Increased Cardiac Performance in Mild Essential Hypertension
Left Ventricular Mechanics

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SUMMARY To analyze cardiovascular dynamics in essential hypertension, 81 subjects with untreated mild essential hypertension (mean blood pressure, 112 ± 14 mm Hg) and 87 normotensive subjects from the same working population were studied by echocardiography and simultaneous blood pressure determination. Hypertensive subjects had significantly higher pulse pressure, stroke volume index, cardiac index, left ventricular internal dimension, end-systolic pressure/volume ratio, end-systolic stress, left ventricular mass index, and relative wall thickness than normotensive subjects. Among both normotensive and hypertensive subjects, cardiac performance was closely dependent on afterload, as indicated by close inverse linear relationships between left ventricular fractional shortening and log end-systolic stress (r = −0.83 and −0.78 respectively; both, p < 0.001). However, 19 of 81 hypertensive patients (23%) fell above the 95% confidence limits of this relationship in normotensive subjects (p < 0.001 compared with that in normotensive subjects), with a bimodal distribution of fractional shortening as a percent of predicted in relation to end-systolic stress among patients with essential hypertension. This subgroup of hypertensive subjects, with increased resting cardiac performance independent of afterload, was similar in age to the remaining hypertensive subjects but had higher fractional shortening (41 ± 5% vs 35 ± 7%; p < 0.001) and cardiac index (4.3 ± 1.3 L/min/m² vs 3.4 ± 1.0 L/min/m²; p < 0.005) and lower total peripheral resistance (1257 ± 502 dyn sec cm⁻⁵ vs 1582 ± 584 dyn sec cm⁻⁵ p < 0.05) and left ventricular relative wall thickness (0.34 ± 0.06 vs 0.42 ± 0.10; p < 0.005). Thus, analysis of cardiac mechanics detected a subset of patients with essential hypertension in whom increased cardiac function cannot be attributed either to relative youth or to supercompensatory left ventricular hypertrophy. (Hypertension 7: 979–988, 1985)

KEY WORDS • echocardiography • essential hypertension • left ventricular function • left ventricular hypertrophy • left ventricular mechanics

STUDIES of the hemodynamics of essential hypertension have yielded conflicting results. Many investigators have found a tendency for patients with mild hypertension (most of whom are young) to have increased cardiac output and normal peripheral resistance,¹-³ while normal or reduced cardiac output and high peripheral resistance have been found in most,¹ ² 5, 8-10 but not all,¹¹-¹³ studies of patients with more severe hypertension. These findings have often been interpreted as representing successive phases in the course of hypertension — what begins as a high output-normal resistance state proceeds to a high resistance state with normal or low cardiac output.¹⁴,¹⁵ However, other studies have suggested that anatomical adaptations in both peripheral vessels and the heart¹⁶-¹⁹ may be more important than duration of hypertension per se in determining the hemodynamic pattern. In addition, limited evidence exists that contractile performance of the heart is supernormal in hypertensive patients with hyperdynamic circulatory states.²₀

A limitation of most available data on cardiac performance and hemodynamics in essential hypertension has been imposed by the need to select subjects whom it is appropriate to study by invasive methods. Subjects
with unusual features of hypertension are likely to be overrepresented, including relatively young subjects or those with extremely severe or labile hypertension. In addition, cardiac catheterization without angiography provides no information about the degree of hypertensive cardiac hypertrophy, an important parameter in assessing the cardiac response to hypertension. The advent of echocardiography has made possible accurate noninvasive evaluation of cardiac anatomy and function in subjects without regional left ventricular wall motion abnormalities.20-25

In this study, we have applied quantitative echocardiography to study untreated patients with mild essential hypertension and normotensive control subjects from the same adult, employed population. Specifically, we have analyzed the relation between left ventricular end-systolic wall stress and fractional shortening to test the hypothesis that a subset of patients with essential hypertension exhibit increased cardiac performance not attributable to supercompensatory left ventricular hypertrophy.

Subjects and Methods

The subjects were prospectively identified through a previously described program in which adult, employed New Yorkers are screened at the worksite.26 Consecutive, newly enrolled, unmedicated patients who met the exclusion criteria given below were studied between January and July 1981 after giving their informed consent. Members of a normotensive cohort stratified to reflect the age, sex, and race composition of the employed population of New York were studied concurrently. All subjects with evidence of cardiac disease other than hypertension by history, physical examination, electrocardiogram, or echocardiogram were excluded. At the time of echocardiographic study, no antihypertensive or other cardioactive medication had been taken for at least 3 weeks and no patients in this series had recently received reserpine or other longer acting medication. The patients who had previously received antihypertensive medications did not differ from the patients who had not with regard to any clinical or echocardiographic variable; therefore, the results from these two subgroups of hypertensive patients were pooled. All hypertensive patients had blood pressure in excess of 160 mm Hg systolic and/or 95 mm Hg diastolic on the average of four determinations in the clinic, whereas normotensive subjects had clinic blood pressures below 140/90 mm Hg at enrollment. Roughly 20% of subjects initially identified in both blood pressure groups were excluded because of technical limitations of the echocardiogram, and a further 10% were excluded because resting blood pressure at the time of echocardiography had been recorded on temporary sheets that were subsequently lost.

Arterial blood pressure was determined by arm cuff and mercury manometry with subjects at rest on at least four clinic visits and at the time of the echocardiographic study. The first and fifth phases of the Korotkoff sounds were used.

The M-mode echocardiograms were performed with the subject in the partial left lateral decubitus position using 13-mm 2.25 MHz transducers. A Picker Echoview 80C (Stamford, CT, USA) was used with a paper speed of 50 mm/sec. Echocardiograms were coded and interpreted blindly and in random order (without knowledge of blood pressure or other findings) by two investigators, as is customary in this laboratory. Left ventricular measurements were made at or just below the tips of the mitral valve leaflets only on high-quality tracings on which the right and left sides of the interventricular septum and the endocardial and epicardial surfaces of the posterior left ventricular wall were recorded continuously through the cardiac cycle. All measurements were made on up to five consecutive cardiac cycles (average, 3), and the means were used for subsequent calculations.

Measurements of interventricular septal thickness (IVST), posterior wall thickness (PWT), and left ventricular internal dimension (LVID) were made at end diastole and end systole according to the recommendation of the American Society of Echocardiography and used for all purposes except determination of left ventricular mass. A second group of end-diastolic measurements was made according to the Penn Convention for calculation of left ventricular mass by the following regression equation validated in two autopsy series:27-28

\[
\text{left ventricular mass} = 1.04 \left( \text{IVST} + \text{LVID} + \text{PWT} \right)^{3} - \left( \text{LVID} \right)^{3} - 14.25
\]

Relative wall thickness was calculated at end diastole by the following ratio: relative wall thickness = 2 \( \text{PWT/IVST} \). The percent change of LVID from end diastole to end systole was calculated (left ventricular fractional systolic shortening) to provide an index of myocardial performance. End-diastolic and end-systolic left ventricular volume were estimated by the cube-function formula from end-diastolic and end-systolic LVID respectively and used to estimate stroke volume and cardiac output.23 As an index of arterial compliance, the ratio of stroke volume to pulse pressure was calculated.

Total peripheral resistance (in dyn sec cm\(^{-5}\)) was estimated as (mean blood pressure \( \times 80 \))/cardiac output. Mean blood pressure was estimated from cuff systolic and diastolic blood pressure as diastolic blood pressure + 0.33 (systolic — diastolic blood pressure). To assess the possible occurrence of altered left ventricular contractility, additional indices were calculated, including the ratio of systolic blood pressure to end-systolic left ventricular volume (P/V ratio).29 End-systolic left ventricular meridional wall stress was calculated from echocardiographic dimensions and blood pressure at the time of echocardiography by a catheterization validated method:30

\[
\text{end-systolic meridional wall stress} = \frac{0.334 \times \text{LVID} \times \text{systolic blood pressure}}{\left( \text{PWT} \times (1 + \text{PWT}/\text{LVID}) \right)}
\]

Urine specimens were collected by the patient during the 24 hours preceding blood renin determinations. Blood was collected into potassium ethylenediaminetetraacetic acid Vacutainers from seated patients after they had been ambulatory for at least 2 hours. Follow-
ing centrifugation at room temperature, the plasma was frozen at -40 °C until assay. The latter was performed according to the methods developed by Sealey and Laragh.30 Urine sodium was determined by flame photometry. Renin values were evaluated with regard to the 24-hour urine sodium excretion using an established nomogram,31 and the renin status was determined as high, low, or normal.

Data are expressed as mean ± standard deviation. The statistical significance of differences between groups was assessed by analyses of variance and the chi-square test with Yates' continuity correction. Least-squares linear regression was performed to assess the relation between continuous variables.

**Results**

As shown in Table 1, the population study consisted of 81 patients with essential hypertension (49 white and 32 black) and 87 normotensive control subjects (42 white and 45 black). Of the hypertensive subjects, 33 (41%) had never been treated whereas 48 (59%) had been withdrawn from antihypertensive medication for periods of 3 weeks to 4 months. No significant difference in any studied variable existed between the previously treated and never treated group. The hypertensive subjects included 49 men and 32 women, ranging in age from 29 to 72 (average, 53 ± 10 yr); the normotensive comprised 50 men and 37 women, ranging in age from 19 to 65 (average, 44 ± 12 yr). None of the cardiac measurements that were analyzed exhibited significant relations to age in either normotensive or hypertensive groups in the present study. Similarly, analysis of the subset of 61 older normotensive subjects matched in age to the hypertensive subjects (average, 64 ± 7 yr) revealed similar results to those obtained with the entire normotensive group (e.g., cardiac index = 3.24 L/min/m² vs 3.08 L/min/m² and fractional shortening = 36 ± 8% vs 36 ± 7%).

Clinical blood pressures of hypertensive and normotensive subjects are given by sex in Table 1. In 57 of 81 patients with essential hypertension (70%) clinic systolic and diastolic pressures exceeded 140 and 90 mm Hg respectively, whereas 21 (26%) had isolated diastolic hypertension and only 3 (4%) had isolated systolic hypertension. Blood pressure at the time of echocardiography was significantly higher in the hypertensive than in the normotensive group (157 ± 18/92 ± 9 mm Hg vs 129 ± 11/79 ± 9 mm Hg for men and 156 ± 22/84 ± 12 mm Hg vs 124 ± 14/75 ± 9 mm Hg for women; p < 0.0001 in each case). Hematocrit was slightly higher in male hypertensive subjects (46.2 ± 3.0% vs 44.8 ± 3.4%; p < 0.05) but not in female hypertensive subjects (40.9 ± 3.8% vs 40.4 ± 3.0%) compared with that in the sex-matched normotensive group.

### Hemodynamic Parameters in Normotensive and Hypertensive Subjects

As may be seen in Table 2, the hypertensive patients exhibited significantly higher stroke volume index, cardiac index, P/V ratio, end-systolic meridional wall stress, and left ventricular end-diastolic dimension, whereas only insignificant increases were seen in left ventricular fractional shortening and total peripheral resistance. Left ventricular mass was increased (103 ± 33 g/m² vs 81 ± 22 g/m²; p < 0.001, as was relative wall thickness (0.40 ± 0.10 vs 0.36 ± 0.07; p < 0.005).

### Analysis of Cardiac Mechanics in Normotensive and Hypertensive Subjects

Among normotensive subjects, a close inverse correlation (r = -0.83) was observed between left ventricular fractional shortening and log end-systolic stress. The individual data points as well as the regression line and lines 2 standard deviations above and below it are shown in Figure 1. Individual patients are shown by the closed dots. As seen, 1 in 87 patients (1%) fell above this range, 3 in 87 (3%) fell below this range, and 83 of 87 (96%) fell within it.

A strong inverse correlation between left ventricular fractional shortening and log end-systolic meridional wall stress also was observed in the hypertensive subjects (r = -0.78, p < 0.001; Figure 2). However, both average fractional shortening and average end-systolic stress were slightly higher in the hypertensive patients than in the normotensive controls, although only the latter reached statistical significance (Table 2). The reason for this is apparent in Figure 2: 19 of 81 (23%) hypertensive subjects fell above the upper limit of the normal fractional shortening-end-systolic meridional wall stress relationship. This proportion was significantly higher than that found in the normotensive subjects (1 in 87 or 1%; p < 0.0001).

It is possible that the increased left ventricular fractional shortening observed in patients with hypertension reflects a simple upward shift of the entire hyper-

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**Table 1. Patient Characteristics**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normotensive</th>
<th>Hypertensive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men (n = 50)</td>
<td>Women (n = 37)</td>
</tr>
<tr>
<td>Clinic BP (mm Hg)</td>
<td>124 ± 11/78 ± 8</td>
<td>119 ± 22/78 ± 16</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>46 ± 11</td>
<td>42 ± 12</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.95 ± 0.16</td>
<td>1.67 ± 0.16</td>
</tr>
</tbody>
</table>

Data are means ± SD.

BP = blood pressure; BSA = body surface area.
TABLE 2. Hemodynamic and Echocardiographic Findings in Normotensive and Hypertensive Subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normotensive</th>
<th>Hypertensive (all)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure (mm Hg)</td>
<td>127 ± 13 / 77 ± 9</td>
<td>157 ± 19 / 89 ± 11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>50 ± 10</td>
<td>68 ± 18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV systolic fractional shortening (%)</td>
<td>35 ± 6</td>
<td>36 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>Stroke volume index (ml/m²)</td>
<td>44 ± 11</td>
<td>51 ± 16</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td>3.08 ± 0.94</td>
<td>3.59 ± 1.14</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>LV end-diastolic dimension (cm)</td>
<td>4.80 ± 0.49</td>
<td>5.00 ± 0.63</td>
<td>&lt;0.025</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>70 ± 14</td>
<td>71 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>Pressure/volume (mm Hg/ml)</td>
<td>5.0 ± 3.2</td>
<td>6.5 ± 6.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>ESS (10³ dyn/cm²)</td>
<td>63.1 ± 19.4</td>
<td>72.8 ± 25.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>TPR (dyn sec cm⁻5)</td>
<td>1445 ± 443</td>
<td>1505 ± 579</td>
<td>NS</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>81 ± 22</td>
<td>103 ± 33</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RWT</td>
<td>0.36 ± 0.07</td>
<td>0.40 ± 0.10</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

LV = left ventricular; NS = not significant; ESS = end-systolic meridional wall stress; TPR = total peripheral resistance; LVMI = left ventricular mass index; RWT = end-diastolic relative wall thickness.

*p values represent comparison of the two groups.

Figure 1. A close relationship exists between the log of meridional left ventricular wall stress at end systole and left ventricular fractional shortening in 87 normotensive subjects (r = 0.83, p < 0.0001). The solid line indicates the regression line, and the dashed lines are 2 SD above and below it; 83 of 87 subjects (95%) fell within these limits.

Figure 2. A close relationship exists between the log of end-systolic stress and fractional shortening in the 81 hypertensive patients (r = -0.78, p < 0.001). The regression line and ±2 SD lines from Figure 1 are shown. The data points for 19 of 81 hypertensive patients fell above the 95% confidence limits from normotensive subjects (compared with 1 of 87 normotensive subjects; p < 0.001).

The distribution of our data is highly suggestive of the existence of a distinct subset of patients with hypertension compared with that in normotensive subjects, or alternatively, our findings might be explained by the existence of two subsets among the patients with essential hypertension. To assess which of these possibilities is more likely we examined the distribution of left ventricular fractional shortening as a percent of predicted (from the regression line of normotensive subjects) in both normotensive and hypertensive groups (Figure 3). As may be seen in Figure 3, the distribution of left ventricular fractional shortening was unimodal among the normotensive subjects, and the peak occurred in the interval between 98 and 102% of the predicted value. In contrast, the distribution of left ventricular fractional shortening among patients with hypertension exhibited two distinct peaks, the higher of which (15 patients) also fell between 98 and 102% of the predicted value, while a second peak (13 patients) fell between 118 and 122% of the predicted value. A total of 24 patients with essential hypertension had fractional shortening 118% of predicted or greater, above the highest value among normotensive subjects, whereas only 4 normotensive subjects had lower fractional shortening than any hypertensive subjects, which provides further evidence against a simple shift of the distribution among hypertensive patients.

Thus, the distribution of our data is highly suggestive of the existence of a distinct subset of patients with
essential hypertension in whom cardiac performance is increased. Because left ventricular fractional shortening was adjusted in this analysis for end-systolic wall stress, the best measure of myocardial afterload, increased left ventricular systolic performance in this group of hypertensive patients is not dependent on either overcompensatory left ventricular hypertrophy or reduced afterload under the restful conditions of echocardiographic study.

Hemodynamic Parameters in Hypertensive Subjects with Normal and Enhanced Cardiac Performance

Hemodynamic parameters and indices of left ventricular hypertrophy are shown separately for the subsets of hypertensive subjects with normal and enhanced systolic performances (hereafter termed Groups 1 and 2) in Table 3. Significant differences appeared in indices of cardiac performance. Fractional shortening was higher in Group 2, as expected from the method of classification (41 ± 5% vs 35 ± 7%; p < 0.001). Other functional parameters were also higher in Group 2, including stroke volume index (63 ± 20 ml/m² vs 47 ± 13 ml/m²; p < 0.001) and cardiac index (4.25 ± 1.33 L/min/m² vs 3.39 ± 1.00 L/min/m²; p < 0.005). Heart rate did not differ significantly. Group 2 exhibited insignificantly higher LVID in diastole (5.2 ± 0.7 cm vs 4.9 ± 0.6 cm; p < 0.1) and P/V ratio (7.3 ± 6.1 mm Hg/ml vs 6.2 ± 6.1 mm Hg/ml).

Mean blood pressure did not differ between the two hypertensive groups, but Group 2 had a wider pulse pressure both in clinic blood pressures (63 ± 20 mm Hg vs 50 ± 15 mm Hg; p < 0.005) and at the time of echocardiography (81 ± 18 mm Hg vs 64 ± 16 mm Hg; p < 0.001) because of a higher systolic and lower
Arterial compliance estimated by the ratio of stroke volume index to pulse pressure was minimally higher in Group 2 hypertensive subjects (mean value = 0.78 ml/m²/mm Hg vs 0.73). Total peripheral resistance was significantly lower in Group 2 hypertensive subjects compared with that in Group 1 hypertensive subjects (1257 ± 502 dyn sec cm⁻⁵ vs 1582 ± 584 dyn sec cm⁻⁵; p < 0.05), but was not statistically different from values in the normotensive subjects (1445 ± 443 dyn sec cm⁻⁵; p < 0.2). The average duration of hypertension was similar in Groups 1 and 2 (4.7 and 4.9 yr respectively).

In Group 2, left ventricular mass index was slightly lower than that in Group 1 (92 ± 26 g/m² vs 106 ± 34 g/m²; p < 0.2), while relative wall thickness was strikingly lower (0.34 ± 0.06 vs 0.42 ± 0.10; p < 0.005). Of note, Group 1 differed significantly from normal in both these indices of left ventricular hypertrophy, whereas Group 2 did not. Individual data of Group 1 and 2 hypertensive subjects for selected parameters of cardiac structure and function are depicted in Figures 4, 5, and 6. The relation between cardiac index and relative wall thickness in the two hypertensive groups is depicted in Figure 7. As may be seen, a closer inverse relationship existed in Group 2 (r = -0.73) than in Group 1 (r = -0.53).

Renin Profile Status

Among the Group 1 hypertensive subjects who had renin determinations, 27 fell into the low renin group, 28 in the normal renin group, and 4 in the high renin group. Of the Group 2 subjects 6 were assigned to the low renin group, 11 to the normal renin group, and 1 to the high renin group. There was no significant difference in the renin status distribution between the two groups (chi square = 0.941, p < 0.50).

Renal Status

Renal function, as judged by serum creatinine levels, did not differ significantly between the two groups, nor did either group's level differ from those found in normotensive subjects. The values were 1.25 ± 0.21 mg/dl for normotensive subjects, 1.26 ± 0.22 mg/dl for Group 1 hypertensive subjects (p = 0.48 compared with normotensive subjects), and 1.33 ± 0.15 mg/dl for Group 2 hypertensive subjects (p = 0.06 compared with normotensive subjects; p = 0.08 compared with Group 1 hypertensive subjects).

Discussion

Evidence of increased cardiac performance has been found in some groups of patients with essential hypertension1-7, 11-13, 20 but not in others.1, 2, 3, 8, 14, 15 One possible explanation for this has been that a proportion of hypertensive patients may acquire an overcompensatory degree of left ventricular hypertrophy, which results in low wall stresses and supernormal systolic performance.22 Our previous echocardiographic studies, which demonstrated a strong inverse relation between left ventricular fractional shortening and end-systolic stress in two independent populations, were compatible with this concept.19, 33 However, use of referred hypertensive patients in previous studies prevented comparison with completely matched control groups and have limited the general applicability of the results. In the present study, we compared cardiac function and hemodynamic findings in representative hypertensive and normotensive subjects from a general adult, employed population.

Noninvasive assessment of resting cardiac mechanics in the present study allowed us to detect an apparent subset of hypertensive patients with supernormal left ventricular function. In contrast to previous sugges-
Figure 6. *Group 1* hypertensive subjects (H-1) showed concentric hypertrophy, as indicated by increased relative wall thickness, compared with that in normotensive subjects (N; \( p < 0.001 \)), whereas *Group 2* patients (H-2) did not.

Figure 7. Relative wall thickness (RWT) and cardiac index (CI) are plotted. There was an excellent inverse linear relation for both groups of hypertensive subjects, which was more pronounced in *Group 2* \( (r = -0.73) \) than in *Group 2* \( (r = -0.53) \).
and contractility resulting in high cardiac output, a set of findings quite distinct from the other hypertensive subjects.

The existence of this subset of hypertensive subjects has been suggested by the occurrence of increases in cardiac output or other indices of cardiac performance in some previous studies of essential hypertensive patients, but it has not been possible previously to separate a subset of patients with evidence of hyperfunction. A similar problem would have occurred in the present study if we had relied on supernormal cardiac output to detect this group, since only 8% of the hypertensive subjects had cardiac output more than 2 SD above the normal mean. Analysis of cardiac mechanics to identify a distinctive subgroup with increased cardiac function in the present study has allowed us to exclude two factors — age and the presence of supercompensatory myocardial hypertrophy — as causes of increased cardiac performance.

The mechanism of the increased cardiac performance in this subset of hypertensive subjects is uncertain. The possibility of anemia as a cause is unlikely, since the Group 2 hypertensive subjects had a slightly, although insignificantly, higher hematocrit (47.0 ± 4.5% vs 45.9 ± 2.3% for men and 42.3 ± 2.1% vs 40.6 ± 4.1% for women) than those in Group 1. It has been previously suggested that causes of a high cardiac output in essential hypertension might include enhanced sympathetic and either or both decreased parasympathetic drive and a resetting of the pressure-natriuresis curve of the kidneys, which results in salt and water retention. Moreover, it has been proposed, that in an attempt to normalize this high output state, total peripheral resistance rises because of structural changes in blood vessels, increased vascular reactivity, or autoregulatory vasoconstriction. Thus, a primary high cardiac output state (caused by increased contractility/or preload or both) associated with normal peripheral resistance state many change into the normal output–high resistance state considered to be typical of established hypertension.

The findings of the present study differ from this proposed schema in two regards. First, the subgroup of hypertensive patients with increased cardiac performance actually had an insignificantly lower peripheral resistance than did the normotensive control subjects. This occurred despite a higher level of mean blood pressure in this group (117 mm Hg in the clinic and 112 mm Hg at rest during echocardiography) than that reported in previous studies of mild or borderline hypertension (100–108 mm Hg). The explanation for this combination of hemodynamic findings is uncertain, but may be related to reports by Lund-Johansen and others that patients with mild hypertension had resting hypermetabolism, as manifested by increased oxygen consumption, with a subsequent chemical vasodilatation and an appropriately increased cardiac output. Second, the age of the patients in the two hypertensive subgroups was virtually identical, and no difference in known duration of hypertension existed between the groups, which reduces the likelihood that they represent sequential stages of disease evolution. However, the fact that the high output group had a lower left ventricular mass index and higher end-systolic stress suggests it is still possible that hypertension was more recent in onset and adequate hypertrophy to reduce wall stress had not yet been achieved. Further follow-up data will be needed to evaluate the frequency with which average patients with essential hypertension evolve from a high output–low resistance state to a normal output–high resistance state.

Several aspects of the present study differ from previous investigations of cardiovascular dynamics in hypertension. Most important among these is evaluation of a representative sample of patients with essential hypertension and normotensive control subjects with similar sex and race distribution drawn from the general adult, working population. Relatively mild hypertension predominated at all ages in the resulting group of patients, with diastolic blood pressure under 105 mm Hg in approximately 80% of patients. This differs from the mix of patients typically studied in many hypertension research units but reflects the distribution of blood pressure levels among patients seen in routine clinical practice. However, examination of the relation between peripheral resistance and cardiac output in our hypertensive and normotensive groups reveals that the hypertensive subjects had substantially higher total peripheral resistance at every level of cardiac output (Figure 8), which is compatible with previous emphasis on

![Figure 8. Total peripheral resistance is plotted against cardiac index for all patients. A higher total peripheral resistance was seen at every level of cardiac output for the hypertensive patients.](http://hyper.ahajournals.org/content/hypertension/7/6/986/F1.large.jpg)
elevated resistance as the classic hemodynamic abnormality of hypertension.

Another important feature of the present study is its reliance on echocardiographic methods, the noninvasive nature of which makes them suitable for study of population samples. An additional advantage of echocardiography for the study of patients with hypertension is its ability to accurately measure left ventricular anatomy. Further, because of its noninvasive nature, its hemodynamic determinations may better reflect subject's baseline cardiac state than do those obtained by an invasive approach. The validity of hemodynamic measurements derived from echocardiographic dimensions in the present study rests on several important precautions we have taken. First, no subject in the present study had either clinical or electrocardiographic evidence of coronary artery disease; nor were there any wall motion abnormalities in visualized segments or echocardiographic findings indicative of dysfunction of nonvisualized segments (including reduced aortic valve ejection time corrected for heart rate, diminished aortic root motion, or increased E point-septal separation). It should be emphasized that the analysis we have performed, using M-mode echocardiographic methods, cannot be validly applied to hypertensive or normotensive subjects with regional left ventricular wall motion abnormalities.

Second, no subject had marked left ventricular dilatation, another known cause of inaccuracy in echocardiographic volume estimates. Nevertheless, despite these precautions the limited visualization of the left ventricle obtained by M-mode echocardiography may result in small errors in volume estimates and probably accounts for the larger standard deviations of hemodynamic parameters in our study population than in similar groups studied invasively. Third, all echocardiographic measurements were made blindly, without knowledge of which subjects were hypertensive or normotensive.

In conclusion, the present study suggests that analysis of cardiac mechanics may provide useful insight into cardiovascular dynamics in patients with essential hypertension. Left ventricular systolic performance (fractional systolic shortening) was elevated in relation to afterload (end-systolic stress) in approximately one-fourth of unselected patients with essential hypertension, with a bimodal distribution suggesting the possible existence of two distinct subgroups. The subgroup with higher resting performance had evidence of higher cardiac output and less marked left ventricular hypertrophy than the remaining hypertensive subjects.

Further study is needed to delineate the characteristics of the subgroup of hypertensive patients with increased cardiac performance and to determine if their increased cardiac output is an appropriate response to supernormal oxygen consumption, as has been suggested by previous studies from other laboratories. Improved characterization of the subgroup of patients with essential hypertension with a hyperdynamic circulatory state would facilitate clarification of the roles of the thyroid and sympathoadrenal axes as well as other metabolic factors in the hemodynamics of hypertension and the evolution of hypertensive heart disease.

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