Determinants of the circadian blood pressure pattern in hospitalized hypertensive patients

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Abstract

Background. Non-dipping hypertension might be associated with increased cardiovascular risk and multiple diseases. The aim of our study was to assess if there are parameters identified in 24-hour ECG-Holter monitoring (ECG-Holter), transthoracic echocardiography (TTE), ECG parameters or laboratory data that allow prediction of circadian blood pressure profile (CBPP).

Material and methods. One hundred and three consecutive patients (male: 50.5%), who underwent 24-hour ambulatory BP measurement and ECG-Holter simultaneously were analyzed. We divided patients into 3 groups: dipping was defined as 10–20% (28.2%), non-dipping as < 10% (50.5%) fall in nocturnal BP and reverse-dipping as higher nocturnal than diurnal BP (21.4%). Additionally, we performed TTE and laboratory check-up in all patients. We built multivariable models for nocturnal fall in systolic BP (SBP) and CBPP.

Results. Multivariable model based on clinical factors was: nocturnal fall in SBP (%) = [13.28 – 0.11 × age – 8.33 × (dilated cardiomyopathy) – 5.95 × PAD – 6.02 × α-adrenolytic]. Multivariable model based on laboratory, echocardiographic and electrocardiographic parameters was: nocturnal fall in SBP (%) = [–27.28 + 1.47 × hemoglobin – 0.14 × CK-MB + 0.14 × maximal heart rate]. Multivariable model for CBPP based on clinical factors included use of β- or α-adrenolytics or torasemide.

Conclusions. We proved that nocturnal fall in SBP and CBPP could be predicted based on ECG-Holter parameters, laboratory data and TTE results, as well as based on detailed medical history. These findings may have implications on care of patients with hypertension.

Key words: ambulatory blood pressure monitoring; circadian rhythm; hypertension; hypotensive therapy

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Introduction

Hypertension is a growing problem, and currently, there are over 1 billion hypertensive individuals worldwide [1, 2]. Some diseases, such as autonomic neuropathies, obstructive sleep apnea, diabetes, autonomic dysfunction, and chronic kidney disease (CKD), also seem to relate to lower fall in nocturnal blood pressure (BP) [3, 4]. Patients affected with secondary or malignant hypertension also have higher prevalence of non-dipping pattern of circadian blood pressure profile (CBPP) [3, 4].

Recently published study suggests that asleep blood pressure is the most important risk factor for cardiovascular events which can be derived from BP values. In the study of Hermida et al., the asleep systolic BP was the most important risk factor for the primary outcome (defined as composite of cardiovascular death, myocardial infarction, coronary revascularization, heart failure and stroke) during 5.1-year median follow-up, and it was regardless of office and awake systolic BP values. The authors emphasize also the fact that attenuation of above-mentioned parameter was the most significant marker of event-free survival [5].

Despite all above-mentioned facts the availability of 24-hour ambulatory blood pressure monitoring (ABPM) is rather low. The awareness about these novel goals in hypertension treatment is also small. Furthermore, little is known about factors either determining or allowing predicting CBPP, besides above-mentioned comorbidities. The aim of the study was to clarify whether there are such factors among 24-hour ECG Holter monitoring (ECG--Holter), transthoracic echocardiography (TTE), ECG parameters or laboratory data.

Material 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 in Warsaw between January 2012 and December 2013. Consecutive hypertensive patients, who simultaneously underwent ABPM and ECG-Holter, were included into analysis. According to nocturnal BP fall pattern we divided the patients into three groups: dippers, non-dippers, reverse-dippers. Dipping was defined as a 10–20% fall in nocturnal systolic BP (SBP), non-dipping as less than 10%, and reverse-dipping as 0% at most fall in nocturnal SBP [6, 7]. Collected data were analyzed retrospectively and Local Ethics Committee gave consent to conduct the study. All procedures were performed in accordance with the Declaration of Helsinki on the treatment of human subjects.

Measurements

We used Tracker NIBP2 SpaceLabs Healthcare and ABP 90217-7Q SpaceLabs Healthcare devices 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 during the night-time. Additionally, all patients underwent TTE with Philips iE-33 and EPIQ ultrasound machines.

Statistical analysis

Statistical analysis was performed with R version 3.1.2 [8]. Continuous variables are presented as number of observations and mean with standard deviation, categorical variables are reported as frequencies and percentages. The distribution of continuous variables was first analyzed with 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. The impact of clinical, laboratory, echocardiographic and electrocardiographic factors on nocturnal fall in SBP and CBPP was analyzed with linear regression analysis and ordinal logistic regression analysis, respectively. Univariable and mutlivariable analyses were performed. Two multivariable models were built, with independent variables chosen from clinical factors and from laboratory, echocardiographic and electrocardiographic parameters for each dependent variable (nocturnal fall in SBP and CBPP). Variables considered in multivariable models were chosen from the set of variables with Wald's test p-value less than 0.25 in univariable analysis and that were classified in expert analysis as having potential influence on dependent variables. Multivariable models were selected with backward elimination procedure. Linear regression coefficients or proportional odds ratios were reported, with 95% confidence interval and Wald's test p-values, respectively.

Results

One hundred three consecutive patients (male: 50.5%) with mean age 63.9 (± 17.7) years who simultaneously underwent ABPM and ECG-Holter were included in further analysis. According to ABPM outcomes 29 (28%) patients were classified as

dippers, 52 (50%) as non-dippers and consecutive 22 (21%) as reverse-dippers. The reverse-dipper group was the oldest, with mean age 74.9 ± 10.9 years ($p < 0.001$). Study population characteristics are presented in Table I. There were significant differences between groups in occurrence of CKD and peripheral artery disease (PAD), with higher prevalence of those diseases in non-dipper and the highest in reverse-dipper group. In Table I we also included comparison of drugs taken by patients' subgroups. We found that reverse-dippers were most likely to take both: β- and α-adrenolytics as well as torasemide. Table I also includes mean values of diurnal and nightly, both systolic and diastolic, BP. In Table II we present laboratory as well as TTE and in Table III ECG-Holter parameters.

The multivariable model for nocturnal fall in SBP based on clinical factors was:

$$\textit{Nocturnal fall in SBP [\%]} = 13.28 - 0.11 \times \textit{age} - 8.33 \times \textit{DCM} - 5.95 \times \textit{PAD} - 6.02 \times \alpha\textit{-adrenolytic}$$

According to the presented model, if a patient's age increases by 1 year, nocturnal fall in SBP decreases by 0.11% [95% confidence interval (CI): –0.187 to –0.029], if a patient has dilated cardiomyopathy (DCM), PAD or uses α-adrenolytics, nocturnal fall in SBP decreases by 8.33% (95% CI: –15.836 to –0.823), 5.95% (95% CI: –10.540 to –1.361) and 6.02% (95% CI: –10.010 to –2.024), respectively (Tab. IV).

Table I. Population characteristics

	Dipper (29 pts)	Non-dipper (52 pts)	Reverse-dipper (22 pts)	
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 = 0.05**
SBP day [mm Hg]	129.72 ± 11.30	127.48 ± 13.81	125.77 ± 15.32	p = 0.58
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 = 0.19
HF-REF	0 (0.00%)	1 (1.92%)	0 (0.00%)	p > 0.999
HF-PEF	6 (20.69%)	15 (28.85%)	10 (47.62%)	p = 0.13
CKD	1 (3.70%)	9 (17.65%)	6 (30.00%)	**p = 0.05**
PAD	1 (4.00%)	3 (5.88%)	5 (26.32%)	**p = 0.03**
DCM	0 (0.00%)	2 (3.85%)	2 (10.00%)	p = 0.18
OSA	7 (25.00%)	13 (25.00%)	2 (9.09%)	p = 0.3
DM	4 (14.29%)	7 (13.73%)	2 (10.00%)	p > 0.999
AF	3 (10.34%)	15 (29.41%)	8 (36.36%)	p = 0.06
β-adrenolytics	15 (57.69%)	32 (69.57%)	19 (95.00%)	**p = 0.01**
α-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 = 0.2
ACE-I	16 (61.54%)	22 (47.83%)	13 (65.00%)	p = 0.39
ARB	3 (11.54%)	14 (30.43%)	6 (30.00%)	p = 0.16
Aldosterone antagonists	0 (0.00%)	7 (15.22%)	1 (5.00%)	p = 0.07
Furosemide	0 (0.00%)	8 (17.39%)	2 (10.00%)	p = 0.06
Torasemide	1 (3.85%)	2 (4.35%)	7 (35.00%)	**p = 0.002**

pts — patients; p — overall p-value for 3-group comparison of means (ANOVA test) or distributions (Kruskal-Wallis test) for continuous variables and percentages (χ^2 test) for categorical variables; SBP — systolic blood pressure; DBP — diastolic blood pressure; HF — heart failure; HF-REF — HF with restricted ejection fraction; HF-PEF — HF with preserved ejection fraction; CKD — chronic kidney disease; PAD — peripheral artery disease; DCM — dilated cardiomyopathy; OSA – obstructive sleep apnea syndrome; DM — diabetes mellitus; AF — atrial fibrillation; ACE-I — angiotensin-converting-enzyme inhibitor; ARB — angiotensin II receptor blocker

Table II. Laboratory and echocardiographic findings

	Dipper (29 pts)	Non-dipper (52 pts)	Reverse-dipper (22 pts)	
HGB [g/dL]	14.30 ± 1.42	13.84 ± 1.57	12.70 ± 1.40	**p = 0.002**
RBC [× $10^6/\mu L$]	4.81 ± 0.56	4.61 ± 0.51	4.28 ± 0.46	**p = 0.003**
HCT (%)	42.24 ± 3.82	41.57 ± 4.15	39.04 ± 3.13	**p = 0.02**
PLT [× 10^9/L]	207.75 ± 46.43	199.96 ± 58.39	212.40 ± 62.87	p = 0.68
MCV [fL]	88.21 ± 3.73	89.97 ± 4.70	91.84 ± 6.20	p = 0.09
MCH [pg]	29.76 ± 1.17	29.99 ± 1.73	30.02 ± 1.97	p = 0.81
MCHC [g/dL]	33.66 ± 1.16	33.36 ± 0.96	32.73 ± 0.97	**p = 0.001**
Urea [mg/dL]	30.21 ± 5.85	41.04 ± 32.72	46.71 ± 21.11	**p = 0.03**
eGFR [mL/min/1.73 m^2]	88.15 ± 18.77	80.35 ± 26.42	76.35 ± 29.04	p = 0.25
Creatinine [mg/dL]	0.84 ± 0.17	1.15 ± 1.70	0.93 ± 0.44	p = 0.47
AST [U/L]	20.86 ± 5.89	26.02 ± 14.03	29.88 ± 30.01	p = 0.4
ALT [U/L]	25.30 ± 13.02	31.49 ± 30.46	23.59 ± 30.22	**p = 0.02**
NT-proBNP [pg/mL]	332.40 ± 359.33	1490.38 ± 2507.92	1414.30 ± 2919.21	p = 0.65
CK [μg/L]	109.56 ± 38.57	92.66 ± 49.09	119.62 ± 65.23	p = 0.1
CK-MB [μg/L]	16.56 ± 5.09	18.75 ± 9.88	24.82 ± 16.82	**p = 0.03**
EF (%)	62.39 ± 4.69	61.35 ± 7.23	59.86 ± 6.23	p = 0.13
LVDd [mm]	50.31 ± 4.43	50.18 ± 5.88	50.10 ± 5.94	p = 0.7
RVDd [mm]	31.50 ± 4.53	32.73 ± 4.86	31.45 ± 6.11	p = 0.48
LAD [mm]	40.21 ± 4.80	43.75 ± 6.15	41.76 ± 5.22	**p = 0.03**
IVSd [mm]	10.59 ± 1.21	10.94 ± 1.77	10.11 ± 3.00	p = 0.68
PWDd [mm]	10.34 ± 1.34	10.76 ± 1.73	10.14 ± 1.53	p = 0.26
TAPSE [mm]	22.00 ± 4.24	23.00 ± 5.89	21.83 ± 2.99	p = 0.9
IVC [mm]	16.00 ± 4.58	20.50 ± 5.45	15.46 ± 7.98	p = 0.66
VHD				
AR	7 (25.00%)	18 (35.29%)	6 (28.57%)	p = 0.63
AS	0 (0.00%)	2 (3.92%)	0 (0.00%)	p = 0.71
MR	20 (68.97%)	36 (70.59%)	18 (85.71%)	p = 0.37
MS	0 (0.00%)	0 (0.00%)	0 (0.00%)	NA
TR	10 (35.71%)	28 (54.90%)	12 (57.14%)	p = 0.24
TS	0 (0%)	2 (3.92%)	0 (0.00%)	p = 0.71
PR	0 (0.00%)	1 (1.96%)	1 (4.76%)	p = 0.45
PS	0 (0.00%)	0 (0.00%)	0 (0.00%)	NA

pts — patients; p — overall p-value for 3-group comparison of means (ANOVA test) or distributions (Kruskal-Wallis test) for continuous variables; HGB — hemoglobin; RBC — red blood cells count; HCT — hematocrit; PLT — platelets count; MCV — mean corpuscular volume; MCH — mean corpuscular hemoglobin; MCHC — mean corpuscular hemoglobin concentration; eGFR — estimated glomerular filtration rate; CRP — C-reactive protein; AST — aspartate transaminase; ALT — alanine transaminase; NT-proBNP — N-terminal of the prohormone brain natriuretic peptide; CK — creatinine kinase; CK-MB — CK MB isoenzyme; 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

The multivariable model for nocturnal fall in SBP based on laboratory, TTE and ECG parameters was:

$$\textit{Nocturnal fall of SBP [\%]} = -27.28 + 1.47 \times HGB - 0.14 \times CK-MB + 0.14 \times HR_{max}$$

According to the presented model, if a patient's hemoglobin concentration (HGB), creatinine kinase MB isoenzyme (CK-MB) or maximal heart rate (HR_{max}) increases by 1 unit, nocturnal fall in SBP increases by 1.47% [95% CI: 0.447 to 2.487), decreases by 0.14% (95% CI: –0.271 to –0.002) or increases by 0.14% (95% CI: 0.051 to 0.223), respectively (Tab. V).

The multivariable model for CBPP based on clinical factors is presented in Table VI. According to that model, if patient uses: β- or α-adrenolytics or

Table III. 24-hour ECG Holter parameters

	Dipper (29 pts)	Non-dipper (52 pts)	Reverse-dipper (22 pts)	
Number of beats	77498.38 ± 16268.83	75538.67 ± 18449.94	82990.00 ± 24477.60	p = 0.89
HR_{mean}	66.66 ± 7.33	68.08 ± 15.63	65.14 ± 6.34	p = 0.8
HR_{max}	109.45 ± 17.21	102.50 ± 18.43	95.77 ± 12.91	**p = 0.02**
HR_{min}	51.31 ± 6.87	52.56 ± 6.78	52.86 ± 6.06	p = 0.65
AF	7.08 ± 38.11	7.84 ± 27.14	9.09 ± 29.42	p = 0.71
Pause	0.24 ± 0.83	2.35 ± 11.32	1.36 ± 4.01	p = 0.78
Pause max (s)	0.24 ± 0.74	0.43 ± 1.11	0.30 ± 0.78	p = 0.78
Bradycardias	0.14 ± 0.74	1.38 ± 4.60	0.14 ± 0.64	p = 0.13
VT	0.03 ± 0.19	0.31 ± 1.55	0.05 ± 0.21	p = 0.5
ExV	482.48 ± 2274.48	426.31 ± 1263.96	259.68 ± 722.38	p = 0.07
ExV/h	70.03 ± 258.05	54.58 ± 132.47	41.18 ± 101.26	**p = 0.05**
ExV/min	3.31 ± 8.70	5.19 ± 8.47	4.91 ± 5.13	**p = 0.03**
Couples	1.66 ± 8.91	0.92 ± 3.09	14.82 ± 67.05	**p = 0.02**
Triplets	0.03 ± 0.19	0.06 ± 0.31	0.05 ± 0.21	p = 0.98
Bigeminy	16.76 ± 79.00	7.31 ± 26.85	0.82 ± 2.44	p = 0.78
ExSV	120.97 ± 426.45	180.27 ± 408.43	1158.27 ± 2947.52	**p = 0.05**
SVT	1.52 ± 4.36	3.25 ± 14.29	39.64 ± 176.11	**p = 0.03**
ExSV/h	27.17 ± 104.68	27.40 ± 63.17	142.86 ± 321.07	p = 0.07
ExSV/min	4.59 ± 7.11	5.40 ± 8.14	10.50 ± 13.91	p = 0.05
Couples	1.64 ± 3.84	5.90 ± 20.13	82.77 ± 335.52	**p = 0.02**
P [ms]	84.86 ± 22.58	80.55 ± 32.14	65.91 ± 39.36	p = 0.29
PQ [ms]	145.17 ± 43.88	149.71 ± 56.68	142.73 ± 80.84	p = 0.75
QRS [ms]	98.28 ± 26.02	120.10 ± 101.88	102.86 ± 23.45	p = 0.5
ST [ms]	138.21 ± 137.70	154.90 ± 143.47	154.55 ± 51.34	**p = 0.04**
ECG HR	73.62 ± 13.04	69.60 ± 15.28	64.73 ± 16.44	p = 0.05
QTc [ms]	445.89 ± 73.36	428.76 ± 60.99	436.19 ± 39.26	p = 0.66

pts — patients; p — overall p-value for 3-group comparison of means (ANOVA test) or distributions (Kruskal-Wallis test) for continuous variables; HR — heart rate; AF — atrial fibrillation; VT — ventricular tachycardia; exV — ventricular extrasystoles; exV/h — exV per hour; exV/min — exVper minute; exSV — supraventricular extrasystoles; SVT — supraventricular tachycardia; exSV/h — exSV per hour; exSV/min — exSV per minute; QTc — corrected QT

Table IV. Multivariable model for nocturnal fall in systolic blood pressure (SBP) based on clinical factors

	Linear regression coefficient (95% CI)	
Age	–0.108 (–0.187 to –0.029)	p = 0.008
DCM	–8.329 (–15.836 to –0.823)	p = 0.03
PAD	–5.951 (–10.540 to –1.361)	p = 0.01
α-adrenolytics	–6.017 (–10.010 to –2.024)	p = 0.004

Variables included in multivariable model for nocturnal fall in SBP based on clinical factors. CI — confidence interval; DCM — dilated cardiomyopathy; PAD — peripheral artery disease

Table V. Multivariable model for nocturnal fall in systolic blood pressure (SBP) based on laboratory, transthoracic echocardiography (TTE) and electrocardiography (ECG) parameters

	Linear regression coefficient (95% CI)	
HGB	1.467 (0.447 to 2.487)	p = 0.006
CK-MB	–0.136 (–0.271 to –0.002)	p = 0.05
HR_{max}	0.137 (0.051 to 0.223)	p = 0.002

Variables included in multivariable model for nocturnal fall in SBP based on all other assessed factors. CI — confidence interval; HGB — hemoglobin; CK-MB — CK MB isoenzyme; HR — heart rate

torasemide, odds ratios of being in non-dipper or reverse dipper group versus dipper group or being in reverse dipper group versus dipper and non-dipper group were 3.26 (95% CI: 1.337 to 8.280), 5.80 (95% CI: 1.678 to 22.110) and 8.69 (95% CI: 1.976 to 48.034), respectively.

Table VI. Multivariable model for circadian blood pressure pattern (CBPP) based on clinical factors

	Proportional OR (95% CI)	
β-adrenolytics	3.264 (1.337 to 8.280)	p = 0.01
α-adrenolytics	5.799 (1.678 to 22.110)	p = 0.007
Torasemide	8.686 (1.976 to 48.034)	p = 0.007

Variables included in multivariable model for CBPP based on clinical factors. OR — odds ratio; CI — confidence interval

Discussion

Millar-Craig et al. described circadian variation of BP in 1978 [9]. They used continuous intra-arterial monitoring. Nowadays ABPM is a noninvasive method to obtain CBPP [10]. Others proved that non-dipping pattern in hypertensive individuals might be associated with increased cardiovascular risk [6]. The lower or lack of fall in nocturnal BP could also cause target organ damage. Literature brings evidence that non-dipping blood pressure profile relates to left ventricle hypertrophy, cardiac functional alternations, renal damage, carotid artery abnormalities and cerebrovascular diseases [11–14].

Our study demonstrated that CBPP could be predicted according to clinical factors as well as laboratory, TTE and ECG parameters. The multivariable model for nocturnal fall in SBP based on clinical factors included age, diagnosis of DCM or PAD and the use of α-adrenolytics. Others described lower sleep-time decline in SBP in older patients and it is known that older patients are more likely to be non-dippers or reverse dippers [15, 16]. Some authors report that dipping status in hypertension influences cardiac structure and function [13, 17, 18]. Although, other studies failed to show statistically significant differences in cardiac structural or functional alternations between dippers and non-dippers [19]. We did not find any associations between CBPP and DCM. Wyss et al. showed hypertension as cardiovascular risk factor, though not associated with severity of PAD in multivariate models [20]. However, Vasunta et al. proved that non-dipping status in hypertension was a risk factor for early atherosclerosis development (intima-media thickness assessment) [21]. In other studies, association between non-dipping pattern of hypertension and early stages of atherosclerosis was also proven and influence of study population age was also emphasized [22]. The fact that the patients who took α-adrenolytics were more likely to have lower fall in nocturnal BP could be explained by the fact that this class of drugs reduces nightly BP and restores dipping CBPP in non-dippers [23]. On the other hand, Pickering et al. showed that doxazosin lowered mostly morning BP and had relatively little influence on nightly BP [24]. According to the current guidelines of arterial hypertension management, β-blockers are recommended as antihypertensive drugs [25]. However, no studies comparing bedtime and morning administration of β-blockers have been conducted so far. These drugs are well known to influence daily BP, with minor influence on nightly BP [26]. There are only a few studies assessing influence of diuretics on dipping pattern. Uzu and Kimura showed that despite the fact that hydrochlorothiazide did not change CBPP in dippers, it changed non-dipping into dipping pattern in salt-sensitive hypertensive patients [27]. We did not find any associations between torasemide administration and CBPP. Furthermore, there is still little data comparing furosemide versus torasemide or other loop diuretics in the treatment of hypertension as well as there is little evidence upon which to base dosing of loop diuretics in the treatment of hypertension. We found no data analyzing different loop diuretics or different groups of diuretics in the context of hypertension dipping status [28].

Among laboratory, TTE and ECG parameters, HGB, CK-MB and HR_{max} seemed to have influence on nocturnal fall in SBP. We could not find any associations between HGB parameters and CBPP. Though lower HGB might be explained with both higher prevalence and severity of CKD among patients with lower decline in nocturnal SBP and higher prevalence of non-dipping pattern of hypertension among patients with CKD [29, 30]. In our study, there was higher prevalence of CKD among non-dippers and reverse-dippers compared with dippers. With the worsening kidney function we observed lower HGB concentration [31]. Non-dipping patients are described to have impairment in autonomic nervous drive (especially parasympathetic inactivity) [32]. No studies regarding either HR_{max} or CK-MB and CBPP were found.

As both, the strength and the limitation of the study we see a relatively big subgroup of reverse-dipper patients in our study population. It may be considered strength, since data regarding reverse-dippers are mostly lacking, and this group of patients is frequently omitted in other studies. Though, it may be seen also as limitation, because we had no data to compare our outcomes. Further limitation of our study could be the fact that we divided patients based only on one ABPM outcome. We also included relatively small group of patients in the study.

Conclusions

We found that detailed medical history could help us to predict not only nocturnal fall in SBP, but also patient's CBPP. These findings may have influence

on care of patients with hypertension, though further studies are needed.

Disclosure of interest

The authors report no conflicts of interest.

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