Echocardiographic Left Ventricular Hypertrophy as Related to Arterial Pressure and Plasma Norepinephrine Concentration in Arterial Hypertension Reversal by Atenolol Treatment

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SUMMARY We tried to assess relationships between echocardiographic left ventricular hypertrophy (LVH), arterial pressure levels, and plasma norepinephrine concentration (NE) in 20 previously untreated stable hypertensive patients with LVH, and in 11 healthy normotensive control subjects. Interventricular septal (IVS) thickness, posterior wall (PW) thickness, and left ventricular mass index (LVMI) were related to arterial pressure levels and to NE by univariate and multivariate regression analyses. In addition, after 18 months of monotherapy with atenolol (carried out in nine of 20 patients), the relationship between echocardiographic changes and degree of pressure reduction was tested. Before treatment, PW thickness weakly correlated with systolic \( r = 0.55; p < 0.01 \) and mean \( r = 0.50; p < 0.05 \) arterial pressure. IVS thickness weakly correlated with NE \( r = 0.53; p < 0.05 \). On this relatively small sample, multivariate regression analysis showed an association of both IVS thickness \( R = 0.57; p < 0.05 \) and PW thickness \( R = 0.58; p < 0.05 \) with mean arterial pressure (MAP) and NE. After atenolol, there was a reduction in IVS thickness (1.15 to 1.02 cm; \( p < 0.01 \)), PW thickness (1.08 to 0.99 cm; \( p < 0.01 \)), and LVMI (136.3 to 113.8 g/m^2; \( p < 0.01 \)), besides a significant reduction in blood pressure and heart rate. The degree of pressure reduction induced by treatment did not correlate the change in IVS or PW thickness. In contrast, the change in diastolic and mean arterial pressure positively correlated the change in LVMI \( r = 0.72 \) and \( r = 0.75 \), respectively; both \( p < 0.05 \). These findings suggest that both arterial pressure levels and NE could influence the degree of LVH in stable arterial hypertension with LVH, and that IVS and PW thickness seem more sensitive indicators of LVH, than LVMI, in the research of subtle relationships with hypothetical pathogenetic factors. In contrast, LVMI seems more suitable than the thickness of either wall in the overall assessment of LVH regression during antihypertensive drug treatment when a possible relationship with pressure reduction is being investigated. (Hypertension 5: 837-843, 1983)

KEY WORDS • hypertension • cardiac hypertrophy • echocardiography • catecholamines • atenolol

In patients with arterial hypertension, left ventricular hypertrophy (LVH) can be diagnosed by echocardiography at an early stage of development, even in the absence of electrocardiographic or radiological evidence.1

As a result, echocardiography can detect LVH in a larger percentage of hypertensive patients than electrocardiographic or radiological techniques.2 Despite some limitations,3-5 echocardiography also offers the opportunity of repeatedly6 and accurately7-7 quantifying the thickness of the ventricular walls as well as the left ventricular mass. Thus, it appears a valuable technique in the study of possible mechanisms involved in the pathogenesis of LVH as well as in its reversal following antihypertensive treatments.

Studies in animals8-15 as well as in hypertensive patients16-18 suggest that other factors besides pressure overload could be involved in the mechanisms responsible for LVH development. In this concern, we sought to evaluate the possible relationships between some echocardiographic signs of LVH interventricular septal (IVS) thickness, posterior wall (PW) thickness, left ventricular mass index (LVMI), and some hypothetical pathogenetic factors (arterial pressure levels, catecholamines, plasma renin activity), in newly diagnosed stable hypertensive patients with echocardiographic evidence of LVH. We also studied some of the patients again after 18 months of monotherapy with atenolol, to test the relationships between change in arterial pressure levels and change in LVH.
Materials and Methods

Trial Population

Twenty hypertensive patients and 11 healthy normotensive subjects (table 1) gave written informed consent to be included in the study. In all hypertensive patients, sphygmomanometric blood pressure levels were constantly found above 150/95 mm Hg during a 3-month period of ambulatory observation, with 2-weekly examinations always at the same time of the day. At the end of the 3rd month of observation, patients were hospitalized for at least 1 week for diagnostic evaluation of the hypertensive state. The normotensive group was composed of subjects hospitalized for clinical evaluation who were eventually found to be healthy.

Essential arterial hypertension was diagnosed according to the criteria established by the World Health Organization (WHO). All patients were in WHO Stage II, with no target organ damage apart from LVH evidenced by echocardiography. In particular, cardiothoracic ratio was normal and cardiac transverse diameter did not exceed 10% above the predicted value. In two patients, electrocardiography revealed signs of "probable LVH," according to the point-score system of Rohmilt and Estes. All patients were at their first diagnosis of hypertension and none of them had received therapy.

Blood pressure was measured by a conventional sphygmomanometer. The appearance of brachial artery sounds and the complete disappearance of the sounds (5th Korotkoff phase) was used for systolic and diastolic pressure recordings, respectively. Measurements were always performed by the same observer, the mean value from three consecutive readings taken at 1-minute interval was recorded. The MAP was derived by the formula: diastolic pressure + 1/3 (systolic pressure – diastolic pressure).

Echocardiography

Echocardiography was performed using a Kontron Irex II System Echograph, with a 2.25 MHz transducer and photorecording at paper speed of 100 mm/sec. All procedures were performed in the same room with the same equipment, on patients who had fasted overnight and were resting supine for at least 1 hour. Smokers were asked to avoid cigarettes for 24 hours before the study. All echocardiograms were carried out and read by the same investigator, with the patient in supine or partial left lateral position, and after placing the transducer on the fourth or fifth intercostal space near the left sternal edge. Echograms were taken at or just below the tips of the mitral valve leaflets, in a position showing continuous echoes of both septum and posterior wall.

End-diastolic left ventricular internal dimensions, IVS thickness, and PW thickness were identified at the peak of the R wave on the simultaneous ECG. Echocardiographic left ventricular mass was calculated according to the Penn Cube formula, which includes the thickness of the endocardial echoes from both IVS and PW in the measurement of ventricular internal dimension, and thus excludes them from measurement of IVS and PW thickness. Left ventricular mass was divided by body surface area to obtain LVMI. Values reported herein are the means of six consecutive echocardiographic readings, three in mild inspiration and three in expiration at lung functional residual capacity.

Table 1. Demographic Characteristics, Blood Pressure, Heart Rate, Echocardiographic and Humoral Findings in Normotensive Subjects at Initial Examination (B) and after 12 to 18 Months (A), and in Hypertensive Patients Basally (B) and after Treatment (Atenolol)

<table>
<thead>
<tr>
<th></th>
<th>Normotensive group (n = 11)</th>
<th>Hypertensive group (n = 20)</th>
<th>Patients undergoing follow up (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>A</td>
<td>B (n = 20)</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>29.2 ± 5.4</td>
<td>30.5 ± 5.2</td>
<td>36.1 ± 7.6</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>8/3</td>
<td>8/3</td>
<td>164</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.74 ± 0.05</td>
<td>1.76 ± 0.08</td>
<td>1.82 ± 0.06</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>121.6 ± 11.2</td>
<td>123.5 ± 11.3</td>
<td>169.0 ± 11.6</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>72.0 ± 8.6</td>
<td>74.0 ± 9.6</td>
<td>107.9 ± 5.6</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>88.7 ± 9.0</td>
<td>91.7 ± 8.5</td>
<td>128.2 ± 4.6</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>72.6 ± 8.7</td>
<td>73.9 ± 8.4</td>
<td>82.4 ± 6.6</td>
</tr>
<tr>
<td>IVS thickness (cm)</td>
<td>0.89 ± 0.09</td>
<td>0.90 ± 0.09</td>
<td>1.15 ± 0.06</td>
</tr>
<tr>
<td>PW thickness (cm)</td>
<td>0.98 ± 0.06</td>
<td>0.98 ± 0.08</td>
<td>1.10 ± 0.06</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>77.0 ± 7.8</td>
<td>77.8 ± 8.6</td>
<td>128.3 ± 10.7</td>
</tr>
<tr>
<td>NE (ng/liter)</td>
<td>141.0 ± 50.3</td>
<td>137.6 ± 41.9</td>
<td>216.0 ± 63.6</td>
</tr>
<tr>
<td>E (ng/liter)</td>
<td>22.1 ± 5.0</td>
<td>20.9 ± 6.6</td>
<td>53.6 ± 19.9</td>
</tr>
<tr>
<td>PRA (ng/ml/hr/angio)</td>
<td>2.1 ± 0.9</td>
<td>2.2 ± 0.8</td>
<td>1.68 ± 0.80</td>
</tr>
</tbody>
</table>

Results expressed as means ± sd. BSA = body surface area; SAP = systolic arterial pressure; DAP = diastolic arterial pressure; MAP = mean arterial pressure; HR = heart rate; IVS = interventricular septum; PW = posterior wall; LVMI = left ventricular mass index; NE = norepinephrine; E = epinephrine; PRA = plasma renin activity.

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Diagnosis of Left Ventricular Hypertrophy

LVH was defined as a left ventricular mass (Penn Cube formula) greater than 215 g. This limit was chosen since, in the validation study by Reichek and Devreux,13 of 14 patients with an echocardiographic left ventricular mass (Penn Cube formula) greater than 215 g also had a postmortem left ventricular weight greater than 215 g, the upper limit of normal left ventricular weight in the same study.2

Plasma Renin Activity and Catecholamines

Venous blood for plasma renin activity (PRA), noradrenaline (NE), and epinephrine (E) determinations was withdrawn during hospitalization when equilibrium state was attained by urinary sodium excretion equaling sodium intake. After subjects rested for 60 minutes supine with an indwelling butterfly needle inserted in an arm vein, samples for PRA22 and catecholamine23 plasma levels determinations were collected.

Follow-Up Period

Normotensive subjects were studied again at 12 to 18 months after the initial study. During this time interval, all subjects had followed a free diet and had not received any drug treatment apart from occasional antipyretics or analgesics. Nine consecutive hypertensive patients (of the original 20) were dismissed from the hospital on atenolol (Tenormin) monotherapy and not received any drug treatment apart from occasional antipyretics or analgesics. Nine consecutive hypertensive patients (of the original 20) were dismissed from the hospital on atenolol (Tenormin) monotherapy and not received any drug treatment apart from occasional antipyretics or analgesics. Nine consecutive hypertensive patients (of the original 20) were dismissed from the hospital on atenolol (Tenormin) monotherapy and not received any drug treatment apart from occasional antipyretics or analgesics. Nine consecutive hypertensive patients (of the original 20) were dismissed from the hospital on atenolol (Tenormin) monotherapy and not received any drug treatment apart from occasional antipyretics or analgesics.

Statistical Analysis

A Hewlett-Packard 41 CV calculator, programmed by the H.P. 00041-15009 application pack as software, was used for statistical analysis. Differences between normotensives and hypertensives on baseline values were tested by one-way analysis of covariance (age as covariate). Comparisons between two quantitative variables were carried out by means of the standard least-square linear regression analysis.

The multiple linear regression of one dependent variable (IVS thickness, PW thickness, LVMI) on two independent variables (basal values of MAP and NE) was analyzed by multiple regression analysis, followed by analysis of variance to test the significance of the calculated regression. Calculation of the standardized partial regression coefficients (the product of the partial regression coefficient of either independent variable and the square root of the ratio between the observed mean sum square of the corresponding independent variable and the sum square of the dependent variable58), allowed a separate assessment of the influence of either independent variable on the dependent one.

Student's t test for paired samples was used to compare pre- with post-follow-up values. A p value less than 0.05 was assumed to be significant.

Results

Baseline Evaluation

Compared to the overall group of hypertensive patients, as well as to the group of patients undergoing follow-up, normotensive subjects showed lower values of systolic arterial pressure (SAP), diastolic arterial pressure (DAP), MAP, heart rate (HR), IVS thickness, PW thickness, LVMI, NE, and E (all p < 0.01); the PRA was similar in both groups (tables 1 and 2). Age was slightly younger in the normotensives compared with the overall group of hypertensives (p < 0.05, analysis of variance), as well as with the patients undergoing follow-up (p < 0.05). Body surface area (BSA) was slightly smaller in the normotensives in respect to the overall hypertensive population (p < 0.05), as well as to the patients undergoing follow-up (p < 0.05).

In the overall hypertensive population, PW thickness positively correlated with SAP (r = 0.37; p < 0.01) and MAP (r = 0.50; p < 0.05), but not with

### Table 2. Coefficients of Linear Correlation between Echocardiographic and Pressure as well as Humoral Data before Treatment in the Hypertensive Group (n = 20)

<table>
<thead>
<tr>
<th>Coefficients of correlation with</th>
<th>Interventricular septal thickness</th>
<th>Posterior wall thickness</th>
<th>Left ventricular mass index</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP</td>
<td>0.21</td>
<td>0.55</td>
<td>0.36</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>DAP</td>
<td>0.17</td>
<td>0.21</td>
<td>0.16</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>MAP</td>
<td>0.31</td>
<td>0.50</td>
<td>0.36</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>NE</td>
<td>0.53</td>
<td>0.37</td>
<td>0.24</td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>E</td>
<td>0.17</td>
<td>0.24</td>
<td>0.25</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>PRA</td>
<td>0.42</td>
<td>0.11</td>
<td>0.31</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

See table 1 for abbreviations.
other parameters. In contrast, IVS thickness positively correlated with NE \( (r = 0.53; \ p < 0.05) \), and its correlation with PRA bordered on statistical significance \( (r = 0.42; \ r_{0.05} = 0.43) \). In this relatively small group of patients \( (n = 20) \), the multiple linear regression of IVS thickness or PW thickness on MAP and NE was significant for both IVS and PW thickness \( (F \text{ values of } 4.19 \ (p < 0.05) \text{ for IVS, and } 4.22 \ (p < 0.05) \text{ for PW thickness}) \). Multiple correlation coefficients\(^{23} \) for IVS and PW thickness were 0.57 and 0.58, respectively. IVS thickness appeared as mainly related to NE \( (\text{standardized partial regression coefficients of } 0.50 \text{ for NE and } 0.38 \text{ for MAP}) \), whereas PW thickness was mainly related to MAP \( (\text{standardized partial regression coefficients of } 0.45 \text{ for MAP and } 0.32 \text{ for NE}) \).

There was no significant correlation between LVMI and each of the other parameters. Also, the multiple linear regression of LVMI on MAP and NE was not significant.

Changes During Follow-Up

Changes in Interventricular Septal Thickness

In the normotensive subjects, IVS thickness did not change during the follow-up period. In the hypertensive patients \( (n = 9) \), the mean IVS thickness was 1.15 cm before treatment and fell to 1.02 cm after treatment \( (p < 0.01) \). This reduction correlated with pretreatment values of DAP \( (r = 0.66; \ p < 0.05) \), and MAP \( (r = 0.68; \ p < 0.05) \), but not with SAP \( (r = 0.58) \), NE \( (r = 0.51) \), or PRA \( (r = 0.08) \) (tables 1 and 3). Reduction in IVS thickness induced by treatment did not show any relationship with the concomitant reduction in SAP \( (r = 0.33) \), DAP \( (r = 0.49) \), or MAP \( (r = 0.27) \). Pretreatment IVS thickness correlated with the degree of reduction following treatment \( (r = 0.79; \ p < 0.01) \) (fig. 1).

Changes in Posterior Wall Thickness

In the normotensive subjects, PW thickness did not change during the follow-up. In the hypertensive patients \( (n = 9) \), the mean PW thickness was 1.08 before treatment and 0.99 after treatment \( (p < 0.01) \) (fig. 2). This reduction correlated with pretreatment values of DAP \( (r = 0.73; \ p < 0.05) \), and MAP \( (r = 0.82; \ p < 0.01) \), but not with SAP \( (r = 0.53) \), NE \( (r = 0.47) \), E \( (r = 0.54) \), and PRA \( (r = 0.11) \). Reduction in PW thickness induced by treatment did not show any relationship with the concomitant reduction in SAP \( (r = 0.54) \), DAP \( (r = 0.54) \), or MAP \( (r = 0.47) \). Pretreatment PW thickness correlated the degree of its reduction following treatment \( (r = 0.72; \ p < 0.05) \).

\[
\begin{array}{|c|c|c|}
\hline
\text{Change in} & \text{Coefficient of correlation with change in} & \\
& \text{Systolic arterial pressure} & \text{Diastolic arterial pressure} & \text{Mean arterial pressure} \\
\hline
\text{IVS thickness} & 0.33 & 0.49 & 0.27 \\
& \text{NS} & \text{NS} & \text{NS} \\
\hline
\text{PW thickness} & 0.54 & 0.54 & 0.47 \\
& \text{NS} & \text{NS} & \text{NS} \\
\hline
\text{LVMI} & 0.17 & 0.72 & 0.75 \\
& \text{NS} & \text{<0.05} & \text{<0.05} \\
\hline
\end{array}
\]

See table 1 for abbreviations.

![Figure 1](http://hyper.ahajournals.org/)

**Figure 1.** Interventricular septal (IVS) thickness before \((B)\) and after \((A)\) 18 months of atenolol treatment in hypertensive patients undergoing follow-up \((n = 9)\). Values expressed as means ± SEM.

![Figure 2](http://hyper.ahajournals.org/)

**Figure 2.** Posterior wall (PW) thickness before \((B)\) and after \((A)\) 18 months of atenolol treatment in hypertensive patients undergoing follow-up \((n = 9)\). Values expressed as means ± SEM.
Changes in Left Ventricular Mass Index

LVMI did not show any significant change during the follow-up in the normotensive subjects. In the hypertensive patients (n = 9), LVMI was 136.3 g/m² before treatment, and fell to 113.8 g/m² during treatment (p < 0.01). This reduction correlated with pretreatment MAP (r = 0.69; p < 0.05), but not with SAP (r = 0.42), DAP (r = 0.62), NE (r = 0.20), and PRA (r = 0.29). Reduction in LVMI following treatment correlated with the concomitant change in DAP (r = 0.72; p < 0.05), and MAP (r = 0.75; p < 0.05), but not with the change in SAP (r = 0.17). No correlation was found between pretreatment LVMI and its change following treatment (r = 0.46).

Changes in Blood Pressure, Heart Rate, and Humoral Parameters

All data are reported in table 1. Normotensive subjects did not show any significant change during follow-up. In contrast, atenolol treatment induced a reduction in SAP (p < 0.01), DAP (p < 0.01), and HR (p < 0.01) in the treated patients (n = 9). NE and E did not change significantly during the follow-up in both normotensive and hypertensive subjects. PRA decreased significantly in the hypertensives following atenolol treatment (p < 0.01), while it did not change in the normotensives.

Unwanted Effects

Two of nine patients complained of mild asthenia during the whole treatment period with atenolol. It was not strong enough to interfere with the normal daily activities, and thus treatment was not discontinued. None of the other patients reported unwanted effects. None of the subjects undergoing follow-up was withdrawn from the study.

Discussion

In this study we sought to evaluate the possible relationships among arterial pressure levels, catecholamines, and plasma renin activity, and the degree of LVH in patients with stable arterial hypertension and LVH. As we used echocardiography to quantify LVH, results of this study are to be considered in the light of all limits of the echocardiographic techniques for LVH assessment. In particular, calculation of left ventricular mass from one-dimensional M-mode echocardiographic data, although supported by strict correlations with corresponding anatomic values, provides results that are difficult to evaluate as accurate actual values. Extrapolating from one-dimensional to three-dimensional data may expose a bias. However, none of the subjects examined in this study was affected by valvular disease, myocardial infarction, impairments of left ventricular wall motion, heart enlargement, or other conditions likely to impair the accuracy of the echocardiographic measurements.

We obtained a positive relationship between left ventricular PW thickness and resting arterial pressure levels (mainly, SAP and MAP), and between IVS thickness and plasma NE concentration. Neither PW thickness showed any relationship with NE, E, or PRA, nor IVS thickness with arterial pressure levels. Results of multivariate regression analysis would indicate a concomitant relationship of MAP and NE to the degree of thickness of both IVS and PW, but this possibility must be considered with caution because of the small sample size, probably less than that needed for two-variable multivariate analysis. Moreover, none of the correlations of LVMI with other pressure or humoral parameters was strong enough to attain statistical significance.

Several echocardiographic studies from independent laboratories are in agreement in indicating a weak but significant positive correlation between arterial pressure levels and the degree of LVH. The rise in peripheral vascular resistance could play an even more important role than the rise in arterial pressure levels in determining the degree of LVH. Moreover, the average pressure value resulting from several measurements over the 24 hours seems to show a closer positive relationship with the degree of LVH in respect to a casual pressure reading.

As opposed to the well-established role of blood pressure, the role of some humoral factors, such as of the renin-angiotensin system or the catecholamines, in the pathogenesis of LVH is still controversial. The possibility that humoral factors may participate in the mechanisms determining LVH in spontaneous hypertensive rats appears supported by strong evidence. As far as the sympathetic nervous system is concerned, a number of experimental studies indicate that catecholamines can induce cardiac hypertrophy. Such an effect seems to be mediated by a catecholamine stimulation of myocardial β-adrenergic receptors. It is uncertain whether cardiac hypertrophy may develop even in the absence of hemodynamic stimuli such as periods of increased cardiac output related to β-adrenergic stimulation. In the last few years, several studies have indicated that hypertensive subjects falling into the borderline category of the WHO classification tend to have an increased IVS thickness compared to normotensive subjects. Such an increase would not be related to the levels of arterial pressure, but rather to those of PRA, or plasma NE, the latter taken as a crude marker of sympathetic activity. On the contrary, PW thickness would still be normal in borderline hypertensive patients, and increased in patients with stable hypertension. In one study carried out in borderline hypertensive patients apparently older in respect to the patients considered in the previous studies, no differences were found between hypertensives and normotensives in terms of IVS thickness. According to results of other studies, neither the renin-angiotensin system nor the sympathetic nervous system would play a major direct role in determining the degree of LVH in unselected patients with arterial hypertension.
These results, although conflicting, would suggest that borderline hypertensive patients should be separated from the stable hypertensives in the study of factors related to the degree of LVH. Moreover, the interventricular septum and the left ventricular posterior wall should be separately considered, since the possibility that IVS hypertrophy begins when PW thickness is still normal would imply that different pathogenetic mechanisms could be involved in their development.

Atenolol treatment induced a significant reduction in left ventricular wall thickness and mass index, thus supporting results of previous reports on LVH regression during treatment with this β-blocking agent. There was no significant correlation between the reduction in thickness of either LV wall and the magnitude of arterial pressure reduction induced by treatment. By contrast, the reduction in LVMI significantly correlated with the magnitude of pressure reduction.

These findings are in line with those obtained in a recent study, in which a significant correlation was observed between SAP, DAP, and MAP reduction following various antihypertensive treatments, and concomitant reduction in LVMI derived by the same formula as in this study. However, discordance between LVMI and LV wall thickness in their relationship with MAP and NE before treatment, as well as the discordance between their change and the pressure change during treatment, was unexpected and raises an interesting point. If it is assumed that the combination of factors determining interventricular septal hypertrophy is not the same as that which influences the hypertrophy of the posterior wall, a discrepancy may be expected when the same factors (i.e., MAP and NE in the present study) are related to the thickness of either wall or to LVMI, which is derived including both of the walls.

Thus, LVMI seems less sensitive than IVS thickness or PW thickness in the study of subtle relationships with possible causal factors. On the contrary, if an overall assessment of LVH is needed and a relationship between changes in LVH and degree of pressure reduction is looked for, LVMI appears to be the most reliable parameter. With larger populations than in our present study, it may be possible to obtain true correlations between LVMI and factors possibly important in the pathogenesis of LVH.

In conclusion, our findings support a possible role of catecholamines as additional stimulus to left ventricular hypertrophy in patients with newly diagnosed stable arterial hypertension. Further studies on larger populations are needed to confirm these data and to clarify whether factors stimulating LVH may differently affect the interventricular septum and the left ventricular posterior wall.

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