Left Ventricular Mechanical Efficiency in Hypertensive Patients With and Without Increased Myocardial Mass and With Normal Pump Function

Alain Nitenberg, Alain Loiseau, and Isabelle Antony

Left ventricular hypertrophy (LVH) is a physiologic process of adaptation of the heart to mechanical load increase. Despite depression of left ventricular contractile performance, mechanical efficiency and ventriculoarterial coupling are preserved in hypertensive patients with LVH. To assess the differences between patients with and without LVH, left ventricular contractile performance and the ventriculoarterial coupling were compared in two groups of hypertensive patients with similar body surface area and arterial pressures, and normal pump function: 30 patients with LVH (group 1) and 23 without LVH (group 2). Left ventricular angiography coupled with simultaneous recording of pressures with a micromanometer were used to determine end-systolic stress-to-volume ratio (ESSVR), end-systolic elastance (Ees), effective arterial elastance (Ea), external work (EW), and pressure–volume area (PVA). Myocardial contractile performance, assessed by Ees normalized by myocardial mass and by ESSVR, was lower in group 1 than in group 2 (1.23 \pm 0.28 \text{ vs } 1.89 \pm 0.48 \text{ mm Hg/mL/100 g}, and 3.85 \pm 0.99 \text{ vs } 5.13 \pm 0.56 \text{ g/cm}^2/\text{mL}, respectively, both P < .001). Ventriculoarterial coupling evaluated through Ea/Ees ratio, and mechanical efficiency evaluated through EW/PVA ratio, were similar in the two groups (0.53 \pm 0.08 \text{ vs } 0.51 \pm 0.05, and 0.78 \pm 0.03 \text{ vs } 0.80 \pm 0.02, respectively, NS). In conclusion, this study shows that ventriculoarterial coupling and mechanical efficiency are comparable in hypertensive patients with and without LVH. These results suggest that in hypertensive patients, the matching between left ventricular performance and arterial load and the energy transfer are preserved either through left ventricular hypertrophy with moderate depression of myocardial contractile performance or through enhancement of myocardial contractile performance in patients with normal left ventricular mass.


Key Words: Hypertension, left ventricular hypertrophy, ventriculoarterial coupling, left ventricular performance, mechanical efficiency.

In arterial hypertension, the adaptation of the heart to mechanical load increase is mainly due to left ventricular hypertrophy (LVH), which is a physiological process that preserves a normal pumping ability. Despite the fact that left ventricular adaptation to hypertension may lead to different patterns of hypertrophy, a compensatory ventricular response to arterial hypertension generally leads to increased wall thickness, which normalizes wall stress and preserves pumping ability. However, numerous hypertensive patients who do not develop LVH despite the increase in arterial load have normal pump function.

Several studies have been dedicated to the relationship between left ventricular contractile performance and hemodynamic load in human arterial hypertension. This relationship can be assessed by the ventriculoarterial coupling, a concept that enables one to evaluate the matching between the left ventricular function and the mechanical properties of the arterial system. In normal subjects it has been demonstrated that ventriculoarterial coupling is set toward maximal mechanical efficiency (ie, maximal external work/myocardial oxygen consumption ratio), and that effective arterial elastance is nearly one half that of ventricular elastance. On the other hand, progressive ventricular dysfunction results in maximization of external work at the expense of mechanical efficiency. We have previously shown that hypertensive left ventricular hypertrophy preserves a normal ventriculoarterial coupling despite a slight depression of myocardial contractile performance.

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The aim of the present study was to compare ventriculoarterial coupling and left ventricular mechanical efficiency in patients with and without left ventricular hypertrophy, as well as normal pump function.

Methods
Patient Selection

The study population comprised 53 patients with a well-established history of elevated blood pressure >140/90 mm Hg, with at least four sets of readings taken at 1-week intervals. Hypertension had been recently diagnosed in 35 patients who had never been treated, and 18 patients were included after discontinuation of all treatment with the exception of nitrates ≥3 weeks before cardiac catheterization. Patients were kept off of therapy because they had no unstable angina and no evidence of severe coronary artery disease on exercise electrocardiogram or thallium. Only long-duration nitrates were given and were discontinued 48 h before the study. During these 3 weeks, patients were informed that they should immediately contact their referring physician in case of any change in their symptoms. The number of patients who had never been treated was not very different in the hypertrophy group (22/35, 63%) and in the normal left ventricular mass group (13/18, 72%). All patients underwent coronary angiography for the diagnosis of chest pain and evidenced abnormal exercise electrocardiography or thallium stress tests consistent with ischemia. Patients with valvular heart disease or diabetes mellitus were excluded. All patients were included in the study on the basis of coronary artery stenosis ≥30% at coronary arteriography. The study protocol was approved by the local institutional review committee. Written informed consent was obtained in all patients before cardiac catheterization.

Left ventricular systolic function, as assessed by two-dimensional and M-mode echocardiography, was normal in all hypertensive patients. Echocardiographically normal systolic function was defined by the absence of segmental wall motion abnormalities and a fractional shortening ≥30%. Left ventricular dimensions and septal and posterior wall thicknesses were measured at end-diastole according to the American Society of Echocardiography guidelines. The left ventricular mass index was calculated at end-diastole using the Penn convention and was used to assign patients in two groups. Based on criteria published by Ganau et al., men with a left ventricular mass index >111 g/m² and women with a left ventricular mass index >106 g/m² composed a group of 30 patients with LVH (group 1). Men with a left ventricular mass index ≤111 g/m² and women with a left ventricular mass index ≤106 g/m² composed a group of 23 patients without LVH (group 2). Patients with concentric remodeling and septal/posterior wall thickness ratio ≥1.2 were excluded.

Catheterization Procedure

Nitrates (when given) were discontinued 48 h before the study. All patients were in the fasting state for ≥12 h before the procedure. No premedication was administered. Lidocaine, 1%, was used for local anesthesia.

After documentation of coronary artery lesions, a 7F double-tipped micromanometer angiographic catheter (Cordis/Sentron Laboratory, Roden, The Netherlands) was placed into the left ventricle through a femoral artery using a 7F sheath. A 15-min delay was observed to eliminate the effects of contrast material. Heart rate, aortic, and left ventricular end-diastolic and systolic pressures were calculated by means of a catheterization data analysis computer system (5600 M, Hewlett-Packard, Andover, MA) that performed on-line analysis on nine beats for averaging out respiratory variations. Left ventricular angiography (50 frames/sec) was obtained in a 30° right anterior oblique projection (35 mL nonionic contrast medium, 12 mL/sec) with simultaneous recording of left ventricular pressure (200 mm/sec) and a frame marker.

Data Analysis and Calculation

Methods used for data analysis and calculation have been previously described in detail. Briefly, left ventricular volumes were calculated from monoplane angiograms by means of the area–length method. The echocardiographic left ventricular mass (LVM) was assumed to be constant and was used to calculate left ventricular myocardial wall volume, end-diastolic (hED), and end-systolic equatorial wall thicknesses (hES) using the long axis measured on left ventricular angiography. Average left ventricular equatorial end-systolic wall stress (ESS) was calculated using the formula of Falsetti et al. for an ellipsoidal geometry of the left ventricle.

Ventriculoarterial coupling was analyzed through the left ventricular output impedance, expressed by the end-systolic elastance (Ees) obtained from the end-systolic pressure–volume relation, and the arterial input impedance expressed by the effective arterial elastance (Ea) obtained from the arterial end-systolic pressure–stroke volume relation. The end-systolic elastance was obtained with the single-beat analysis method described by Takeuchi et al., which assumes that the end-systolic pressure–volume relation is roughly linear for physiological conditions when myocardial contractility does not change. The linear relation was obtained from the determination of two points (Fig. 1), the first point corresponding to the smaller left ventricular volume and the second one corresponding to a theoretical left ventricular isovolumic contraction (without ejection) on the end-diastolic volume (EDV). To determine the peak isovolumic pressure (PIP), a nonlinear least-squares approximation was used by measuring pressure each 0.005 sec during the isovolumic contraction (from the end-diastolic pressure to the pressure corresponding to the peak positive dP/dt), and during the isovolumic relaxation (from the pressure corresponding to the
peak negative dP/dt to a pressure equal to the left ventricular end-diastolic pressure). The line determined by the PIP-EDV and the end-systolic pressure–volume points on the pressure–volume loop defined the left ventricular end-systolic pressure–volume line, the slope of which represents the end-systolic elastance (Ees) (Fig. 1).

The slope of the arterial end-systolic pressure–stroke volume relation (termed arterial effective elastance [Ea]) served to represent arterial properties. Arterial effective elastance was determined by the line between the left ventricular end-systolic pressure–volume point and the left ventricular end-diastolic volume point on the volume axis (Fig. 1).

Left ventricular pressure–volume loop represents the external work (EW) of the left ventricle. For simplicity, the pressure–volume loop was regarded as a trapezoid, the width of which was the stroke volume (SV) and the heights of which were respectively represented by the end-systolic pressure (ESP) and the end-systolic pressure minus end-diastolic pressure (EDP) (Fig. 1). As myocardial oxygen consumption could not be measured because of ethical considerations (right heart and coronary sinus catheterizations were not allowed in these patients), left ventricular mechanical efficiency was defined by the external work/pressure–volume area ratio (EW/PVA).

**Statistical Analysis**

In each group, data were expressed as means ± SD. The Student t-test for unpaired samples was used to compare data between the two groups of patients. Simple linear regression analysis was used to examine relations between parameters. Values of P < .05 were considered to be statistically significant.

**Results**

**Patient Characteristics**

The two groups of patients were not statistically significantly different with regard to age, gender, body surface area, and body mass index. Aortic systolic pressure was significantly higher and aortic diastolic pressure significantly lower in patients with LVH than in patients without LVH. Pulse pressure was higher in the former group, and conversely mean aortic pressure was higher in the latter group. Echocardiographic left ventricular mass index was increased in LVH group and was within normal values in nonhypertrophied patients (Table 1).

<table>
<thead>
<tr>
<th></th>
<th>LVH (n = 30)</th>
<th>Normal LVM (n = 23)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>59 ± 3</td>
<td>50 ± 9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>20/10</td>
<td>16/7</td>
<td>NS</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.84 ± 0.12</td>
<td>1.88 ± 0.18</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.7 ± 4.2</td>
<td>26.4 ± 4.0</td>
<td>NS</td>
</tr>
<tr>
<td>SP&lt;sub&gt;Pa&lt;/sub&gt; (mm Hg)</td>
<td>187 ± 10</td>
<td>171 ± 9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>DP&lt;sub&gt;Pa&lt;/sub&gt; (mm Hg)</td>
<td>93 ± 6</td>
<td>103 ± 10</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MAOP (mm Hg)</td>
<td>124 ± 9</td>
<td>131 ± 11</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>PP&lt;sub&gt;Pa&lt;/sub&gt; (mm Hg)</td>
<td>94 ± 8</td>
<td>68 ± 9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVM&lt;sub&gt;i&lt;/sub&gt; (g/m²)</td>
<td>141 ± 16</td>
<td>90 ± 8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVM&lt;sub&gt;i&lt;/sub&gt; (g/m²²)</td>
<td>58 ± 8</td>
<td>38 ± 5</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

LVH = left ventricular hypertrophy; LVM = left ventricular mass index; NS = not significant; BSA = body surface area; BMI = body mass index; SP<sub>Pa</sub> = systolic aortic pressure; DP<sub>Pa</sub> = diastolic aortic pressure; MAOP = mean aortic pressure; PP<sub>Pa</sub> = aortic pulse pressure.
Angiographic data (Table 2) show that heart rate was comparable in the two groups. Left ventricular end-diastolic and end-systolic volumes as well as ejection fraction were within normal range in both groups. However, volumes were significantly higher and ejection fraction significantly lower in patients with LVH, which resulted in similar stroke volumes in the two groups. Left ventricular end-diastolic pressure was significantly higher in the LVH group, but end-systolic pressure and left ventricular peak isovolumic pressure were comparable in the two groups (Table 2). Patients with LVH evidenced lower end-systolic wall stress than did patients without LVH, but showed similar peak isovolumic wall stress (which was calculated using peak isovolumic pressure, left ventricular end-diastolic dimensions, and wall-thickness). Relative wall thicknesses at end-diastole and end-systole were significantly higher in patients with LVH (Table 2), and were significantly correlated with pulse pressure (Fig. 2), as previously shown.\(^\text{20}\)

### Table 2. Hemodynamic and angiographic data (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>LVH ((n = 30))</th>
<th>Normal LVM ((n = 23))</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>72 ± 6</td>
<td>75 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>EDV (mL/m²)</td>
<td>86 ± 11</td>
<td>80 ± 8</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>ESV (mL/m²)</td>
<td>30 ± 7</td>
<td>24 ± 5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SV (mL/m²)</td>
<td>55 ± 5</td>
<td>55 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>EF (%)</td>
<td>65 ± 5</td>
<td>70 ± 4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>h/r(_{ED}) (mm)</td>
<td>0.43 ± 0.06</td>
<td>0.39 ± 0.05</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>h/r(_{ES}) (mm)</td>
<td>1.03 ± 0.19</td>
<td>0.89 ± 0.13</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>14 ± 4</td>
<td>11 ± 2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SP (mm Hg)</td>
<td>187 ± 10</td>
<td>171 ± 9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>ESP (mm Hg)</td>
<td>165 ± 10</td>
<td>161 ± 8</td>
<td>NS</td>
</tr>
<tr>
<td>PIP (mm Hg)</td>
<td>485 ± 53</td>
<td>481 ± 44</td>
<td>NS</td>
</tr>
<tr>
<td>ESS (g/cm²)</td>
<td>184 ± 42</td>
<td>228 ± 36</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SS(_{max}) (g/cm²)</td>
<td>1,349 ± 406</td>
<td>1,484 ± 185</td>
<td>NS</td>
</tr>
<tr>
<td>ESSVR (g/cm²/mL)</td>
<td>3.85 ± 0.99</td>
<td>5.13 ± 0.56</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Ees (mm Hg/mL/100 g)</td>
<td>1.23 ± 0.28</td>
<td>1.89 ± 0.48</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

LVH = left ventricular mass; HR = heart rate; EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction; h/r\(_{ED}\) = end-diastolic wall thickness to radius ratio; h/r\(_{ES}\) = end-systolic wall thickness-to-radius ratio; EDP = end-diastolic pressure; SP = systolic pressure; ESP = end-systolic pressure; PIP = peak isovolumic pressure; ESS = end-systolic stress; SS\(_{max}\) = peak isovolumic wall stress; ESSVR = end-systolic stress-to-volume ratio; Ees = end-systolic elastance; other abbreviations as in Table 1.

### Left Ventricular Contractile Performance

Left ventricular contractile performance was estimated using end-systolic wall stress-to-end-systolic volume ratio (ESSVR)\(^\text{21}\) and end-systolic elastance (Ees).\(^\text{13,19,22}\) End-systolic stress-to-volume ratio was significantly higher in patients without LVH (Table 2). On the other hand, end-systolic elastance normalized by body surface area was comparable in the two groups (Table 3). However, when normalized by left ventricular mass, Ees was higher in the group without LVH (Table 2). Moreover, these two indices were negatively correlated to left ventricular mass index (Fig. 3). Finally, a fair correlation was found between the two indices of left ventricular contractile performance, ESSVR, and Ees normalized by left ventricular mass (Fig. 4).
### Parameters of Ventricleoarterial Coupling and Left Ventricular Mechanical Efficiency

The volume axis intercept (Vo) of the end-systolic pressure-volume line was not different in the two groups (Table 3). Effective arterial elastance, which is representative of arterial vascular load,23 end-systolic elastance, and the Ea/Ees ratio, which represents the ventriculoarterial coupling10,13 were similar in both groups of hypertensive patients (Table 3). This diagrammatic representation of ventriculoarterial coupling allows to calculate the external work (EW) and the pressure-volume area (PVA), 24 which were comparable in the two groups (Fig. 1). Conversely, when indexed to myocardial mass to normalize the energy delivered by the myocardium, results show that EW and PVA were significantly higher in patients without LVH (Table 3).

As myocardial oxygen consumption could not be calculated in our patients, left ventricular mechanical efficiency was estimated through the ratio of external work-to-pressure-volume area.24 As shown in Table 3, comparisons between the two groups of patients did not show any difference.

### Discussion

This study, which was dedicated to compare the coupling between the ventricular pump and the arterial load in terms of matching between effective arterial elastance and left ventricular end-systolic elastance in hypertensive patients with and without LVH and normal pump function, shows that despite a slight depression of left ventricular contractile performance in patients with LVH, mechanical efficiency, and ventriculoarterial coupling are comparable to that observed in patients without LVH.

### Table 3. Parameters of ventriculoarterial coupling (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>LVH (n = 30)</th>
<th>Normal LVM (n = 23)</th>
<th>P Value</th>
</tr>
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<tbody>
<tr>
<td>Vo (mL)</td>
<td>-3 ± 11</td>
<td>-8 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>Ea (mm Hg/mL)</td>
<td>1.70 ± 0.29</td>
<td>1.56 ± 0.19</td>
<td>NS</td>
</tr>
<tr>
<td>Ees (mm Hg/mL)</td>
<td>3.30 ± 0.65</td>
<td>3.12 ± 0.57</td>
<td>NS</td>
</tr>
<tr>
<td>Ea/Ees</td>
<td>0.52 ± 0.08</td>
<td>0.50 ± 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>EW (J)</td>
<td>2.10 ± 0.39</td>
<td>2.19 ± 0.24</td>
<td>NS</td>
</tr>
<tr>
<td>EW (J/100g)</td>
<td>0.84 ± 0.15</td>
<td>1.32 ± 0.20</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PVA (J)</td>
<td>2.69 ± 0.51</td>
<td>2.75 ± 0.31</td>
<td>NS</td>
</tr>
<tr>
<td>PVA (J/100g)</td>
<td>1.08 ± 0.20</td>
<td>1.66 ± 0.24</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EW/PVA</td>
<td>0.78 ± 0.03</td>
<td>0.80 ± 0.02</td>
<td>NS</td>
</tr>
</tbody>
</table>

Ea = effective arterial elastance; EW = external work; EW/PVA = mechanical efficiency; PVA = pressure-volume area; Vo = volume axis intercept of the end-systolic pressure-volume relationship (see Fig. 1). Other abbreviations as in Tables 1 and 2.

### Left Ventricular Mass and Contractile Performance

In this study, arterial elastance was comparable in the two groups and, despite similar mean aortic pressure and left ventricular end-systolic and peak isovolumic pressures
into account left ventricular mass have been made. Then, lower values in patients with LVH. Because it has been emphasized that Ees should be normalized to enable comparisons of contractile function among patients, and because the main consequence of arterial hypertension is left ventricular hypertrophy, we thought that normalization by left ventricular mass might be more appropriate for comparisons between the two groups. Similar attempts to take ventricular mass into account left ventricular mass have been made. Then, results showed lower values in hypertensive patients with LVH (1.23 ± 0.28 mm Hg/mL/100 g in group 1 v 1.89 ± 0.48 in group 2, P < .001, Fig. 4). Moreover, a close negative relationship was observed between the two indices of left ventricular performance and left ventricular mass (Fig. 3), and a close positive relationship between these two indices of left ventricular contractile performance was evidenced (Fig. 4), which strengthen the conclusion that the more elevated is the left ventricular mass, the more the contractile performance is depressed, and that increased contractile force developed by the ventricle results either from the increased ventricular mass in group 1 or from an enhanced contractility in group 2.

Ventriculoarterial Coupling and Energy Transfer
In this study, we studied the matching of the left ventricular contractile properties (Ees) with the arterial load properties (Ea). It has been demonstrated that, under normal conditions, Ea is nearly one half of Ees, and that progression of myocardial depression leads to reduced Ees and increased Ea so that the Ea/Ees ratio is progressively increased. It has also been postulated that when afterload is increased, external work tends to be maximized when mechanical efficiency declines. This reduction of mechanical efficiency is also observed when left ventricular contractile performance is reduced.

In the two groups of patients, Ea and Ees were comparable despite the depression of left ventricular contractile performance in group 1, which resulted in Ea/Ees ratios (Table 3) comparable to those observed in normal subjects, which supports the conclusion that LVH in group 1 and myocardial contractility in group 2 are responsible for the elevated developed force.

It has been shown that the mechanical efficiency of the left ventricle is defined as the ratio of external work to myocardial oxygen consumption and that the energy transfer from PVA to EW represents the left ventricular work efficiency. In our study, efficiency of energy transfer was evaluated by determining the EW/PVA ratio, as coronary sinus catheterization was not allowed because of ethical considerations and myocardial oxygen consumption therefore could not be calculated. Thus, mechanical efficiency was comparable in the two groups, with values within the normal range of that reported in the literature. Nevertheless, although EW and PVA were comparable in the two groups, values indexed to myocardial mass were significantly lower in group 1, showing that LVH is used to maintain a normal pump function when contractility is depressed. This normal level of mechanical efficiency (despite depression of left ventricular contractile performance in group 1) agrees with previously published data in patients with moderately depressed left ventricular pump function suggesting that LVH might be a better means to preserve left ventricular pump function with less energetic cost than increased myocardial contractility, the means used by a left ventricle without hypertrophy.

Methodologic Considerations
Patients with left ventricular hypertrophy were older than patients without left ventricular hypertrophy, probably because of a longer duration of arterial hypertension in older patients. This may influence our results: in particular, left ventricular hypertrophy and aortic structure and function. Nevertheless, the actual duration of the disease was unknown because most of our patients (35/53) had recently diagnosed hypertension. However it must be pointed out that whatever the condition of the patients, the ventriculoarterial coupling was preserved, which was the main conclusion of this study.

The two groups have been considered to have normal systolic function. However, results show that patients with LVH had higher end-diastolic and end-systolic volumes and lower ejection fraction than patients without LVH. Nevertheless, the values were within the normal range, and stroke volumes were similar in the two groups.

In this study, we used the echocardiographic left ventricular mass to calculate wall thickness on angiographic volumes. This method was chosen because determination of end-diastolic wall thickness on angiograms may be difficult and may lead to some inaccuracy. Because patients with concentric remodeling have small cavity dimensions and normal left ventricular mass, they were excluded from this study. Finally, left ventricular hypertrophy was determined using criteria published by Ganau et al, which are currently used in the literature. Normalizing left ventricular mass by height (Table 1) did not significantly change the results; only two male and two female patients may have been misclassified in the left ventricular hypertrophy group accordingly to the upper limits given by De Simone et al.
Coronary arteriography may have missed patients with microvascular disease, which is frequently observed in hypertensive patients, especially when left ventricular mass is increased. In our study, coronary flow reserve was reduced in both groups, when compared with normal values we had previously published (5.2 ± 0.8 vs 3.0 ± 0.9 in patients with left ventricular hypertrophy, and 3.8 ± 0.7 in patients without left ventricular hypertrophy). However, it has never been demonstrated that a coronary microvascular disease could depress left ventricular contractile performance at rest.

As pressures decline rather rapidly from peak systolic pressure, there could be errors in end-systolic pressure if assumed at the time of minimum left ventricular volume. Furthermore, the assumptions made for the determination of left ventricular geometry could also induce some errors in the stress calculation.

A single-beat estimation of Ees was used in this study. Takeuchi et al have shown that values of estimated peak isovolumic pressure were not significantly different from true values. This method avoids pharmacologic manipulations, which have been demonstrated to misjudge Ees and allows determination of Ees from one pressure-volume loop. It also avoids construction of the end-systolic pressure-volume line using the end-systolic point and the origin of the two axes, which may considerably change the actual slope of the line because volume axis intercept Vo may vary from −24 to +22 mL (Table 3). Finally, the curvilinearity of the end-systolic pressure-volume relationship does not prevent end-systolic elastance from being an accurate index of contractility.

We calculated the EW of the left ventricle as a trapezoid area constructed from the end-systolic and end-diastolic pressures and the stroke volume. This assumption may have produced some quantitative errors but could not have affected the main results of the study.

In summary, in this study in which the concept of ventriculoarterial coupling was used to evaluate left ventricular contractile performance and energy transfer, results show that in hypertensive patients, preservation of the matching between left ventricular performance and arterial load and the energy transfer can be achieved through two different mechanisms: 1) left ventricular hypertrophy, despite a moderate depression of myocardial contractile performance; and 2) myocardial contractile performance in patients with normal left ventricular mass. However, further studies using methods allowing the non-invasive measurement of actual myocardial oxygen consumption are warranted to precisely measure the energy cost of each means of adaptation to pressure overload due to arterial hypertension.

References


