Longitudinal Mitral Annulus Velocities Are Reduced in Hypertensive Subjects With or Without Left Ventricle Hypertrophy

Maria Cândida C. Borges, Roberta C.R. Colombo, José Geraldo F. Gonçalves, José de Oliveira Ferreira, Kleber G. Franchini

Abstract—Normotensive and hypertensive subjects with and without left ventricular (LV) hypertrophy (LV mass index [LVMI] >51 g/m²²) were examined by conventional echocardiography and tissue Doppler imaging of mitral annulus motion. The subgroups included nonobese normotensive subjects (n=16; age 51±9 years; 11 female; systolic blood pressure [SBP] 109±11 mm Hg, body mass index [BMI] 24±2.7 kg/m²; LVMI 32±5.5 g/m²²), nonobese hypertensive subjects without LV hypertrophy (n=16; age 54±12 years; 12 female; SBP 166±15 mm Hg; BMI 25±2.7 kg/m²; LVMI 42±5.5 g/m²²), and hypertensive subjects with LV hypertrophy (n=22; age 56±10 years; 10 female; SBP 181±19 mm Hg; BMI 26±2.3 kg/m²; LVMI 69±16 g/m²²). Ejection fraction was comparable among the subgroups, but midwall fractional shortening was reduced in hypertensive subjects with LV hypertrophy (≈26%). Isovolumic relaxation time was increased in subjects with LV hypertrophy, whereas mitral wave A velocity was increased in hypertensive subjects with and without LV hypertrophy. Tissue Doppler imaging mitral annulus systolic (Sₘ) and diastolic (Eₘ) velocities were reduced in subjects with and without LV hypertrophy compared with normotensive subjects. There was a positive correlation between Sₘ and Eₘ (r=0.68; P<0.0001) and negative correlations between these 2 variables and LVMI (Sₘ, r=−0.41; P=0.002; Eₘ, r=−0.56; P<0.0001). Thus, reductions in mitral annulus systolic and diastolic velocities parallel increases in LV mass in hypertensive subjects, beginning at LV mass within the clinically defined normal values. (Hypertension. 2006;47:854-860.)

Key Words: cardiac function | myocardium | echocardiography

Epidemiological studies have established a continuous relationship between the left ventricular (LV) mass and cardiovascular risk in the general and hypertensive population.¹ ² The poorer prognosis of hypertensive subjects with major increases in LV mass has been, in part, attributed to myocardial dysfunction,³ ⁴ but it remains unknown whether hypertensive subjects without clinically defined LV hypertrophy have subtle abnormalities of myocardial function. Echocardiographic indices derived from LV chamber dimensions and Doppler measurements of flow velocities have been used routinely for the assessment of systolic and diastolic function. However, such indices have been proved to be generally nonspecific and insensitive for the detection of minor abnormalities of cardiac relaxation and contraction.⁵ ¹² Tissue Doppler imaging (TDI) has been advocated as a reliable, rapid, and efficient method to assess myocardial function.¹³ TDI derived from early systolic and diastolic velocities of the mitral annulus longitudinal motion have been shown to correlate with contractile and relaxing activities of LV subendocardial layers.¹⁴ ¹⁷ Several studies have shown the usefulness of TDI mitral annulus motion velocities for the detection of LV subclinical myocardial dysfunction in patients with cardiomyopathy.¹⁸ ²¹ In hypertensive patients with LV hypertrophy, TDI mitral annulus systolic and diastolic velocities have been shown to be reduced.²² ²⁴ However, a comprehensive TDI approach has not been reported in hypertensive subjects without or with minor increases in LV mass. The present study examines whether, by using TDI early systolic and diastolic velocities, one might identify changes in LV myocardial contraction and relaxation in subsets of hypertensive subjects with and without clear-cut, clinically defined LV hypertrophy and normal LV ejection fraction (EF).

Methods

Study Population

The study included 16 normotensive and 38 untreated hypertensive subjects selected from the primary care unit of School of Medicine, Minas Gerais, Brazil; and Departments of Internal Medicine (K.G.F.) and Nursing (R.C.R.C.), School of Medicine, State University of Campinas, Campinas, Sao Paulo, Brazil. Correspondence to Kleber G. Franchini, Departamento de Clínica Médica, Faculdade de Ciências Médicas, Universidade Estadual de Campinas, Cidade Universitária “Zeferino Vaz,” 13081-970 Campinas, Sao Paulo, Brazil. E-mail franchin@unicamp.br

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Federal University of Triângulo Mineiro, based on body mass index (BMI) <27 kg/m². The subjects underwent resting transthoracic, Doppler, and tissue Doppler echocardiography as part of the clinical evaluation. After the initial imaging acquisition, hypertensive patients with and without LV hypertrophy were given 50 mg PO of captopril and rested for 2 hours before being examined in a new echocardiographic imaging acquisition session. All 54 of the subjects were asymptomatic; had no clinical evidence of diabetes, coronary artery, renal or cerebrovascular disease; were without Q-wave myocardial infarction or bundle branch block on their EKG; and were in sinus rhythm. Each study participant gave written informed consent. The study was approved by the local scientific ethics committee.

**Echocardiographic Examination**

The echocardiographic examination was performed by a skilled physician with 2.5- to 3.5-MHz transducers. Readings were performed by a second physician who was blinded to clinical characteristics of participants. LV dimensions and mass were assessed from 2D guided M-mode tracings according to American Society of Echocardiography recommendations. M-mode measurements were averaged from 5 cycles. LV end-systolic, end-diastolic, and stroke volumes were calculated with the use of Teichholz’s method. LV hypertrophy was defined with the use of cutoff points LV mass/height^{2.7} >51 for men and women. Relative wall thickness was calculated as twice the posterior wall in diastole divided by internal diastolic diameter and was used to estimate the LV geometry. Midwall fractional shortening (MWS) was assessed as described previously.

Mitral inflow velocity was examined with pulsed Doppler from the 4-chamber apical view according to a method described previously. The following indices describing the mitral inflow velocity curve were evaluated: peak early inflow velocity (E), peak atrial inflow velocity (A), and peak early/atrial velocity ratio (E/A). Deceleration time of early diastolic flow was measured from the peak E wave to the intercept of its descending limb with the velocity baseline. The isovolumic relaxation time (IVRT) was measured from the continuous wave Doppler at the level of LV outflow tract as the time from aortic valve closure to the onset of mitral inflow. Pulmonary vein flow velocities were obtained by placing the sample volume into the orifice of right upper pulmonary vein during the transthoracic examination. Velocities of systolic (S) and diastolic (D) pulmonary venous flow were measured and used to calculate the S/D ratio. The velocity and duration of pulmonary venous flow reversal during atrial contraction were also determined. Measurements were made in 3 cardiac cycles and averaged.

TDI was obtained by using the apical window at apical 4-chamber, apical 2-chamber, and long apical views for evaluating the septal, lateral, inferior, anterior, and posterior walls according to the previously described standard pulse-wave tissue Doppler technique. The sample volume was placed at the basal portion of the referred walls. The lowest possible wall filter settings and the lowest filter setting were used as recommended by the manufacturer. Peak spectral longitudinal contraction (S_m), initial (E_m), and final (A_m) diastolic velocities for 3 consecutive beats were analyzed, and E_m/A_m ratio was calculated.

The reproducibility of both acquiring and measuring S_m and E_m were determined in recordings obtained from 10 subjects (6 normotensive and 4 hypertensive). Samples (5 per patient) were repeatedly analyzed to assess the variability of S_m and E_m measurements. The results found by the same observer on different occasions (intrabserver variability) or by an independent observer (interobserver variability) were compared by using Bland-Altman analysis. Intrabserver S_m and E_m variability were <7%, whereas interobserver variabilities of these variables were <11%.

**Statistical Analysis**

Continuous variables are expressed as mean±SD. One-way ANOVA followed by the Bonferroni post hoc test was used to compare the clinical and echocardiographic data among the groups of subjects. Paired t test was used in the comparisons of blood pressure, heart rate, and echocardiographic variable intragroup changes. Univariate relations between variables were assessed by Pearson correlation coefficients. Hemodynamic and LV geometric and functional variables as determined by echocardiography that were significantly related to S_m and E_m in univariate analyses and did not exhibit excessive collinearity with each other were considered as potential independent variables in multiple linear regression analyses. Statistical significance was set at a 2-tailed value of P<0.05.

**Results**

**Clinical and Echocardiographic Structural Characteristics**

The clinical characteristics of the study subgroups are shown in Table 1. Subgroups consisted of normotensive subjects (n=16) and nonobese hypertensive subjects with (n=22) and without LV hypertrophy (n=16). Mean age, BMI, and gender distribution did not differ among the subgroups. Systolic blood pressure was significantly higher in the subjects with than in those without LV hypertrophy (9%), whereas heart rate did not differ among the subgroups of the present study (Table 2). The average LVMI (31%) and posterior wall thickness (22%), although within the range of clinically normal values, were significantly greater in the hypertensive subjects without LV hypertrophy than in normotensive subjects. Notably, of the 16 subjects enrolled in a subgroup of hypertensive subjects without LV hypertrophy, 4 were classified as having LV concentric remodeling, and 12 had normal LV geometry. In addition, the average relative wall thickness of hypertensive subjects with LV hypertrophy was higher than the cutoff value of 0.45. In this subgroup, 11 of 22 subjects had a concentric type of LV hypertrophy. In univariate analysis, LVMI and LV wall thickness were positively related to higher systolic (r=0.66 and r=0.55; P<0.0001) and diastolic (r=0.63 and r=0.56; P<0.0001) blood pressure. The average systolic and diastolic LV diameters of hypertensive subjects were within the clinically defined normal range but trended toward an increase in

<table>
<thead>
<tr>
<th>TABLE 1. Characteristics of the Study Group</th>
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<td>Characteristic</td>
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<tr>
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<tr>
<td>Age, y</td>
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<tr>
<td>Gender, F</td>
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<tr>
<td>BMI, kg/h²</td>
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<tr>
<td>LV mass, g</td>
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<td>LVMI, g/h²</td>
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<td>PWTh, mm</td>
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<td>LVDD, mm</td>
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<td>LVSd, mm</td>
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<td>LAD, mm</td>
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</table>

NT indicates normotensive subjects; LVH⁺, hypertensive subjects without LV hypertrophy; LVH⁺, hypertensive subjects with LV hypertrophy; PWTh, LV posterior wall thickness; RWT, relative wall thickness; LVDD, LV end diastolic diameter; LVSD, LV end systolic diameter; LAD, left atrium diameter. Values are mean±SD.

*P<0.05 vs NT subjects.
†P<0.05 vs LVH⁺.
Systolic Parameters

Echocardiographic parameters of LV systolic function at baseline and after blood pressure fall by captopril are shown in Table 2. LV EF was within the normal range and with comparable values among the subgroups of this study. The averaged MWS was reduced in the subjects with LV hypertrophy, as compared with values of normotensive or hypertensive subjects without LV hypertrophy. In analyses that included all of the subjects enrolled in this study, MWS correlated negatively with LVMI (r = 0.75; P < 0.0001), LV posterior wall thickness (r = −0.84; P < 0.0001), and systolic blood pressure (r = −0.51; P < 0.0001).

We next quantified LV systolic function by determining TDI mitral annular peak systolic velocity (Sm) at 5 different regions of the left ventricle. Figure 1A shows the regional peak systolic wall motion velocities in the 3 subgroups of the present study. In hypertensive subjects without LV hypertrophy the Sm was found to be depressed in anterior and posterior walls, whereas in subjects with LV hypertrophy, a significant depression was found in anterior, lateral, and posterior LV walls. Figure 1B shows the distribution of the average Sm in the 3 subgroups of the present study. A significant negative correlation (r = −0.41; P = 0.002) was found between the averaged Sm and LVMI (Figure 1C), but no correlation was found between Sm and LV geometry. In univariate analysis, Sm was negatively related to systolic blood pressure (r = −0.60; P < 0.0001) and LVMI (r = −0.43; P = 0.001). In the multivariate analysis, only systolic blood pressure was independently predictive of lower Sm. LVMI approached but did not attain statistical significance.

We next assessed LV systolic function and Sm of hypertensive subjects 2 hours after blood pressure fall by captopril (Table 2). Reductions of systolic and diastolic blood pressure were similar in hypertensive subjects without (systolic blood pressure, 27 ± 3.9; diastolic blood pressure, 18 ± 3.8 mm Hg) and with (systolic blood pressure, 31 ± 3.5; diastolic blood pressure, 16 ± 2.4 mm Hg) LV hypertrophy. These reductions of blood pressure were accompanied by increases in EF and MWS. However, no significant change was observed in the values of Sm after the reductions of blood pressure in hypertensive subjects.

Diastolic Parameters

LV diastolic function was evaluated with transmitral and pulmonary vein Doppler echocardiography. As shown in Table 2, no difference was found in the peak E velocity among the subgroups of the present study. The peak A velocity was significantly higher in the 2 subgroups of hypertensive as compared with normotensive subjects, but the E/A ratio was significantly lower in both subgroups of hypertensive as compared with normotensive subjects. IVRT was significantly longer in hypertensive subjects compared with subjects without LV hypertrophy or normotensive subjects. In univariate analysis, peak A velocity correlated positively with higher systolic blood pressure (r = 0.50; P < 0.0001), and IVRT was positively related to LVMI (r = 0.48; P = 0.0002) and systolic blood pressure (r = 0.38; P = 0.004). Indices derived from Doppler echocardiography

TABLE 2. Blood Pressure, Heart Rate, and LV Function

<table>
<thead>
<tr>
<th>Variable</th>
<th>NT Baseline</th>
<th>Baseline Captopril</th>
<th>Baseline Captopril</th>
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</thead>
<tbody>
<tr>
<td>SBP, mm Hg</td>
<td>109 ± 11</td>
<td>166 ± 15*</td>
<td>140 ± 12‡</td>
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<tr>
<td>DBP, mm Hg</td>
<td>71 ± 5</td>
<td>102 ± 11*</td>
<td>88 ± 8‡</td>
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<tr>
<td>HR, bpm</td>
<td>58 ± 6.6</td>
<td>63 ± 9.1</td>
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**Conventional echocardiography indices**

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<th>Baseline Captopril</th>
<th>Baseline Captopril</th>
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<tbody>
<tr>
<td>EF, %</td>
<td>68 ± 5.2</td>
<td>68 ± 6.5</td>
<td>74 ± 6.9‡</td>
</tr>
<tr>
<td>MWS, %</td>
<td>19 ± 1.9</td>
<td>16.7 ± 2.6</td>
<td>18.1 ± 2.5‡</td>
</tr>
<tr>
<td>Peak E, cm/s</td>
<td>69 ± 13</td>
<td>69 ± 14</td>
<td>67 ± 13</td>
</tr>
<tr>
<td>Peak A, cm/s</td>
<td>46 ± 9</td>
<td>70 ± 18*</td>
<td>67 ± 23*</td>
</tr>
<tr>
<td>Ratio E/A</td>
<td>1.5 ± 0.4</td>
<td>1.1 ± 1.6*</td>
<td>1.2 ± 1.2*</td>
</tr>
<tr>
<td>IVRT, ms</td>
<td>89 ± 12</td>
<td>111 ± 18</td>
<td>113 ± 19*</td>
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**TDI indices**

<table>
<thead>
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<th>Variable</th>
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<th>Baseline Captopril</th>
<th>Baseline Captopril</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sm, cm/s</td>
<td>8.1 ± 1.2</td>
<td>6.5 ± 1.5*</td>
<td>7.1 ± 1.4</td>
</tr>
<tr>
<td>Em, cm/s</td>
<td>11.9 ± 2</td>
<td>8.5 ± 2.5*</td>
<td>9 ± 2.9</td>
</tr>
<tr>
<td>Am, cm/s</td>
<td>8.3 ± 1.2</td>
<td>9.7 ± 1.9</td>
<td>9.5 ± 2.3</td>
</tr>
</tbody>
</table>

**SBP** indicates systolic blood pressure; **DBP**, diastolic blood pressure; **HR**, heart rate; **FS**, fractional shortening; **CWS**, circumferential end-systolic wall stress.

*P < 0.05 vs normotensive subjects.

†P < 0.05 vs LVH.

‡P < 0.05 vs baseline.

hypertensive subjects with LV hypertrophy in comparison with the values of normotensive subjects. The diameter of the left atrium was significantly higher in hypertensive subjects with LV hypertrophy, although still within the clinically defined normal range.
of pulmonary venous flow did not differ among the subgroups of the present study.

The averaged EM velocity (Figure 2A) was reduced at all 5 of the assessed LV regions of hypertensive subjects with or without LV hypertrophy as compared with normotensive subjects. Figure 2B shows the distribution and mean values of averaged EM of the 3 subgroups. Significant negative correlation was found among the averaged EM velocity, systolic blood pressure ($r=-0.72; P<0.0001$), and LVMI ($r=-0.56; P<0.0001$; Figure 2C). In multivariate analysis, systolic blood pressure was the strongest determinant for the EM followed by LVMI. Univariate analysis of EM with Doppler echocardiographic data indicated that EM was correlated with peak A ($r=-0.63; P<0.0001$) and IVRT ($r=0.62; P<0.0001$). No correlation was found between EM and peak E index.

The average values of AM were comparable in the subgroups of the present study (Table 2). Reductions of blood pressure by captopril did not change conventional or TDI-derived indices of LV diastolic function.

Next, the relationship between the early peak of systolic and diastolic mitral annulus motion was tested by regression analysis. A significant positive correlation ($r=0.68, P<0.0001$) was found between SM and EM (Figure 3).
MWS, SM was significantly reduced in the hypertensive without clinically defined LV hypertrophy. Distinct from of mitral annulus systolic motion, in hypertensive patients subtle reduction in LV systolic function, as assessed by TDI hypotrophy. Our present study extends these data to show dysfunction may occur in hypertensive patients with mild LV systolic function by MWS have indicated that LV systolic demonstrate of abnormalities in regional and global dia-
muscle is dependent on LV loading conditions than the LV geometry-
tions in LV mass after treatment with losartan have been
anterolateral and inferior scars. The abnormalities of TDI mitral annulus motion were correlated with LVMI. Notably, hypertensive subjects without clinically defined LV hypertrophy had higher LVMI than the normotensive subjects, implying that even mild subclinical increases in LV mass might be associated with reductions in the velocities of mitral annulus motion. Factors such as fibrosis and reductions in subendocardial flow, as well as pressure overload might be involved in the reductions of TDI mitral annulus motion of the hypertensive subjects. The available data indicate that fibrosis and limitations in subendocardial flow reserve appear early in the evolution of hypertensive heart disease and could possibly reduce the systolic and diastolic mitral annulus motion. In addition, our findings that SM and EM were correlated with blood pressure in either univariate or multivariate analysis suggest that the reduced TDI indices seen in hypertensive subjects might be related to pressure overload. Alternatively, one might argue that the lack of change of SM and EM after blood pressure reduction by captopril would indicate that TDI indices are independent of LV loading conditions. However, the fact that treatment with captopril did not bring blood pressure to the levels of normotensive subjects makes it possible that the lack of change in SM or EM was caused by the residual increases in LV load. Thus, further studies are necessary to clarify whether high blood pressure levels, per se, contribute to the reductions of contractile and relaxing activities in hypertensive subjects with minor changes in LV mass. In this context, it is important to note that it remains controversial whether TDI indices are dependent or not on load conditions.16,37

A recent study24 has shown the value of TDI-derived mitral annulus systolic and diastolic velocities to the prognosis of unselected groups of hypertensive subjects. Moreover, reductions in LV mass after treatment with losartan have been shown to be followed by significant increases of SM and EM.38 It would be interesting to investigate whether long-term antihypertensive treatment is accompanied by normalization

Discussion

The data obtained in the present study indicated that systolic and early diastolic longitudinal mitral annulus velocities are reduced in untreated nonobese hypertensive subjects with and without clinically defined LV hypertrophy compared with age- and BMI-matched normotensive subjects. Reductions of TDI longitudinal early systolic and diastolic velocities of mitral annulus parallel the increases in LV mass, beginning at LV mass levels within the normal range, as defined by current echocardiographic criteria. Overall, these data indicate that both the contractile and relaxing myocardial function may already be depressed in hypertensive subjects with mild or no increases in LV mass, supporting the potential value of using TDI velocities in the evaluation of cardiac function in hypertensive subjects.

Several lines of evidence indicate an association between LV hypertrophy and systolic dysfunction. Although such association is well defined in patients with major increases in LV mass, data provided by studies8,9,12 that assessed LV systolic function by MWS have indicated that LV systolic dysfunction may occur in hypertensive patients with mild LV hypertrophy. Our present study extends these data to show subtle reduction in LV systolic function, as assessed by TDI of mitral annulus systolic motion, in hypertensive patients without clinically defined LV hypertrophy. Distinct from MWS, SM was significantly reduced in the hypertensive subjects without LV hypertrophy. In addition, MWS and EF, but not SM, were significantly increased after blood pressure reduction with captopril, suggesting that SM might be less dependent on LV loading conditions than the LV geometry-based indices of systolic function.

In addition, myocardial relaxation, as detected by TDI of mitral annulus diastolic motion, was depressed in all segments of the left ventricle in hypertensive patients with or without LV hypertrophy. These results confirm previous demonstrations of abnormalities in regional and global diastolic indices in hypertensive patients with or without LV hypertrophy.22–24,30 The reductions in EM were accompanied by an enlargement of the left atrium in the hypertensive subjects with LV hypertrophy and a trend toward an enlargement in those without LV hypertrophy. This agrees with previous data31 indicating the left atrial enlargement as an early sign of reduced diastolic LV function. In contrast with the reductions of myocardial systolic function, which were restricted to the anterior and posterior walls in hypertensive subjects without LV hypertrophy, reductions of EM were uniformly distributed along the LV wall. This might indicate that the impairment of myocardial diastolic function in hypertensive patients is either more intense or appears earlier than the impairment of myocardial systolic function. However, our data showing the significant reductions of SM and EM in hypertensive patients with or without LV hypertrophy, as well as the correlation between these 2 parameters, suggest that depression of myocardial relaxation is paralleled by depression in contractile activity, beginning at levels of LV mass below the cutoff values. Accordingly, it has been shown previously32 that LV contractile activity, as assessed by TDI, correlates with relaxation, reflecting the fact that early myocardial LV relaxation is closely coupled to the previous systolic phase of the cardiac cycle.

The findings of the present study bring into discussion the issue of systolic and diastolic myocardial dysfunction in hypertensive patients with mild or without LV hypertrophy. The abnormalities of TDI mitral annulus motion were correlated with LVMI. Notably, hypertensive subjects without clinically defined LV hypertrophy had higher LVMI than the normotensive subjects, implying that even mild subclinical increases in LV mass might be associated with reductions in the velocities of mitral annulus motion. Factors such as fibrosis and reductions in subendocardial flow, as well as pressure overload might be involved in the reductions of TDI mitral annulus motion of the hypertensive subjects. The available data indicate that fibrosis and limitations in subendocardial flow reserve start early in the evolution of hypertensive heart disease and could possibly reduce the systolic and diastolic mitral annulus motion. In addition, our findings that SM and EM were correlated with blood pressure in either univariate or multivariate analysis suggest that the reduced TDI indices seen in hypertensive subjects might be related to pressure overload. Alternatively, one might argue that the lack of change of SM and EM after blood pressure reduction by captopril would indicate that TDI indices are independent of LV loading conditions. However, the fact that treatment with captopril did not bring blood pressure to the levels of normotensive subjects makes it possible that the lack of change in SM or EM was caused by the residual increases in LV load. Thus, further studies are necessary to clarify whether high blood pressure levels, per se, contribute to the reductions of contractile and relaxing activities in hypertensive subjects with minor changes in LV mass. In this context, it is important to note that it remains controversial whether TDI indices are dependent or not on load conditions.16,37

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of \( S_M \) or \( E_M \), as well as whether this would improve the prognosis of hypertensive subjects with LV mass values lower than the traditional upper normal limits.

### Perspectives

The present study provides evidence that there are close correlations between LVMI and longitudinal mitral annulus systolic and diastolic velocities, reflecting unsuspected reductions of contractile and relaxing activities in hypertensive adults with mild or no increases in LV mass. The use of TDI mitral annulus velocities may aid in the identification of hypertensive patients at high risk of future congestive heart failure. We speculate that reductions in TDI indices should be investigated as additional risk markers in hypertensive subjects without major cardiac structural abnormality.

### Acknowledgments

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