Effect of dipping and nondipping pattern of blood pressure on subclinical left ventricular dysfunction assessed by two-dimensional speckle tracking in hypertensive patients

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Objective **The aim of this study was to evaluate the left ventricular (LV) function by conventional two-dimensional speckle tracking echocardiography (2D STE) to detect subclinical LV systolic dysfunction in patients with dipper and nondipper hypertension.**

Methods **One hundred consecutive patients with hypertension were included in our study. Clinical evaluation, baseline laboratory investigations, 24 ambulatory blood pressure monitoring 2D echocardiographic examination and 2D STE were performed for all patients. Patients were classified as dippers and nondippers according to their nighttime MAP (mean arterial blood pressure) reduction rate of ≥10 or <10%, respectively.**

Results **Of 100 patients, 71% were nondippers while 29% were dippers. Nondippers had a significantly lower global longitudinal strain (LS) value (−22.45 ± 3.26 vs. −18.2 ± 3.3, $P < 0.001$), global circumferential strain (CS) value (−24.23 ± 3.56 vs. −19.16 ± 8.25, $P < 0.001$) and global radial strain (RS) value (35.04 ± 11.16 vs. 29.58 ± 8.44, $P = 0.009$). It was found that nondipper status was associated with worsening of LS by 2.737, ($P = 0.001$), CS by 3.446, ($P = 0.002$), RS by −3.256, ($P = 0.158$) and DM also was found associated with worsening of LS by 1.849, ($P = 0.062$), CS by 3.284 ($P = 0.018$), RS by −2.499 ($P = 0.381$).**

Conclusion **The nondipping hypertension pattern is associated with subclinical LV systolic dysfunction as shown by the impaired global myocardial strain in all three directions.** ***Blood Press Monit*** **XXX: 000–000**

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Keywords: dipping pattern, hypertension, left ventricular function, myocardial strain, nondipping pattern, speckle tracking echocardiography, two-dimensional echocardiography

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Introduction

Hypertension is reportedly associated with negative cardiovascular events and mortality [1]. Thus, it is important to identify outpatients who are at high risk for hypertension before adverse events occur.

Ambulatory blood pressure monitoring (ABPM) is currently recommended by several guidelines for both the diagnosis and follow-up of hypertensive patients [2,3]. Independent of the degree of hypertension, a nondipping pattern of blood pressure (BP) is a risk factor for the development of left ventricular hypertrophy (LVH), heart failure and other cardiovascular complications [4–6].

The early detection of left ventricular (LV) dysfunction before the development of left ventricular structural changes, such as LVH, may represent a clinical finding that would justify aggressive management to reduce cardiovascular morbidity and mortality [7].

Speckle tracking echocardiography (STE) is now being used with increasing frequency to detect subclinical LV systolic dysfunction that can be assessed by quantifying the myocardial strain [8].

This study aimed to evaluate LV systolic function by STE in hypertensive patients with dipper and nondipper patterns.

Patient and methods

Study protocol

This study complied with the Declaration of Helsinki and was approved by the Ethics Committee of the Sultan Qaboos Hospital (Salalah, Oman). Informed consent was taken from all participants included in the study.

One hundred and thirty-three consecutive patients with essential hypertension were recruited from an outpatient clinic from July 2019 to July 2020. After the exclusion of

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patients through the exclusion criteria, 100 patients were included in our study. Hypertension was defined as systolic blood pressure (SBP) of >140 mmHg and/or diastolic blood pressure (DBP) of >90 mmHg, which is equivalent to a 24-h ambulatory mean BP of ≥130/80 mmHg [2], or if the subject was prescribed antihypertensive medications. All patients with a previous history of cardiovascular disease (including myocardial infarction, coronary artery disease, congenital heart disease, valvular disease, atrial fibrillation/flutter, frequent premature beats, left bundle branch block, heart failure, stroke, low left ventricular ejection fraction (LVEF) <50%), chronic renal or liver disease, and those with poor quality echocardiographic images, insufficient ABPM readings or who refused to give written informed consent were excluded from the study.

Patients were divided as dippers or nondippers according to the dipping pattern of hypertension. An arbitrary cut-off was used to classify patients as dippers, that is, if their nocturnal BP decreased by >10% of the average daytime BP value [9]. The baseline characteristics, electrocardiogram and laboratory values (glycated hemoglobin, lipid profile and serum creatinine) were obtained for all patients.

Ambulatory blood pressure monitoring

A portable noninvasive recording ABPM device (GE Tonoport Healthcare, Berlin, Germany) was used for 24-h BP recording; all patients were instructed to practice their normal daily routine activities but to avoid strenuous exercise. They were also given a diary to report their activities during the day, including the times they woke up and slept. The measurements were taken from the nondominant arm with patients instructed to keep their arms straight during the measurements. The device was calibrated, fully automated, and programmed to take measurements every 30 min during the daytime (from 6 a.m. to 10 p.m.) and every hour during nighttime (from 10 p.m. to 6 a.m.). The recorded data were analyzed using ABPM management software (GE cardio soft v 6.73, Berlin, Germany). The ambulatory blood pressure monitoring was accepted only if at least 80% of the measurements were recorded. The values considered normal for mean daytime, nighttime and 24-h BP were <135/85 mmHg, <120/70 mmHg and <130/80 mmHg, respectively [2]. Patients were arbitrarily defined as dippers or nondippers if they had a decline of >10% or <10%, respectively, in the average nighttime BP compared to the average daytime BP [9,10]. We defined patients as nondippers if the nighttime MAP decreased by <10% from the daytime MAP using the following formula:

$$\frac{\text{Daytime MAP - nighttime MAP}}{\text{Daytime MAP}} \times 100$$

Transthoracic echocardiography

The transthoracic echocardiography was performed by a single operator in the left lateral decubitus using the Vivid-E9 General electric (GE Vingmed Ultrasound AS, Horten, Norway) machine with tissue Doppler and speckle tracking imaging capability. All findings were obtained by the M5S probe at a frequency of 2–4.5 MHz with simultaneous ECG triggering. The frame rate was adjusted at 60–90 frames/s for the acquisition of images and cine-loops to display the endocardium clearly and to avoid foreshortening. The following views were obtained during three consecutive cardiac cycles: LV apical four chamber, apical three chamber, apical two chamber, long axis and short-axis views (at mitral valve level, papillary muscle level and apical level). All images and cine-loops were transferred to a workstation and the EchoPAC v. 202 software (GE Vingmed Ultrasound AS, Horten, Norway) was used for off-line analysis. We followed the guidelines and standards of chamber quantifications to obtain all parameters [11].

Conventional transthoracic echocardiographic examination

In every patient, we assessed the left ventricular end-diastolic dimension (LVEDD), left ventricular end-systolic dimension (LVESD), interventricular septal diameter (IVSD) and left ventricular posterior wall diameter (LVPWD) in both M-mode and 2D images. The left ventricular ejection fraction (LVEF) and left ventricular volumes were assessed by Simpson's method. Left ventricular mass (LVM) and left ventricular mass index (LVMI) were calculated using linear measurements derived from the M-mode measurements by the application of the cube formula: $LVM = 0.8 \times 1.04 \{(IVSD + LVEDD + LVPWD)^3 - LVEDD^3\} + 0.6$ gm. The value for LVM was then indexed to the body surface area to obtain the LVMI [11]. An LVMI between 49–115 and 43–95 g/m^2 was considered normal in men and women, respectively [11]. The relative wall thickness (RWT) was calculated by using the formula: RWT = 2 × posterior wall thickness (PWD)/left ventricular dimension in diastole (LVEDD). The values for increased and normal RWT were defined as an RWT >0.42 and ≤0.42, respectively [11]. The RWT and LVMI were used to categorize subjects into four categories: those with normal geometry (normal RWT and normal LVMI), concentric remodeling (increased RWT and normal LVMI), eccentric hypertrophy (normal RWT and increased LVMI) and concentric hypertrophy (increased LVMI and increased RWT) [11]. The LV diastolic function was assessed according to the American Society of Echocardiography guidelines [12]. A pulsed-wave Doppler of the mitral valve inflow at the level of the mitral leaflets was used to measure the peak early diastolic velocity (E) and the peak late diastolic velocity (A), as well as for calculating the E/A ratio. Tissue Doppler imaging was used to measure the early diastolic velocity (E′) at the septal, lateral annulus and mean (E′). The E/E′ ratio was calculated to estimate the LV filling pressure.

Left ventricular speckle tracking echocardiography

A commercially available speckle tracking analysis software (2D-Strain, EchoPac. PC version 202, GE

Healthcare, Horten, Norway) was used for off-line analysis. A single operator blinded to the ABPM data performed the strain assessment, and the best-acquired apical three-chamber view was chosen for measuring the average global longitudinal strain (LS). By manually delineating the endocardium at the level of basal anterior, basal inferolateral and apical segments, the software automatically traced the rest of the endocardium and depicted the region of interest (ROI), including the LV subendocardium, mid-myocardium and subepicardium. If the system failed to trace the ROI correctly, the tracing could be adjusted manually. The software automatically defined the aortic valve closure time according to the ECG tracing and generated the strain values and curves for each segment. This process was repeated for the apical 4 and apical 2 chamber views to obtain the LS of the LV 17 segments, bull's eye view and corresponding curves. The average LS was the mean of the LS of the LV 17 segments. The global circumferential strain (CS) and global radial strain (RS) were obtained in the same way using the short-axis views at the level of the mitral valve, papillary muscle, and apical segments (Fig. 1).

Statistical analysis

Data management and statistical analysis were done using SPSS v. 25 (IBM, Armonk, New York, USA). Numerical data were summarized as means and standard deviations or medians and ranges. Categorical data were summarized as numbers and percentages. Comparisons between both groups were performed using an independent *t* test or the Mann–Whitney *U* test for normally and nonnormally distributed numerical data, respectively. Categorical data were compared using the Chi-square test or Fisher's exact test if appropriate. Linear regression analysis was done for the prediction of LS, CS and RS. Regression coefficients with 95% confidence intervals were calculated. All *P* values were two-sided. *P* values less than 0.05 were considered statistically significant.

Results

Baseline demographic and clinical data

One hundred hypertensive patients were finally included in our study. The mean age of our study population was 50.8 + 9.8 years, of which 67% were men, 21% were diabetics and 38% were smokers. Patients were divided according to their hypertension dipping status into the dipper group (29%) and the nondipper group (71%). Baseline characteristics of both groups are shown in Table 1. No significant differences in baseline demographic or clinical characteristics between both groups were found. Similarly, there were no differences in the basic laboratory investigations between both groups.

Ambulatory blood pressure monitoring data

Mean office BP measurement was similar in both groups; however, the nondipper group had a significantly higher nighttime SBP, DBP and MAP, as well as a higher daytime SBP, 24-h mean SBP and 24-h MAP as shown in Table 2.

Echocardiographic data

A comparison of the echocardiographic and speckle tracking parameters between the dipper and nondipper groups is shown in Table 3. The IVSD, LVPWD, RWT and LVMI were significantly higher in the nondipper group than in the dipper group, and as a result, normal LV geometry was observed less frequently in the nondipper group than in the dipper group (39.4 vs. 75.9%, $P = 0.006$). The E/E′ ratio was significantly higher in the nondipper group than in the dipper group (8.48 ± 1.97 vs. 6.81 ± 1.51, $P < 0.001$).

2D-strain results

Furthermore, the global longitudinal, circumferential and RSs were all significantly lower in the nondipper group than in the dipper group (–18.2 ± 3.3 vs. –22.45 ± 3.26, $P < 0.001$; –19.16 ± 8.25 vs. –24.23 ± 3.56, $P < 0.001$; 29.58 ± 8.44 vs. 35.04 ± 11.6, $P = 0.009$, respectively). An example of the strain pattern in a dipper and a nondipper patient is shown in Fig. 1. A linear regression analysis (after adjusting for age, sex, BMI, smoking, hypertension duration, diabetes mellitus, dyslipidemia and antihypertensive medications, 24-h MAP) was performed for the prediction of LS, CS and RS. It was revealed that the nondipper status was associated with worsening of LS by 2.737, ($P = 0.001$), CS by 3.446, ($P = 0.002$), RS by –3.256, ($P = 0.158$) as shown in Table 4 and Fig. 2. DM also was found associated with worsening of LS by 1.849, ($P = 0.062$), CS by 3.284 ($P = 0.018$), RS by –2.499 ($P = 0.381$).

Dipping status based on systolic blood pressure

When we reclassified the nondipper status of our study population on the basis of <10% nighttime reduction of SBP, we found that nondippers and dippers were 73 and 23 patients, respectively. LS, CS and RS were all also lower in nondippers than in dippers when we used SBP for reclassification (–18.64 ± 3.32 vs. –21.35 ± 3.27, $P = 0.001$; –21.03 ± 4.80 vs. –22.61 ± 3.81, $P = 0.085$; 30.25 ± 8.87 vs. 34.43 ± 9.24, $P = 0.049$, respectively). Furthermore, linear regression analysis (after adjusting for age, sex, BMI, smoking, hypertension duration, diabetes mellitus, dyslipidemia, antihypertensive medications, 24-h mean SBP) showed that nondipper status was associated with worsening of LS by 2.798, ($P = 0.001$), CS by 1.682, ($P = 0.158$), RS by –3.346, ($P = 0.146$) as shown in Table 5. DM also was associated with worsening of LS by 1.837, ($P = 0.064$), CS by 3.337 ($P = 0.022$), RS by –2.480 ($P = 0.384$).

Intraobserver variability

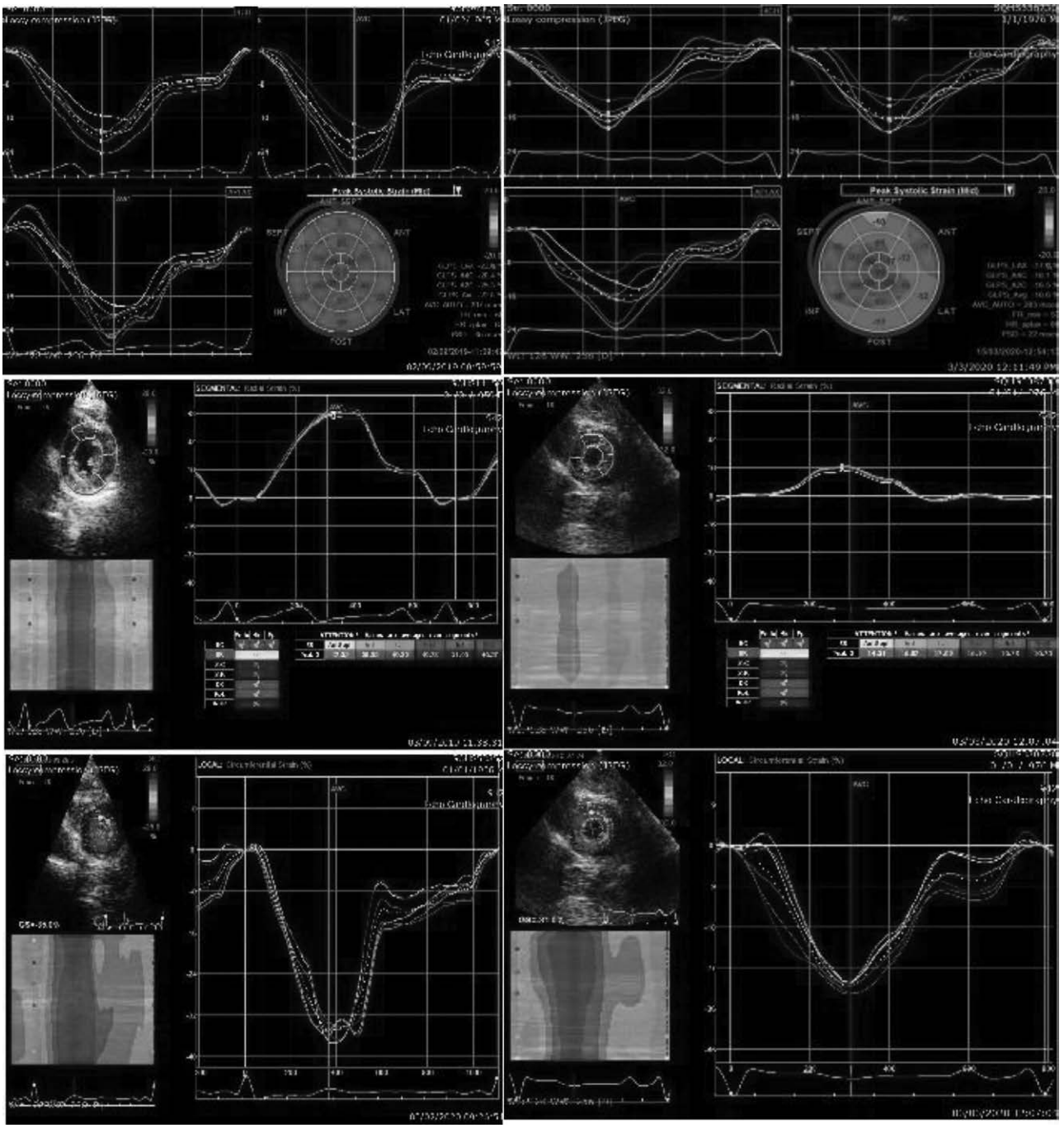
The intraclass correlation was 0.867, with a *P* value <0.001 for LS; 0.800 with a *P* value <0.001 for CS; and 0.788 with a *P* value <0.001 for RS.

Discussion

Our study demonstrated that patients with a nondipper hypertension pattern have a higher LVMI and a higher E/E′ ratio, with abnormal LV geometry observed more frequently than in patients with the dipper pattern hypertension. There was a significant difference in the LS, CS and RS between the dipper and nondipper groups, representing a subclinical LV dysfunction independent of LVEF.

Blood pressure variability occurs normally throughout the day and night independent of behavioral and mechanical changes; it may be related to stimuli originating within

Fig. 1



Representative example of the 2D-strain results of global LS, CS and RS in a dipper (left panel) and nondipper (right panel) patient. CS, circumferential strain; LS, longitudinal strain; RS, radial strain.

Table 1. Baseline demographic and clinical characteristics of dipper and nondipper groups

		Nondipper ($n=1$)	Dipper ($n=29$)	*P* value
Demographic data				
Age (years)	Mean ±SD	51 ± 11	51 ± 9	0.925
Sex	Men	49 (69.0%)	18 (62.1%)	0.503
	Women	22 (31.0%)	11 (37.9%)	
BMI (kg/m^2)	Mean±SD	33.8 ± 6.3	32.5 ± 7.5	0.37
BSA (m^2)	Mean±SD	1.96 ± 0.17	1.91 ± 0.17	0.149
Medical history				
HTN duration (years)	Median (range)	3 (1–16)	3 (1–10)	0.179
DM	N(%)	17 (23.9)	4 (13.8)	0.258
Dyslipidemia	N(%)	32 (45.1)	12 (41.4)	0.736
Smoking	N(%)	31 (43.7)	7 (24.1)	0.068
Medication history	N(%)			
ACEIs	N(%)	16 (22.5)	8 (27.6)	0.592
ARBs	N(%)	41 (57.7)	13 (44.8)	0.24
Beta-blockers	N(%)	22 (31.0)	8 (27.6)	0.736
CCB	N(%)	33 (46.5)	9 (31.0)	0.156
Laboratory investigations				
HbA1C (%)	Mean±SD	5.7 ± 1.28	5.29 ± 0.98	0.129
Serum creatinine (mg/dl)	Mean±SD	0.97 ± 0.2	0.94 ± 0.2	0.455

ACEIs, converting enzyme inhibitors; ARBs, angiotensin receptor blockers; BMI body mass index, BSA; body surface area; CCB, calcium channel blocker; DM, diabetes mellitus; HbA1C, hemoglobin A1C; HTN, hypertension.

Table 2. Office BP and 24-h ABPM measurements in the study groups

BP parameter		Nondipper ($n=71$)	Dipper ($n=29$)	*P* value
Office SBP (mmHg)	Mean±SD	142 ± 15	140 ± 12	0.495
Office DBP (mmHg)	Mean±SD	86 ± 8	83 ± 9	0.177
Daytime SBP (mmHg)	Mean±SD	145 ± 16	137 ± 20	**0.036**
Daytime DBP (mmHg)	Mean±SD	92 ± 19	87 ± 18	0.28
Nighttime SBP (mmHg)	Mean±SD	138 ± 17	119 ± 13	**<0.001**
Nighttime DBP (mmHg)	Mean±SD	84 ± 13	71 ± 9	**<0.001**
24-h mean SBP (mmHg)	Mean±SD	138 ± 15	129 ± 9	**<0.001**
24-h mean DBP (mmHg)	Mean±SD	82 ± 11	79 ± 8	0.192
Daytime MAP (mmHg)	Mean±SD	101.1 ± 12	98.1 ± 8	0.153
Nighttime MAP (mmHg)	Mean±SD	98.1 ± 11.6	83.5 ± 6.7	**<0.001**
24-h MAP (mmHg)	Mean±SD	102.2 ± 12.4	96.3 ± 8.1	**0.006**
24-h mean HR (bpm)	Mean±SD	87 ± 15	86 ± 8	0.78

Significant *P* values are shown in bold.
ABPM, ambulatory blood pressure monitoring; BP, blood pressure; bpm, beat per minute; DBP, diastolic blood pressure; HR, heart rate; MAP, mean artery pressure; SBP, systolic blood pressure; SD, standard deviation.

the brain [13]. The dipping and nondipping patterns in hypertensive patients were originally introduced by O'Brein *et al.* and Pickering *et al.* [14,15]. They defined a dipping pattern as a decrease of ≥10% in the average nighttime BP values compared to the average daytime values, while a BP decline of <10% was called a nondipping pattern. This definition was widely accepted and since then, the nondipper state was linked to cardiovascular and cerebrovascular complications in multiple studies with robust evidence [4–6,16–19]. The left ventricular myocardial strain value is now used with increasing frequency to detect subtle subclinical LV systolic dysfunction independent of LVEF in hypertensive patients [20,21].

Table 3. Echocardiographic parameters in dipper and nondipper groups

		Nondipper ($n=71$)	Dipper ($n=29$)	*P* value
IVSd (cm)	Median (range)	1 (0.7–1.1)	0.9 (0.7–1.3)	0.008
LVEDD (cm)	Mean±SD	4.8 ± 0.5	4.7 ± 0.5	0.395
LVPWD (cm)	Mean±SD	1.02 ± 0.18	0.89 ± 0.12	**<0.001**
RWT	Mean±SD	0.43 ± 0.08	0.39 ± 0.07	**0.02**
LVMI (gm/m^2)	Mean±SD	91 ± 28	76 ± 20	**0.01**
LV geometry	Normal	28 (39.4)	22 (75.9)	**0.006**
	Concentric remodeling	25 (35.2)	6 (20.7)	
	Concentric hypertrophy	14 (19.7)	1 (3.4)	
	Eccentric hypertrophy	4 (5.6)	0 (0.0)	
LVESV (ml)	Mean±SD	42 ± 16	40 ± 13	0.55
LVEDV (ml)	Mean±SD	117 ± 40	115 ± 31	0.829
LVEF (%)	Mean±SD	62 ± 6	63 ± 4	0.175
E/A ratio	Mean±SD	0.99 ± 0.29	1.05 ± 0.28	0.335
E/E' ratio	Mean±SD	8.48 ± 1.97	6.81 ± 1.51	**<0.001**
LS (%)	Mean±SD	−18.2 ± 3.3	−22.45 ± 3.26	**<0.001**
CS (%)	Mean±SD	−19.16 ± 8.25	−24.23 ± 3.56	**0.002**
RS (%)	Mean±SD	29.58 ± 8.44	35.04 ± 11.16	**0.009**

Significant *P* values are shown in bold.
CS, circumferential strain; IVSD, interventricular septum diameter; LS, longitudinal strain; LVEDD, left ventricular diastolic diameter; LVEDV, left ventricular diastolic volume; LVESV, left ventricular systolic volume; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; RS, radial strain; RWT, relative wall thickness.

Table 4. Linear regression analysis for using nondipper status[a] as a predictor of LS, CS and RS

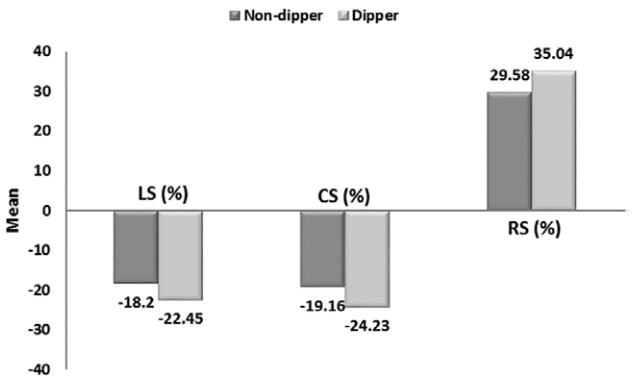
	B	SE	BETA	95% CI for *B*		*P* value
Nondipper status for prediction of LS	2.737	0.790	0.353	1.166	4.308	0.001
Nondipper status for prediction of CS	3.446	1.104	0.333	1.250	5.643	0.002
Nondipper status for prediction of RS	−3.256	2.294	−0.032	−7.822	1.303	0.158

Results were adjusted for age, sex, BMI, HTN duration, DM, dyslipidemia, smoking, ACEIs, ARBs, beta-blockers, CCB, 24-h MAP. B, regression coefficient; 95% CI, 95% confidence interval for B, ACEIs, converting enzyme inhibitors.
ARBs, angiotensin receptor blockers; BMI body mass index; BSA, body surface area; CCB, calcium channel blocker; DM, diabetes mellitus; CS, circumferential strain; LS, longitudinal strain; RS, radial strain; 24-h MAP, 24 h mean arterial blood pressure.
[a]Based on <10 % nighttime reduction of the mean arterial pressure.

Our results are consistent with the growing evidence that correlates the nondipping pattern of hypertension with the impairment of the global myocardial strain. Seo *et al.* [22] showed that the nondipper pattern was associated with decreased tissue Doppler systolic S wave velocity, peak strain and strain rate in newly diagnosed (<1 year) hypertensive patients. Tadic *et al.* [23] showed, in a group with newly diagnosed hypertensive patients and those with untreated hypertension, that only 2D LS, but not CS or RS, was lower in nondippers while 3D STE could reveal a significant difference in the three directions in the nondipper group. This may be due to the recent diagnosis of hypertension and the early stage of LV dysfunction in these patients.

Longitudinal myocardial fibers are located in the subendocardium, and LS is the earliest strain parameter to be

Fig. 2



Bar chart showing the mean global LS, CS and RS in the dipper and nondipper groups. CS, circumferential strain; LS, longitudinal strain; RS, radial strain.

Table 5. Linear regression analysis for using nondipper status[a] as a predictor of LS, CS and RS

	B	SE	BETA	95% CI for B		*P* value
Nondipper status for prediction of LS	2.798	0.807	0.352	1.193	4.403	0.001
Nondipper status for prediction of CS	1.682	1.179	0.228	-0.662	4.027	0.158
Nondipper status for prediction of RS	−3.346	2.342	−0.164	−8.095	1.222	0.146

Results were adjusted for age, sex, BMI, HTN duration, DM, dyslipidemia, smoking, ACEIs, ARBs, Beta-blockers, CCB, 24-h mean SBP. B, regression coefficient; 95% CI, 95% confidence interval for B.

ACEIs, converting enzyme inhibitors; ARBs, angiotensin receptor blockers; BMI body mass index; BSA, body surface area; CCB, calcium channel blocker; DM, diabetes mellitus; CS, circumferential strain; LS, longitudinal strain; RS, radial strain; 24-h mean SBP, 24 h mean systolic blood pressure.

[a]Based on <10% nighttime reduction of the systolic blood pressure.

affected, while CS and RS are preserved in the early stages of LV systolic dysfunction when assessed by 2D STE [24]. Kalaycıoğlu *et al*. showed that the nondipper pattern was associated with a decreased LS and the LS rate in a hypertensive diabetic group of patients with preserved LV systolic function [25]. Kalaycıoğlu *et al*. also demonstrated that osteoprotegerin, which is a soluble member of the tumor necrosis factor receptor and is linked to the pathogenesis of heart failure, was an independent predictor of LS in hypertensive diabetic patients [26]. The subclinical impairment of the LS in nondippers was also confirmed in newly diagnosed hypertensive patients [27]. In a treated cohort of patients with variable duration of hypertension, Chen *et al*. [28] also showed impairment of global myocardial strain in the three directions by using 2D STE.

Nighttime reduction rate of SBP, DBP, MAP, night/day SBP ratio and night/day DBP ratio were all suggested for differentiation between dippers and nondippers [2]. However, to the best of our knowledge, there is no study that showed any differences between these indices. SBP and DBP behave differently during physical activity which may influence the classification of the dipping state whenever one of them is considered [29]. It was suggested that nighttime MAP reduction might be considered as a better index for the classification of dipping and nondipping state [30]. Nighttime reduction of SBP was used to define dipping/nondipping pattern in some similar studies [22,23,27] while nighttime reduction of MAP was used in other studies [25,28]. In our analysis, we defined the dipping/nondipping state according to the nighttime reduction in MAP. When we redefined the dipping/nondipping pattern according to nighttime reduction of SBP, we obtained similar results.

The long-term prognostic implications of these findings need larger studies with a long-term follow-up. The LS has been suggested to be the most sensitive, easily measured and widely available parameter to assess subclinical LV systolic dysfunction in hypertensive patients [31]; thus it may be recommended, according to the available body of evidence, to assess at least LS and treat hypertension more aggressively in these patients. The finding of subclinical LV systolic dysfunction may also be included in patient counseling, especially for those who are not compliant in taking antihypertensive medications.

Study limitations

Our study has some limitations due to the relatively small number of patients and the cross-sectional nature of the study. There was also no control group of normotensive patients.

Conclusion

In this study, we demonstrated the impact of the nondipping pattern of hypertension on subclinical LV systolic dysfunction as shown by the impairment of the global myocardial strain. This highlights the importance of recognizing dipping and nondipping states of hypertension for refinement of the risk stratification process for these patients.

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Conflicts of interest

There are no conflicts of interest.

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