Disparate Relationships Between Blood Pressure and Left Ventricular Mass in Patients With and Without Left Ventricular Hypertrophy

JAN I. M. DRAYER, JULIUS M. GARDIN, DEBORAH D. BREWER, AND MICHAEL A. WEBER

SUMMARY The relationship between casual blood pressure and left ventricular (LV) mass has been reported to be fairly weak in hypertensive patients. In this study we analyzed this relationship using noninvasive devices to monitor blood pressure for 24 hours in ambulatory patients and M-mode echocardiography to determine LV mass. Among the 33 patients with hypertension, 21 had echocardiographic LV hypertrophy (LV mass >250 g). Patients with LV hypertrophy did not differ significantly from patients without hypertrophy with respect to age or casual systolic or diastolic blood pressure. The averages of whole-day systolic and diastolic blood pressures were 146 ± 17 (SD) over 90 ± 12 and 136 ± 16 over 89 ± 12 mm Hg, respectively. The relationship between whole-day average systolic blood pressure and LV mass was significantly positive (r = 0.66, p<0.05) in patients without hypertrophy but was not significant in patients with LV hypertrophy (r = -0.24). Similarly, the relationship between whole-day average diastolic blood pressure and LV mass was significantly positive in the former group (r = 0.64, p<0.05) but significantly negative in hypertensive patients with LV hypertrophy (r = -0.67, p< 0.01).

Thus, blood pressure correlates positively with LV mass only in patients without cardiac hypertrophy. In hypertensive patients with LV hypertrophy, factors additional to the high blood pressure itself must participate in the regulation of LV mass. (Hypertension 9 [Suppl II]: II-61-II-64, 1987)

KEY WORDS • ambulatory blood pressure monitoring • casual blood pressure • hypertension • left ventricular hypertrophy • echocardiography

RECENT studies have revealed that new noninvasive techniques have great value in evaluating the relationship between blood pressure and left ventricular (LV) mass. The correlations between casual systolic or casual diastolic blood pressure and echocardiographic LV mass are reported to be relatively weak. However, it seems well established that the relationship between casual systolic blood pressure and LV mass is stronger than that between casual diastolic blood pressure and LV mass (Table 1). In addition, the data from the same studies show that the relationship between blood pressure and LV mass is more significant when blood pressure is expressed as the average of all blood pressures obtained during a 24-hour period of continuous monitoring than when casual blood pressures are used. Most patients included in the studies mentioned here did not have signs of LV hypertrophy, however. None of the patients studied by Rowlands et al., and fewer than 25% of the patients studied by Devereux et al., or by our group had echocardiographic LV hypertrophy. The lack of patients with clear hypertension-induced end-organ damage may have affected the outcome of these studies.

Raftery et al. and Messerli et al. have demonstrated that the 24-hour blood pressure pattern in hypertensive patients with LV hypertrophy is significantly different from that found in hypertensive patients without LV hypertrophy. The diurnal pattern of blood pressure in patients with electrocardiographic or echocardiographic LV hypertrophy was shifted in a parallel fashion to a higher level than that found in hypertensive patients without LV hypertrophy. In the present study, we have further analyzed the relationship between blood pressure and LV mass in hypertensive patients with and without hypertrophy.

Methods

The study was performed in 33 male patients with mild essential hypertension. None of the patients had known coronary artery disease or a history of a recent myocardial infarction. None of the patients had evidence for other forms of heart disease such as cardiomyopathy. Antihypertensive medication had been discontinued for at least 2 weeks prior to the date of the study. A routine blood test, standard casual blood pressure and heart rate, and an M-mode echocardiogram were obtained on the study day. Ambulatory blood pressure monitoring was performed in all patients during 24 hours using a wholly automated device. In 12 patients, the Pressurometer III (Del Mar Avionics, Irvine, CA, USA) was used to monitor blood pressure at 7.5-minute intervals for 24 hours. In the remaining 21 patients, the ICR monitor (Spacelabs, Bellevue, WA, USA) was used to monitor blood pressure at 15-minute intervals for 24 hours. A successful and complete monitoring was obtained in 26 of the patients. The averages of systolic and diastolic blood pressures...
recorded during the 24-hour day were calculated in these patients. The techniques used to edit the data have been reported previously. From the two-dimensional guided M-mode echocardiogram, LV mass was calculated using the averages of the thickness of the interventricular septum, the LV posterior free wall, and the transverse dimension of the left ventricle obtained during diastole. These parameters were measured in at least three beats using the criteria of the American Society of Echocardiography. The relationship between blood pressure and LV mass was analyzed using standard statistical tests for the group of patients as a whole (n = 33), and in subgroups of patients without echocardiographic LV hypertrophy (LV mass < 250 g, n = 12) and patients with echocardiographic LV hypertrophy (LV mass > 250 g, n = 21).

Results

The relevant clinical and biochemical characteristics of the 12 male patients with hypertension alone and the 21 male patients with hypertension and LV hypertrophy are given in Table 2. The two subgroups of patients did not differ with respect to their mean age, casual blood pressures, and heart rate, averages of whole-day blood pressures and heart rates, serum creatinine, or venous blood hematocrit. The body surface area was 2.07 ± 0.15 (SD) m² in patients with hypertrophy and 2.01 ± 0.13 m² in patients without hypertrophy. The whole-day averages of blood pressure were lower than casual blood pressures in both subgroups, but the difference between casual and average 24-hour systolic blood pressure did not reach statistical significance in the hypertensive patients with LV hypertrophy.

Septal thickness, posterior wall thickness, LV internal dimension, and LV mass were all significantly greater in hypertensive patients with, than in those without, echocardiographic LV hypertrophy (p < 0.05; Table 3). The systolic ejection fraction was 0.80 ± 0.24 (SD) in patients with hypertrophy and 0.80 ± 0.18 in patients without hypertrophy. The relationship between blood pressure and echocardiographic LV mass is depicted in Figures 1 and 2.

The correlation coefficient between casual systolic blood pressure and LV mass was −0.02 for the group of patients as a whole, but in patients whose LV mass was less than 250 g, a highly significant positive correlation (r = 0.63, p < 0.05) was found between casual systolic blood pressure and LV mass. In contrast, this relationship was not significant (r = −0.23) in patients with an LV mass greater than 250 g (see Figure 1). Similarly, the correlation coefficient between casual diastolic blood pressure and LV mass was not significant for the group as a whole (r = −0.17) but was significantly positive within the group without LV hypertrophy (r = 0.62, p < 0.05). The correlation was negative in hypertensive patients with LV hypertrophy (r = −0.33).

The relationship between whole-day average systolic or diastolic blood pressure and LV mass was not significant for the group as a whole (r = 0.18 and r = −0.20, respectively). However, subgroup analysis revealed a significantly positive relationship between blood pressure and LV mass in hypertensive patients without hypertrophy and a negative correlation in patients with LV hypertrophy (see Figure 2). The slopes of the regression lines for the relationships between blood pressures and LV mass are given in Table 4.

Discussion

Data from the 33 patients with essential hypertension reported in this study reveal that the relationship between casual systolic or diastolic blood pressure and left ventricular mass is weak. In fact, the correlation coefficient between systolic blood pressure and LV mass in this study was smaller than in previously reported studies. The relationship between blood pressure and LV mass did not improve when 24-hour ambulatory blood pressure monitoring techniques were used to establish the degree of hypertension in our patients. This finding contrasts with previous reports (see Table 1). In previous reports, averages of whole-day blood pressures were found to have a closer association with LV mass than casual blood pressures.

We attempted to analyze this discrepancy by subdividing the patients into those with and those without echocardiographic evidence for LV hypertrophy. A comparison of the clinical characteristics of the two subgroups revealed that blood pressures were comparable between the two groups. Only the whole-day average systolic blood pressure was slightly, but not significantly, higher in patients with LV hypertrophy than in those without hypertrophy. This finding confirms the relatively weak relationship between blood pressure and LV mass found in
the group as a whole. Others have reported that the diurnal pattern of blood pressure in hypertensive patients with LV hypertrophy is different from those with a normal LV mass. In these studies blood pressure was significantly higher in patients with hypertrophy, especially during the night. Data from Devereux et al. and the data presented here are not in agree-

**FIGURE 1.** Relationship between casual systolic (A) or diastolic (B) blood pressure and echocardiographic left ventricular mass in hypertension.

**FIGURE 2.** Relationship between whole-day systolic (A) or diastolic (B) blood pressure averages and echocardiographic left ventricular mass in hypertension.
TABLE 4.  Relationships Between Blood Pressure and Left Ventricular Mass

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Without hypertrophy</th>
<th>With hypertrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 12</td>
<td>n = 21</td>
</tr>
<tr>
<td>Casual systolic</td>
<td>slope = +0.80</td>
<td>slope = -0.80*</td>
</tr>
<tr>
<td>Casual diastolic</td>
<td>slope = +1.45</td>
<td>slope = -1.96†</td>
</tr>
<tr>
<td>24-hr systolic</td>
<td>slope = +0.94</td>
<td>slope = -0.95†</td>
</tr>
<tr>
<td>24-hr diastolic</td>
<td>slope = +1.28</td>
<td>slope = -3.67†</td>
</tr>
</tbody>
</table>

*p < 0.05, †p < 0.01, significance of difference of slopes between the subgroups.

In fact, evaluation of the relationship between blood pressure and LV mass in subgroups of patients with and without echocardiographic LV hypertrophy showed that this positive relationship between blood pressure and LV mass was absent in the former group but present in the latter group. Relatively strong, direct correlation coefficients were found between systolic or diastolic blood pressure and LV mass in patients without cardiac hypertrophy. Once hypertrophy is present, however, the relationship tends to become a negative one. This disparate relationship between blood pressure and LV mass for the two subgroups of patients was most impressive for the whole-day average of diastolic blood pressures. The correlation coefficient between whole-day average diastolic blood pressure and LV mass was 0.64 (p < 0.05) in patients without hypertrophy and -0.67 (p < 0.01) in hypertensive patients with LV hypertrophy.

The positive relationship between blood pressure and LV mass reported here for patients without cardiac hypertrophy agrees with data published previously. The results probably indicate that blood pressure is one of the important factors in the development of LV hypertrophy in hypertensive patients. Once cardiac hypertrophy has developed, however, factors other than blood pressure must be involved in the regulation of cardiac muscle mass. Alternatively, one could argue that patients with marked hypertrophy may not be able to maintain the high levels of blood pressure that have caused the increase in LV mass. Decreases in cardiac function associated with marked increases in LV mass may be responsible for this phenomenon. However, a significant decrease in systolic function using ejection fraction measured by echocardiography was not observed in our patients with hypertrophy. Thus, further evaluation of the changing relationship between blood pressure and LV mass in hypertensive patients seems warranted.

References

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Hypertension. 1987;9:II61
doi: 10.1161/01.HYP.9.2_Pt_2.II61

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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