

# Left ventricular deformation in relation to the geometric pattern in hypertensive patients

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## Abstract

This study aimed to evaluate left ventricular deformation in relation to the geometric pattern in hypertensive patients with normal left ventricular ejection fraction using speckle tracking echocardiography (STE).

Transthoracic echocardiography was performed in 80 hypertensive patients and 50 age- and gender-matched normotensive subjects. Left ventricular geometric pattern was defined according to left ventricular mass index and relative wall thickness as normal geometry, concentric remodeling, concentric hypertrophy, and eccentric hypertrophy, respectively. Quantitative measurements of longitudinal, circumferential, and radial strain were performed for endocardial, middle, and epicardial layers of the left ventricular wall at each segment.

The longitudinal strain in hypertension was lower for all 3 layers in concentric (n=20) and eccentric hypertrophy (n=20) than normotensive subjects (n=50,  $P < .01$ ). It was also significantly lower for the endocardial layer in concentric remodeling (n=20,  $P = .04$  vs normotensive subjects). The circumferential strain in hypertension was higher in normal geometry or concentric remodeling, lower in concentric hypertrophy, and at similar level in eccentric hypertrophy, in comparison with normotensive subjects. The difference from normotensive subjects was statistically significant for the endocardial and middle layers in normal geometry ( $P < .03$ ), for the endocardial layer in concentric remodeling ( $P < .02$ ), and for the middle and epicardial layers in concentric hypertrophy ( $P \leq .001$ ). The radial strain and twist did not differ between normotensive and hypertensive subjects ( $P > .08$ ).

Left ventricular deformation in hypertension occurs with various geometric patterns disproportionately in the endocardial, middle and epicardial layers and differently in the longitudinal and circumferential orientations.

**Abbreviations:** A = late transmitral flow velocities, E = early transmitral flow velocities, E/A = the ratio of early-to-late peak velocities, E' = early diastolic velocity of mitral annulus, IVS = interventricular septal, LAD = left atrial diameters, LVDD = left ventricular diastolic diameter, LVMI = left ventricular mass index, LVPW = left ventricular posterior wall, RWT = relative wall thickness, STE = speckle tracking echocardiography, TDI = tissue Doppler imaging.

**Keywords:** hypertension, left ventricular geometry, left ventricular layer function, two-dimensional strain

## 1. Introduction

With the conventional echocardiographic technique, left ventricular structure and function can be measured. For the left ventricular structure, 4 geometric patterns have been recognized: normal geometry, concentric remodeling, and concentric and eccentric hypertrophy.<sup>[1]</sup> For the left ventricular function,

ejection fraction is used for the evaluation of systolic function, and the ratio of peak early-to-late transmitral velocities (E/A) or the ratio of early diastolic transmitral to mitral annulus velocity (E/E') for the evaluation of diastolic function. The abnormal left ventricular geometric patterns have strong prognostic significance and may predict the incidence of cardiac failure and other cardiovascular diseases.<sup>[1,2]</sup> However, the functional relevance of various left ventricular geometric patterns to systolic and diastolic dysfunction of the left ventricle remains under investigation.

The speckle tracking echocardiography (STE) may help analyze the structural with functional disturbances of the left ventricle by detecting early functional changes in the myocardial wall, which is a complex, multilayered, and heterogeneous structure.<sup>[3]</sup> The conventional echocardiography technique measures global myocardial function. STE, as an angle-independent technique, allows for the quantification of myocardial function in the endocardial, middle, and epicardial layers.<sup>[4]</sup> These regional cardiac functional changes occur in the early stages of hypertension.<sup>[5]</sup> In our previous research with the 2D STE, we found that hypertensive patients had more prominent deformation and functional changes than normotensive subjects.<sup>[6]</sup> In the present study, we investigated with the 2D STE the deformation and function of the left ventricle according to the 4 geometric patterns defined by the conventional echocardiography in hypertensive patients.

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**2. Methods**

**2.1. Study population**

Our study included a group of hypertensive patients and a group of gender- and age-matched normotensive subjects. The hypertensive group included patients with primary hypertension, diagnosed with a blood pressure  $\geq 140/90$  mm Hg or the use of antihypertensive medication. Normotensive subjects also had normal electrocardiogram and echocardiography, and had no history of cardiovascular disease or risk factors. We excluded those with valvular heart disease, atrial fibrillation, or poor quality echocardiographic images. The Ethics Committee of Ruijin Hospital, Shanghai Jiaotong University School of Medicine, China approved the study protocol. All participants provided written informed consent.

**2.2. Standard conventional echocardiography**

A Vivid E9 ultrasound scanner (GE HealthCare, Milwaukee, WI) with a M5S-D Phased Array Transducer (GE HealthCare) was used to perform the 2D transthoracic echocardiography on patients in the left lateral decubitus position. Data were acquired from 3 standard apical and parasternal short- and long-axis views through 3 consecutive cardiac cycles each view. Left ventricular evaluation was carried out on optimized grayscale recordings (frame rate: 50–80 frames/s). Tissue Doppler imaging (TDI) measurements were performed to assess mitral annulus excursion. Pulsed-wave sample volume, which was positioned at the septal corner of the mitral valve annulus, was used to document early diastolic velocity of mitral annulus (E').

Interventricular septal (IVS) and left ventricular posterior wall (LVPW) thickness, left ventricular end-diastolic diameter (LVDD), and left atrial diameter (LAD) were obtained through analysis of 2D imaging. The formula used to compute left ventricular mass (g) from M-mode echocardiograms was described by Devereux et al.<sup>[7]</sup> Left ventricular mass =  $0.8 \times \{1.04 \times [(LVDD + LVPW + IVS)^3 - (LVDD)^3]\} + 0.6$ . Left ventricular mass index (LVMI, g/m<sup>2</sup>)

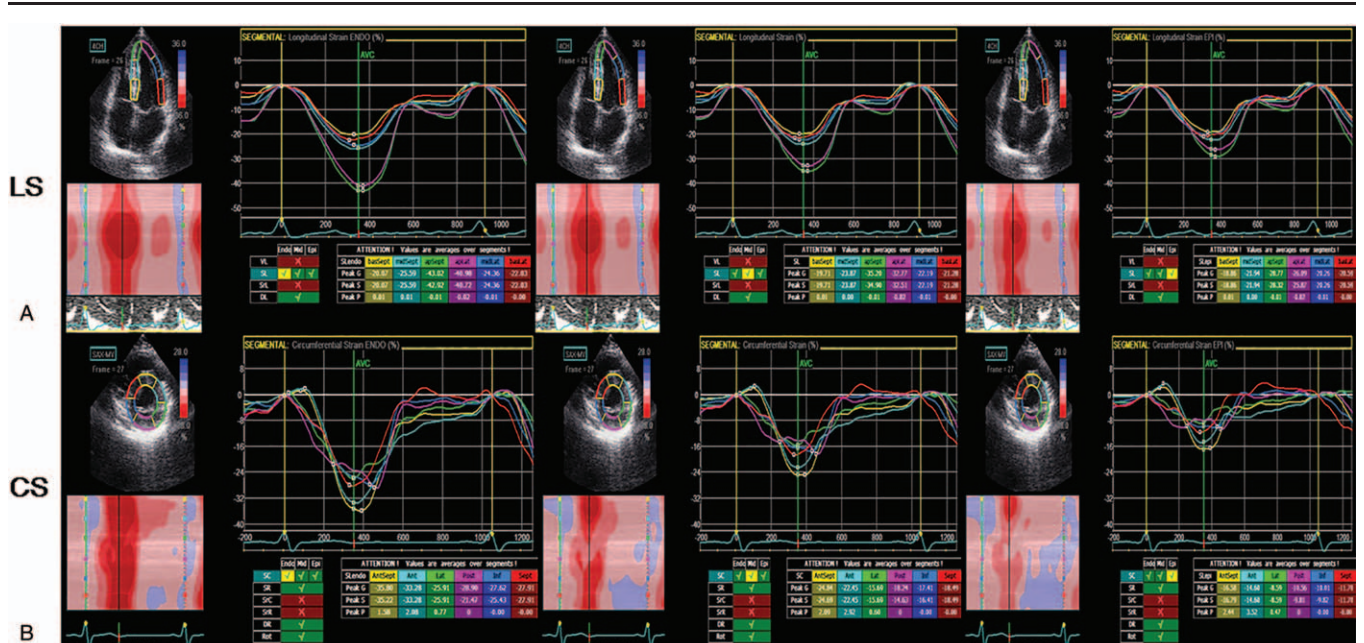
was the ratio of left ventricular mass to body surface area. Relative wall thickness (RWT) was computed as the ratio of the sum of anteroseptal and LVPW thickness to LVDD. Left ventricular hypertrophy was defined as a LVMI  $\geq 95$  g/m<sup>2</sup> in women and 115 g/m<sup>2</sup> in men. Concentric geometry was present when RWT  $> 0.42$ . The biplane 2D Simpson's method was used to measure left ventricular volume.

Hypertensive subjects were classified into 4 subgroups of the left ventricular geometric pattern defined by LVMI and RWT as described previously:<sup>[8]</sup> normal geometry, normal LVMI, and RWT  $\leq 0.42$ ; concentric remodeling, normal LVMI, and RWT  $> 0.42$ ; concentric hypertrophy, increased LVMI, and RWT  $> 0.42$ ; and eccentric hypertrophy, increased LVMI, and RWT  $\leq 0.42$ .

The apical 4-chamber view was used to acquire mitral inflow velocity by placing a pulsed-wave Doppler sample volume between mitral leaflet tips during diastole. The peak early (E) and late (A) transmitral flow velocities were measured, and E/A was calculated. From TDI, the E/E' ratio was computed.

**2.3. 2D STE**

2D strain parameters were analyzed as previously described.<sup>[6]</sup> Briefly, automated software (EchoPAC PC, Version113; GE HealthCare, Milwaukee, WI) was used to analyze all 3 cardiac layers of speckle tracking data. Three endocardial markers were positioned at the apical 4-, 2-, and 3-chamber views, respectively, to analyze 2D longitudinal data. Appropriate tracking was corroborated in real time and adjusted for the area of interest or manually to ensure accurate tracking. The 2D three-layer longitudinal and circumferential strains were analyzed from apical (Fig. 1A) and parasternal short axis views (Fig. 1B), respectively. The average left ventricular global longitudinal and circumferential strains were computed in 18 segments (6 basal, 6 middle, and 6 apical) based on its relation to the strain magnitude at the aortic valve closure. With the strain definition, systolic indices had a negative value. The left ventricular basal and apex rotations were determined from parasternal mitral valve and



**Figure 1.** The 3 myocardial layers of left ventricular segmental longitudinal strain curves (A), and circumferential strain curves (B).

apical levels. The left ventricular twist was acquired from the software.

### 2.4. Other clinical measurements

A physician administered a standardized questionnaire, inquiring into each subject’s smoking and drinking habits. Body height (meters) was measured using a commercially available stadiometer. Body weight (kilograms, rounded to the nearest integer value) was measured using a weighing scale when the patient was in light clothing without shoes. The body surface area was calculated using the previously reported function:<sup>[9]</sup> Body surface area = Weight<sup>0.425</sup> × Height<sup>0.725</sup> × 0.007184. After subjects had fasted overnight, venous blood samples were drawn and analyzed by automated enzymatic methods for serum total cholesterol and blood glucose. Blood pressure was measured twice consecutively using a conventional mercury sphygmomanometer. An average of these 2 blood pressure readings was used for analysis.

### 2.5. Statistical analysis

Data management and statistical analyses were performed with the Statistical Analysis Software (SAS), version 9.1.3 (SAS Institute Inc, Cary, NC). Values were expressed as either mean ± standard deviation (SD) or number (percentages) as appropriate.

A chi square ( $\chi^2$ ) test was used for the comparison of categorical variables between groups. Analysis of variance (ANOVA) was performed for the comparison of continuous variables between groups with an option of the Bonferroni post-hoc test. Multivariable regression analyses were performed to search for determinants of longitudinal or circumferential stain, which was used as a dependent variable in the subsequent regression analyses. The *P* values for explanatory variables to enter and stay in the models were set at .10. We considered the following covariables: sex, age, body surface area, current smoking and alcohol intake, plasma glucose, serum total cholesterol, LVMI, heart rate, and mean arterial pressure. A *P* value of less than .05 was considered statistically significant.

## 3. Results

### 3.1. Clinical and conventional echocardiographic characteristics of the study participants

Table 1 shows the clinical and conventional echocardiographic characteristics of 50 normotensive subjects and 80 hypertensive patients with normal cardiac geometry (n=20), concentric remodeling (n=20), or concentric (n=20) or eccentric hypertrophy (n=20). Age and gender were not different between these groups (*P* > .05), but body surface area was higher in concentric remodeling and left ventricular hypertrophy groups. The

**Table 1**  
Demographic, clinical, and echocardiographic characteristics of participants.

	Normotensive control (n=50)	Hypertensive patients (n=80)			
		Normal geometry (n=20)	Concentric remodeling (n=20)	Concentric hypertrophy (n=20)	Eccentric hypertrophy (n=20)
Demographics and clinical characteristics					
Age (y)	52.6 ± 9.7	52.3 ± 13.7	54.4 ± 14.0	54.5 ± 13.9	55.5 ± 11.2
Men (%)	24 (48)	9 (45)	11 (55)	11 (55)	9 (45)
Body surface area (m <sup>2</sup> )	1.5 ± 0.1	1.5 ± 0.2	1.7 ± 0.2 <sup>*,†</sup>	1.8 ± 0.3 <sup>*,†</sup>	1.7 ± 0.2 <sup>*,†</sup>
Systolic blood pressure, mm Hg	116.9 ± 10.8	136.1 ± 9.2 <sup>*</sup>	144.6 ± 8.4 <sup>*</sup>	157.6 ± 22.0 <sup>*,†,‡</sup>	161.6 ± 20.0 <sup>*,†,‡</sup>
Diastolic blood pressure, mm Hg	74.3 ± 8.4	84.1 ± 6.8 <sup>*</sup>	85.2 ± 7.1 <sup>*</sup>	93.9 ± 19.2 <sup>*,†</sup>	91.0 ± 11.4 <sup>*,†</sup>
Heart rate (beats/min)	68.7 ± 6.1	71.6 ± 11.4	70.1 ± 13.0	71.1 ± 8.4	66.7 ± 9.8
Plasma glucose, mmol/L	5.0 ± 0.8	5.4 ± 0.6	5.2 ± 0.8	5.8 ± 2.0 <sup>*</sup>	5.6 ± 1.2 <sup>*</sup>
Serum total cholesterol, mmol/L	4.9 ± 0.8	5.0 ± 0.7	5.2 ± 1.0	5.8 ± 1.2 <sup>*,†,‡</sup>	5.5 ± 1.3 <sup>*</sup>
Smoker, n (%)	5 (10)	2 (10)	4 (20)	8 (40) <sup>*,†</sup>	7 (35) <sup>*,†</sup>
Drinking alcohol, n (%)	3 (6)	3 (15)	3 (15)	4 (20)	7 (35) <sup>*</sup>
Echocardiographic 2D characteristics					
Aortic diameter (mm)	30.4 ± 3.2	29.2 ± 2.8	32.7 ± 2.7 <sup>*,†</sup>	34.7 ± 3.5 <sup>*,†</sup>	32.6 ± 3.7 <sup>*,†</sup>
Left atrial diameter (mm)	34.7 ± 3.3	34.0 ± 3.2	40.9 ± 4.0 <sup>*,†</sup>	39.8 ± 5.1 <sup>*,†</sup>	40.7 ± 4.7 <sup>*,†</sup>
LV septal thickness (mm)	8.5 ± 0.7	8.8 ± 0.6	10.6 ± 1.4 <sup>*,†</sup>	12.9 ± 2.2 <sup>*,†,‡</sup>	10.1 ± 1.2 <sup>*,†</sup>
LV end diastolic diameter (mm)	48.5 ± 3.2	45.6 ± 2.8 <sup>*</sup>	43.6 ± 3.7 <sup>*</sup>	49.5 ± 5.5 <sup>†,‡</sup>	52.6 ± 3.1 <sup>*,†,‡</sup>
LV posterior wall thickness (mm)	8.5 ± 0.7	8.5 ± 0.6	9.9 ± 1.2 <sup>*,†</sup>	12.1 ± 1.6 <sup>*,†,‡</sup>	9.6 ± 1.1 <sup>*,†</sup>
LV relative wall thickness (mm)	0.35 ± 0.02	0.37 ± 0.03 <sup>*</sup>	0.47 ± 0.04 <sup>*,†</sup>	0.51 ± 0.06 <sup>*,†,‡</sup>	0.37 ± 0.04 <sup>*,†</sup>
LV mass index (g/m <sup>2</sup> )	83.5 ± 2.6	84.4 ± 11.4	86.8 ± 16.7	136.8 ± 30.4 <sup>*,†,‡</sup>	115.7 ± 21.5 <sup>*,†,‡</sup>
LV ejection fraction, %	64.4 ± 6.4	65.7 ± 6.0	66.5 ± 5.4	63.8 ± 8.8	64.2 ± 5.9
Doppler					
E/A ratio					
Mean ± SD	1.3 ± 0.5	1.0 ± 0.3	0.9 ± 0.2	1.0 ± 0.4	1.0 ± 0.3
<1.0, n (%)	11 (22%)	10 (50%) <sup>*</sup>	15 (75%) <sup>*,†</sup>	11 (55%) <sup>*,‡</sup>	14 (70%) <sup>*</sup>
Septal E/E' ratio					
Mean ± SD	9.7 ± 2.6	10.9 ± 2.8	11.2 ± 2.5 <sup>*</sup>	12.2 ± 3.2 <sup>*</sup>	12.7 ± 3.3 <sup>*</sup>
>15, n (%)	0	0	0	5 (25%)	4 (20%)

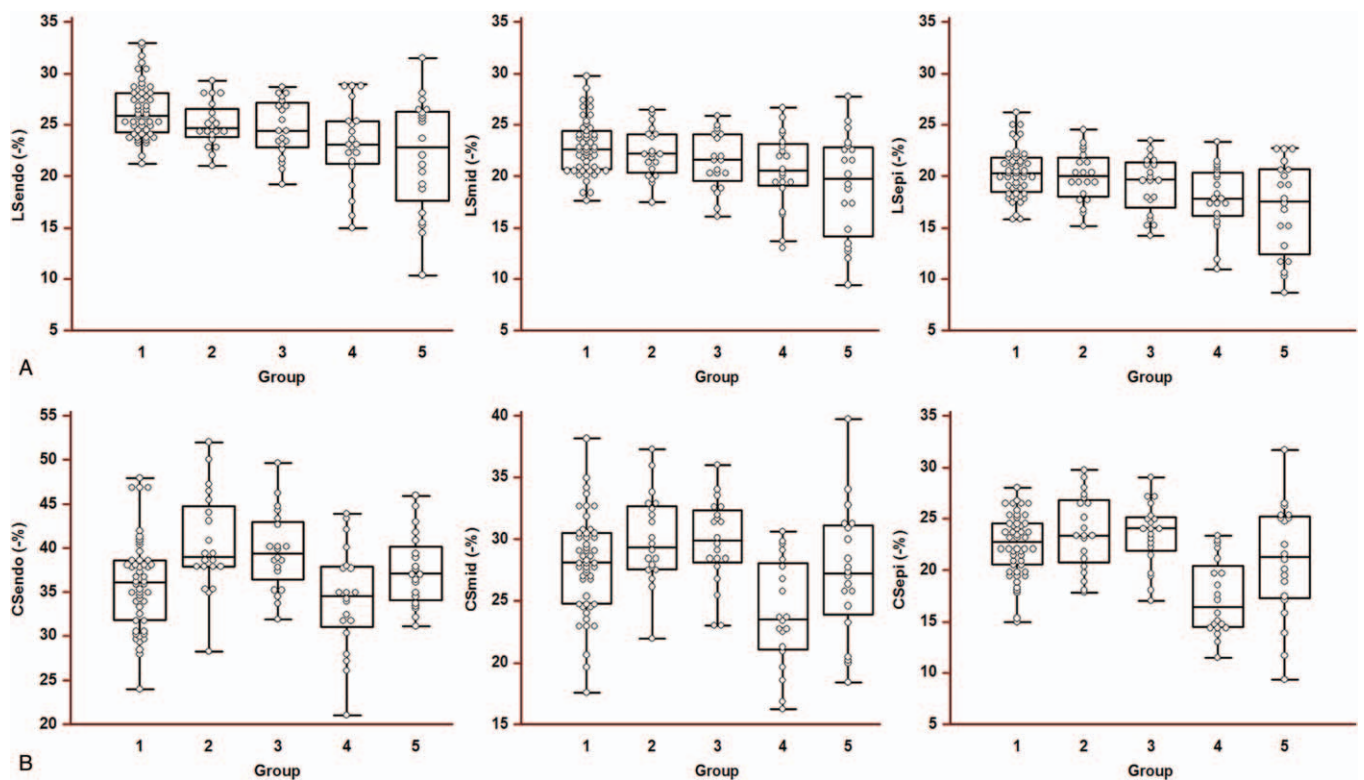
Values are expressed as mean ± standard deviation (SD), or number of subjects (%).

A=late transmitral flow velocities, E' =early diastolic velocity of mitral annulus, E=early transmitral flow velocities, LV=left ventricular.

\* *P* < .05 versus control.

† *P* < .05 versus normal geometry.

‡ *P* < .05 versus concentric remodeling.



**Figure 2.** The distribution of layer-specific strain analysis among groups, including longitudinal strain (A), and circumferential strain (B). Group 1: Normotensive control, group 2: Normal geometry, group 3: Concentric remodeling, group 4: Concentric hypertrophy, group 5: Eccentric hypertrophy.

corresponding proportions of current smoking/ drinking were 10%/6%, 10%/15%, 20%/15%, 40%/20%, and 35%/35%, respectively.

These hypertensive and normotensive subjects had normal range (55–75%) and similar mean levels ( $\pm$ SD) of left ventricular ejection fraction ( $65.0 \pm 6.5\%$ ,  $P = .54$ ). They also had similar mean levels of E/A ratio ( $P = .18$ ). However, hypertensive patients compared with normotensive subjects had higher mean levels of septal E/E' ratio ( $P \leq .05$ ) and higher proportions of a E/E' ratio  $> 15$  ( $P < .001$ ).

### 3.2. Myocardial strain parameters according to the left ventricular geometric pattern

The distribution of layer-specific strain analysis among groups has been shown (Fig. 2). Myocardial strain parameters showed that both longitudinal and circumferential strain measurements decreased from endocardium to epicardium ( $P < .01$ ), and that the circumferential strain was greater than the longitudinal strain for all 3 layers of myocardium ( $P < .01$ ) in normotensive subjects as well as hypertensive patients (Table 2).

**Table 2**

**Myocardial strain as assessed by speckle tracking echocardiography in hypertensive patients with various geometric patterns and normotensive subjects.**

Strain and twist		Normotensive control (n=50)	Hypertensive patients (n=80)			
			Normal geometry (n=20)	Concentric remodeling (n=20)	Concentric hypertrophy (n=20)	Eccentric hypertrophy (n=20)
Longitudinal strain	Endocardial	$-26.5 \pm 2.7$	$-25.0 \pm 2.5$	$-24.7 \pm 2.8^*$	$-22.8 \pm 4.7^{*,\ddagger}$	$-23.0 \pm 5.1^{*,\ddagger}$
	Middle	$-23.1 \pm 3.2$	$-22.3 \pm 2.4$	$-21.5 \pm 2.8$	$-20.3 \pm 4.4^{*,\ddagger}$	$-20.0 \pm 4.8^{*,\ddagger}$
	Epicardial	$-20.3 \pm 2.4$	$-20.0 \pm 2.5$	$-19.2 \pm 2.7$	$-17.6 \pm 3.7^{*,\ddagger}$	$-17.9 \pm 4.1^{*,\ddagger}$
	Global	$-23.3 \pm 2.7$	$-22.4 \pm 2.6$	$-21.8 \pm 2.5$	$-20.3 \pm 4.1^{*,\ddagger}$	$-20.3 \pm 4.6^{*,\ddagger}$
Circumferential strain	Endocardial	$-36.0 \pm 5.2$	$-40.7 \pm 6.1^*$	$-39.7 \pm 4.5^*$	$-33.7 \pm 6.9^{\ddagger}$	$-37.7 \pm 4.6$
	Middle	$-28.0 \pm 4.0$	$-30.5 \pm 4.5^*$	$-29.9 \pm 4.1$	$-24.0 \pm 4.4^{*,\ddagger,\S}$	$-27.3 \pm 5.3^\ddagger$
	Epicardial	$-22.4 \pm 3.4$	$-23.9 \pm 4.0$	$-23.3 \pm 3.9$	$-17.3 \pm 3.7^{*,\ddagger,\S}$	$-20.9 \pm 5.6^\ddagger$
	Global	$-28.8 \pm 4.0$	$-31.7 \pm 4.6^*$	$-31.0 \pm 3.8^*$	$-25.0 \pm 4.5^{*,\ddagger,\S}$	$-28.6 \pm 4.8^\ddagger$
Radial strain		$33.1 \pm 8.1$	$33.8 \pm 9.1$	$32.3 \pm 12.9$	$30.9 \pm 9.9$	$31.5 \pm 11.4$
Twist ( $^\circ$ )		$17.2 \pm 5.6$	$19.1 \pm 7.3$	$19.0 \pm 6.0$	$19.6 \pm 7.0$	$19.8 \pm 6.1$

Values are mean  $\pm$  standard deviation.

\*  $P < .05$  versus control.

$^\ddagger$   $P < .05$  versus normal geometry.

$^\S$   $P < .05$  versus concentric remodeling.

The longitudinal strain was lower in hypertensive patients than normotensive subjects. The difference reached statistical significance for all 3 layers of myocardial strain in hypertensive patients with concentric and eccentric left ventricular hypertrophy ( $P < .01$ ) and for the endocardial ( $P = .04$ ) but not the middle and epicardial layers of strain in patients with concentric remodeling ( $P \geq .1$ ).

The circumferential strain was higher in hypertensive patients with normal geometry or concentric remodeling than normotensive subjects, with a statistically significant difference for the endocardial and middle layers of myocardium in patients with normal geometry ( $P < .03$ ) and for the endocardial layer of myocardium in patients with concentric remodeling ( $P < .02$ ). However, it was significantly ( $P < .05$ ) lower in patients with concentric hypertrophy and at similar level ( $P \geq .16$ ) in patients with eccentric hypertrophy in comparison with normotensive subjects. The radial strain and twist did not differ between normotensive and hypertensive subjects ( $P > .08$ ).

The amplitude of endocardial longitudinal strain negatively correlated with LVMI ( $r = -0.36$ ,  $P < .01$ ), while the endocardial circumferential strain positively correlated with ejection fraction ( $r = 0.30$ ,  $P < .01$ ). In multivariable adjustment models considered age, sex, body surface area, current smoking and alcohol intake, plasma glucose, serum total cholesterol, LVMI, heart rate, and mean arterial pressure, LVMI ( $\beta = -0.23$ ,  $P = .03$ ) was significantly associated with the longitudinal strain, and age ( $\beta = -0.21$ ,  $P = .02$ ) and LVMI ( $\beta = -0.25$ ,  $P = .03$ ) were significantly associated with the circumferential strain.

#### 4. Discussion

In the present study, we used the 2D layer-specific speckle tracking technique to investigate left ventricular deformation in relation to various left ventricular geometric patterns in hypertensive patients. The principal findings are as follows. In the presence of hypertension and normal geometry, longitudinal strain remains normal, but circumferential strain increases, especially in the endocardial and middle layers of myocardium. With the structural remodeling, longitudinal strain decreases and circumferential strain increases, both in the endocardial layer. In the presence of left ventricular concentric hypertrophy, longitudinal strain decreases in all 3 layers, and circumferential strain decreases in the middle and epicardial layers of myocardium. With the increasing left ventricular volume in eccentric hypertrophy, longitudinal strain decreases similarly as concentric hypertrophy, but circumferential strain remains at similar level as normotensive subjects.

Our findings on the left ventricular deformation in relation to the geometric patterns in hypertensive patients measured by 2D layer-specific speckle tracking analysis extend previous observations on this topic.<sup>[10,11]</sup> Two previous studies examined longitudinal and circumferential strain of myocardial layers in hypertensive subjects with or without left ventricular hypertrophy. In keeping with our present finding, these studies revealed that longitudinal strain in all 3 layers of myocardium decreased in left ventricular hypertrophy, whereas circumferential strain was in the descending order of hypertensive normal geometry, normotensive subjects, and left ventricular hypertrophy.<sup>[10,11]</sup> Our study further differentiated eccentric from concentric hypertrophy in hypertensive patients, and showed that patients with concentric and eccentric hypertrophy had similarly decreased levels of longitudinal strain, but only the former, not the latter, had significantly decreased circumferential strain.

Our observation on the disproportionate changes in longitudinal and circumferential strain across the 3 layers of myocardium with various left ventricular geometric patterns may have straightforward explanations. According to the theory postulated by Torrent-Guasp et al,<sup>[3]</sup> the myocardial band consists of 2 helices. A right-handed helix from the subendocardium changes gradually into a left-handed helix in the subepicardium. The subendocardium contains longitudinally oriented fibers with an angle of approximately  $80^\circ$  in regards to the circumferential orientation. However, the angle reduces toward the mid-wall, where the fibers are circumferentially oriented. The angle further diminishes to an oblique direction of approximately  $60^\circ$  in the subepicardium. The myocardial fiber organization predominantly defines the elements of myocardial deformation.<sup>[12]</sup> Thus, the endocardial region predominately contributes to the longitudinal mechanism of the left ventricle, whereas the middle and epicardial regions predominately contribute to the circumferential motion and twist.<sup>[13]</sup>

In addition, the indexes of left ventricular myocardial strain, which depict ventricular deformation at the regional level, are more accurate and sensitive in revealing left ventricular functional damages as opposed to the global performance of the heart.<sup>[14,15]</sup> Echocardiographic estimation of left ventricular function is routinely accomplished through visual interpretation of endocardial motion and myocardial thickening. However, this approach is subjective and operator-dependent, and it is difficult to accurately evaluate left ventricular layer function. In our study, there was no difference in left ventricular ejection fraction between these groups, whereas the left ventricular longitudinal strain in concentric remodeling was already reduced. Among the 3 layers of the myocardium, longitudinal subendocardial fibers are the most susceptible to adverse effects linked to ischemia, high blood pressure, and age-related interstitial fibrosis.<sup>[16]</sup> Myocardial ischemia usually extends from the endocardium to the epicardium. A previous study showed that longitudinal strain declined by approximately 50% when myocardial perfusion gradually decreased.<sup>[17]</sup> Therefore, longitudinal dysfunction may serve as a marker at an early stage of myocardial damage. In addition, progressive remodeling consists of both an increase in the size of the cardiomyocytes, and accumulation of fibrosis in the extracellular matrix, which are accompanied by increased left ventricular wall thickness and dimension. With the structural remodeling, longitudinal strain decreases and the circumferential strain increases to maintain normal ejection fraction. In the presence of hypertrophy, longitudinal strain decreases progressively, but the compensation in circumferential strain disappears. Our results suggest that left ventricular hypertrophy as a result of hypertension may lead to endocardial injury in the early phase, while sparing the middle and epicardium.

Several limitations of our study should be acknowledged. First, the sample size was small. Hence, future studies with a larger sample size are needed to corroborate our findings. Second, the data obtained from this study were cross-sectional and did not include a longitudinal observational follow-up to further elucidate the hypotheses. Third, some of our hypertensive patients used antihypertensive drugs. However, the small sample size did not allow investigation for the potential effects of antihypertensive treatment.

In conclusion, left ventricular deformation in hypertension occurs with various geometric patterns disproportionately in the endocardial, middle, and epicardial layers of myocardium and differently in the longitudinal and circumferential orientations. A clinical implication of our finding is that the strain measurement

might help in the refined division of cardiac function and in the early detection of cardiac dysfunction and failure. Prospective follow-up studies with regularly repeated 2D STE strain measurements are required to delineate the prognostic value of these early functional evaluations.

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### Author contributions

**Conceptualization:** Ting-Yan Xu.

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**Methodology:** Yan Yang.

**Project administration:** Yan Li.

**Supervision:** Ji-Guang Wang.

**Writing – original draft:** Ting-Yan Xu.

**Writing – review & editing:** Ji-Guang Wang.

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