Left Ventricular Hypertrophy in Hypertension
Prevalence and Relationship to Pathophysiologic Variables

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JEFFREY S. BORER, AND JOHN H. LARAGH

SUMMARY In less than a decade since development of echocardiographic measurement of left ventricular muscle mass, studies using this technique have provided considerable information about the prevalence and pathophysiology of left ventricular hypertrophy in human hypertension. Increased left ventricular mass has been found in a significant minority of patients with systemic hypertension, with the exact prevalence dependent both on how a population is selected and on the sex, race, and possibly age composition of its members. All published studies have reported that left ventricular hypertrophy is more closely related to blood pressure recorded in the patient's natural setting during normal activity or exercise — whether measured by portable recorder or home manometer — than to blood pressure measured by the physician. In addition, studies indicate that the classic hypertensive abnormalities of concentric left ventricular hypertrophy and increased peripheral resistance are interrelated, while left ventricular hypertrophy is absent in a subgroup of patients with mild essential hypertension who exhibit high cardiac output and evidence of supernormal myocardial contractility. Conversely, the left ventricular functional response to exercise is inversely related to the degree of hypertrophy. High levels of blood viscosity, which would tend to blunt the reduction in peripheral resistance expected during sleep or exercise, have also been associated with left ventricular hypertrophy in patients with essential hypertension. Echocardiographic studies have provided evidence both for and against the hypothesis that activity of the sympathetic or renin-angiotensin systems plays a direct role in causing hypertensive cardiac hypertrophy. These findings demonstrate the useful role that echocardiographic assessment of left ventricular structure and function may play in hypertension research. (Hypertension 9 [Suppl II]: II-53-II-60, 1987)

Key Words • blood viscosity • echocardiography • hypertension • left ventricular hypertrophy • blood pressure

Left ventricular hypertrophy (LVH) plays a dual role in patients with systemic hypertension — being both a necessary adaptation to pump a normal amount of blood against the increased pressure load and a pathologic manifestation of hypertensive cardiovascular disease. For many years, knowledge of the complex status of the heart in human hypertension advanced slowly because of the lack of suitable means of measuring cardiac anatomy and function in unselected hypertensive patients or of following their natural history. The development over the last decade of accurate echocardiographic methods for detection of LVH and characterization of ventricular contractile performance has catalyzed an explosion of information about the heart in hypertension.

In this review, we summarize the available data to provide preliminary answers to the following questions: Do all patients with systemic hypertension exhibit cardiac involvement? Is hypertensive cardiac hypertrophy closely related to the level of blood pressure, or does it convey independent information about disease severity? Are cardiac findings related to cardiovascular dynamics in patients with hypertension? Do neural or endocrine factors directly influence the heart — beyond their influence on hemodynamics?

Prevalence of Left Ventricular Hypertrophy in Hypertension

Detection of LVH by echocardiogram depends not only on the established accuracy of the method in reflecting anatomic findings1-4 but also on use of correct normal limits. Although considerable progress has been made in establishing statistically reliable and reproducible normal limits,3,4 no single criterion is yet completely validated and universally accepted. Despite this, tentative conclusions may be drawn about the prevalence of LVH among patients with hypertension by examining the results of studies in which hypertensive patients and normoten-
This approach can be applied in four available clinical studies of patients with essential hypertension that include comparison groups of apparently normal subjects. In these studies the overall prevalence of LVH was determined by applying identical cutoffs for left ventricular (LV) mass indexed for body size (e.g., LV mass/body surface area > 120 g/m²) to both patients and control populations. The prevalence of LVH was 23 to 48% in hypertensive patients and 0 to 10% in normal subjects (Figure 1). Overall, 189 of 450 (42%) of hypertensive patients and 9 of 251 (3.6%) of controls exhibited LVH (chi square test = 117.1, p < 10⁻⁴). These data establish that LVH occurs in a substantial minority of patients with mild to moderately severe essential hypertension evaluated in a referral center. The finding that LVH was detected by echocardiogram in a slightly higher percentage of apparently normal subjects than had been expected statistically for upper 95% confidence limits (3.6% vs 2.5%) may reflect a slight random fluctuation of results in a moderate sample size or admixture of a few individuals with clinically undetected heart disease. A recent study indicates that the prevalence of LVH by the same echocardiographic criteria is somewhat lower — 17% and 21%, respectively — among unselected patients with uncomplicated borderline or established hypertension drawn from an employed population.

Further information about the prevalence of LVH in patients with hypertension has been derived from more recent studies of similar age, duration of hypertension, and prior treatment status. In one group, the greater degree of concentric LVH among black patients was associated with a modest elevation of peripheral resistance (Table 1), whereas white patients from the same population exhibited an increased cardiac output without a significant increase in peripheral resistance. This evidence of greater concentric LVH and a hemodynamic pattern felt to characterize more advanced hypertension in black as compared to white patients makes an interesting parallel with the known higher incidence of cardiovascular morbidity in black hypertensive persons, although it has not yet been established whether the excess morbidity among blacks is accounted for by the subset with concentric LVH.

**Effect of Sex**

The prevalence of LVH found in men and women with essential hypertension is strikingly dependent on whether one uses such sex-specific criteria or a single criterion of LV mass indexed for body surface area. When a single cutoff value for hypertension is applied to both sexes, the prevalence of LVH is consistently higher among men (26-56%) than women (18-42%) in clinical studies. When sex-specific criteria are used, however, a higher proportion of female (43-61%) than male (18-41%) hypertensive subjects exhibit LVH (Figure 2B). We have recently obtained similar results (i.e., a higher prevalence of LVH in women by sex-specific criteria and in men by unified criteria) in a study of patients with hypertension in an employed population. The reasons for this discrepancy between men and women with hypertension are not clear but might include either a selective reduction in physical activity among men with hypertension or a substantial prevalence of clinically inapparent heart disease, associated with mild LVH, among apparently normal men. Longitudinal studies of physical activity, cardiac status, and cardiovascular morbidity will be needed to determine the cause.

**Influence of Race**

No difference has been detected between normal black and white subjects in any echocardiographic measurement of LV anatomy or function. In contrast, both Dunn et al. and our group have reported that black patients with essential hypertension have a greater degree of LVH than white patients with similar levels of clinically measured blood pressure, but this was not found in a previous study. Our findings demonstrate a significant increase in relative wall thickness, an index of concentric LVH, in black patients compared to white patients of similar age, duration of hypertension, and prior treatment status identified through the same worksite clinics (Table 1). In the same group, the greater degree of concentric LVH among black patients was associated with a modest elevation of peripheral resistance (Table 1), whereas white patients from the same population exhibited an increased cardiac output without a significant increase in peripheral resistance. This evidence of greater concentric LVH and a hemodynamic pattern felt to characterize more advanced hypertension in black as compared to white patients makes an interesting parallel with the known higher incidence of cardiovascular morbidity in black hypertensive persons, although it has not yet been established whether the excess morbidity among blacks is accounted for by the subset with concentric LVH.

**Age and Hypertensive Cardiac Hypertrophy**

Both increasing age and duration of hypertension would logically be expected to be associated with a higher prevalence and greater severity of hypertensive cardiac hypertrophy, but we have not been able to demonstrate this in cross-sectional studies of large clinical or unselected populations of patients with systemic hypertension. Evidence does suggest, however, that a small proportion of elderly hypertensive patients develop severe LVH associated with symptoms of cardiac dysfunction. Topol et al. recently reported 21 elderly patients with systemic hypertension, predominantly black women, with a mean age of 73 years, who exhibited severe concentric LVH, normal LV systolic function, and severely impaired early diastolic LV filling. In an echocardiographic study of the original Framingham cohort (mean age, 70 ± 7 years), Savage et al. found that 27 of 1620 (1.7%) exhibited disproportionate thickness of the interventricular septum, associated in nearly all such subjects with a history of at least mild hypertension as well as a high prevalence of heart murmurs and cardiac symptoms. By their design, neither of these reports permits calculation of the prevalence of cardiac hypertrophy among elderly patients in whom systemic...
hypertension has been diagnosed by conventional clinical criteria although the Framingham Study has an optimal data base in which to do so.3

Hypertensive Cardiac Hypertrophy and Blood Pressure

Although early studies of highly selected patients suggested that heart weight was closely related to the level of arterial blood pressure,21 it is now clear that this is not normally the case. In several groups of patients with uncomplicated essential hypertension, physician measurements of systolic blood pressure have been only weakly related to echocardiographic LV mass, with correlation coefficients of 0.24 to 0.45.7, 13, 22-23 Even weaker correlations were observed in these studies between diastolic arterial pressure and LV mass. A similarly modest relationship \((r = 0.43)\) was observed by Abi-Samra et al. 24 in a study of 74 patients with systemic hypertension.

Ambulatory Blood Pressure

Twenty years ago Sokolow et al.25 reported that evidence of cardiovascular damage in patients with hypertension was more closely related to blood pressure measured by a portable recorder than by physicians. More recently, the same group has reported that ambulatory blood pressure measurements were better predictors than casual determinations of subsequent morbid events in patients with hypertension.26 Both these studies, however, used an ambulatory recording system that was patient-activated, thus precluding complete 24-hour recordings, and also employed indirect means of detecting LVH, such as the electrocardiogram.

Recording blood pressure through the entire 24-hour period has been made possible by development of invasive27 and noninvasive28-29 systems with acceptable accuracy. Because of their greater acceptability and safety, noninvasive systems have been most widely used and have provided important information about blood pressure variability30 with implications for patient management.31 Three studies have compared the relationships between echocardiographically determined LV mass and physician or 24-hour blood pressure measurement.7, 32-33 In each study average 24-hour systolic blood pressure was the closest correlate of LV mass (Table 2). To gain further insight into the relationship between cardiac hypertrophy and blood pressure during normal activity, we categorized ambulatory blood pressures by the setting in which they were recorded (e.g., physician’s office, occupational workplace, home, and sleep). The closest relationships were observed between LV mass index and average workplace systolic blood pressure \((r = 0.50, p<0.001)\) and between end-diastolic relative wall thickness and average workplace diastolic blood pressure \((r = 0.59, p<0.001)\). In this study LV mass was less closely related to peak blood pressure than to the average workplace blood pressure. These data suggest that the blood pressure response to regularly recurring stress may have particular importance in the pathogenesis of hypertensive cardiac hypertrophy.

### Table 1. Cardiac Anatomy and Function in Black and White Normotensive and Hypertensive Subjects

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Normotensive</th>
<th>Hypertensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blacks (n = 70)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>163.2 ± 62.6 NS</td>
<td>207.2 ± 56 NS</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.36 ± 0.08 NS</td>
<td>0.43 ± 0.11 &lt;0.05</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.63 ± 1.91 NS</td>
<td>5.80 ± 2.16 &lt;0.01</td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>(10^3) dyn cm sec^-1</td>
<td>1.43 ± 0.47 NS</td>
</tr>
<tr>
<td>Whites (n = 62)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>163.8 ± 49.2 NS</td>
<td>203.8 ± 76.6 NS</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.34 ± 0.06 NS</td>
<td>0.37 ± 0.07</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.93 ± 2.05 NS</td>
<td>7.25 ± 2.35</td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>(10^3) dyn cm sec^-1</td>
<td>1.36 ± 0.45</td>
</tr>
<tr>
<td>Hypertensive (n = 30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>207.2 ± 56 NS</td>
<td>203.8 ± 76.6 NS</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.43 ± 0.11 &lt;0.05</td>
<td>0.37 ± 0.07</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.80 ± 2.16 &lt;0.01</td>
<td>7.25 ± 2.35</td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>(10^3) dyn cm sec^-1</td>
<td>1.85 ± 0.72 &lt;0.01</td>
</tr>
<tr>
<td>Whites (n = 45)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are means ± SD. NS = not significant. Adapted from Hammond et al.13
Other Blood Pressure Measurements

Because obtaining precise 24-hour ambulatory blood pressure recordings is cumbersome and technically difficult, considerable attention has been devoted to finding other methods of obtaining a reliable estimate of patients' average blood pressure. Two recent studies have yielded promising results. In the first, we demonstrated that home blood pressure recordings by trained patients not only provided a better estimate of average 24-hour blood pressure than did physician measurements but also were more closely correlated to indices of LVH. The second study, by Ren et al., reported a substantially closer relationship of LV mass to maximum systolic arterial pressure during treadmill exercise testing than to blood pressure at rest prior to exercise (see Table 2).

### Table 2. Correlation Between Blood Pressure Measurements and Left Ventricular Mass

<table>
<thead>
<tr>
<th>Physician</th>
<th>Portable recorder</th>
<th>Self</th>
<th></th>
<th>Physican</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference</td>
<td>n</td>
<td>Systolic</td>
<td>Diastolic</td>
<td>Systolic</td>
</tr>
<tr>
<td>Devereux et al.</td>
<td>100</td>
<td>0.24±0.20</td>
<td>0.38±0.31</td>
<td>0.40±0.30</td>
</tr>
<tr>
<td>Kleiner et al.</td>
<td>32</td>
<td>0.22</td>
<td>0.26</td>
<td>0.24</td>
</tr>
<tr>
<td>Rowlands et al.</td>
<td>32</td>
<td>0.45±0.46</td>
<td>0.60±0.35</td>
<td>0.60±0.35</td>
</tr>
<tr>
<td>Drayer et al.</td>
<td>12</td>
<td>0.55</td>
<td>0.56</td>
<td>0.56</td>
</tr>
<tr>
<td>Ren et al.</td>
<td>67</td>
<td>0.16</td>
<td>0.02</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Data are correlation coefficients between left ventricular mass and the specified type of blood pressure measurement except for the study of Kleiner et al., in which end-diastolic relative wall thickness was the index of left ventricular hypertrophy. * = p < 0.001; ** = p < 0.05; *** = p < 0.01. Other correlations are not significant.

### Hypertensive Cardiac Hypertrophy and Cardiovascular Dynamics

Because cardiac hypertrophy is felt to be an important adaptive response to hypertension, consideration must be given to the degree to which LVH is matched to the hemodynamic load and is successful in maintaining cardiac performance in hypertension. In the first studies in this regard, performed by Follow in experimental animals, the severity of cardiac hypertrophy tended to parallel the severity of peripheral vascular resistance. In a subsequent study by Shihvatsayba and coworkers, echocardiographically determined LV mass correlated significantly (r = 0.60, p < 0.001) with vascular resistance in the calf, measured by plethysmography during maximal vasodilation.

Extending this line of investigation to 100 patients with essential hypertension, we examined the relationships between systemic hemodynamics and the pattern of LV anatomy. A significant positive correlation (r = 0.52, p < 0.001) was observed between total peripheral resistance and end-diastolic LV relative wall thickness (Figure 3A). Furthermore, cardiac index was inversely related to relative wall thickness (r = -0.47, p < 0.001) (Figure 3B). Taken together, these experimental and clinical studies suggest that the cardiac pattern of concentric LVH and the hemodynamic pattern of elevated peripheral resistance with low cardiac output are pathophysiologically interrelated.

### Left Ventricular Performance

To investigate further the relationships between cardiac structure and function, we have performed an additional series of studies. In order to exclude the possibility that excessive or inadequate degrees of LVH in relation to blood pressure load accounted for differences in LV function, we measured myocardial afterload by calculating end-systolic LV wall stress with a catheterization-validated formula. As predicted from basic principles of cardiac mechanics, a close inverse relationship existed in 87 normotensive subjects between end-systolic stress and LV fractional shortening, an echocardiographic index of systolic ventricular performance. A significant inverse relationship between these variables was also observed in 81 unmedicated patients with essential hypertension (r = -0.78, p < 0.001). When the data points from the hypertensive patients were superimposed on 95% confidence limits derived from the normal subjects (Figure 4), a significant proportion of the hypertensive patients exhibited high fractional shortening in relationship to wall stress (19 of 81 or 23%; p < 0.001 vs 1 of 87 normotensive subjects).

Subdivision of hypertensive patients into groups with normal and increased LV performance based on this analysis of cardiac mechanics revealed striking differences in both systemic hemodynamics and LVH (Table 3). Of note, the patients with increased ventricular performance exhibited substantially increased cardiac output, lower peripheral resistance, and an absence of LVH compared to the hypertensive patients with normal ventricular performance. In a more recent study from our laboratory, hypertensive patients who had high fractional shortening in relation to end-systolic stress on baseline measurements exhibited a significantly higher (p < 0.005) slope of the end-systolic force-length line during nitroglycerin-induced reduction in hemodynamic load than hypertensive patients who fell into the normal range of fractional shortening in relation to end-systolic stress. Taken together, these studies suggest that in the majority of mildly hypertensive patients the heart plays a secondary role, undergoing adaptive hypertrophy in proportion to the elevation of blood pressure. In a significant minority of such patients, however, increased myocardial contractility may have pathogenetic importance by allowing the heart to pump an increased cardiac output without need for any hypertrophy.

Since an important capacity of the normal heart is the ability to sustain a strikingly increased hemodynamic load during normal activity, assessment of the heart in hypertension is not complete without evaluation of the cardiac responses to exercise. This has been directly evaluated by means of radionuclide cineangiography at rest and during exercise in several recent studies. Among hypertensive patients with no evidence of coronary artery disease, the prevalence of abnormal LV ejection fraction responses to exercise has ranged from 9 of 37 patients (24%) to 15 of 20 patients (75%). Our studies indicate that a small proportion — approximately 10% — of the hypertensive patients who exhibit abnormal LV functional reserve can be identified by evidence of impaired myocardial contractility (low echocardiographic fractional shortening in relation to end-systolic stress) at rest; whereas in the remainder, LV dysfunction is only revealed by imposition of exercise stress. A preliminary study from our laboratory suggests that LV dysfunction during exercise and LVH are closely linked, with an inverse linear relationship (r = -0.50, p < 0.01) between echo-
cardiographically determined LV muscle mass and the change in LV ejection fraction from rest to exercise observed among hypertensive patients with LVH.

The ability of the left ventricle to sustain a normal or increased workload is also dependent on its diastolic performance characteristics. Abnormalities of LV diastolic time intervals and filling rates have been well documented in patients with systemic hypertension and may be a more sensitive marker of hypertensive cardiac involvement than echocardiographic LVH. These diastolic abnormalities appear to be manifestations of so-called pathologic LVH since subjects with a similar degree of exercise-induced physiologic hypertrophy exhibited normal LV diastolic properties in one study.

Blood Viscosity
Several lines of evidence suggest that altered blood rheology may be importantly related to cardiac findings in hypertension. Hematocrit and blood viscosity have been found to be higher in hypertensive than normotensive individuals. Recently we reported a modest direct relationship between blood pressure and whole blood viscosity in both hypertensive and normoten-tive subjects. We found that increased whole blood viscosity in unselected patients with mild essential hypertension accounted for the entire increase in peripheral resistance in them as compared with normotensive subjects drawn from the same employed population. Finally, high rates of cardiovascular morbidity have been reported in patients with hypertensive cardiac involvement.

Therefore, we recently undertook a study to examine the relationships among arterial blood pressure, whole blood viscosity, and LVH in 24 patients with essential hypertension and 13 age- and sex-matched control subjects. A significant correlation was observed between mean arterial blood pressure and whole blood viscosity, and LVH in 24 patients with essential hypertension and 13 age- and sex-matched control subjects. A significant correlation was observed between mean arterial blood pressure and whole blood viscosity in this study population, and in the normotensive subjects.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patients with normal LV performance</th>
<th>Patients with increased LV performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>53 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>111 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td>3.39 ± 1.00</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Total peripheral resistance (dyn·cm⁻²·sec⁻¹)</td>
<td>1,582 ± 584</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.42 ± 0.10</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

LV = left ventricular; NS = not significant.
Adapted from Lutas et al.

FIGURE 3. A. Left ventricular (LV) relative wall thickness at end diastole (vertical axis) is directly related to total peripheral resistance (horizontal axis) in 100 unmedicated patients. B. Cardiac index (vertical axis) is inversely related to LV relative wall thickness at end diastole (horizontal axis). (Reprinted from Devereux et al. with permission.)

FIGURE 4. Data points for left ventricular fractional shortening and end-systolic stress in 81 untreated patients with essential hypertension are superimposed on the solid regression line and dashed 95% confidence interval of this relationship in 87 normal subjects. A close inverse relation is observed (r = 0.47, p < 0.001), and 19 of 81 hypertensive patients exhibit increased ventricular function in relation to wall stress (compared to 1 of 87 normotensive subjects, p < 0.0001). (Reprinted from Lutas et al. with permission of the American Heart Association.)
5A). A close correlation was observed (Figure 5B) between whole blood viscosity at the high shear rate of 104 sec$^{-1}$ and LV mass in the hypertensive patients ($r = 0.80$, $p < 0.001$), which was significantly closer than the correlation between mean blood pressure and LV mass ($p < 0.02$). Furthermore, as shown in Figure 5B, the relationship between viscosity and LV mass in normotensive subjects closely resembled that in the hypertensive patients with normal whole blood viscosity.

**Neurohumoral Factors and Hypertensive Cardiac Hypertrophy**

Although hemodynamic factors have received the most attention in studies of the pathogenesis of hypertensive cardiac hypertrophy, considerable scatter clearly exists in the relationships between the best available measures of blood pressure and LV muscle mass. The search for nonhemodynamic causes of LVH in hypertensive patients has focused mostly on neurohumoral factors, principally the sympathetic and renin–angiotensin systems.

Evidence in favor of the so-called catecholamine hypothesis of LVH was originally derived from studies using sympathetic agonists$^{59}$ and antagonists$^{60}$ in intact animals. More recently, Simpson and co-workers$^{57,58}$ have provided evidence that induction of protein synthesis by norepinephrine in tissue-cultured cardiac myocytes is an $\alpha_1$-receptor–mediated phenomenon. Limited studies in patients with essential hypertension have suggested a positive relationship between plasma norepinephrine concentration and LV mass$^{59}$ and a greater reduction in blood pressure, especially in patients with low-renin forms of essential hypertension, than age-, sex- and blood pressure-matched patients with essential hypertension.$^{70}$

**Acknowledgment**

We thank Miss Virginia Burns for her assistance in preparation of this manuscript.

**References**

6. Devereux RB, Savage DD, Drayer JIM, Laragh JH. Left ventricular hypertrophy and function in high, normal, and low-renin forms of essential hypertension. Hypertension 1982;4:524–531
8. Reichek N, Devereux RB. Reliable estimation of peak left ventricular
33. Devereux RB, Savage DD, Sachs I, Laragh JH. Relation of hemodynamic load to left ventricular hypertrophy and performance in hypertensive patients. Hypertension 1986;3:574–578
42. Gibson DG, Trailf TA, Hall RJ, Devogu D. Echocardiographic features of secondary left ventricular hypertrophy. Br Heart J 1979;41:54–59
55. Karliner J, Simpson P, Moazed D, Park B. Norepinephrine and T3 induced myocardial cell hypertrophy is associated with down regulation of alpha-1 adrenergic receptors [Abstract]. Circulation 1984;70(suppl 2):110
57. Devereux RB, Savage DD, Sechla I, Laragh JH. Effect of blood pressure control on left ventricular hypertrophy and function in hypertension [Abstract]. Circulation 1980;62(suppl 3):36
64. Robertson AL Jr, Khairallah PA. Angiotensin II: rapid localization in nuclei of smooth and cardiac muscle. Science 1971;172:1138-1139
70. Vensel LA, Devereux RB, Pickering TG, Herrold E McM, Borer JS, Laragh JH. Cardiac structure and function in renovascular hypertension produced by unilateral and bilateral renal artery stenosis. Am J Cardiol 1986;58:575-582
Left ventricular hypertrophy in hypertension. Prevalence and relationship to pathophysiologic variables.
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Hypertension. 1987;9:II53
doi: 10.1161/01.HYP.9.2_Pt_2.II53

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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