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# Valvular heart disease and different circadian blood pressure profiles

## Wady zastawkowe serca i różne profile dobowe ciśnienia tętniczego

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### Abstract

**Introduction.** Valvular heart diseases (VHD) increase the risk of cardiovascular morbidity and mortality. Little is known about the correlation between circadian blood pressure profile (CBPP) and VHD. The study aimed to clarify the association between CBPP and VHD prevalence.

**Material and methods.** 103 consecutive patients (male: 50.5%), who underwent 24-hour ambulatory blood pressure measurement (ABPM) and Holter electrocardiography simultaneously were analysed. Patients were divided into 3 groups: dipping was defined as 10–20% (28.2%), non-dipping as < 10% (50.5%) fall in nocturnal blood pressure (BP) and reverse-dipping as higher nocturnal than diurnal BP (21.4%). VHD was assessed by transthoracic echocardiography and described as mild, moderate or severe regurgitation or stenosis accordingly. Further, the severity of VHD, nocturnal fall pattern and ABPM features in all groups were compared.

**Results.** The authors found no statistically significant association between severity of VHD and dipping status. The presented study showed some correlations between VHD severity and different ABPM parameters.

**Concusions.** Though severity of VHD did not influence dipping status obtained by ABPM, there were associations between VHD and ABPM outcomes. Further studies are needed.

Key words: circadian rhythm, hypertension, valvular heart disease

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## Introduction

Hypertension is a growing problem, and currently, there are over 1 billion hypertensive individuals worldwide [1, 2]. In 1978 Millar-Craig et al. [3] described circadian variation of blood pressure (BP) using continuous intra-arterial monitoring. Nowadays ambulatory blood pressure measurement (ABPM) is a non-invasive method to obtain circadian blood pressure profile (CBPP) [4].

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Studies prove that non-dipping pattern in hypertensive individuals might be associated with increased cardiovascular risk [5–7]. The lower or lack of fall in nocturnal BP values could also cause target organ damage. Literature brings evidence that the non-dipping pressure profile is connected with left ventricle hypertrophy (LVH), cardiac functional alteration, renal damage, carotid artery abnormalities and cerebrovascular diseases [8–15]. Valvular heart disease (VHD) may be not as common as coronary artery disease (CAD) or heart failure but remains significant since it frequently requires interventions [16, 17]. The study aimed to assess if there are associations between VHD prevalence as well as the occurrence and either CBPP or ABPM parameters.

## Materials and methods

### Study population

This was a retrospective study analyzing data of 103 patients hospitalized in the Department of Cardiology and Hypertension in Central Research Hospital of the Ministry of Interior and Administration in Warsaw between January 2012 and December 2013. All consecutive patients, who simultaneously underwent ABPM and 24-hours Holter electrocardiography (ECG-Holter), were included in further analysis. According to nocturnal fall pattern, patients were divided into three groups. Dipping was defined as a 10–20% fall in nocturnal systolic BP (SBP), non-dipping as less than 10%, and reverse-dipping as higher average SBP during the night than during the day [6, 18]. Collected data were analyzed retrospectively and Local Ethics Committee gave consent to conduct the trial.

### Measurements

In the study, Treacker NIBP2 SpaceLabs Healthcare and ABP 90217-7Q SpaceLabs Healthcare devices were used to obtain ABPM and Lifecard CF Reynolds Medical device to assess ECG-Holter. Measurements of BP were performed every 10 minutes during awake hours and every 30 minutes at night. Additionally, all patients had transthoracic echocardiography (TTE) using Phillips IE-33 and EPIQ Ultrasound machines and rest-ECG performed. In TTE VHD severity was described as none (0), mild (+), moderate (++) or severe (+++).

### Statistics

Statistical analysis was performed on R version 3.1.2 [19]. Continuous variables are presented as several observations and mean with standard deviation; categorical variables are reported as frequencies and percentages. The distribution of continuous variables was first analysed with the Shapiro-Wilk test of normality and then according to the results ANOVA test or Kruskal-Wallis test were used. Categorical variables were compared using Fisher's exact test. The significance level was set at 0.05.

## Results

One hundred three consecutive patients (male: 50.5%) with mean age 63.9 ( ± 17.7) years simultaneously underwent ABPM and ECG-Holter were included in further analysis. According to ABPM outcomes, 29 (28%) patients were dippers, 52 (50%) were non-dippers and 22 (21%) were reverse-dippers respectively. The reverse dipper group was the oldest, with mean age 74.9 ± 10.9 years, and differences in age between groups were statistically significant ($p < 0.001$). Study population characteristic is presented in Table 1. There were significant differences between groups in the occurrence of diagnosis of chronic kidney disease (CKD) and peripheral artery disease (PAD), with the higher prevalence of those diseases in non–dipper and the highest in the reverse-dipper group. Mean systolic and diastolic pressure, both diurnal and nocturnal, which differed statistically significantly between subgroups in our study population, is presented also in Table 1.

Echocardiography parameters assessed in our study population are given in Table 2. From those results only left atrium diameter (LAD) differed significantly regarding CBPP (40.2 ± 4.8 mm; 43.8 ± 6.2 mm; 41.8 ± 5.2 mm; $p = 0.026$; dippers, non-dippers, reverse-dippers, respectively). We found no statistically significant difference between neither prevalence nor occurrence of VHD regarding CBPP in our study population. Parameters are given in Table 2 and Table 3.

List of drugs administered in this study population is given in Table 4. Both: β- and α-adrenolytics were more commonly used in non-dipper and reverse-dipper than in dipper population (α-adrenolytics: 0%; 13.04%; 30%; $p = 0.009$; β-adrenolytics: 57.69%; 69.57%; 95%; $p = 0.011$; dippers, non-dippers, reverse dippers, respectively). There were no statistically significant differences in drugs doses (Table 5).

This study showed that severity of aortic stenosis (AS) correlated positively with maximal nocturnal systolic blood pressure (SBP) ($\rho = 0.208$; $p = 0.038$) and that severity of aortic regurgitation (AR) correlated negatively with diastolic blood pressure (DBP) during the nighttime ($\rho = -0.214$; $p = 0.033$). Additionally it was found that severity of AR was connected with lower maximal heart rate (HR) at night ($\rho = -0.197$; $p = 0.050$), while AS correlated with lower maximal HR during awake hours ($\rho = -0.202$; $p = 0.044$). Tricuspid stenosis correlated negatively with both awake ($\rho = -0.199$; $p = 0.047$) and nightly DBP ($\rho = -0.207$; $p = 0.039$) and with maximal DBP during awake hours ($\rho = -0.198$; $p = 0.048$). During awake hours both minimal SBP ($\rho = -0.197$; $p = 0.050$) and maximal DBP ($\rho = -0.236$; $p = 0.018$) correlated negatively with pulmonary regurgitation.

**Table 1.** Population characteristics

| Variable | Dipper | Non-dipper | Reverse dipper | p* |
|---|---|---|---|---|
| Age [years] | 55.24 ± 17.83 | 63.98 ± 17.46 | 74.91 ± 10.86 | **p < 0.001** |
| Gender [M/F] | 16/13 | 30/22 | 6/16 | **p = 0.052** |
| SBP day [mm Hg] | 129.72 ± 11.30 | 127.48 ± 13.81 | 125.77 ± 15.32 | p = 0.575 |
| SBP night [mm Hg] | 111.55 ± 9.16 | 121.58 ± 13.73 | 133.00 ± 18.04 | p < 0.001 |
| SBP fall | 13.93 ± 2.83 | 4.63 ± 2.91 | –5.61 ± 3.71 | **p < 0.001** |
| DBP day [mm Hg] | 74.41 ± 8.19 | 71.23 ± 8.67 | 67.45 ± 6.53 | **p = 0.009** |
| DBP night [mm Hg] | 61.34 ± 6.67 | 65.29 ± 7.87 | 67.86 ± 8.05 | **p = 0.009** |
| DBP fall | 17.46 ± 3.93 | 8.21 ± 5.32 | –0.53 ± 5.35 | **p < 0.001** |
| HF | 8 (27.59%) | 17 (32.69%) | 11 (52.38%) | p = 0.188 |
| HFrEF | 0 (0.00%) | 1 (1.92%) | 0 (0.00%) | p > 0.999 |
| HFpEF | 6 (20.69%) | 15 (28.85%) | 10 (47.62%) | p = 0.126 |
| CKD | 1 (3.70%) | 9 (17.65%) | 6 (30.00%) | **p = 0.045** |
| PAD | 1 (4.00%) | 3 (5.88%) | 5 (26.32%) | **p = 0.031** |
| DCM | 0 (0.00%) | 2 (3.85%) | 2 (10.00%) | p = 0.175 |
| CAD | 6 (20.69%) | 8 (15.38%) | 7 (35.00%) | p = 0.178 |
| Post MI | 2 (7.14%) | 2 (3.85%) | 3 (15.79%) | p = 0.212 |
| Post CABG | 0 (0.00%) | 0 (0.00%) | 1 (5.26%) | p = 0.192 |
| OSAS | 7 (25.00%) | 13 (25.00%) | 2 (9.09%) | p = 0.302 |
| DM | 4 (14.29%) | 7 (13.73%) | 2 (10.00%) | p > 0.999 |
| Hyperlipidemia | 14 (50.00%) | 30 (57.69%) | 13 (61.90%) | p = 0.720 |
| COPD | 0 (0.00%) | 0 (0.00%) | 1 (5.00%) | p = 0.213 |
| AF | 3 (10.34%) | 15 (29.41%) | 8 (36.36%) | p = 0.055 |
| NT-proBNP [pg/mL] | 332.40 ± 359.33 | 1490.38 ± 2507.92 | 1414.30 ± 2919.21 | p = 0.650 |
| CK [µg/L] | 109.56 ± 38.57 | 92.66 ± 49.09 | 119.62 ± 65.23 | p = 0.103 |
| CK-MB [µg/L] | 16.56 ± 5.09 | 18.75 ± 9.88 | 24.82 ± 16.82 | **p = 0.030** |

*p – overall p-value for 3-group comparison of means (ANOVA test) or distributions (Kruskal-Wallis test) for continuous variables and percentages ($\chi^2$ test) for categorical variables; M – male; F – female; SBP – systolic blood pressure; DBP – diastolic blood pressure; HF – heart failure; HFrEF – HF with reduced ejection fraction; HFpEF – HF with preserved ejection fraction; CKD – chronic kidney disease; PAD – peripheral artery disease; DCM – dilated cardiomyopathy; CAD – coronary artery disease; post MI – post myocardial infarction; post CABG – post coronary artery bypass grafting; OSAS – obstructive sleep apnea syndrome; DM – diabetes mellitus; COPD – chronic obstructive pulmonary disease; AF – atrial fibrillation; NT-proBNP – N-terminal pro-B-type natriuretic peptide; CK – creatine kinase; CK-MB – creatine kinase myocardial bound

## Discussion

The heart is one of the organs damaged by hypertension. Some authors described LVH and higher left ventricle mass index (LVMI) in non-dipper patients group compared to dippers [8, 20–22]. Those data are inconclusive because others failed to prove those outcomes [23–25]. It may be due to different methods used in those studies, i.e. Cuspidi et al. [8], who proved statistically significant differences in LVH prevalence and higher LVMI in never-treated non-dippers with a reproducible non-dipper pattern of hypertension comparing to those with reproducible dipper pattern of hypertension, used multiple ABPM measurements to divide patients into groups, while others used only 1 measurement. Cuspidi et al. [9] described that fact as the reason for different outcomes in comparison with other studies. This fact may be relevant, but other authors reported high reproducibility of ABPM outcomes regarding CBPP in their study population (hemodialysis patients) after 6 and 12 months [20]. Ferrara et al. [26] reported that differences in echocardiography outcomes may be significant in dippers comparing to non-dippers only in recently discovered hypertension and showed similar changes in both groups in long-standing hypertension. Additionally, Sokmen et al. [27] found that there was no statistically significant difference between dippers and non-dippers hypertensive patients, who had adequate BP control regarding LVH, LVMI. We also assessed the presence of LVH and in our population, we found that non-dippers had higher both posterior wall end-diastolic diameter (PWDd) and interventricular septal end-diastolic dimension (IVSd). Additionally, it was found that there was an inverse tendency

**Table 2.** Echocardiography parameters

| Variable | Dipper | Non–dipper | Reverse dipper | p* |
|---|---|---|---|---|
| EF [%] | 62.39 ± 4.69 | 61.35 ± 7.23 | 59.86 ± 6.23 | p = 0.127 |
| LVDd [mm] | 50.31 ± 4.43 | 50.18 ± 5.88 | 50.10 ± 5.94 | p = 0.700 |
| RVDd [mm] | 31.50 ± 4.53 | 32.73 ± 4.86 | 31.45 ± 6.11 | p = 0.475 |
| LAD [mm] | 40.21 ± 4.80 | 43.75 ± 6.15 | 41.76 ± 5.22 | **p = 0.026** |
| IVSd [mm] | 10.59 ± 1.21 | 10.94 ± 1.77 | 10.11 ± 3.00 | p = 0.676 |
| PWDd [mm] | 10.34 ± 1.34 | 10.76 ± 1.73 | 10.14 ± 1.53 | p = 0.260 |
| TAPSE [mm] | 22.00 ± 4.24 | 23.00 ± 5.89 | 21.83 ± 2.99 | p = 0.901 |
| IVC [mm] | 16.00 ± 4.58 | 20.50 ± 5.45 | 15.46 ± 7.98 | p = 0.663 |
| **VHD** | | | | |
| AR | 7 (25.00%) | 18 (35.29%) | 6 (28.57%) | p = 0.632 |
| AS | 0 (0.00%) | 2 (3.92%) | 0 (0.00%) | p = 0.717 |
| MR | 20 (68.97%) | 36 (70.59%) | 18 (85.71%) | p = 0.370 |
| MS | 0 (0.00%) | 0 (0.00%) | 0 (0.00%) | NA |
| TR | 10 (35.71%) | 28 (54.90%) | 12 (57.14%) | p = 0.236 |
| TS | 0 (0.00%) | 2 (3.92%) | 0 (0.00%) | p = 0.712 |
| PR | 0 (0.00%) | 1 (1.96%) | 1 (4.76%) | p = 0.454 |
| PS | 0 (0.00%) | 0 (0.00%) | 0 (0.00%) | NA |

*p – overall p-value for 3-group comparison of means (ANOVA test) or distributions (Kruskal-Wallis test) for continuous variables and percentages ($\chi^2$ test) for categorical variables; EF – ejection fraction; LVDd – left ventricle end diastolic dimension; RVDd – right ventricle end diastolic dimension; LAD – left atrium diameter; IVSd – intraventricular septum end-diastolic diameter; PWDd – posterior wall end-diastolic diameter; TAPSE – tricuspid annular plane systolic excursion; IVC – inferior vena cava; VHD – valvular heart disease; AR – aortic regurgitation; AS – aortic stenosis; MR – mitral regurgitation; MS – mitral stenosis; TR – tricuspid regurgitation; TS – tricuspid stenosis; PR – pulmonary regurgitation; PS – pulmonary stenosis

**Table 3.** Valvular heart disease severity

| Variable | Severity | Dipper | Non-dipper | Reverse dipper | p* |
|---|---|---|---|---|---|
| AR | 0 | 21 (75.00%) | 33 (64.71%) | 15 (71.43%) | p = 0.869 |
| | 1 | 7 (25.00%) | 17 (33.33%) | 6 (28.57%) | |
| | 2 | 0 (0.00%) | 1 (1.96%) | 0 (0.00%) | |
| AS | 0 | 28 (100.00%) | 49 (96.08%) | 21 (100.00%) | p > 0.999 |
| | 1 | 0 (0.00%) | 1 (1.96%) | 0 (0.00%) | |
| | 3 | 0 (0.00%) | 1 (1.96%) | 0 (0.00%) | |
| MR | 0 | 9 (31.03%) | 15 (29.41%) | 3 (14.29%) | p = 0.422 |
| | 1 | 19 (65.52%) | 28 (54.9%) | 15 (71.43%) | |
| | 2 | 1 (3.45%) | 7 (13.73%) | 2 (9.52%) | |
| | 3 | 0 (0.00%) | 1 (1.96%) | 1 (4.76%) | |
| TS | 0 | 28 (100.00%) | 49 (96.08%) | 21 (100.00%) | p = 0.712 |
| | 1 | 0 (0.00%) | 2 (3.92%) | 0 (0.00%) | |
| TR | 0 | 18 (64.29%) | 23 (45.10%) | 9 (42.86%) | p = 0.280 |
| | 1 | 10 (35.71%) | 23 (45.10%) | 10 (47.62%) | |
| | 2 | 0 (0.00%) | 5 (9.80%) | 2 (9.52%) | |
| PR | 0 | 28 (100.00%) | 50 (98.04%) | 20 (95.24%) | p = 0.454 |
| | 1 | 0 (0.00%) | 1 (1.96%) | 1 (4.76%) | |

*p – overall p-value for 3-group comparison of percentages ($\chi^2$ test) for categorical variables; AR – aortic regurgitation; AS – aortic stenosis; MR – mitral regurgitation; TR – tricuspid regurgitation; TS – tricuspid stenosis; PR – pulmonary regurgitation

**Table 4.** Drugs administered in the study population

| Variable | Dipper | Non–dipper | Reverse dipper | p* |
|---|---|---|---|---|
| Beta-adrenolytics | 15 (57.69%) | 32 (69.57%) | 19 (95.00%) | **p = 0.011** |
| Alpha-adrenolytics | 0 (0.00%) | 6 (13.04%) | 6 (30.00%) | **p = 0.009** |
| Ca-antagonists | 9 (34.62%) | 26 (56.52%) | 9 (45.00%) | p = 0.196 |
| ACEI | 16 (61.54%) | 22 (47.83%) | 13 (65.00%) | p = 0.388 |
| ARB | 3 (11.54%) | 14 (30.43%) | 6 (30.00%) | p = 0.164 |
| Amiodarone | 0 (0.00%) | 1 (2.17%) | 0 (0.00%) | p > 0.999 |
| Digoxine | 0 (0.00%) | 2 (4.35%) | 0 (0.00%) | p = 0.714 |
| Aldosterone antagonists | 0 (0.00%) | 7 (15.22%) | 1 (5.00%) | p = 0.073 |
| Furosemide | 0 (0.00%) | 8 (17.39%) | 2 (10.00%) | p = 0.057 |
| Torasemide | 1 (3.85%) | 2 (4.35%) | 7 (35.00%) | **p = 0.002** |
| Kaldyum | 0 (0.00%) | 6 (13.04%) | 0 (0.00%) | **p = 0.047** |
| Kalipoz | 4 (15.38%) | 19 (41.3%) | 12 (60.00%) | **p = 0.006** |
| Hydrochlorotiazide | 1 (3.85%) | 8 (17.02%) | 0 (0.00%) | p = 0.065 |
| Indapamide | 5 (19.23%) | 9 (19.57%) | 3 (15.00%) | p = 0.941 |
| ASA | 7 (26.92%) | 12 (26.09%) | 8 (40.00%) | p = 0.524 |
| VKA | 3 (11.54%) | 10 (21.74%) | 3 (15.00%) | p = 0.593 |
| LMWH | 0 (0.00%) | 1 (2.17%) | 1 (5.00%) | p = 0.467 |
| Statin | 18 (69.23%) | 30 (65.22%) | 15 (75.00%) | p = 0.732 |

*p – overall p value for 3-group comparison of percentages ($\chi^2$ test) for categorical variables; Ca – calcium; ACEI – angiotensin converting-enzyme inhibitor; ARB – angiotensin II receptor blocker; ASA – acetylsalicylic acid; VKA – vitamin K antagonist; LMWH – low-molecular-weight heparin

**Table 5.** Drugs doses in the study population

| Variable | Doses | Dipper | Non-dipper | Reverse dipper | p* |
|---|---|---|---|---|---|
| Beta-adrenolytics | 0 | 11 (44.00%) | 14 (31.11%) | 1 (5.26%) | p = 0.082 |
| | 1 | 9 (36.00%) | 22 (48.89%) | 12 (63.16%) | |
| | 2 | 5 (20.00%) | 7 (15.56%) | 5 (26.32%) | |
| | 3 | 0 (0.00%) | 2 (4.44%) | 1 (5.26%) | |
| ACEI | 0 | 10 (38.46%) | 24 (52.17%) | 7 (35.00%) | p = 0.073 |
| | 1 | 0 (0.00%) | 5 (10.87%) | 3 (15.00%) | |
| | 2 | 6 (23.08%) | 2 (4.35%) | 4 (20.00%) | |
| | 3 | 10 (38.46%) | 15 (32.61%) | 6 (30.00%) | |
| ARB | 0 | 23 (88.46%) | 32 (71.11%) | 14 (70.00%) | p = 0.612 |
| | 1 | 1 (3.85%) | 2 (4.44%) | 1 (5.00%) | |
| | 2 | 2 (7.69%) | 7 (15.56%) | 3 (15.00%) | |
| | 3 | 0 (0.00%) | 4 (8.89%) | 2 (10.00%) | |

*p – overall p-value for 3-group comparison of percentages ($\chi^2$ test) for categorical variables 1 – small doses; 2 – medium doses; 3 – high doses; ACEI – angiotensin-converting-enzyme inhibitor; ARB – angiotensin II receptor blocker

in a reverse-dipper group, what was in contraposition to Wang et al. [28] results. Although, in all those parameters a statistical significance was not reached, what might be due to the relatively smaller study population than in cited studies. Additionally, in another original article recently submitted for publication, factors determining CBPP and nightly fall of SBP were analysed. It was found that older patients diagnosed with peripheral artery disease or dilated cardiomyopathy and who used α-adrenolytics had lower fall in nocturnal SBP. Also, patients who had lower haemoglobin concentration, higher CK-MB values or lower maximal heart rate had lower fall in nocturnal SBP. The authors assessed a higher prevalence of altered CBPP in patients who used β- or α-adrenolytics or torasemide.

Though according to these results none of TTE parameters (including LVH) determined CBPP and nightly fall of SBP. Looking further into the analysis of TTE measurements, in the context of CBPP larger LAD, was already described by others in the non-dipper group [9]. These results proved those outcomes regarding differences between dippers and non-dippers. Surprisingly it was found that this tendency was not continued in the reverse-dipper group, although no studies to compare these outcomes with were found. Regarding the detailed analysis of VHD in the context of CBPP, only one study in which authors assessed VHD (in that particular study only aortic valve disease was considered) influence on CBPP was found. Authors found that aortic valve disease was connected with altered CBPP. The outcomes of the presented study did not prove that fact. It may be due to the smaller population in the cited Jensen et al. study (13 patients with either aortic regurgitation or aortic stenosis) [29].

Above-mentioned Jensen's study may also be considered as one of the reasons why there was a correlation between AS severity and higher maximal SBP at night. They found that there was higher activity of the renin-angiotensin system in patients with aortic valve disease [29]. Others described higher sympathetic nervous system activity in patients diagnosed with AS [30]. Further studies are needed because the outcomes of this study regarding other correlations were not proved by other studies.

As already mentioned, the limitation of this study may be the fact that the authors used only one ABPM outcome to define and divide the study population. Additionally, relatively large reverse dipper population may be considered both, limitation and strength of this study. It could be a limitation because there are not enough data regarding reverse dipper population in literature, so the authors had little opportunity to compare their results. It may be considered a strength because it gives information about the population, which has not been precisely described so far.

## Conclusion

To conclude, the influence of neither occurrence nor severity of VHD on CBPP in the study population was found. Some interesting associations between ABPM parameters and severity of VHD were found though. Further studies are needed, but these outcomes may have implications on the care of patients with hypertension.

## Conflict of interest

Authors declare no conflict of interest.


### Streszczenie

**Wstęp.** Wady zastawkowe serca (VHD) zwiększają ryzyko zachorowań i zgonów z przyczyn sercowo-naczyniowych. Niewiele wiadomo na temat zależności między profilem dobowym ciśnienia tętniczego (CBPP) a VHD. Celem tej pracy było wyjaśnienie związku między CBPP a VHD.

**Materiał i meody.** Do badania włączono 103 kolejnych pacjentów (mężczyźni 50,5%), u których równocześnie wykonano całodobowy pomiar ciśnienia tętniczego (ABPM) i 24-godzinny zapis elektrokardiograficzny metodą Holtera. Podzielono ich na trzy grupy: *dippers* — zdefiniowanych jako osoby z ciśnieniem tętniczym (BP) w nocy o 10–20% niższym niż w ciągu dnia (28,2%), *non-dippers* — osoby ze spadkiem BP w nocy mniejszym niż 10% (50,5%), *reverse-dippers* — osoby z wyższymi wartościami BP w nocy niż w ciągu dnia (21,4%). Metodą echokardiografii przezklatkowej oceniano VHD jako małą, umiarkowaną lub ciężką. Następnie porównywano ciężkość VHD, CBPP i dane z ABPM we wszystkich grupach.

**Wyniki.** Nie znaleziono istotnej statystycznie zależności między ciężkością VHD a CBPP. Zaobserwowano korelację między ciężkością VHD a niektórymi parametrami ocenianymi w trakcie ABPM.

**Wnioski.** Choć ciężkość VHD nie wpływała na CBPP, to istnieją zależności między wynikami VHD i ABPM. Konieczne są dalsze badania.


Słowa kluczowe: rytm okołodobowy, nadciśnienie tętnicze, wady zastawkowe serca

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