

ORIGINAL ARTICLE

Assessment of left ventricular dyssynchrony in dipper and non-dipper hypertension

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Abstract

Background. Ventricular dyssynchrony is an co-determinant of progression and exacerbation of heart failure (HF). The co-existence of ventricular dyssynchrony with hypertension (HT) and HF were shown, however there is no data regarding the effect of circadian rhythm of blood pressure (BP) on ventricular synchrony. Therefore, we aimed to study the left ventricular synchrony in dipper and non-dipper normotensive and hypertensive participants. **Methods.** Participants ($n = 142$) were categorized into four groups as “Normotensive-Dipper” (NT-D) ($n = 40$), “Normotensive-Non-dipper” (NT-ND) ($n = 30$), “Hypertensive-Dipper” (HT-D) ($n = 38$) and “Hypertensive-Non-dipper” (HT-ND) ($n = 34$). Left ventricular dyssynchrony was investigated by color-coded tissue Doppler imaging. **Results.** Non-dippers had higher 24-h and night-time BP both in normotensives and hypertensives. The incidence of ventricular dyssynchrony (a Ts-SD-12 > 34.4 ms) was higher in the hypertensive group (47.2% vs 24.3%, $p = 0.005$). The frequency of ventricular dyssynchrony was higher in the HT-ND group than the HT-D group (58.8% vs 36.8%, $p = 0.05$); however, the frequency of ventricular dyssynchrony was similar among the normotensives (26.7% vs 22.5%, $p = 0.45$). Ts-SD-12 and Ts-12 were higher in NT-ND group than the NT-D group. **Conclusions.** Non-dipping BP pattern was associated with impaired left ventricular contraction synchrony in both normotensive and hypertensive participants, which may be related with short- and long-term effects of HT on myocardium.

Key Words: Diastolic dysfunction, non-dipper hypertension, ventricular dyssynchrony

Introduction

Hypertension (HT) is one of the greatest risk factors for diastolic and later systolic dysfunction, associated with worse cardiovascular outcomes and may lead to subsequent heart failure (HF) through a variety of mechanisms, including increased afterload, left ventricular (LV) hypertrophy, myocardial fibrosis and impaired diastolic filling (1). Blood pressure (BP) levels show a circadian pattern in which both the systolic (SBP) and diastolic BP (DBP) decrease more than 10% in the night. Non-dipping HT is defined as lack of nocturnal fall in the circadian BP pattern, which was also shown to be associated with higher risk of cardiovascular, renal and cerebrovascular diseases (2–4).

Ventricular contraction dyssynchrony is defined as the loss of contraction coordination within or

between the ventricles (5). Dyssynchrony has been first emphasized in systolic HF patients with wide QRS and low ejection fraction (EF) for the selection of candidates of cardiac resynchronization therapy (6,7). However, recent studies have shown that it was associated with systolic dysfunction, diastolic dysfunction with preserved EF, diabetes mellitus (DM), acute myocardial infarction (AMI), and hypo- and hyperthyroidism (5,8–13). Dyssynchrony was also found to be associated with newly diagnosed HT (14), however there is no data about changes of contraction synchrony according to the circadian rhythm of the BP. Therefore, we aimed to study the LV contraction synchrony in dipper and non-dipper normotensive and newly diagnosed hypertensive participants.

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Materials and methods

A total of 142 participants who admitted to cardiology outpatient clinic were taken in this study. Seventy-two newly diagnosed hypertensive participants according to the Joint National Committee (JNC) 7 criteria were evaluated by ambulatory BP monitoring (ABPM) (Suntech Medical Inc., Morrisville, NC, USA) for the dipping and non-dipping pattern and 70 age- and sex- matched outpatient participants without HT were taken as the control group. The patients were categorized into four, grouped as "Normotensive-Dipper" (NT-D) group, "Normotensive-Non-dipper" (NT-ND) group, "Hypertensive-Dipper" (HT-D) group and "Hypertensive-Non-dipper" (HT-ND) group. For the 24-h period, SBP, DBP and heart rate were recorded every 15 min during the daytime (07:00 to 23:00 h) and every 30 min during the night-time (23:00 to 07:00 h). As defined by Verdecchia et al. (15), a greater than 10% fall both in the nocturnal SBP and DBP than those of the daytime values was defined as dipping pattern. Subjects with less than 10% decrease in either SBP or DBP was regarded as non-dipper HT.

Patients with known coronary artery disease, secondary HT, renal failure, hepatic failure, chronic obstructive lung disease and/or manifest heart disease, such as cardiac failure (LV ejection fraction, LVEF, <50%), atrial fibrillation, and moderate to severe cardiac valve disease, a QRS duration greater than 120 ms were excluded from the study. All the participants were informed about the study, and their oral and written consents were taken.

After a detailed clinical history, comprehensive physical examination was performed to all participants. By dividing weight in kilograms by height in meters squared (kg/m^2), the body mass index (BMI) was calculated. Complete blood count, serum glucose, urea, creatinine, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and triglycerides were measured from the venous blood samples taken in the morning after 8-h fasting. Our study was approved by the local ethics committee.

Echocardiographic measurements

All echocardiographic examinations were done by GE Vivid 7 system (GE Vingmed Ultrasound AS, Horten, Norway) with a 3.5-MHz transducer. All data were transferred to a workstation for further offline analysis (EchoPAC PC; GE Vingmed Ultrasound AS). M-mode measurements were performed according to recommendations of the American Society of Echocardiography. LVEF was calculated using the Simpson's formula from measurement of end-diastolic and end-systolic volumes on apical four-chamber views (16). Left atrium (LA) posterior-anterior dimension was

measured from the parasternal long axis (LAX) view. Mitral flow velocity was recorded in the apical four-chamber view, by placing the sample volume of pulsed-wave Doppler between the tips of the mitral leaflets. Then, peak A and E waves, and E deceleration time (Dt) were measured. LV mass (LVM) and LV mass index (LVMI) were calculated using the formula (17):

$$\text{LVM (g)} = 0.8 \left(1.04 \left[\text{septum} + \text{posterior wall} + \text{LV internal diastolic dimension} \right]^3 - [\text{LV diastolic dimension}]^3 \right) + 0.6.$$

Diastolic dysfunction was defined as Grade 1, Grade 2 or Grade 3 dysfunction according to the E/A ratio. Grade 1 diastolic dysfunction was defined as an E/A ratio <1.0 and/or a Dt >240 ms. Grade 2 diastolic dysfunction was defined as an E/A ratio of >1.0 and an E/Em ratio of >10 (18). Grade 3 diastolic dysfunction was defined as an E/A >2, Dt <140 ms and E/Em >10 (19). All images were evaluated by two experienced cardiologists who were blinded to the patients' clinical characteristics.

Tissue Doppler imaging (TDI)

TDI indices were taken by using color-coded-TDI from the four-chamber apical view. The sample volume was placed at the septal mitral annulus and lateral mitral annulus. Peak systolic mitral annular velocity (Sm), early diastolic mitral annular velocity (Em) and late diastolic mitral annular velocity (Am) were recorded (Figure 1). The high pass filter was bypassed and gains were minimized to acquire optimal tissue velocities. The Nyquist limit was adjusted to a velocity range of -24 to 24 cm/s and monitor sweep speed was increased to 50-100 mm/s for the optimum spectral display of myocardial velocities. Temporal resolution was prioritized by using >250 frames/s to achieve high sensitivity during the analysis. Three consecutive cycles were recorded. The time from QRS onset to peak systolic velocity (Ts) was recorded in six segments at the cardiac base and six at the midventricular level (anterior, anterior septum, septum, inferior, lateral, posterior) (Figure 1). Parameters for systolic dyssynchrony included:

- standard deviation of Ts of the 12 LV segments (Ts-SD-12);
- standard deviation of Ts of the six basal LV segments (Ts-SD-6);
- maximal difference in Ts between any two of the 12 LV segments (Ts-12);
- maximal difference in Ts between any two of the six basal LV segments (Ts-6).

A Ts-SD-12 value greater than 34.4 ms was accepted as LV systolic dyssynchrony, as Yu et al. defined (20). The intra- and interobserver variability for Ts-12-SD were found to be 5% and 7%, respectively.

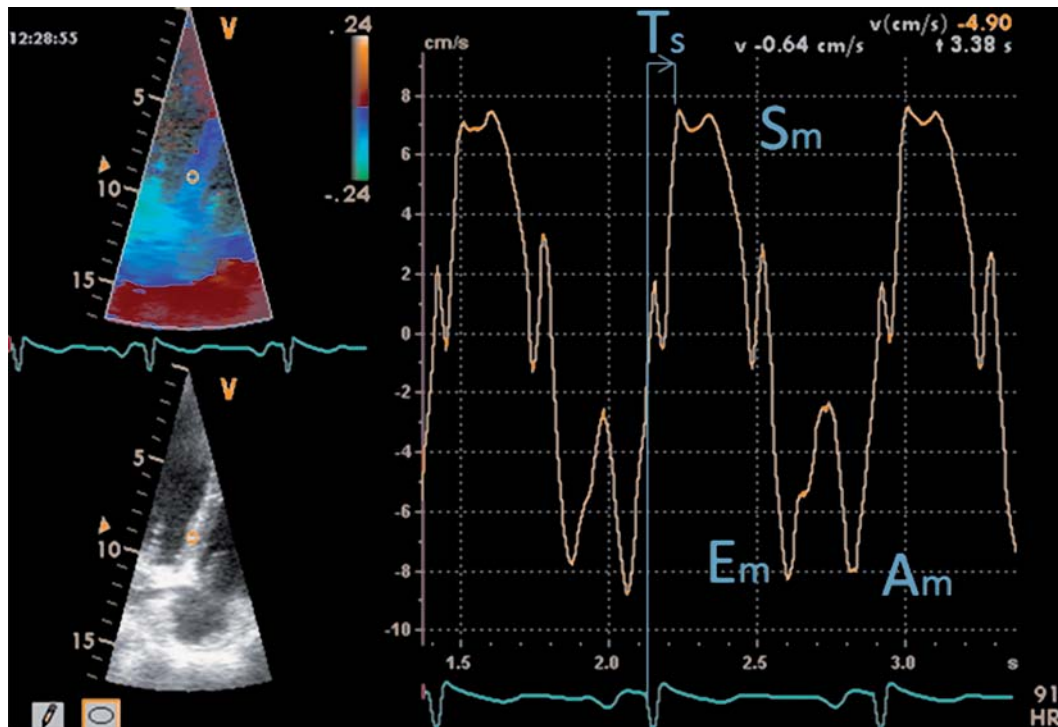


Figure 1. Echocardiographic analysis of tissue velocities using color-coded tissue Doppler.

Statistical analyses

Statistical analyses were performed using SPSS software package program (SPSS Inc., Chicago, IL, USA), version 17.0 for Windows. Continuous variables were expressed as mean $\pm$ SD and categorical variables were expressed as percentages. After employing normality tests, to compare continuous variables, the Mann-Whitney *U* or Kruskal-Wallis tests were used. For categorical variables, χ^2 test was used. Correlation between variables were tested by Spearman correlation tests where appropriate. A *p*-value less than 0.05 was accepted as significant. For the inter- and intra-observer variability for dyssynchrony parameters, a Bland-Altman analysis was performed.

Results

One hundred and forty-two participants (41.6 ± 3.0 years, 64.2% male) were included in the study. Baseline clinical and laboratory characteristics of the participants according to the dipping and non-dipping status were shown in Table I. Age, sex, BMI were no different between the groups. In the normotensive group, dippers and non-dippers had similar daytime SBP and DBP (113.3 ± 5.9 mmHg vs 113.2 ± 8.5 mmHg, *p* = 0.71; 72.6 ± 5.5 mmHg vs 74.4 ± 8.0 mmHg, *p* = 0.38), however non-dippers have significantly higher night-time SBP and DBP (109.3 ± 8.0 mmHg vs 95.5 ± 7.9 mmHg, *p* < 0.001; 69.3 ± 7.9 mmHg vs 56.2 ± 7.5 mmHg, *p* < 0.001) and 24-h mean SBP and DBP (119.9 ± 7.9 mmHg vs

107.5 ± 6.3 mmHg, *p* = 0.026; 72.7 ± 7.4 mmHg vs 67.2 ± 5.9 mmHg, *p* = 0.003). In the hypertensive patients, however, while daytime SBP and DBP (158.9 ± 11.3 mmHg vs 157.6 ± 10.5 mmHg, *p* = 0.74; 97.4 ± 6.8 mmHg vs 96.7 ± 5.9 mmHg, *p* = 0.61) were similar in the dippers and non-dippers, the hypertensive non-dippers had higher 24-h mean SBP and DBP (156.8 ± 11.3 mmHg vs 150.5 ± 10.1 mmHg, *p* = 0.013; 95.2 ± 6.2 mmHg vs 90.6 ± 5.1 mmHg, *p* = 0.001) and night-time SBP and DBP (152.7 ± 11.5 mmHg vs 136.3 ± 9.8 mmHg, *p* < 0.001; 90.1 ± 5.8 mmHg vs 78.5 ± 5.6 mmHg, *p* < 0.001) (Table II). Daytime and night-time heart rate values were similar in all groups.

The conventional echocardiography and dyssynchrony parameters of the participants were shown in Table III. In the normotensive group, there were no differences between conventional parameters. Hypertensive patients had higher LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), interventricular septum (IVS) thickness, posterior wall (PW) thickness, LVMI compared with the normotensive participants (48.5 ± 4.5 mm vs 45.9 ± 3.4 mmHg, *p* < 0.001 for LVEDD; 28.6 ± 3.9 mmHg vs 26.5 ± 3.2 mmHg, *p* < 0.001 for LVESD; 9.8 ± 1.2 mmHg vs 8.5 ± 0.9 mmHg, *p* < 0.001 for IVS; 9.5 ± 1.0 mmHg vs 8.5 ± 0.9 mmHg, *p* < 0.001 for PW; 90.0 ± 20.8 g/m² vs 70.2 ± 11.2 g/m², *p* < 0.001 for LVMI). LVEF values were no different between the groups. In the hypertensive group, 28 patients of HT-ND group and 29 patients of HT-D group has diastolic dysfunction. However, there was no significant difference between HT-D and HT-ND groups

Table I. Baseline clinical and laboratory characteristics of patient groups.

	Normotensive-Dipper	Normotensive-Non-dipper	Hypertensive-Dipper	Hypertensive-Non-dipper	
Variables	(n = 40)	(n = 30)	(n = 38)	(n = 34)	p
Age (years)	41.6 ± 3.8	41.7 ± 2.5	40.8 ± 3.0	42.3 ± 3.1	0.93
Sex, male (%)	26 (65%)	18 (60%)	21 (55.5%)	26 (76.5%)	0.27
BMI (kg/m ²)	25.7 ± 2.5	25.4 ± 4.7	28.3 ± 6.1	25.6 ± 5.2	0.068
Smoking (%)	17 (42.5%)	11 (36.7%)	21 (55.3%)	17 (50%)	0.46
Glucose (mg/dL)	85.2 ± 11.6	86.6 ± 22.0	84.4 ± 16.1	89.2 ± 15.1	0.31
HDL-C (mg/dL)	31.9 ± 7.9	34.5 ± 9.7	36.6 ± 8.5	34.8 ± 9.5	0.12
LDL-C (mg/dL)	111.1 ± 31.4	125.2 ± 29.0	127.5 ± 37.0	129.4 ± 32.0	0.10
TG (mg/dL)	170.2 ± 87.9	159.9 ± 107.9	165.9 ± 58.1	153.4 ± 53.6	0.38
Creatinine (mg/dL)	0.92 ± 0.33	0.93 ± 0.25	0.91 ± 0.22	0.95 ± 0.23	0.91
TSH (mU/L)	1.69 ± 1.1	1.47 ± 1.3	1.29 ± 0.79	1.37 ± 0.84	0.62

Continuous data were expressed as mean ± standard deviation and categorical data were expressed as percentages. Comparison between groups was done by χ^2 or Kruskal–Wallis where appropriate. $p < 0.05$ was accepted as significant. BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TSH, thyroid stimulating hormone.

regarding diastolic dysfunction prevalence. HT-ND group had higher LVMI, IVS, E/Em, Dt values and lower E/A values compared with the HT-D group (Table III).

LV systolic dyssynchrony parameters including Ts-SD-12, Ts-12, Ts-SD-6, and Ts-6 were found to be higher in the hypertensive participants compared with controls (Table III). The participants with ventricular dyssynchrony (a Ts-SD-12 > 34.4 ms, as Yu et al. described) were higher in the hypertensive subjects than the control subjects (47.2% vs 24.3%, $p = 0.005$). The number of participants with ventricular dyssynchrony within the hypertensive participants were higher in the HT-ND group than the HT-D group (58.8% vs 36.8%, $p = 0.05$) however, the number of participants with ventricular dyssynchrony was similar among the normotensive participants (26.7% vs 22.5%, $p = 0.45$). On the other hand, Ts-SD-12 and Ts-12 were higher in NT-ND group than the NT-D group (Table III). In the correlation analysis, systolic dyssynchrony parameters were found to be correlated with LVMI, E/A, day-time SBP, day-time DBP, night-time SBP, night-time DBP, 24-h DBP and 24-h DBP, as shown in Table IV.

Discussion

In this particular study, we investigated the relationship between LV contraction synchrony and dipping and non-dipping patterns of BP both in normotensive and hypertensive participants. Our study results demonstrated that, non-dipping BP pattern was associated with impaired LV contraction synchrony in both normotensive and hypertensive participants, which may be related with short- and long-term effects of HT on myocardium.

HT is associated with worse cardiovascular outcomes, and may lead to systolic and diastolic HF by increased afterload, LV hypertrophy, myocardial fibrosis and impaired diastolic filling (1). Besides that, in patients with newly diagnosed HT, evidence of LV dyssynchrony was found. Ventricular dyssynchrony, defined as loss of contraction coordination within or between ventricles, was first emphasized in the early studies regarding the utility of cardiac resynchronization therapy devices in systolic HF patients with low EF and wide QRS complex (21). However, it is now known that LV dyssynchrony associates to many clinical conditions such as HT, DM, AMI, hypo- and hyperthyroidism, even in the

Table II. Ambulatory blood pressure monitoring results of dippers and non-dippers among individuals with normal and high 24-h blood pressure.

	Normotensive subjects			Hypertensive subjects		
	Dipper	Non-dipper	p	Dipper	Non-dipper	p
24-h systolic	107.5 ± 6.3	111.9 ± 7.9	0.026	150.5 ± 10.1	156.8 ± 11.3	0.013
24-h diastolic	67.2 ± 5.9	72.7 ± 7.4	0.003	90.6 ± 5.1	95.2 ± 6.2	0.001
Day-time systolic	113.3 ± 5.9	113.2 ± 8.5	0.71	157.6 ± 10.5	158.9 ± 11.3	0.74
Day-time diastolic	72.6 ± 5.5	74.4 ± 8.0	0.38	96.7 ± 5.9	97.4 ± 6.8	0.61
Night-time systolic	95.5 ± 7.9	109.3 ± 8.0	<0.001	136.3 ± 9.8	152.7 ± 11.5	<0.001
Night-time diastolic	56.2 ± 7.5	69.3 ± 7.9	<0.001	78.5 ± 5.6	90.1 ± 5.8	<0.001
24-h heart rate	71.4 ± 3.8	73.2 ± 4.4	0.32	66.8 ± 5.5	70.2 ± 4.2	0.66

Data are expressed as mean ± SD. Statistical significance was tested by Mann–Whitney *U* test for continuous variables. $p < 0.05$ was accepted as significant.

Table III. Comparison of conventional echocardiography and dyssynchrony parameters.

	Normotensive subjects			hypertensive subjects		
	Dipper	Non-dipper	<i>p</i>	Dipper	Non-dipper	<i>p</i>
LVEDD (mm)	46.0 ± 3.3	45.8 ± 3.6	0.88	47.3 ± 4.5	49.8 ± 4.2	0.21
LVEDS (mm)	26.7 ± 3.2	26.4 ± 3.3	0.65	29.3 ± 3.7	27.9 ± 4.1	0.34
IVS (mm)	8.8 ± 1.0	8.7 ± 0.8	0.37	9.4 ± 1.0	10.2 ± 0.1	0.01
PW (mm)	8.7 ± 1.0	8.3 ± 0.7	0.18	9.3 ± 0.9	9.8 ± 0.1	0.18
LVMI (g/m ²)	71.5 ± 11.5	68.3 ± 10.8	0.24	82.0 ± 15.2	99.1 ± 22.7	0.003
LVEF (%)	63.2 ± 2.1	62.5 ± 2.5	0.34	61.4 ± 4.1	60.3 ± 3.2	0.35
E/A	1.21 ± 0.2	1.23 ± 0.22	0.78	0.96 ± 0.2	0.84 ± 0.2	0.003
Dt (ms)	189 ± 25	180 ± 24	0.15	247 ± 29	265 ± 27	0.014
E/Em	7.5 ± 1.7	7.4 ± 1.7	0.86	10.9 ± 1.0	11.9 ± 1.4	0.037
Ts-6 (ms)	60.7 ± 25.1	71.6 ± 27.1	0.09	75.1 ± 26.5	93.4 ± 25.6	0.009
Ts-SD-6 (ms)	23.2 ± 9.1	26.9 ± 9.9	0.12	28.8 ± 9.6	36.1 ± 9.9	0.002
Ts-12 (ms)	75.5 ± 24.8	91.2 ± 19.1	0.017	93.6 ± 24.2	106.8 ± 17.0	0.01
Ts-SD-12 (ms)	24.8 ± 8.3	29.5 ± 6.3	0.012	30.3 ± 7.0	35.1 ± 6.5	0.009
Systolic dyssynchrony	9 (22.5%)	8 (26.7%)	0.45	14 (36.8%)	20 (58.8%)	0.05

Values were given as mean ± standard deviation or numbers (percentages). Comparison between groups were done by Mann-Whitney *U* test and χ^2 test where appropriate. *p* < 0.05 was considered significant. LV, left ventricle; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVEDS, left ventricular end-systolic diameter; LVMI, left ventricular mass index; PW, posterior wall; Ts, time to peak tissue velocity; SD, standard deviation; Ts-SD-6, standard deviation of Ts of the six LV segments; Ts-12, maximal difference in Ts between any two of the 12 LV segments; Ts-SD-6, standard deviation of Ts of the six basal LV segments; Ts-6, maximal difference in Ts between any two of the six basal LV segments.

patients with normal EF and narrow QRS (8–12). The prognostic significance of this coexistence for most of these conditions is not clear for the time being, however it is a known fact that systolic dyssynchrony is an important co-determinant of progression and exacerbation of HF and associated with adverse outcomes in patients with systolic HF (22,23).

There is a circadian pattern for BP levels in which both the SBP and DBP decrease more than 10% in the night. Non-dipping HT is defined as lack of nocturnal fall in the BP (2,24). This daily nocturnal BP fall is seen not only in hypertensive patients but also in normotensive people (24). The importance of dipping/non-dipping status was shown by several

studies that the non-dipping of BP means higher risk for cardiovascular diseases for both hypertensive and normotensive people (24–26).

The co-existence of ventricular dyssynchrony with HT and HF were shown, there is no data regarding the circadian rhythm of BP on ventricular contraction synchrony in normotensive and hypertensive subjects. In our study, we found that in the HT group, more patients with ventricular dyssynchrony were detected compared with the control group and in a substantial number of hypertensive patients, diastolic dysfunction accompanied. Moreover, the presence ventricular dyssynchrony was higher in the “non-dipper” hypertensives compared with the “dipper” hypertensives. One possible explanation for

Table IV. The correlation table of systolic dyssynchrony parameters with echocardiography and ambulatory blood pressure monitoring parameters.

	LVMI	E/A	E/Em	Day SBP	Day DBP	Night SBP	Night DBP	24-h SBP	24-h DBP
Ts-SD-12									
Rho	0.16	−0.22	0.06	0.28	0.37	0.37	0.39	0.33	0.39
<i>P</i>	0.054	0.008	0.75	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Ts-12									
Rho	0.16	−0.19	0.19	0.29	0.36	0.40	0.41	0.34	0.39
<i>P</i>	0.22	0.02	0.92	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Ts-SD-6									
Rho	0.19	−0.22	0.18	0.29	0.29	0.36	0.37	0.33	0.34
<i>P</i>	0.02	0.007	0.35	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Ts-6									
Rho	0.18	−0.23	0.15	0.28	0.31	0.36	0.31	0.32	0.34
<i>P</i>	0.035	0.007	0.44	0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Correlation analysis was done by Spearman Correlation Test. *p* < 0.05 was considered significant. Em, early diastolic mitral annular velocity; LVMI, left ventricle mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Ts, time to peak tissue velocity; SD, standard deviation; Ts-SD-6, standard deviation of Ts of the six LV segments; Ts-12, maximal difference in Ts between any two of the 12 LV segments; Ts-SD-6, standard deviation of Ts of the six basal LV segments; Ts-6, maximal difference in Ts between any two of the six basal LV segments.

the hypertensives having more ventricular dyssynchrony may be the impaired coordination of contraction resulting from increased LV mass. Tan et al. reported in a study that LV systolic synchronicity was impaired in hypertensive patients with LV hypertrophy (27). Our results have shown that there was a correlation between dyssynchrony parameters and LVMI, which supports the aforementioned explanation. Another possible mechanism may be the myocardial hypertrophy and fibrosis caused by HT. The hypertrophy and fibrosis may not affect the myocardium homogeneously, resulting in impairment of myocyte contraction and impulse propagation and therefore loss of contraction synchrony. In addition to that, increased filling pressures and LV mass caused by HT may lead to supply/demand mismatch in the myocardium, which may lead to ischemia. Ischemia may impair both systolic and diastolic functions, contributing to LV dyssynchrony. In our study, we found out that non-dipper hypertensives had more ventricular dyssynchrony compared with dippers. As we mentioned in the results, non-dippers have higher 24-h SBP and DBP which means higher filling pressures, higher LV mass and higher end organ damage. Higher BP may be reason for the impairment of contraction synchrony. It is also well established that HT has an inflammatory background (28). The inflammatory markers were found to be higher in non-dippers, reflecting a higher inflammatory status (29). Inflammation causes endothelial dysfunction, which may interfere with myocardial functions. More inflammation may mean more impairment in myocyte function, which may be the reason for the increased ventricular dyssynchrony in the non-dippers. In the normotensive group, although the number of participants with ventricular dyssynchrony was not significantly different between dipper and non-dipper groups, the dyssynchrony parameters were significantly higher in the non-dipper group, which may be related with higher inflammatory background of non-dipping status, as discussed above. With this study, the relationship between the dipping/non-dipping pattern of BP with ventricular dyssynchrony was exposed for the first time in the literature.

Limitations

A major limitation of the current study is the small sample size. It would be better to include more patients in order to increase the statistical power. Besides, the ventricular dyssynchrony was not assessed after the normalization of BP in hypertensives, which would provide valuable information. One point that should be taken into consideration that, the participants were categorized as dippers and non-dippers according to results of one day "24-h ABPM". Daily variation in 24-h ABPM results might have been underestimated since we had only 1 day

of 24-h ABPM results. One other limitation is that we did not include the patients with prehypertension. Because there is lack of data for long-term follow-up, there is no prognostic data in terms of future cardiovascular events and progression to HF, which is another limitation of the study.

Conclusion

In this study, we investigated the relationship between LV contraction synchrony and dipping and non-dipping patterns of BP both in normotensive and hypertensive participants. Our study results demonstrated that, non-dipping BP pattern was associated with impaired LV contraction synchrony in both normotensive and hypertensive participants which may be related with short- and long-term effects of HT on myocardium.

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Conflict of interest

None declared.

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