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Circadian blood pressure rhythm in normotensive offspring of hypertensive parents

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Abstract

Background: *The aim of this study was to explore the circadian blood pressure (BP) rhythm using ambulatory BP monitoring (ABPM) in normotensive children with a family history of essential hypertension.*

Methods: *Group 1 consisted of children with hypertensive mothers and/or fathers (n = 20), Group 2 consisted of children with hypertensive grandparents (n = 20), and Group 3 consisted of children with normotensive parents (n = 20). All participating children underwent a 24-h ABPM and echocardiography.*

Results: *Significantly higher systolic burden was found in children with hypertensive parents ($p < 0.05$) and grandparents ($p < 0.05$) compared to controls. Ambulatory BP measurements had a higher daytime systolic BP in Group 1 compared to controls ($p < 0.05$). While left ventricular (LV) posterior wall thickness was similar in Group 1 and Group 2, it was significantly higher in both of these groups compared to the controls. The LV mass index (LVMI) was significantly higher in Group 1 than in controls ($p < 0.05$). However, diastolic BP was significantly higher in dippers compared to non-dippers ($p < 0.05$). LV posterior wall thickness, interventricular septum thickness and LVMI were significantly higher among non-dippers compared to dippers ($p < 0.05$). In children with a family history of hypertension, a positive correlation between nocturnal systolic BP and LVMI was found, and increasing nocturnal BP values were associated with increasing LVMI ($p < 0.01$).*

Conclusions: *In children with a family history of hypertension, target-organ damage may precede the clinical detection of hypertension, and in those with a nocturnal non-dipper status, a more marked effect on LVMI may occur.* (Cardiol J 2015; 22, 2: 172–178)

Key words: blood pressure, offspring, hypertensive parents

Introduction

Essential hypertension represents the most common cause of hypertension in adults. Conversely, hypertension in pediatric populations is generally due to a detectable cause and is mostly associated with renal conditions [1]. Family history is considered to represent an important risk factor for essential hypertension in children. Compared to the offspring of normotensive individuals, children of hypertensive parents have been reported to have higher blood pressure (BP) values [2, 3].

Ambulatory BP measurements that monitor the changes in BP during a 24–48 h period are particularly useful for the diagnosis of hypertension. Advances in ambulatory BP monitoring (ABPM) techniques that permitted a non-invasive follow-up of the circadian rhythm of BP made possible studies that have evaluated nocturnal BP values both in normotensive and hypertensive individuals [4].

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Studies in normotensive individuals have demonstrated a reduction in BP values during the night. Despite individual variability in the extent of the nocturnal reduction in BP, percent reduction has been shown to be between 10% and 20% in the majority of the populations studied. The terms "dippers", "extreme dippers", and "non-dippers" are used to define those persons with a nocturnal reduction of BP between 10% and 20%, a reduction of BP greater than 20%, and < 10%, respectively. Those whose BP is higher during the night than during the day are referred to as "reverse dippers" [5]. Studies involving hypertensive patients have shown significantly increased end-organ damage in "non-dippers". Additionally, prospective studies have detected the presence of independent risk factors for cardiovascular disease in "non-dippers" and/or reverse dippers [6]. It has been previously reported that "non-dipper" status is associated with increased left ventricular (LV) mass (LVM) in adult populations [7] and with a higher risk of LV hypertrophy compared to "dippers" among normotensive populations [8].

Few studies exist that have examined the association between ambulatory BP and target organ damage in hypertensive children or in normotensive children with risk factors for hypertension. Therefore, in this study our objective was to explore the circadian BP rhythm using ABPM in normotensive children with a family history of essential hypertension; to assess the association between this rhythm and target organ damage; and to determine its predictive role for the development of hypertension in adulthood.

Methods

A total of 40 normotensive children with hypertensive parents who were followed in the Cardiology Department with a diagnosis of essential hypertension and 20 normotensive healthy children with normotensive parents seen in the Department of Pediatrics were included the study. Parents of the participants were provided information on the nature and content of the study, and informed written consent was obtained. The study protocol was approved by the Ethics Committee, Faculty of Medicine, Eskisehir Osmangazi University on 16 December 2009 (No: 2009/14).

A detailed medical and family history was obtained from the children. Those with a history of chronic disease, anemia, or other conditions requiring medical treatment were excluded. A physical examination was performed and patients with normal findings were included.

A total of three groups were defined as follows: children with hypertensive mothers and/or fathers (Group 1, n = 20); children with hypertensive grandparents (Group 2, n = 20); and children with normotensive parents and grandparents (control group; n = 20). For each child, BP measurements were performed 3 times using appropriately sized cuffs with a mercury sphygmomanometer after 10 min of rest. The cuff was inflated 20 mm Hg above the level of the abolished brachial pulse, and then deflated at a rate of 2–3 mm Hg/s while Korotkoff sounds were recorded. Korotkoff phase 1 was accepted as the systolic BP (SBP), Korotkoff phase 5 was recorded as the diastolic BP (DBP).

Ambulatory BP measurements:

All participating children underwent a 24-h ABPM monitoring in the Department of Pediatric Cardiology using a *Scanlight* II ABPMS system. The system used for ABPM consisted of an appropriately sized cuff, a battery charged recording unit, and software for data analysis. It uses an oscillometric method to detect a BP range of 60–290 mm Hg and 45–180 mm Hg for the systolic and diastolic measurements, respectively. The reported standard error margin for the system is ± 3 mm Hg. The dimensions of the recording unit are 13 × 8 × × 2.8 cm with a weight of 220 g. The measurements were performed using appropriately sized standard cuffs on the non-dominant arm. Subjects were asked to continue their usual activities, with the arm in a comfortable position during the measurements. Parents also were instructed to record the sleeping and waking hours. BP measurements were performed automatically every 20 min and 30 min. Subjects with a less than a 10% BP drop during the night were categorized as non-dippers, those with a drop between 10% and 20% were categorized as dippers. Data obtained through ABPM were analyzed in a digital environment. Children were grouped as dippers and non-dippers. For each child, the following values were recorded: the average 24-h SBP and DBP; the nocturnal average SBP and DBP; systolic, diastolic and average BP burden; and the circadian rhythm (dipper vs. non-dipper). The BP burden was defined as the ratio between the number of higher BP value and the total number of measurements. A BP burden of 25% indicates the presence of hypertension. The difference between the SBP and DBP is referred to as the "pulse pressure". The average pulse pressure is defined as the average of the total number of pulse pressure values measured during a certain duration of time.

Echocardiographic assessment

All participating children underwent a 2-dimensional Doppler echocardiography by the same experienced physician in the Department of Pediatric Cardiology using a Hewlett Packard Sonos (Model 5500) and 2–4 MHz and 4–8 MHz broadband probes. LV diastolic diameter (LVDD), LV posterior wall thickness (LVPWT), interventricular septum (IVS) thickness, the LVM, and LVM index (LVMI) were calculated: $\mathbf{LVM = 0.8 (1.04 \{ (LVDD + IVS + PW)^3 - (LVDD)^3 \}) + + 0.6}$, $\mathbf{LVMI = LVM / BSA}$ [9]. The body surface area (BSA) was calculated with the following formula: $\mathbf{BSA = (body\ weight^{0.425} \times height^{0.725}) \times 0.007184}$.

Statistical analyses

SPSS for Windows 15.0, NCSS 2007 package software was used for statistical analyses. The probability of fit to normal distribution was tested using the Shapiro-Wilk test. For parameters with a normal distribution, the between-group comparisons were performed using the t-test, and the multiple comparisons were performed using ANOVA. Based on the results of ANOVA, the post-hoc tests (Tukey or Tamhane tests) were used to detect the difference between the groups. For parameters without a normal distribution, the Mann-Whitney U test was used for pairwise group comparisons, while multiple comparisons were performed by the Kruskal-Wallis test. The cross-table analysis was based on the χ^2 test. The power and direction of the correlation between variables was tested using the Pearson correlation analysis. NCSS package software was used to obtain age-adjusted LVMI. The LVMI was accepted as a dependent variable, while ambulatory BP parameters were accepted as independent variables. The association between dependent and independent variables was tested using Ridge regression analysis. Data are shown as mean ± standard deviation, and for all tests a $p < 0.05$ was considered significant.

Results

A total of 60 healthy normotensive girls (n = 29) and boys (n = 31) between 8 and 22 years of age were included in the study. The mean age for the girls and boys was 14 ± 3.5 years and 15 ± 4.5 years, respectively. A total of 40 normotensive children with a family history of hypertension and 20 children with no family history of hypertension were studied in three groups. Group 1 consisted of children with hypertensive mothers and/or fathers (n = 20); Group 2 consisted of children with hypertensive grandparents (n = 20); and Group 3 consisted of children with normotensive parents (Control Group; n = 20).

The age, gender, body mass index (BMI), 24-h average SBP and DBP, nocturnal and daytime SBP and DBP, average pulse pressure, and systolic and diastolic burden are shown in Table 1. Children with a family history of hypertension did not differ significantly from the children in the Control Group in terms of age, gender, BMI, 24-h average SBP and DBP, nocturnal SBP, nocturnal and daytime SBP, diastolic burden, and the percentage of dippers or non-dippers ($p > 0.05$). However, significantly higher systolic burden was found in children with hypertensive parents ($p < 0.05$) and in children with hypertensive grandparents ($p < 0.05$) compared to controls. Additionally, ambulatory BP measurements showed higher daytime SBP in Group 1 compared to controls ($p < 0.05$).

A comparison of echocardiographic variables between study groups and control subjects is depicted in Table 1. The three groups were similar in terms of LVDD and IVS thickness ($p > 0.05$). While LVPWT was similar in Group 1 and Group 2 ($p > 0.05$), it was significantly higher than the Control Group ($p < 0.05$; $p < 0.05$). The LVMI was significantly higher in Group 1 than in controls ($p < 0.05$). On the other hand, in Group 2 had a higher LVMI compared to controls, though this difference was not significant ($p > 0.05$).

Normotensive children with a family history of hypertension were categorized into two groups according to dippers (n = 19) and non-dippers (n = 21). The dippers and non-dippers did not differ significantly with regard to age, gender, BMI, and SBP ($p > 0.05$) (Table 2). However, DBP was significantly higher in non-dippers than in dippers ($p < 0.05$, Table 2). Increased LVDD, isovolumetric contraction time, and ejection time were not different in non-dippers vs. dippers ($p > 0.05$, Table 2). LVPWT, IVS thickness and LVMI were significantly higher among non-dippers compared to dippers ($p < 0.05$, Table 2).

The children with hypertensive parents were categorized into three age groups: 8–14 years of age (n = 25); 15–19 years of age (n = 8); and 20–22 years of age (n = 7). Age groups were compared in terms of gender distribution, BMI, and ambulatory BP data (Table 3). There were significant differences between the age groups in terms of gender distribution, BMI, average DBP, daytime SBP, average pulse pressure, systolic burden, and diastolic burden ($p > 0.05$). A nocturnal non-dipper status

Table 1. The comparison of study and control groups according to anthropometric measurement, echocardiography and ambulatory blood pressure variables.

	Group 1 (n = 20)	Group 2 (n = 20)	Group 3 (n = 20)	P
Age [year]	15 ± 6	15 ± 6	15 ± 4	p1, p2, p3 > 0.05
Female/male	9/11	10/10	10/10	p1, p2, p3 > 0.05
BMI [kg/m^2]	19.5 ± 3	19 ± 3	19 ± 3	p1, p2, p3 > 0.05
IVSd	9 ± 2	8 ± 2	7 ± 2	p1, p2, p3 > 0.05
LVPW [mm]	7 ± 1	7 ± 2	5 ± 1	p1 > 0.05, **p2 < 0.05**, **p3 < 0.05**
LVEDd [mm]	44 ± 6	43 ± 4	42 ± 6	p1, p2, p3 > 0.05
LVED mass [g/m^2]	77 ± 15	71 ± 15	61 ± 13	**p2 < 0.05**
24-h average SBP	116 ± 7	110 ± 7	111±10	p1, p2, p3 > 0.05
24-h average DBP	64 ± 6	63 ± 5	62 ± 5	p1, p2, p3 > 0.05
Daytime SBP	121 ± 8	114 ± 8	116 ± 10	p1 > 0.05, **p2 < 0.05**, p3 > 0.05
Daytime DBP	68 ± 6	66 ± 6	66 ± 6	p1, p2, p3 > 0.05
Nocturnal SBP	106 ± 8	102 ± 7	102 ± 9	p1, p2, p3 > 0.05
Nocturnal DBP	57 ± 7	56 ± 4	56 ± 6	p1, p2, p3 > 0.05
Systolic burden [%]	10 ± 11	9 ± 8	5 ± 6	p1 > 0.05, **p2 < 0.05**, **p3 < 0.05**
Diastolic burden [%]	2 ± 3	2 ± 3	1 ± 2	p1, p2, p3 > 0.05
Pulse pressure	52 ± 5	48 ± 5	49 ± 7	p1, p2, p3 > 0.05
Dipper	9 (45%)	10 (50%)	12 (60%)	p1, p2, p3 > 0.05
Non-dipper	11 (55%)	10 (50%)	8 (40%)	p1, p2, p3 > 0.05

BMI — body mass index; IVSd — diastolic interventricular septum diameter; LVPW — left ventricular posterior wall diastolic diameter; LVEDd — left ventricular end-diastolic diameter; SBP — systolic blood pressure; DBP — diastolic blood pressure

p1 — comparison between group 1 and group 2; p2 — comparison between group 1 and group 3; p3 —comparison between group 2 and group 3

Table 2. The comparison of age groups according to anthropometric measurement, echocardiography and ambulatory blood pressure variables.

	8–14 years (n = 25)	15–19 years (n = 8)	20–22 years (n = 7)	P
Female/male	12/13	5/3	2/5	p1, p2, p3 > 0.05
BMI [kg/m^2]	18 ± 3	20 ± 3	21 ± 3	p1, p2, p3 > 0.05
IVSd	7.7 ± 1.4	8.1 ± 1.6	9.9 ± 2.9	p1 > 0,05, **p2 < 0.05, p3 < 0.05**
LVPW [mm]	6.5 ± 0.8	6.9 ± 1.5	8.6 ± 1.4	p1 > 0.05, **p2 < 0.05, p3 < 0.05**
LVEDd [mm]	42 ± 4	45 ± 6	46 ± 6	p1, p2, p3 > 0.05
LVED mass [g/m^2]	66 ± 15	73 ±12	88 ± 17	p1 > 0.05, **p2 < 0.05**, **p3 < 0.05**
24-h average SBP	112 ± 8	113 ± 10	113 ± 5	p1, p2, p3 > 0.05
24-h average DBP	62 ± 5	65 ± 5	65 ± 7	p1, p2, p3 > 0.05
Daytime SBP	117 ± 9	117 ± 6	121 ± 12	p1, p2, p3 > 0.05
Daytime DBP	65 ± 5	69 ± 6	71 ± 7	p1 > 0.05, **p2 < 0.05,** p3 > 0.05
Nocturnal SBP	104 ± 8	100 ± 9	108 ± 5	p1 > 0.05, **p2 < 0.05,** p3 > 0.05
Nocturnal DBP	56 ± 5	55 ± 8	58 ± 4	p1 > 0.05, **p2 < 0.05,** p3 > 0.05
Systolic burden [%]	7 ± 7	6 ± 9	7 ± 9	p1, p2, p3 > 0.05
Diastolic burden [%]	1.2 ± 2.3	0.8 ± 1.4	2.9 ± 3.1	p1, p2, p3 > 0.05
Pulse pressure	48 ± 5	49 ± 6	51 ± 6	p1, p2, p3 > 0.05
Dipper	14 (56%)	4 (50%)	1 (14%)	p1 > 0.05, **p2 < 0.05**, **p3 < 0.05**
Non-dipper	11 (44%)	4 (50%)	6 (86%)	p1 > 0.05, **p2 < 0.05**, **p3 < 0.05**

Abbreviations as in Table 1

p1 — comparison between group 1 and group 2; p2 — comparison between group 1 and group 3; p3 — comparison between group 2 and group 3

Table 3. The comparison of dippers and non-dippers according to anthropometric measurement, echocardiography and ambulatory blood pressure variables.

	Dipper (n = 19)	Non-dipper (n = 21)	P
Age [year]	14 ± 4	15 ± 4	
Female/male	12/7	10/11	
BMI [kg/m^2]	20 ± 3	19 ± 3	
IVSd	7.3 ± 1.3	8.9 ± 2	**< 0.05**
LVPW [mm]	6.5 ± 0.9	7.7 ± 1.7	**< 0.05**
LVEDd [mm]	43 ± 5	44 ± 5	
LVED mass [g/m^2]	84 ± 11	62 ± 8	**< 0.001**
24-h average SBP	113 ± 8	112 ± 7	
24-h average DBP	62 ± 4	64 ± 6	**< 0.05**
Daytime SBP	116 ± 8	120 ± 9	
Daytime DBP	65 ± 5	69 ± 7	**< 0.05**
Nocturnal SBP	101 ± 7	107 ± 7	**< 0.05**
Nocturnal DBP	56 ± 7	57 ± 4	
Systolic burden [%]	5 ± 7	7 ± 7	
Diastolic burden [%]	1.1 ± 1.95	1.8 ± 2.7	
Pulse pressure	49 ± 5	51 ± 6	

Abbreviations as in Table 1

was significantly more common in those between 20 and 22 years of age compared to other age groups ($p < 0.05$). Additionally, higher daytime BP and nocturnal SBP and DBP values were recorded in subjects between 20 and 22 years of age ($p < 0.05$). Despite increased LVDD in the 20–22 year age group, the difference was not statistically significant ($p > 0.05$). LVPWT, IVS thickness, and LVMI were increased in subjects between 20 and 22 years of age, and the difference was significant compared to those between 15 and 19 years of age ($p < 0.05$).

A correlation analysis was performed to identify the factors that may be associated with an increased LVMI. In children with a family history of hypertension, a positive correlation between nocturnal SBP and LVMI was found, and increasing nocturnal BP values were associated with increasing LVMI ($p < 0.01$). In Ridge regression analysis, the age-adjusted LVMI was found to change with nocturnal SBP ($p < 0.001$), daytime SBP ($p < 0.01$) and nocturnal DBP ($p = 0.029$).

Discussion

Compared to offspring of normotensive individuals, children of hypertensive parents have been shown to have higher BP values [2, 3]. In the study by Robinson et al. [10], the reported incidence of a positive family history among children with essential hypertension was 51%, while in the study by Flynn it amounted to 86.2% [1].

Despite being in the normal SBP and DBP ranges, the BP in children with a family history of hypertension has been found to be higher compared to controls, though the difference was not significant ($p > 0.05$). Similarly, previous studies have shown significantly higher BP values among normotensive young adults [11, 12] or children [13, 14] with a positive family history of hypertension compared to controls, leading to the conclusion that family history of hypertension represents an important risk factor. The absence of a significant increase in BP in children with a family history of hypertension compared to controls in our study may be due to small sample size.

In our study, daytime SBP and systolic burden were significantly higher in children with a family history of hypertension compared to controls, while there were no significant differences in daytime DBP, nocturnal SBP and DBP, average SBP and DBP, and average pulse pressure between the groups. In the study by Ravogli et al. [12], involving young adults, and in the study by Alpay et al. [15], involving children with a positive family history of hypertension, nocturnal and average SBP values were significantly higher in these populations compared to those with a negative family history for hypertension. In these studies, the authors emphasized the fact that in healthy children with a positive family history of hypertension, ABPM allows detection of early changes. Consistent with the literature, in our study normotensive children

with hypertensive parents had a higher systolic burden and daytime SBP. Further, a family history emerged as an important risk factor for the development of hypertension, and ABPM was able to detect early changes in BP.

Additionally, echocardiographic findings were compared between children with a family history of hypertension and controls. This comparison showed statistically significant increases in the LVPWT and LVMI among children with a family history of hypertension. In line with the previous observations [12, 16, 17], children with hypertensive parents had increased LVPWT and LVMI that preceded the development of hypertension, suggesting that a family history of hypertension was an important risk factor for target-organ damage.

In essential hypertension, a non-dipper status for nocturnal BP has been linked to target-organ damage [18]. In our study, patients with and without a family history of hypertension did not differ significantly in terms of the percentage of dippers and non-dippers ($p > 0.05$). Despite previous studies examining the nocturnal reduction in ambulatory BP in healthy adults and in subjects with hypertension, chronic renal failure, renal transplantation, or pheochromocytoma [19–21], our literature search did not reveal any studies examining the percentage of dippers and non-dippers among normotensive children with a family history of hypertension.

In our study, children with a family history of hypertension were categorized into two following groups based on the nocturnal reduction in BP as evidenced by ambulatory BP monitoring: "dippers" and "non-dippers". Prospective studies previously suggested that a nocturnal "non-dipper" status or elevated BP during night hours could be independent risk factors for cardiovascular disease [18]. In our study, non-dippers had significantly higher LVPWT, IVS thickness and LVMI. In studies involving hypertensive [8, 22] or normotensive [7] adults, nocturnal non-dipper status was associated with a higher LVMI suggesting that non-dipper status was an independent risk factor for cardiovascular disease.

In our study, non-dippers had higher 24-h diastolic, daytime diastolic, and nocturnal SBP compared to dippers. In addition, average systolic, daytime systolic and nocturnal DBP were higher in non-dippers vs. dippers, although the differences were not significant. Sorof et al. [21], in their study involving hypertensive children, observed higher daytime, nighttime, and average SBP values in those with target-organ damage compared to those without it. In a study by Fagart et al. [23] of adult hypertensives, a positive correlation was found between average SBP, daytime average SBP, and LVMI. Findings regarding the association between LVMI and daytime and nighttime BP have been inconsistent. Several studies have shown a stronger association between LVMI and ambulatory SBP compared to DBP [24]. In our study, the nocturnal SBP in children with a family history of hypertension had a significant positive correlation with LVMI, and increasing nocturnal BP values were associated with increasing LVMI ($p > 0.05$). In the study by Balcı et al. [25], 24-h, daytime and nocturnal SBP had a stronger correlation with LVMI compared to diastolic measurements involving the same periods. Additionally, these authors have reported that maximum daytime SBP was an independent risk factor and the strongest predictor for the LVMI. In the study by Soylu et al. [7], where normotensive adults were involved, a positive correlation between LVMI and average systolic and diastolic, and nocturnal SBP was reported. Sorof et al. [21] performed ABPM in a total of 37 newly diagnosed, treatment-naive hypertensive children, and found higher average systolic, daytime systolic, and nocturnal SBP among those with increased LVMI. Richey et al. [26] found a significant correlation between SBP burden and LVMI in a total of 106 normotensive children between 6 and 18 years of age who had risk factors for hypertension. In our subjects with a family history of hypertension, an increase in LVMI was observed in association with increases in nocturnal SBP, suggesting that the absence of a nocturnal drop in BP could be particularly important in terms of target-organ damage. Additionally, a stronger correlation between ambulatory SBP and LVMI was found compared to DBP. These findings are in line with previously reported data.

In our study, ABPM revealed a particular increase in daytime diastolic, nocturnal systolic, and nocturnal DBP in subjects between 20 and 22 years of age. Additionally, the increase in BP continued into night hours. A significant increase in LVPWT and IVS thickness was observed in patients between 20 and 22 years of age. A significantly higher percentage of nocturnal non-dippers in subjects between 20 and 22 years of age suggests that target-organ damage associated with high BP may start as early as 20 years of age.

Conclusions

In conclusion, in children with a family history of hypertension, target-organ damage may precede the clinical detection of hypertension,

and in those with a nocturnal non-dipper status, a more marked effect on LVMI may occur. In normotensive children of hypertensive parents, a nocturnal non-dipper status may be observed after the second decade of life, and an effect on LVMI can be detected.

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Conflict of interest: None declared

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