LVOT VEs exhibited this phenomenon. These differences may reflect the nature of excitation propagation during VEs, even though the RVOT and LVOT are anatomically close.

The mean CI of VEs did not differ significantly between different arrhythmogenic substrate localizations. Factors such as a large number of interpolated VEs or evident parasystole with classical signs could have influenced the analysis. However, excluding such patients from the sample did not substantially alter the results.

In the second stage of the analysis, SBP, DBP, and PBP during VEs were evaluated as indicators that largely characterize the hemodynamic significance of ectopic contractions. Significant reductions in SBP and non-significant increases in DBP during VEs were observed. The most important characteristic of VEs, likely determining their hemodynamic significance, was the CI. No correlation was found with other parameters, such as arrhythmogenic substrate localization, QRS width, QRS fragmentation, or the presence of organic heart disease. The reduction in the integral PBP during VEs was minimally related to the increase in DBP and was primarily determined by the decrease in SBP.

A correlation between diastolic blood pressure during premature ventricular contractionsq and the number of VEs was established, indicating that DBP VE increases as the VE count rises. This correlation is logical, as a higher number of VEs reduces the overall relaxation and filling time of the heart. However, the observed inverse relationship between LVEF and both systolic blood pressure during VEs (SBP VE) and pulse blood pressure during VEs (PBP VE) is less straightforward. It appears that higher LVEF reduces the need for compensatory mechanisms, such as sympathetic nervous system activation, which might otherwise increase SBP.

The third stage of analysis explored the phenomenon of PESP. An interesting finding was the correlation between the mean CI and the duration of the compensatory pause; longer CIs were associated with longer pauses. This may be due to ventriculo-atrial conduction of VEs and the discharge or non-discharge of the sinus node.

Analysis of post-VE SBP, DBP, and PBP revealed that the presence of PESP should be interpreted cautiously. Post-VE SBP was slightly lower than the SBP of sinus beats preceding VEs. Post-VE DBP decreased modestly but not markedly. Post-VE PBP increased significantly; however, unlike VE beats, its changes were equally influenced by both post-VE SBP and DBP (with DBP having a slightly greater impact). As with VE-related SBP, DBP, and PBP, PESP characteristics showed no dependence on the localization of the arrhythmogenic substrate, QRS width or fragmentation, or the presence of structural heart disease. Post-VE SBP increased with longer compensatory pauses, which is consistent with physiological expectations. A weak but significant correlation was observed between reduced post-VE SBP and paired VEs, likely because two consecutive VEs share a single compensatory pause.

The correlation between post-VE DBP and DBP VE is straightforward and expected, whereas its positive association with CI suggests reflexive sympathetic activation due to baroreceptor stimulation. This could increase vascular tone, elevating post-VE DBP. However, while a significant relationship between CI and the compensatory pause duration was observed, post-VE DBP did not show a statistically significant link to the pause duration.

The relationship between post-VE DBP and the number of VEs (including VE percentages during Holter monitoring and the presence of non-sustained ventricular tachycardia) is logical and reflects insufficient ventricular relaxation during diastole. Consequently, the integral indi-

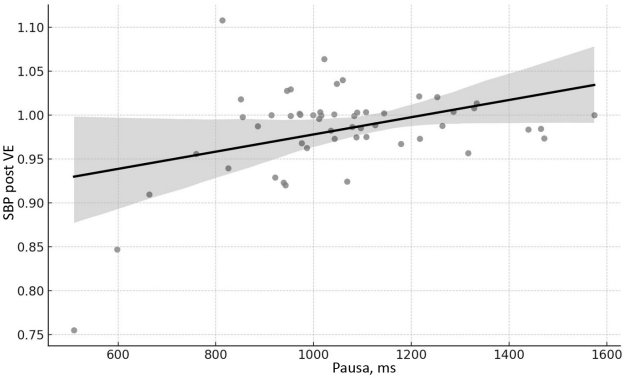

Figure 3. Relationship between post VE SBP and the duration of the compensatory pause.

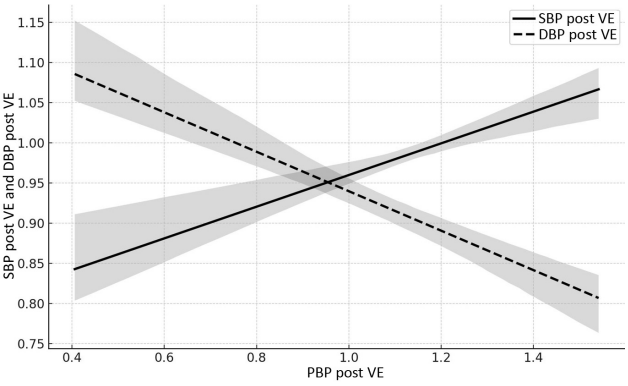

Figure 4. Relationship among post VE SBP, post VE DBP, and post VE PBP.

cator post-VE PBP correlates with the VE count and compensatory pause duration.

The study's foundation was the non-invasive determination of SBP, DBP, and PBP for each heartbeat. This enabled the assessment of hemodynamic properties of both ectopic and post-compensatory beats, providing insights into the PESP phenomenon. A notable feature influencing the study's outcomes was the selection of patients with no or minimal structural heart changes. Therefore, the findings should not be extrapolated to patients with clinically significant heart failure, previous myocardial infarction, severe LV hypertrophy, or other serious organic heart conditions. This important limitation is also a strength of the study, as it provides reference hemodynamic characteristics for VEs (SBP, DBP, PBP, post-VE SBP, DBP, and PBP) applicable to patients with frequent VEs. The most significant findings of this research can be summarized as follows.

CONCLUSION

1. Premature ventricular contractions, as hemodynamically ineffective cardiac beats, are characterised by a reduction in systolic blood pressure and, to a lesser extent, an increase in diastolic blood pressure.
2. SBP VE and pulse blood pressure during VEs decrease as the coupling interval shortens; no significant correlation was found between CI and DBP VE.
3. Post-VE SBP and DBP are lower, whereas post-VE PBP is higher than the corresponding values of sinus beats preceding VEs; the differences are small but statistically significant.
4. The examined characteristics (SBP VE, DBP VE, PBP VE, post-VE SBP, post-VE DBP, and post-VE PBP) were not dependent on QRS width or fragmentation or the localization of the arrhythmogenic substrate.

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