Left Ventricular Hypertrophy and Function in High, Normal, and Low-Renin Forms of Essential Hypertension

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SUMMARY To assess the relative importance of the level of blood pressure (BP) and renin profile status as determinants of hypertensive left ventricular hypertrophy (LVH) and dysfunction, we studied, by quantitative echocardiography, 118 hypertensive patients off medication. The 19 high-renin patients were younger (31 ± 13 years; p < 0.01) but had hypertension of severity (152 ± 13/95 ± 11 mg Hg) similar to the 79 normal-renin patients (42 ± 14 years; 152 ± 17/98 ± 12 mg Hg) and 20 low-renin patients (49 ± 13 yrs; 157 ± 17/95 ± 11 mm Hg). Left ventricular (LV) mass index (normal = 70 ± 25 g/m²) was similar in the high- (113 ± 21 g/m², p < 0.001), normal = (114 ± 31 g/m², p < 0.001), and low-renin patients (115 ± 18 g/m², p < 0.01). End-diastolic relative wall thickness (nl = 0.32 ± 0.05) was equally elevated in high- (0.41 ± 0.09), normal (0.42 ± 0.08) and low-renin groups (0.41 ± 0.08) (all p < 0.001). In the entire population, there was a closer correlation of relative wall thickness with total peripheral resistance (r = 0.54, p < 0.001) than with mean blood pressure (r = 0.31, p < 0.05). LV dysfunction (LV fractional systolic shortening < 26%) occurred only in two high-renin patients, whereas LV fractional shortening was significantly increased in the low-renin subgroup (p < 0.01). We conclude that the degree of LVH is similar in low-, normal-, and high-renin hypertensives and is proportional to the degree of hypertension, even though the high-renin patients were significantly younger; but that the low-renin patients with similar level of blood pressure, even though significantly older and with a longer duration of hypertension than the other patient groups, manifest increased LV function compared to normals. (Hypertension 4:524-531, 1982)

KEY WORDS • left ventricular hypertrophy • hypertension • renin • echocardiography

HYPERTENSION commonly causes cardiac hypertrophy and failure, the frequency and severity of which are related to the degree of blood pressure (BP) elevation, and to the duration of hypertension. Cardiac size may differ strikingly, however, between human patients or spontaneously hypertensive rats (SHR) with the same level of blood pressure. It has been suggested, therefore, that factors in addition to BP itself may play a role in the pathogenesis of left ventricular hypertrophy (LVH) and dysfunction in hypertension.

Because of its central importance in normal BP regulation and in many forms of human hypertension, the renin-angiotensin-aldosterone system has received considerable attention as a factor in hypertensive heart disease. Brunner et al. reported that high-renin patients were at greater risk of stroke or heart attack than either normal-renin or low-renin hypertensives, a finding that has been supported by some studies but not others. Indirect evidence for a role of the renin-angiotensin system in hypertensive LVH has been provided by studies by Sen et al. of the cardiac effects of controlling hypertension by use of different drugs in the SHR. Treatment with alpha-methyl dopa, propranolol, or the angiotensin-converting enzyme inhibitor (captopril), which inhibit the renin-angiotensin system at different points, either prevented LVH from developing or caused partial reversal of established LVH. We have recently found, similarly, that control of essential hypertension by beta-blockers and alpha-methyldopa caused partial reversal of LVH. In contrast, BP control in SHR with either hydralazine or minoxidil, which caused elevation of plasma renin levels, had no effect on LVH.
Despite this evidence suggesting that activity of the renin-angiotensin system may contribute to hypertensive LVH, this question has not been studied in human subjects with hypertension. Accordingly, we undertook the present study to evaluate the relationship of BP and renin-profile status to LVH and function in humans.

Methods

Patients

We studied 119 outpatients with essential hypertension who had technically excellent echocardiograms within 1 week of renin-sodium profile determination. We excluded 46 additional patients because of suboptimal echocardiographic studies (in 26 patients) or because our review of clinical records revealed angina pectoris, prior myocardial infarction, valvular heart disease, atrial fibrillation, or previous congestive heart failure. There was no significant difference between the studied and excluded patients with regard to renin profile status or outpatient BP. Of the 118 patients 27 had never received antihypertensive medication; of the 91 who had been treated previously, eight had been off therapy for 1 to 7 years, seven for 6 to 11 months, 16 for 3 to 5 months, and 60 for 3 to 12 weeks. Previous treatment consisted solely of antisympathetic drugs (beta-blockers, alpha-methyldopa, reserpine, clonidine) in 12 patients, diuretics in 28 patients, antisympathetic drugs and diuretics in 33, and vasodilators (hydralazine or prazosin) in combination with other drugs in 10. Eight patients were unable to specify their previous treatment.

Blood pressure was determined by standard sphygmomanometric methods; the average of all measurements during the period while the patient was off treatment before echocardiographic study (up to 3 months) was used for this portion of the study. Blood pressure exceeded 140 mm Hg systolic and/or 90 mm Hg diastolic in all patients. Control echocardiographic data was obtained from 79 normal subjects studied in this laboratory.

Echocardiographic Measurements

All patients were studied in the left decubitus position using standard echocardiographic technique. Studies were performed using 2.25 megahertz transducers with a Picker 80c echograph, a Smith Kline 20A echograph, or a custom-built amplifier with a Hewlett-Packard X-Y display; Honeywell 1856 fiberoptic recorders were used with all machines.

The echocardiograms were coded and interpreted blindly and independently by two investigators (R.B.D. and D.D.S.) to minimize variance in echocardiographic measurements. The LV 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 surface of the posterior LV wall were recorded continuously through the cardiac cycle. Measurements of interventricular septal thickness (IVST) and posterior wall thickness (PWT) and of left ventricular internal dimension (LVID) were made at end-diastolic and end-systole according to the recommendation of the American Society of Echocardiography and used for all purposes, except measurement of LV mass and detection of asymmetric septal hypertrophy. Additional measurements of IVST and PWT and of LVID were made at end-diastole according to the Penn Convention for calculation of LV mass by the following autopsy-validated regression equation:

$$LVM = 1.04 \left[ (IVST_p + LVID_p + PWT_p)^3 - (LVID_p)^3 \right] - 13.6 \text{ g.}$$

The IVST and PWT were also measured in mid-diastole before the onset of atrial systole, to determine the presence of asymmetric septal hypertrophy.

Relative wall thickness (RWT) was calculated at end-diastolic and end-systolic by the ratio: $RWT = \frac{LVID}{PWT}$. The percent change of LVID from end-diastolic to end-systolic was calculated (LV fractional systolic shortening) to provide an index of myocardial performance. End-diastolic and end-systolic LV volume were derived by the cube-function formula from end-diastolic and end-systolic LVID respectively and used to estimate stroke volume and cardiac output. Total peripheral resistance (TPR) in dyne·cm·sec$^{-3}$ was estimated as:

$$TPR = \frac{\text{diastolic pressure} + 0.33 \times \text{systolic-diastolic pressure)} \times 80}{\text{cardiac output}}$$

Measurements of cardiac dimensions by two investigators were averaged when they differed by no more than 1 mm for wall thickness of 2 mm for LVID. Echocardiograms that resulted in greater differences in measurements were reviewed jointly and the disagreement resolved before the code was broken.

Renin Profile Determination

Patients were instructed in the collection of 24-hour urine specimens, which they collected during the day prior to the drawing of blood for renin measurement. Blood was drawn into K$_2$EDTA Vacutainers from seated patients who had been ambulatory for at least 2 hours. The blood was centrifuged at room temperature and the plasma frozen at $-40^\circ\text{C}$ until the renin assay was performed. Plasma renin activity was measured by the radioimmunoassay method of Sealey and Laragh. Sodium and potassium were measured in the urine by flame photometry, and the 24-hour urine aldosterone excretion was measured by radioimmunoassay. The renin values were evaluated in relation to the 24-hour urine sodium excretion using a nomogram, and the patients were then classified into low-, normal-, and high-renin subgroups. Since renin-sodium profiling is not precise in subjects ingesting a high sodium intake, patients who excreted more than 140 mEq sodium per day were asked to ingest a moderately restricted sodium diet for 1 week, and the measurements were then repeated.
Statistical Methods
The data were analyzed using least squares linear regression and Student's t test, with correction for multiple comparison.28

Results
Subjects
Patients were classified by renin-sodium profile, using the nomogram,27 into high-renin, (n = 19), normal-renin (n = 79), and low-renin (n = 20) groups. Patient characteristics are listed in table 1. High-renin patients were similar in age to normals, and younger than the other two hypertensive groups. Males predominated in the normal-renin and high-renin subgroups, which accordingly had higher body surface areas. There was no significant difference among hypertensive groups in the frequency of previous treatment and no significant effect of type of previous treatment on LV mass (fig. 1). This may be because many patients had been off therapy for prolonged periods, up to 7 years. Blood pressure was elevated to a moderate, similar degree in all three groups. Low-renin patients had a longer duration of hypertension than either normal- or high-renin patients (table 1).

Echocardiographic Findings
Echocardiographic findings in the patients in each renin subgroup as well as control values from 79 normal subjects studied in this laboratory are shown in table 2 and figure 2.

Left Ventricular Hypertrophy
In each subgroup of hypertensives, IVST and PWT at end-diastole were significantly increased while LVID was minimally elevated. As a result, mild concentric LVH was present, as indicated by a significant increase in diastolic relative wall thickness (RWTd) (normal = 0.32 ± 0.05) in high-renin (0.41 ± 0.09, p < 0.01 vs normals), normal-renin (0.42 ± 0.08, p < 0.001), and low-renin hypertensive patients (0.41 ± 0.08, p < 0.001). In the entire population, there was a moderate correlation of RWTd with total peripheral resistance (r = 0.54, p < 0.001), which was closer than the correlation of RWTd with mean BP (r = 0.31, p < 0.05). There was similar, moderate LVH, as measured by LV mass index (normal = 70 ± 25 g/m²), in all three groups (113 ± 21 to 116 ± 33 g/m², p < 0.001 for each group vs normal). Left ventricular mass was not significantly related to age in either the normal subjects or any of the renin profile groups (p > 0.2 for all groups); LV mass index was similar in 29 normal subjects with a mean age of 48 ± 10 years (71 ± 20 g/m²). Therefore, age stratification of the data has not been performed.

Since LVH in subjects with mild hypertension has been reported to affect the interventricular septum more than the posterior wall of the LV, the ratio of IVST to PWT at end-diastole by American Society of Echocardiography criteria was calculated and found to be the same (1.1) in our control patients and each hypertensive subgroup. However, in only four patients (three normal-renin, and one low-renin) was dispro-
### Table 1. Clinical Findings

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects</th>
<th>Hypertensive subjects</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>High-renin</td>
<td>Normal-renin</td>
<td>Low-renin</td>
</tr>
<tr>
<td></td>
<td>79</td>
<td>19</td>
<td>79</td>
<td>20</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>32 ± 13</td>
<td>31 ± 13</td>
<td>42 ± 14</td>
<td>50 ± 13</td>
</tr>
<tr>
<td>Sex</td>
<td>27M, 52F</td>
<td>16M, 3F</td>
<td>54M, 25F</td>
<td>10M, 10F</td>
</tr>
<tr>
<td>Plasma renin</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension duration (yrs)</td>
<td></td>
<td>5.4 ± 7.8</td>
<td>5.4 ± 7.3</td>
<td>7.7 ± 8.9</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>119 ± 12</td>
<td>152 ± 13*</td>
<td>152 ± 17*</td>
<td>157 ± 17*</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>73 ± 8</td>
<td>96 ± 11*</td>
<td>98 ± 12*</td>
<td>95 ± 11*</td>
</tr>
<tr>
<td>Previously treated (%)</td>
<td>0</td>
<td>68</td>
<td>77</td>
<td>85</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.80 ± 0.15</td>
<td>1.93 ± 0.17</td>
<td>1.93 ± 0.16</td>
<td>1.79 ± 0.14</td>
</tr>
</tbody>
</table>

*p < 0.001 vs normals.  
BSA = body surface area; BP = blood pressure; M = male; F = female.

### Table 2. Echocardiographic Findings in High-, Normal-, and Low-Renin Hypertension

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects (79)</th>
<th>Hypertensive subjects</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>High-renin (19)</td>
<td>Normal-renin (79)</td>
<td>Low-renin (20)</td>
</tr>
<tr>
<td>IVSd (cm)</td>
<td>0.9 ± 0.2</td>
<td>1.1 ± 0.24</td>
<td>1.2 ± 0.3†</td>
<td>1.2 ± 0.3†</td>
</tr>
<tr>
<td>LVIDd (cm)</td>
<td>4.8 ± 0.5</td>
<td>5.1 ± 0.6</td>
<td>5.0 ± 0.7</td>
<td>5.0 ± 0.6</td>
</tr>
<tr>
<td>PWTd (cm)</td>
<td>0.8 ± 0.2</td>
<td>1.0 ± 0.3*</td>
<td>1.1 ± 0.2‡</td>
<td>1.0 ± 0.3*</td>
</tr>
<tr>
<td>IVSd/PWTd</td>
<td>1.1 ± 0.1</td>
<td>1.1 ± 0.1</td>
<td>1.1 ± 0.2</td>
<td>1.1 ± 0.2</td>
</tr>
<tr>
<td>LV mass (g/m²)</td>
<td>70 ± 25</td>
<td>113 ± 21‡</td>
<td>114 ± 31§</td>
<td>116 ± 33§</td>
</tr>
<tr>
<td>RWTd</td>
<td>0.32 ± 0.05</td>
<td>0.41 ± 0.09‡</td>
<td>0.42 ± 0.08§</td>
<td>0.41 ± 0.08§</td>
</tr>
<tr>
<td>IVSs (cm)</td>
<td>1.4 ± 0.3</td>
<td>1.6 ± 0.3*</td>
<td>1.6 ± 0.2‡</td>
<td>1.6 ± 0.3‡</td>
</tr>
<tr>
<td>LVIDs (cm)</td>
<td>3.1 ± 0.5</td>
<td>3.2 ± 0.6</td>
<td>3.1 ± 0.5</td>
<td>3.0 ± 0.5</td>
</tr>
<tr>
<td>PWTs (cm)</td>
<td>1.4 ± 0.3</td>
<td>1.6 ± 0.5†</td>
<td>1.6 ± 0.3§</td>
<td>1.6 ± 0.4†</td>
</tr>
<tr>
<td>RWTs</td>
<td>0.90 ± 0.12</td>
<td>0.99 ± 0.21</td>
<td>1.07 ± 0.24§</td>
<td>1.09 ± 0.27†</td>
</tr>
<tr>
<td>Δ LVID (%)</td>
<td>35 ± 5</td>
<td>36 ± 7</td>
<td>37 ± 6</td>
<td>41 ± 8‡</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>3.15 ± 1.0</td>
<td>3.37 ± 1.0</td>
<td>3.33 ± 0.95</td>
<td>3.77 ± 1.00</td>
</tr>
<tr>
<td>Peripheral resistance (dyne-cm)</td>
<td>1,243 ± 235</td>
<td>1,509 ± 502§</td>
<td>1,573 ± 429§</td>
<td>1,571 ± 375‡</td>
</tr>
<tr>
<td>Peripheral resistance index (dyne-cm/m²)</td>
<td>2,237 ± 423</td>
<td>2,912 ± 960*</td>
<td>3,036 ± 828§</td>
<td>2,812 ± 671†</td>
</tr>
</tbody>
</table>

Values are means ± sd. Significance (p) is vs normal subjects.  
*p < 0.05.  
†p < 0.02.  
‡p < 0.01.  
§p < 0.001.  
||p < 0.05 < p < 0.10.  
Abbreviations used: LV = left ventricular; IVSd = interventricular septal thickness (end diastole); LVIDd = LV internal dimension; PWTd = posterior wall thickness; RWTd = end-diastolic relative wall thickness; IVSs = interventricular septal thickness (end-systole).
Normal subjects (N = 79)  
Low-renin hypertensives (N = 20)  
Normal-renin hypertensives (N = 79)  
High-renin hypertensives (N = 19)

FIGURE 2. Measurements (mean ± standard deviation) of left ventricular hypertrophy and function are depicted for normal subjects, and patients with low-, normal-, and high-renin forms of essential hypertension. A. Left ventricular (LV) mass index is significantly increased in all three hypertensive groups. B. End-diastolic LV relative wall thickness is similarly increased in all three hypertensive groups. C. LV fractional systolic shortening is increased above normal only in the low-renin group.

Portionate septal thickening was present by NIH criteria. There was no consistent relationship between the IVST/PWT ratio by either method of measurement and BP or age.

Left Ventricular Function

In all normal- and low-renin patients, LV fractional shortening was normal (≥ 26%). Only in two high-renin patients was LV fractional shortening mildly depressed (to 23% and 24% respectively). In each hypertensive group, mean LV fractional shortening was above the mean for normal patients in this laboratory, but this was statistically significant only in the low-renin group (41% ± 8%, p < 0.01 vs normal of 35% ± 5%). The cardiac index was almost identical to normal in high-renin patients and normal-renin patients, but was mildly elevated in low-renin patients (3.77 ± 1.0 liter/min/m² vs normal of 3.15 ± 1.0 liter/min/m²; p = 0.06) despite their greater age; cardiac index was inversely related to age in all three subgroups, but only in low-renin patients was this statistically significant (r = −0.62, p < 0.005). Because of the similarity in BP and cardiac output, estimated total peripheral resistance was similarly about 20% above normal in all groups. However, when total peripheral resistance was indexed to take into account differences among groups in body size, the resistance index was slightly lower in the low-renin hypertensives (2812 ± 671 dyne-cm-sec⁻³) than in either the normal-renin patients (3036 ± 828) or high-renin patients (2912 ± 969).

Relationship of Left Ventricular Hypertrophy to Function

If LVH induced by hypertension is physiologic, that is, serves as a successful compensatory mechanism for the increased afterload, then the pumping ability of the LV should be maintained. Pumping ability might even be enhanced in patients with increased preload due to elevated central blood volumes. To assess whether LVH served to maintain pumping ability in the three renin groups, cardiac index was related to LV mass index. There was a strong positive correlation between LV mass index and cardiac index in the 19 high-renin patients (r = 0.68, p < 0.01) and a moderate positive correlation in the 79 normal-renin patients (r = 0.30, p < 0.02). In contrast, in low-renin patients...
there was a trend toward a lower cardiac index as LV mass index rose \( r = -0.43, p = 0.06 \), possibly due to these subjects older age and longer duration of known hypertension. Since the level of total peripheral resistance (TPR) is the major determinant of LV pressure afterload in hypertension, TPR was related to end-diastolic relative wall thickness as a measure of the severity of concentric LVH. There was a highly significant correlation in normal-renin patients \( r = 0.54, p < 0.001 \) and low-renin patients \( r = 0.61, p < 0.01 \) but only a modest association in high-renin patients \( r = 0.31, p < 0.1 \).

Discussion

During the past decade, the possibility has been raised that the renin-angiotensin system may be important not only as a mechanism for the pathogenesis of hypertension, but also as a determinant of hypertensive LVH\(^{14-16. 31}\) and cardiovascular disease,\(^{11-12. 35}\) above and beyond the effects of renin-mediated BP elevation. The present study indicates that, in patients with similar levels of mild to moderate hypertension, the severity of hypertensive LVH is equivalent in high-, normal- and low-renin patients, and suggests that differences among renin groups in hemodynamic profile and cardiac performance are relatively slight.

The previous studies that have implicated the renin-angiotensin system in cardiac hypertrophy have relied on indirect evidence. It was initially shown that angiotensin II increases protein synthesis in myocardial and smooth-muscle cells.\(^{31}\) The hypothesis that the renin-angiotensin system potentiated hypertensive LVH gained further support from the demonstration that control of hypertension by hydralazine\(^{14}. 16 \) or minoxidil,\(^{15}\) both of which substantially increase plasma renin levels, failed to reverse LVH, whereas similar or even lesser degrees of BP control by medications that interrupt the renin-angiotensin system caused LVH to regress.\(^{14-16. 33}\) However, these studies may also be taken to indicate a primary catecholamine effect in causing cardiac hypertrophy, in accord with experiments showing induction of LVH by catecholamines\(^{34. 35}\) and complete prevention or partial reversal of LVH by antisypathetic interventions.\(^{37. 38}\) Furthermore, the cited studies of the renin-angiotensin hypothesis of LVH in hypertension have not adequately excluded a hemodynamic mechanism for their findings, since treatment of hypertension with vasodilators alone causes a significant increase in cardiac output, substituting volume overload for pressure overload as a stimulus for LVH.

When hemodynamic factors that might account for myocardial hypertrophy are excluded, as in a recent study in which Sen et al.\(^{39}\) administered angiotensin antagonists to normotensive rats without altering BP, the findings have suggested, at most, an indirect rather than direct effect of angiotensin in myocardial hypertrophy. Two different angiotensin antagonists that increased myocardial catecholamines were found to induce myocardial hypertrophy, whereas another angiotensin antagonist that had no effect on catecholamines did not produce hypertrophy.\(^{39}\)

Our data, which show an identical degree of concentric LVH in low-, normal-, and high-renin patients with essential hypertension, provide the first clear evidence that the renin-angiotensin system does not play a major independent role in hypertensive LVH in humans. While patients in the high-renin group were substantially younger than the others and therefore might have had hypertension for a shorter time, it is not clear that these patients actually had an accelerated development of LVH. Both in experimental animals\(^{40}\) and in humans,\(^{41}\) myocardial hypertrophy has been shown to develop or regress within weeks of induced hemodynamic changes. However, LV mass has been shown to increase modestly with age in previous echocardiographic studies of hypertensive adults.\(^{18}\)

The renin subgroups did differ, however, in other aspects of cardiovascular performance. Low-renin patients had a small increase in cardiac index as compared to either controls or the normal- or high-renin hypertensive groups, in accord with the vasoconstrictor-volume model,\(^{42}. 43\) with a parallel increase in pulse pressure and in LV fractional systolic shortening. This in accord with the finding of other investigators that patients with an increased plasma volume have suppressed renin levels,\(^{44. 45}\) and that there is a correlation between plasma volume, and especially cardiopulmonary blood volume and cardiac output.\(^{46}\) The difference between our findings and the previous report by Schalekamp et al.,\(^{47}\) in which low-renin hypertensives had lower cardiac outputs than normal-renin hypertensives, may be due to their having studied a group of low-renin patients with much higher mean BP (134 mm Hg) than those in our present study (117 mm Hg). Similar to our results, Esler et al.\(^{48}\) found that cardiac output was normal in high- and normal-renin hypertensives; they did not study low-renin patients.

The patients in our present study predominantly had relatively mild hypertension, enabling them to be off antihypertensive medication for a period of at least 3 weeks prior to renin-profiling and echocardiography. Further, patients with previous clinical congestive heart failure were excluded from the study population. Therefore, the absence of LV dysfunction in the vast majority of patients is not surprising. In fact, there was a tendency for resting LV function to be slightly above that of controls in our hypertensive patients, as has been noted by Blafox et al.\(^{49}\) However, it is of note that the two patients with mildly depressed systolic LV performance, men aged 25 to 29 years, were in the high-renin group. Both patients had above average BP (185/118 and 155/110 mm Hg) with below average increases in LV mass index (103 g/m² and 105 g/m² respectively). In these two, the conjunction of moderately severe hypertension with minimal compensatory LVH would lead to abnormally high LV wall stresses during systole. Previous studies in patients with hypertension\(^{50-52}\) or aortic stenosis\(^{53-54}\) have indeed shown an inverse relationship between LV systolic function and LV wall stress. Thus, depressed LV function in these...
two patients may reflect a hypertrophic response that is insufficient, in relation to their degree of hypertension, to normalize LV wall stresses, rather than intrinsic myocardial disease.

In summary, we have found that patients with high-, normal-, and low-renin forms of essential hypertension of similar severity have almost identical degrees of LVH. Although these results do not support a major direct role of angiotensin in hypertensive LVH, the high-renin hypertensives were younger than the normal- or low-renin hypertensives and thus may have developed cardiac hypertrophy at a slightly accelerated rate. Further study of larger populations of unselected hypertensive patients will be needed to answer this question, and to determine whether the hemodynamic profile of the different renin subgroups evolve in characteristic patterns.

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R B Devereux, D D Savage, J I Drayer and J H Laragh

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