Original Article

The Relation between Left Ventricular Geometric Patterns and Left Ventricular Midwall Mechanics in Hypertensive Patients

Yinong JIANG, Peng QU*, Yanchun DING*, Daozi XIA*, Hongyan WANG*, and Xiaohong TIAN*

To evaluate the alteration of myocardial contractility in hypertensive patients with different left ventricular geometric patterns by the end-systolic stress-midwall fractional shortening relation. Echocardiography was applied to study the left ventricular geometry and cardiac function among 117 cases of essential hypertension, with 45 normal cases as control(s). Left ventricular mass index (LVMI) and relative wall thickness (RWT) were calculated using echocardiographic data. All patients were divided into four kinds of left ventricular geometry pattern based on LVMI and RWT. Patients of the eccentric hypertrophy group suffered the most serious damage of left ventricular systolic function. Myocardial contractility shown by end-systolic stress-midwall fractional shortening relation was significantly decreased in the concentric remodeling group, eccentric hypertrophy group and concentric hypertrophy group, and those with concentric hypertrophy showed the worst contractility. The degree of myocardial contractility damage was different in patients with different left ventricular geometric patterns. Geometric changes may have compensated for the reduction of myocardial contractility in some phases in order to maintain the normal pump function. (Hypertens Res 2002; 25: 191–195)

Key Words: hypertension, left ventricular geometry, myocardial contractility

Introduction

Theoretically, the left ventricle adapts to a sustained pressure overload through a concentric hypertrophic process that results in an increased left ventricular (LV) wall thickness while maintaining a normal chamber volume (1). Thus, despite high intracavitary pressures, systolic wall stress (afterload) is not increased and fiber shortening (ejection fraction) is preserved. But in humans, during the development of hypertension there is not only the concentric hypertrophy but also other types of LV geometric patterns. In humans, however, the development of hypertension is characterized not only by concentric hypertrophy but also by other types of LV geometric patterns. The LV geometry changes followed by the alteration of myocardial contractility. And patients with different LV geometry patterns tend to have different prognoses (2–5). In some studies, LV ejection fraction (EF) and LV fractional shortening (FS) have been measured at the endocardium, reflecting chamber dynamics but not necessarily providing a direct measurement of myocardial fiber shortening (6, 7). Such a method might overestimate myocardial function in the presence of LV hypertrophy.

The midwall fractional shortening (mFS) and mFS/end-systolic wall stress relation is more accurate and more appropriate for estimating the myocardial contractility (8, 9). Accordingly, we assessed the midwall left ventricular mechanics in normotensive and hypertensive subjects in order to...
Table 1. Clinical Profiles and Echocardiographic Characteristics in Normotensive and Hypertensive Subjects

<table>
<thead>
<tr>
<th></th>
<th>NG (n = 37)</th>
<th>CR (n = 30)</th>
<th>EH (n = 20)</th>
<th>CH (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51.9 ± 6.7</td>
<td>54.6 ± 8.2</td>
<td>55.2 ± 8.3</td>
<td>56.1 ± 9.4</td>
</tr>
<tr>
<td>Male/Female</td>
<td>22/23</td>
<td>20/17</td>
<td>15/15</td>
<td>12/8</td>
</tr>
<tr>
<td>Duration (years)</td>
<td>3.7 ± 4.6</td>
<td>5.7 ± 6.5</td>
<td>2.3 ± 3.1</td>
<td>2.5 ± 3.3</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.80 ± 0.18</td>
<td>1.81 ± 0.16</td>
<td>1.81 ± 0.14</td>
<td>1.85 ± 0.16</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>71 ± 7</td>
<td>71 ± 11</td>
<td>73 ± 10</td>
<td>73 ± 9</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120 ± 9</td>
<td>163 ± 13*</td>
<td>175 ± 15*</td>
<td>165 ± 15*</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>72 ± 5</td>
<td>97 ± 12*</td>
<td>98 ± 12*</td>
<td>99 ± 13*</td>
</tr>
<tr>
<td>LAD (cm)</td>
<td>2.58 ± 0.25</td>
<td>2.90 ± 0.46</td>
<td>3.02 ± 0.43*</td>
<td>3.15 ± 0.51*</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>4.65 ± 0.18</td>
<td>4.71 ± 0.16</td>
<td>4.30 ± 0.27*</td>
<td>5.39 ± 0.25*</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>3.15 ± 0.23</td>
<td>3.35 ± 0.21*</td>
<td>3.10 ± 0.19*</td>
<td>4.13 ± 0.28*</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>95.2 ± 11.8</td>
<td>102.9 ± 8.2*</td>
<td>103.7 ± 11.2*</td>
<td>163.8 ± 24.0*</td>
</tr>
<tr>
<td>RWT</td>
<td>0.40 ± 0.03</td>
<td>0.42 ± 0.02</td>
<td>0.51 ± 0.04*</td>
<td>0.43 ± 0.02*</td>
</tr>
</tbody>
</table>

NG, normal geometry; CR, concentric remodeling; EH, eccentric hypertrophy; CH, concentric hypertrophy; BSA, body surface area; HR, hear rate; SBP, systolic blood pressure; DBP, diatolic blood pressure; LAD, left atrial dimension; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LVMI, left ventricular mass index; RWT, relative wall thickness. *p < 0.05, vs. control; †p < 0.05, vs. NG; ‡p < 0.05, vs. CR; ‡p < 0.05, vs. EH.

evaluate LV contractility and its relation to the different LV geometry patterns.

Subjects and Methods

Study Population

One-hundred-and-seventeen hypertensive patients and forty-five normotensive controls participated in this study. Informed consent for all procedures was obtained from each patient. All hypertensive patients were untreated or had been free from medication for at least two weeks before the investigation. Blood pressure was determined by sphygmomanometer before Echo-Doppler examination.

Echocardiographic LV Measurement

Echocardiography was performed using an Ultramark 9 echocardiograph machine with a 2.5MHz transducer (ATL Inc., Seattle, USA) according to the Penn convention (10). The interventricular septal end-diastolic thickness (IVSTd) and end-systolic thickness (IVSTs), left ventricular posterior wall end-diastolic thickness (LVPWTd) and end-systolic thickness (LVPWTs), left ventricular end-diastolic dimension (LVEDD) and end-systolic dimension (LVESD) were measured by five averaged cardiac cycles.

Left ventricular mass (LVM) was estimated using the formula from Devereux and Reichek (11): LVM(g) = 1.04 [(LVEDD + LVPWTd + IVSTd) - LVEDD] - 13.6. The left ventricular mass index (LVMI) = LVM/BSA, where BSA (body surface area) (g/m²) = 71.84 height (cm)0.425 weight (kg)0.125 10^-4. Relative wall thickness (RWT) was calculated as follows: RWT = (LVPWTd + IVSTd)/LVEDD (12). A normal left ventricular mass index was defined as 125 and 120g/m² (13) in males and females, respectively. A partition value of smaller than 0.45 for RWT was used both for normal males and females (12). All patients were divided into four groups based on LVMI and RWT according to the method of Ganau et al. (14). Patients with increased LVMI and increased RWT were considered to have concentric hypertrophy, and those with increased LVMI and normal RWT were considered to have eccentric hypertrophy. Those with normal LVMI and increased or normal RWT were considered to have concentric remodeling or normal geometry, respectively.

Calculation of Midwall Fractional Shortening and LV End-Systolic Wall Stress

The ejection fraction (EF) was calculated using a standard formula. Endocardial shortening was calculated as the difference between end-diastolic and end-systolic short-axis dimension divided by end-diastolic dimension: FS = (LVEDD - LVESD)/LVEDD 100%. Midwall fractional shortening was calculated by the formula of de Simone (9): mFS = [(LVEDD + LVPWTd + IVSTd) - LVEDD] / (LVEDD + LVPWTd + IVSTd) 100%.

LV meridional end-systolic stress (MESS) was calculated: MESS = 0.334 SBP LVESD / [LVPWTs [1 + (LVPWTs/LVEDD)].

Statistical Analysis

All values are expressed as the mean ± SD. Differences be-
between and within groups of normotensive and hypertensive subjects were assessed using one-factor ANOVA with post hoc comparisons by Fisher’s protected least significant difference test. Correlation coefficients were obtained using linear regression. A probability value less than 0.05 was defined as indicating statistical significance. SPSS statistical software, version 8.0 was used to perform the analyses.

### Results

#### LV Geometry Patterns

Table 1 summarizes the clinical profiles and echocardiographic characteristics in normotensive controls and hypertensive patients. There were no significant differences in sex, BSA, or heart rate (HR) among the five groups. There were no significant differences in SBP or DBP among the four different LV geometry patterns of hypertensive patients.
LV Midwall Performance

LV midwall performance in hypertensive patients with different patterns of LV geometry is shown in Table 2 and Table 3. The systolic function expressed as EF, FS, and mFS was significantly decreased in hypertensive patients with left ventricular concentric remodeling, eccentric hypertrophy and concentric hypertrophy, and the after-load expressed as MESS was increased. There were no significant differences of cardiac systolic function between normotensive control and normal geometry hypertensive patients.

Midwall Fractional Shortening and LV Meridional End-Systolic Stress

There was a significant inverse relation between mFS and LVMI in all the patients with hypertension. The hypertrophied hearts exhibited depressed midwall fiber shortening (Fig. 1). The relation of midwall fractional shortening to meridional end-systolic stress was significant in both normal and hypertensive subjects. Figure 2 shows the relation of mFS to MESS in normotensive subjects \( y = -0.061x + 32.5, R^2 = 0.376, p < 0.001 \).

The ratios of observed left ventricular midwall fractional shortening to the values predicted by meridional end-systolic stress in normotensive subjects were calculated in hypertensive patients with different left ventricular geometry (Table 3). The ratio of observed to predicted midwall fractional shortening in normotensive subjects was 100.07 ± 9.89. In the normal geometric hypertensive patients, this ratio was significantly lower than that in normotensive subjects \( p < 0.05 \). In the left ventricular concentric remodeling hypertensive patients, the ratio was significantly reduced \( p < 0.05 \) compared with that for the normal left ventricular geometric hypertensive patients. The ratio in the left ventricular concentric hypertrophy geometry group was greatly reduced compared with those of the other left ventricular geometric groups.

Discussion

Left ventricular ejection fraction (EF) and endocardial fractional shortening (FS) reflected the left ventricular pump function and did not directly reflect the ventricular contractility. They were influenced by ventricular pre-load and after-load and were not sufficiently sensitive for the evaluation of myocardial damage \((6, 7)\). It has been reported that use of the relation of stress to FS could rule out the influence of ventricular after-load and reflect the myocardial contractility \((6)\). However, some researchers have also reported that using the relation of stress to FS might overestimate the myocardial contractility in hypertensive subjects with normal or hypertrophied left ventricular geometry \((15, 16)\). These might be artificially influenced by relating endocardial shortening to the average value of end-systolic stress across the thickness of the left ventricular wall. Some studies have reported that midwall fractional shortening \( (mFS) \) was physiologically more appropriate than endocardial fractional shortening for evaluating the systolic function of hypertensive patients, especially when left ventricular hypertrophy existed \((8, 9)\). Using the relation of mFS and meridional end-systolic stress \( (MESS) \) would be more appropriate for the evaluation of myocardial contractility in hypertensive patients. And the decreased mFS might be an independent risk factor for the prediction of cardiovascular events in hypertensive patients \((17)\).

The systolic function of the heart was related not only to ventricular pre-load and after-load, but also to the myocardial contractility. In the present study, there were no differences of systolic and diastolic blood pressure among the different LV geometric patterns of hypertensive patients. The correlation equation of mFS to MESS in the normotensive subjects was used to calculate the predicted values of mFS for the different LV geometric hypertensive subjects. The results showed that the predicted values of mFS in LV eccentric hypertrophy and concentric hypertrophy hypertensive subjects were significantly reduced and were lower than the actually observed values, and so were those in the normal geometry and concentric remodeling hypertensive patients. Because the predicted mFS was independent of the ventricular after-load and reflected myocardial contractility, the results of the present study suggested that the myocardial contractility was damaged in hypertensive patients with LV hypertrophy or remodeling. The ratio of observed to predicted mFS in patients with the concentric hypertrophic pattern was less than that in other LV geometric patterns of hypertension, indicating that the damage of myocardial contractility in concentric LV hypertrophic patients was the most severe.

During the development of hypertension, to assist with the adaptation to the increasing pressure and volume load, left ventricular geometry alteration and left ventricular hypertrophy occur \((18)\). Consequently, the metabolites of the hypertrophied myocardium are changed, the proportion of the myosin light chain to the myosin heavy chain is imbalanced, and the activity of the myosin isoenzyme is decreased, all of which could contribute to the decreased contractility of the myocardium that ultimately results in the decreasing heartpump function.

In the present study, an inverse relation between LVMI and mFS was found. This suggested that, along with the development of left ventricular hypertrophy, the systolic function of the left ventricle is deteriorated. According to the principle of Laplace, stress is positively related to intra-ventricular pressure and the dimensions of the left ventricle, and inversely related to the thickness of the ventricular wall. The increasing of RWT could decrease the left ventricular wall stress, i.e., the after-load. The decreased after-load might improve left ventricular systolic function. The geometric change of the left ventricle during the development of hypertension might play some role in compensating and maintain-
ing left ventricular pump function during the decrease in myocardial contractility. These results might partly explain why the contractility in LV concentric hypertrophy patients was significantly decreased compared to that in the left ventricular concentric remodeling patients, and why there was no significant difference of the left ventricular pump function between them.

In conclusion, our study demonstrated that the degree of myocardial contractility damage was different in the patients with different left ventricular geometric patterns. Geometric changes may have compensated for the reduction of myocardial contractility in some phases in order to maintain normal pump function.

References

2. Koren MJ, Devereux RB, Casale PN, *et al*: Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Ann Intern Med* 1991; 114: 345–352.