Contractile performance of hypertrophied left ventricle may be depressed in arterial hypertension. Ventriculoarterial coupling is impaired when myocardial contractile performance is reduced and when afterload is increased. The left ventricular contractile performance and the ventriculoarterial coupling were evaluated in 30 hypertensive patients with moderate left ventricular hypertrophy and 20 control subjects. Left ventricular angiography coupled with the simultaneous recording of pressures with a micromanometer were used to determine end-systolic stress/volume index, the slope of end-systolic pressure-volume relationship, ie, end-systolic elastance, effective arterial elastance, external work, and pressure-volume area. In hypertensive patients, left ventricular contractile performance, as assessed by end-systolic elastance/100 g myocardial mass, was depressed (4.35 ± 1.13 vs 5.21 ± 1.89 mm Hg/mL/100 g in control subjects, P < .02), when end-systolic stress-to-volume ratio was comparable in the two groups (3.85 ± 0.99 g/cm²/mL in hypertensive patients versus 3.51 ± 0.77 g/cm²/mL in control subjects). Ventriculoarterial coupling, evaluated through effective arterial elastance/end-systolic elastance ratio, was slightly higher in hypertensive patients (0.53 ± 0.08 vs 0.48 ± 0.09 mm Hg/mL in control subjects, P < .05), and work efficiency (external work/pressure-volume area) was similar in the two groups (0.78 ± 0.04 mm Hg/mL in hypertensive patients versus 0.80 ± 0.03 mm Hg/mL in control subjects). This study shows that despite a slight depression of left ventricular contractile performance, work efficiency is preserved and ventriculoarterial coupling is almost normal in hypertensive patients with left ventricular hypertrophy. Thus, it appears that left ventricular hypertrophy might be a useful means of preserving the match between left ventricle and arterial receptor with minimal energy cost. Am J Hypertens 1998;11:1188–1198 © 1998 American Journal of Hypertension, Ltd.

KEY WORDS: Hypertension, left ventricular hypertrophy, ventriculoarterial coupling, left ventricular performance, work efficiency.
Left ventricular hypertrophy is a physiologic process of adaptation of the heart to mechanical load increase. Despite that left ventricular adaptation to hypertension may lead to different patterns of hypertrophy, compensatory ventricular response to arterial hypertension generally leads to increased wall thickness, which normalizes wall stress, preserves pumping ability, and results in a normal myocardial oxygen consumption per unit mass in the basal state. Although several studies have been dedicated to the relation between left ventricular hypertrophy, contractile performance, and hemodynamic load in human arterial hypertension, no study has investigated ventriculoarterial coupling and left ventricular efficiency in this setting.

The ventriculoarterial concept is built from the properties of the arterial system, represented by the slope of end-systolic pressure-stroke volume relation (effective arterial elastance, \( E_{a} \)), and left ventricular mechanical properties, represented by the end-systolic pressure-volume relation (end-systolic elastance, \( E_{es} \)). This concept enables the evaluation of the matching between the left ventricular function and the mechanical properties of the arterial system: 1) the left ventricular mechanical efficiency, which is defined as the ratio of external work to myocardial oxygen consumption per beat, is maximal when arterial impedance is about one-half the cardiac output impedance, and 2) the pressure-volume area, which represents the total mechanical energy of contraction, is correlated with the myocardial oxygen consumption per beat. 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 ventricular elastance. On the other hand, progressive ventricular dysfunction results in maximization of external work at the expense of mechanical efficiency. Lastly, the correlation between pressure-volume area and myocardial oxygen consumption has been validated in human left ventricle.

The aim of the present study was to examine in hypertensive patients with moderate left ventricular hypertrophy the relation existing between the left ventricular contractile performance, the ventriculoarterial coupling, and the left ventricular efficiency. Because the slope of the end-systolic pressure-volume relationship is usually constructed by producing stepwise afterload variations that may incorrectly estimate end-systolic elastance, a single-beat estimation of end-systolic elastance has been used in this study.}

**METHODS**

**Patient Selection** The study population comprised 50 patients undergoing coronary arteriography for the diagnosis of chest pain. All patients evidenced abnormal exercise electrocardiogram 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 \( \leq 30\% \) at coronary arteriography.

**Hypertensive Patients** Thirty 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 were included. Hypertension had been recently diagnosed in 18 patients who had never been treated and 12 additional patients were included after discontinuing all treatment, with the exception of nitrates, at least 3 weeks before cardiac catheterization. Left ventricular systolic function 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 \( \geq 30\% \). Left ventricular dimensions and septal and posterior wall thicknesses were measured at end-diastole, in accordance with the American Society of Echocardiography guidelines. The left ventricular mass index was calculated at end-diastole by using the Penn convention.

Men with left ventricular mass index (LVMI) \( > 111 \) g/m\(^2\) and women with LVMI \( > 106 \) g/m\(^2\) were included. Patients with septal/posterior wall thickness ratio \( \geq 1.2 \) were excluded.

**Control Subjects** Twenty subjects with a supine systolic blood pressure \( < 140 \) mm Hg and a diastolic blood pressure \( < 90 \) mm Hg composed the control group. Left ventricular systolic function and left ventricular dimensions assessed by two-dimensional and M-mode echocardiography were normal. Septal and posterior wall thicknesses were \( < 12 \) and \( < 10 \) mm, respectively. Men with LVMI \( \leq 111 \) g/m\(^2\) and women with a LVMI \( \leq 106 \) g/m\(^2\) were included.

**Catheterization Procedure** Nitrates, when given, were discontinued 48 h before the study. All patients were in the fasting state for \( \geq 12 \) h before the procedure. No premedication was administered, and 1% lidocaine was used for local anesthesia.

After documentation of coronary artery lesions, a 7F double-tipped micromanometer angiographic catheter (Sentron, Cordis 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 and aortic, left ventricular end-diastolic, and systolic pressures were calculated by means of a catheterization data analysis computer system (Hewlett-Packard 5600 M, Andover, MA) that performed online 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 Calculation of Left Ventricular Volume, Mass, and End-Systolic Wall Stress

Left ventricular volumes were calculated from monoplane angiograms by means of the area-length method.\textsuperscript{34} The echocardiographic left ventricular mass (LVM) was assumed to be constant and was used to calculate left ventricular myocardial wall volume as LVM/1.05. Left ventricular myocardial wall volume was then applied to the left ventricular end-diastolic and end-systolic angiographic volumes to calculate the end-diastolic (h\textsubscript{ED}) and end-systolic equatorial wall thicknesses (h\textsubscript{ES}) by solving the equation of Rackley et al.\textsuperscript{35}

Average left ventricular equatorial end-systolic wall stress (LV\textsubscript{WS\textsubscript{ES}}) was calculated using the Falsetti formula for an ellipsoidal geometry of the left ventricle:\textsuperscript{36}

\[
\text{LVWS}_{\text{ES}} = \left[\frac{(\text{LVESP} \cdot b)}{h_{\text{ES}}} \right] \cdot \left[\frac{(2a^2 - b^2)/(2a^2 + b \cdot h_{\text{ES}})}{a^2 + b^2} \right].
\]

where LVESP (in mm Hg) is the left ventricular end-systolic pressure (the pressure corresponding to the smaller ventricular volume), \(a\) (in centimeters) is the major semiaxis, \(b\) (in centimeters) is the minor semiaxis. To obtain values of LVWS in grams/centimeters squared, LVESP expressed in mm Hg was multiplied by 1.36.

Ventriculoarterial Coupling

Ventriculoarterial coupling was analyzed through the left ventricular output impedance, expressed by the end-systolic elastance (E\textsubscript{es}) obtained from the end-systolic pressure-volume relation, and the arterial input impedance expressed by the effective arterial elastance (E\textsubscript{a}) obtained from the arterial end-systolic pressure-stroke volume relation.

Determination of End-Systolic Elastance

The end-systolic elastance was obtained with the single-beat analysis method described by Takeuchi et al.\textsuperscript{30} assuming that the end-systolic pressure-volume relation is roughly linear for physiologic conditions when myocardial contractility does not change.\textsuperscript{16} The linear relation was obtained from the determination of two points (Figure 1). The first point was determined by the left ventricular pressure corresponding to the smaller left ventricular volume. The second point was obtained on the same beat by determination of a systolic pressure (estimated peak isovolumic pressure) corresponding to a theoretical left ventricular isovolumic contraction (without ejection) on the end-diastolic volume. To construct the pressure curve P(t) of such an isovolumic contraction on the left ventricular end-diastolic volume, a nonlinear least-squares approximation was used:

\[
P(t) = 1/2 \text{PIP}[1 - \cos(\omega t + C)] + \text{LVEDP},
\]

where PIP represents the peak isovolumic pressure, \(\omega\) is the angular frequency, C is the phase shift angle of the sinusoidal curve, and LVEDP is the left ventricular end-diastolic pressure. The P(t) curve was obtained 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...
pressure). The line determined by the PIP-LVEDP and the end-systolic pressure-volume points on the pressure-volume loop defined the left ventricular end-systolic pressure-volume line, whose slope represents the end-systolic elastance Ees (Figure 1).

**Determination of Arterial Effective Elastance**

Arterial properties can be represented as a first approximation by the slope of the arterial end-systolic pressure-stroke volume relation called arterial effective elastance. As the equilibrium point between pressure and volume that should exist when the left ventricle is coupled with the aorta can be obtained by the intersection between left ventricular and aortic end-systolic pressure-volume relation lines, the 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).

**External Work and Pressure-Volume Area of the Left Ventricle**

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 whose width was the stroke volume (SV) and whose heights, respectively, represented the end-systolic pressure (ESP) and the end-systolic pressure minus end-diastolic pressure (EDP) (Figure 1):

\[
EW = SV \cdot [ESP + (ESP - EDP)] / 2.
\]

The triangle comprised between EW area and the Ees line represents the end-systolic potential energy of the ventricle (PE) (Figure 1). The sum of PE and EW is called the pressure-volume area (PVA) and represents the total mechanical energy of the ejecting contraction.

**Estimate of Left Ventricular Efficiency**

Because myocardial oxygen consumption could not be measured because of ethical considerations (right heart and coronary sinus catheterization were not allowed in these patients), mechanical efficiency, the EW/myocardial oxygen consumption ratio, could not be calculated. However, as myocardial oxygen consumption per beat is related to the pressure-volume area (PVA), work efficiency, defined by the external work/pressure-volume area ratio (EW/PVA), was used as an estimate of left ventricular mechanical efficiency.

**Statistical Analysis**

In each group, data were expressed as mean ± SD. Student’s 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 significant.

**RESULTS**

**Patient Characteristics**

The two groups of patients were not statistically different for age, gender, body surface area, and body mass index. Arterial pressures and echocardiographic left ventricular mass index were significantly higher in hypertensive patients (Table 1). Angiographic data (Table 2) show that heart rate, left ventricular end-diastolic and end-systolic volumes, and ejection fraction were comparable in the two groups. Left ventricular pressures (systolic, end-diastolic, and end-systolic) (Table 2) were significantly higher in the hypertensive patients. Similarly, left ventricular peak isovolumic pressure was higher in hypertensive patients, but end-systolic wall stress and peak isovolumic wall stress (calculated using peak isovolumic pressure, left ventricular end-diastolic di-

\[\text{TABLE 1. CHARACTERISTICS OF THE STUDY POPULATION (MEAN ± SD)}\]

<table>
<thead>
<tr>
<th></th>
<th>Age (year)</th>
<th>Sex (M/F)</th>
<th>BSA (m²)</th>
<th>BMI (kg/m²)</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>LVMi (g/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control subjects (n = 20)</td>
<td>56.5 ± 4.8</td>
<td>14/6</td>
<td>1.86 ± 0.14</td>
<td>29.0 ± 4.0</td>
<td>128 ± 8</td>
<td>71 ± 5</td>
<td>89 ± 10</td>
</tr>
<tr>
<td>Hypertensive patients (n = 30)</td>
<td>58.3 ± 3.0</td>
<td>19/11</td>
<td>1.83 ± 0.12</td>
<td>27.8 ± 4.2</td>
<td>182 ± 10</td>
<td>96 ± 6</td>
<td>138 ± 17</td>
</tr>
</tbody>
</table>

*BSA, body surface area; BMI, body mass index; DBP, diastolic blood pressure; LVMi, left ventricular mass index; SBP, systolic blood pressure.*

\[\text{TABLE 2. LEFT VENTRICULAR ANGIOGRAPHIC AND HEMODYNAMIC DATA (MEAN ± SD)}\]

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects (n = 20)</th>
<th>Hypertensive Patients (n = 30)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>68 ± 7</td>
<td>70 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>EDVi (mL/m²)</td>
<td>81 ± 10</td>
<td>83 ± 14</td>
<td>NS</td>
</tr>
<tr>
<td>ESVi (mL/m²)</td>
<td>26 ± 5</td>
<td>28 ± 8</td>
<td>NS</td>
</tr>
<tr>
<td>EF (%)</td>
<td>68 ± 5</td>
<td>67 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>h/rEd (mm)</td>
<td>0.32 ± 0.03</td>
<td>0.44 ± 0.06</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>h/rEs (mm)</td>
<td>0.84 ± 0.14</td>
<td>1.09 ± 0.23</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>9 ± 1</td>
<td>14 ± 4</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>SP (mm Hg)</td>
<td>132 ± 7</td>
<td>187 ± 9</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>ESP (mm Hg)</td>
<td>117 ± 10</td>
<td>165 ± 9</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

*EDP, end-diastolic pressure; EDVi, end-diastolic volume index; ESVi, end-systolic volume index; EF, ejection fraction; ESP, end-systolic pressure; ESVi, end-systolic volume index; HR, heart rate; h/rEd, end-diastolic wall thickness/radius; h/rEs, end-systolic wall thickness/radius; SP, peak systolic pressure.*
dimensions and wall thickness) were comparable in the two groups (Figure 2). Relative wall thickness at end-diastole and end-systole was significantly higher in hypertensive patients and was positively correlated to the peak isovolumic pressure (Figure 3).

**Left Ventricular Contractile Performance** Left ventricular contractile performance was estimated using end-systolic wall stress–to–end-systolic volume ratio and end-systolic elastance. End-systolic stress-to-volume ratio was comparable in both groups (3.85 ± 0.99 g/cm²/mL in hypertensive patients versus 3.51 ± 0.77 g/cm²/mL in control subjects) (Figure 4A).

End-systolic elastance, which is another index of contractile performance, was higher in hypertensive patients than in control subjects (Figure 4B), and there was a positive relation between end-systolic elastance and left ventricular mass (Figure 5A). Conversely, when normalized for left ventricular mass, the end-systolic elastance value was lower in the hypertensive group (Figure 4C). However, a fair correlation was found between end-systolic elastance normalized by left ventricular mass and end-systolic stress-to-end-systolic volume ratio (Figure 5B). On the other hand, end-systolic stress-to-volume ratio was negatively correlated to left ventricular mass in both groups but the two groups had different negative correlations. In hypertensive patients, for a level of end-systolic stress-to-volume ratio similar to control subjects, there was a higher left ventricular mass (Figure 5C). Lastly, end-systolic elastance/100 g myocardial mass was negatively correlated to left ventricular mass (Figures 5C and D).

**Parameters of Ventriculoarterial Coupling** The volume axis intercept (Vo) of the end-systolic pressure volume line was not different in the two groups (Table 3). However, it must be pointed out that the standard deviation for Vo was large in the two groups and that Vo value ranged from −22 to 20 mL. Effective arterial elastance, Ea, and end-systolic elastance, Ees, were both significantly higher in the hypertensive patients (Table 3). Effective arterial elastance, which is representative of arterial vascular load, was positively related to left ventricular mass and end-diastolic and end-systolic wall thickness (Figure 6). However, the Ea/Ees ratio, which represents the ventriculoarterial coupling, was only slightly higher in the hypertensive group (Table 3).

**Energy Conversion Efficiency** As shown in Table 3, comparisons between control subjects and hypertensive patients using absolute values show that potential energy, external work, and, consequently, pressure-volume area were higher in the latter group.

Conversely, comparisons using values indexed to myocardial mass showed comparable results in the two groups for potential energy and pressure-volume area.
area, only the external work being slightly lower in the hypertensive patients (Table 3). Work efficiency (external work/pressure-volume area) was comparable in the two groups.

**DISCUSSION**

To our knowledge, this study is the first one investigating in hypertensive patients with left ventricular hypertrophy the coupling between the ventricular pump and the arterial afterload in terms of matching between effective arterial elastance and left ventricular end-systolic elastance. The principal findings of this study are that despite a slight depression of left ventricular contractile performance, work efficiency, used as an estimate of mechanical efficiency, is preserved, and ventriculoarterial coupling, evaluated through the effective arterial elastance/end-systolic elastance ratio, is almost normal in hypertensive patients with left ventricular hypertrophy. Thus, it appears that left ventricular hypertrophy might be a useful means of preserving the match between left ventricle and arterial receptor with minimal energy cost.

**Left Ventricular Mass and Contractile Performance**

The characterization of the left ventricular contractile performance of hypertrophied ventricles remains a difficult challenge. Our results suggest that left ventricular hypertrophy in our hypertensive patients was matched to ventricular load. Indeed, end-systolic wall stress was similar in hypertensive patients and in control subjects (Figure 2), even when end-systolic pressure was much higher in the hypertensive patients (Table 2). Also, the calculation of peak isovolumic wall stress showed no differences between the two groups when peak isovolumic pressure was significantly higher in hypertensive patients (Figure 2). Moreover, peak isovolumic pressure was positively related to both diastolic and systolic wall-to-thickness ratios (Figure 3). Lastly, effective arterial elastance, which was used as an index of arterial vascular load, was related to all the indices of left ventricular hypertrophy, ie, left ventricular mass and wall-to-thickness ratios (Figure 6).

Data about left ventricular contractile performance in hypertensive patients with left ventricular hyper-

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**FIGURE 4.** Comparisons of left ventricular contractile performance indices between the two groups show that end-systolic stress/end-systolic volume ratio is similar in the two groups (A). End-systolic elastance is higher in hypertensive patients (B) when end-systolic elastance indexed to myocardial mass is lower in these patients (C).

**FIGURE 5.** End-systolic elastance is positively related to left ventricular myocardial mass (A) and to end-systolic stress/end-systolic volume ratio (B). End-systolic stress/end-systolic volume ratio is negatively related to myocardial mass in the two groups of patients, but the regression lines are different in each group (C). End-systolic elastance indexed to myocardial mass is negatively related to myocardial mass (D). Open circles, control subjects; closed circles, hypertensive patients.
trophy are conflicting. An increased or normal myocardial contractility has been suggested by Hartford et al and by Lutas et al. De Simone et al, using end-systolic stress-to-volume ratio, did not find any evidence of a difference with control subjects. Conversely, other studies have shown that contractile performance was depressed, and, more recently, De Simone et al have demonstrated that midwall fractional shortening/end-systolic stress reduction in patients with concentric hypertrophy or remodeling is a predictor of cardiovascular risk in hypertensive patients. Takahashi et al, using end-systolic stress, have shown that contractile performance was normal in patients with moderate hypertrophy and depressed in patients with advanced hypertrophy. Ganau et al have shown that left ventricular mass was inversely related to contractility estimated through the end-systolic stress-to-volume index ratio. In our study, the two indices of contractile performance that have been used to evaluate left ventricular contractile performance show results depending on the way the data were expressed, ie, absolute values or values indexed to myocardial mass.

The end-systolic stress-to-volume ratio, which has been established as a reasonably load independent index of myocardial performance, was not different in the two groups of patients (Figure 4A). However, because it has been shown that this ratio was dependent on the end-diastolic size of the left ventricle, the calculation made without the four hypertensive patients with left ventricular end-diastolic volume index ≥ 65 mL/m² did not show any difference between the two groups. In addition, the ratio was negatively related to left ventricular mass (Figure 5C) in control subjects and in hypertensive patients. Moreover, results show that the relation between the two parameters was displaced to higher values of left ventricular mass in hypertensive patients, which shows that the left ventricular contractile performance is depressed when left ventricular mass increases, at which time the decline of the ratio with the increase of end-diastolic volume was comparable in the two groups (Figure 7).

The end-systolic elastance, that is, the slope of the end-systolic pressure-volume relationship, has also been demonstrated to be relatively independent from ventricular load. The higher ratio observed by Lutas et al in hypertensive patients led these authors to conclude that contractile performance was increased. However, Suga et al have reported the size dependence of this index and it has since been emphasized that this index should be normalized to enable comparisons of contractile function among patients. Indeed in our study, absolute value of end-systolic elastance was higher in hypertensive patients (Figure 4B) and was related positively to left ventricular mass (Figure 5A). This relation could lead to the conclusion that contractile performance is increased in hypertensive patients with left ventricular hypertrophy. Thus, 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. However, our results showed lower values in hypertensive patients (4.35 ± 1.13 v 5.21 ± 1.89 mm Hg/mL/100 g in control subjects, P < .02, Figure 4C) and a negative relation with left ventricular mass comparable to the negative relation observed between end-systolic stress-to-volume ratio and left ventricular mass was obtained (Figure 5D). In addition, we also observed a close positive relationship between these two indices of left ventricular contractile performance, which strengthens the conclusion that the more elevated is the left ventricular mass, the more the contractile performance is depressed, and that the increased contractile force developed by the left ventricle results from the increased left ventricular mass rather than an enhanced myocardial contractility. The fact that we found a negative relation between pressure-volume area per unit mass, an estimate of myocardial oxygen consumption per beat, and left ventricular mass (Figure 8) is another argument for mild myocardial depression in hypertensive patients.

### Ventrículoarterial Coupling

We studied the matching of the left ventricular properties quantified by the slope of end-systolic pressure-volume relation (end-systolic elastance), Ees, with the arterial load properties expressed by the slope of the end-systolic press-
sure-stroke volume relation, which is called effective arterial elastance, Ea. It has been experimentally demonstrated that the left ventricle produces its maximal mechanical efficiency when effective arterial elastance is nearly one-half end-systolic elastance, and maximal external work when effective arterial elastance equals end-systolic elastance. The concept of ventriculoarterial coupling has been validated in humans by Asanoi et al., who found similar coupling conditions in the normal left ventricle, and who have shown that progression of myocardial depression leads to reduced end-systolic elastance and increased effective arterial elastance. The concept of ventriculoarterial coupling has been validated in humans by Asanoi et al., who found similar coupling conditions in the normal left ventricle, and who have shown that progression of myocardial depression leads to reduced end-systolic elastance and increased effective arterial elastance. Thus, the effective arterial elastance/end-systolic elastance ratio is progressively increased. It has also been postulated that when the 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. As arterial hypertension with left ventricular hypertrophy is characterized both by an elevation of afterload, and a decline of left ventricular contractile performance one should expect a deterioration of ventriculoarterial coupling and mechanical efficiency.

Our data show that if effective arterial elastance was increased in hypertensive patients, end-systolic elastance (expressed in mm Hg/milliliter) was also increased despite the slight reduction of left ventricular contractile performance (see above), resulting in an almost normal effective arterial elastance/end-systolic elastance ratio, i.e., ventriculoarterial coupling (Table 3), with values comparable to those observed in normal subjects by others. This result also supports the conclusion that left ventricular hypertrophy is responsible for the elevated developed force rather than an increased inotropic state.

Energy Transfer It has been shown by Suga et al. 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 pressure-volume area to external work represents the left ventricular work efficiency. In our study, the efficiency of energy transfer of the left ventricle was studied by determining the external work and the pressure-volume area, the latter representing the total mechanical energy of an ejecting contraction. In this study, coronary sinus catheterization was not allowed for ethical considerations; consequently, myocardial oxygen consumption could not be calculated. Nevertheless, as it has been demonstrated that the pressure-volume area is linearly related to myocardial oxygen consumption, mechanical efficiency was estimated through the external work/pressure-volume area ratio (called work efficiency). Because of increased arterial pressure, it is not surprising that the analysis of the components of mechanical energy of the left ventricle shows absolute values of potential energy, external work, and pressure-volume area significantly higher in hypertensive patients than in control subjects. Conversely, values of potential energy and pressure-volume area indexed to ventricular mass were similar in hypertensive patients and in control subjects, but external work was slightly lower in hypertensive patients (Table 3). Nevertheless, despite the slightly lower value of external work, work efficiency (external work/pressure-volume area) was comparable in the two groups with values within the normal range given in the literature. This normal level of mechanical efficiency, despite the slight depression of left ventricular contractile performance and the increased afterload, is in accordance with previously published data in patients with moderately depressed left ventricular pump function. Thus, a hypertrophied left ventricle might be a better means to preserve left ventricular pump function with less energetic cost than a left ventricle without hypertrophy.
that maintains its pump function, ie, delivers its external work, through an energetically costly increased myocardial contractility.\textsuperscript{9,37,51,52}

Methodologic Considerations In this study, we have 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 lead to some inaccuracy. In addition, because calculation of left ventricular mass by echocardiography takes into account the septal wall thickness, patients with septal/posterior wall thickness ratio \( \geq 1.2 \) have been excluded from the study. Lastly, left ventricular hypertrophy of our hypertensive patients was moderate and our results cannot be extended to patients with severe left ventricular hypertrophy.

As pressures decline rather rapidly from peak systolic pressure, there could be errors induced 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.

The end-systolic pressure-volume relationship was obtained by single-beat estimation, according to the method validated in humans by Takeuchi et al.\textsuperscript{30} This method avoids pharmacologic manipulations that have been demonstrated to overestimate end-systolic elastance when afterload is increased by angiotensin II and to underestimate its value when afterload is reduced by sodium nitroprusside.\textsuperscript{29} This method allows determination of end-systolic elastance from one pressure-volume loop with one measurement of left ventricular end-diastolic volume and an estimated peak isovolumic pressure. Takeuchi et al have shown that the curvilinearity of the true end-systolic pressure-volume relation at its extremities does not produce values of estimated peak isovolumic pressure significantly different from true values.\textsuperscript{30} In our study, values of estimated peak isovolumic pressure in the control group were similar to those published by Takeuchi et al.\textsuperscript{30} On the other hand, the curvilinearity of the true end-systolic pressure-volume relation at its extremities\textsuperscript{20,53,54} might also influence determination of end-systolic elastance when the volume axis intercept (Vo) is obtained by linear extrapolation of the end-systolic pressure-volume relationship. However, it has been shown that the curvilinearity of the end-systolic pressure-volume relationship does not prevent end-systolic elastance from being an accurate index of contractility.\textsuperscript{55,56}

We calculated the external work of the left ventricle as a trapezoid area constructed from the end-systolic and end-diastolic pressures and stroke volume. This assumption might produce some quantitative errors, but we think that these errors could not affect the main results provided by this study. Indeed, our values of potential energy, external work, and pressure-volume area in control subjects are quite similar to values previously published.\textsuperscript{27,28,47}

Myocardial oxygen consumption was not measured in this study, which lead us to use work efficiency in place of mechanical efficiency to evaluate the energy transfer of the left ventricle. Although this method does not allow us to know the true myocardial oxygen consumption, it provides a noninvasive means of estimating energy transfer. Finally, this study was realized on patients whose basal contractile state was unknown. Although contractile state could have an influence on the actual basal myocardial oxygen consumption,\textsuperscript{37} our results are relatively homogeneous, leading us to assume that the data presented are reliable and contain limited quantitative errors.

Lastly, coronary arteriography might have missed patients with microvascular disease, which is frequently observed in hypertensive patients where coronary reserve is reduced, especially when left ventricular mass is increased.\textsuperscript{57} However, it has never been demonstrated that a coronary microvascular disease...
could depress left ventricular contractile performance at rest.

In summary, in this study where the concept of ventriculoarterial coupling was used to evaluate left ventricular contractility and energy transfer. Our results show that, in hypertensive patients with left ventricular hypertrophy, the elevated contractile force is produced through left ventricular hypertrophy despite a moderate depression of myocardial contractile performance. Also, there is a preservation of the matching between left ventricular performance and arterial load and an optimal energy transfer. However, further investigation using methods allowing us to measure actual myocardial oxygen consumption noninvasively are warranted to confirm these results.

REFERENCES


