Left Ventricular Concentric Geometry During Treatment Adversely Affects Cardiovascular Prognosis in Hypertensive Patients

Maria Lorenza Muiesan, Massimo Salvetti, Cristina Monteduro, Bianca Bonzi, Anna Paini, Sara Viola, Paolo Poisa, Damiano Rizzoni, Maurizio Castellano, Enrico Agabiti-Rosei

Abstract—Left ventricular (LV) mass and geometry predict risk for cardiovascular events in hypertension. Regression of LV hypertrophy (LVH) may imply an important prognostic significance. The relation between changes in LV geometry during antihypertensive treatment and subsequent prognosis has not yet been determined. A total of 436 prospectively identified uncomplicated hypertensive subjects with a baseline and follow-up echocardiogram (last examination 72±38 months apart) were followed for an additional 42±16 months. Their family doctor gave antihypertensive treatment. After the last follow-up echocardiogram, a first cardiovascular event occurred in 71 patients. Persistence of LVH from baseline to follow-up was confirmed as an independent predictor of cardiovascular events. Cardiovascular morbidity and mortality were significantly greater in patients with concentric (relative wall thickness ≥0.44) than in those with eccentric geometry (relative wall thickness <0.44) in patients presenting with LVH (P=0.002) and in those without LVH (P=0.002) at the follow-up echocardiogram. The incidence of cardiovascular events progressively increased from the first to the third tertile of LV mass index at follow-up (partition values 91 and 117 g/m²), but for a similar value of LV mass index it was significantly greater in those with concentric geometry (OR: 4.07; 95% CI: 1.49 to 11.14; P=0.004 in the second tertile; OR: 3.45; 95% CI: 1.62 to 7.32; P=0.001 in the third tertile; P<0.0001 in concentric versus eccentric geometry). Persistence or development of concentric geometry during follow-up may have additional prognostic significance in hypertensive patients with and without LVH. (Hypertension. 2004;43:1-8.)

Key Words: hypertension • remodeling • cardiovascular disease

In hypertension, left ventricular hypertrophy (LVH) is initially a useful compensatory process to abnormal loading conditions but it is also the first step toward the development of overt clinical disease, such as congestive heart failure, ischemic heart disease, cardiac dysrhythmias, and stroke.1–4 The Framingham study has clearly demonstrated that once LVH is recognized clinically by electrocardiography5 or echocardiography,6 it represents a strong predictor for cardiovascular disease. Echocardiographic studies revealed that antihypertensive treatment can induce reversal of LVH in hypertensive patients,7 and the reduction of echocardiographically determined LV mass has been associated with a reduction in risk for subsequent cardiovascular disease.8,9

LV adaptation to arterial hypertension can yield different geometric responses with corresponding hemodynamic patterns related to the influence that, for a given level of blood pressure (BP), might have increased cardiac output or higher peripheral resistance.10,11 The assessment of LV mass and geometry may be useful in stratifying cardiovascular risk, and hypertensive patients with concentric hypertrophy have the greatest likelihood of having a cardiovascular event.12–15

Echocardiographic studies evaluating LVH regression by antihypertensive treatment have usually reported changes in LV mass index, and changes in relative wall thickness have not been properly assessed. The prognostic significance of changes in LV geometry has not been evaluated. Therefore, the purpose of the present study was to investigate the role of changes in LV geometric patterns in the prediction of cardiovascular events in a large group of prospectively identified essential hypertensive patients with and without LVH undergoing usual medical treatment.

Methods

We prospectively identified uncomplicated hypertensive subjects selected from an ongoing prospective registry of morbidity and mortality according to the following criteria. At entry, all patients had clinic systolic BP ≥140 and/or diastolic BP ≥90 mm Hg and no clinical or laboratory evidence of cardiovascular complications, secondary hypertension, or severe medical illness. Family doctor gave antihypertensive treatment and followed-up all patients. All patients were invited to repeat the visit and the echocardiographic examination at least every year after the initial study. Direct contact or...
telephone interviews were undertaken to ascertain the incidence of major medical events or complications. All subjects gave informed consent to the study. The institution committee on human research approved the study protocol.

Complications
Cardiovascular complications were considered sudden death, fatal and non-fatal stroke, myocardial infarction, transient cerebral ischemic attack, new-onset angina, progressive heart failure requiring hospitalization, coronary artery bypass or angioplasty, and renal failure requiring dialysis. Hospital records and all other available source documents collected were reviewed by all authors. Two independent medical members of the local Ethical Committee, unaware of the echocardiographic data, decided the attribution of cardiovascular events to patients on the basis of available documents.

Clinical and Echocardiographic Evaluation
At baseline and follow-up visits, patients underwent a thorough clinical examination, cardiovascular risk factors assessment, and a documented clinical history was collected. Seated BP was measured by sphygmomanometer as the mean of 3 measurements after the echocardiographic examination. M-mode echocardiographic tracings, obtained under 2-dimensional control, were measured by 2 different readers in a blinded manner according to the recommendations of the American Society of Echocardiography. Concentric geometry was defined as a relative wall thickness (RWT) ≥0.44.12 LV mass was calculated according to the Penn Convention.17 LVH was prospectively defined as a value of LV mass index (LVMI) ≥125 g/m².18,19 LV function was estimated by endocardial and mid-wall fractional shortening, as previously reported.20

Statistical Analysis
The calculation of sample size power confirmed an adequate number of patients for each subgroup study as compared with patients with normal LV mass and geometry. The statistical power ranged from 63% to 95%, considering a mean follow-up of 9 years.

Event-free survival analysis was performed with the Kaplan-Meier method and the groups were compared using the Mantel test. The relative importance of each prognostic factor, adjusting for the others, was assessed using Cox proportional hazard model with stepwise procedure.

Data were stored and analyzed with the SPSS 10.1 Statistical software package. All data are expressed as mean ± SD. In addition, confidence intervals of differences between groups with different echocardiographic geometric patterns are given as a surrogate information of statistical power of the study.21–23 Differences among prospectively defined subgroups were analyzed by ANOVA (Scheffé post-hoc) and χ² tests when appropriate; P<0.05 was considered statistically significant.

Results
A total of 436 patients (249 men and 187 women, age range 18 to 71 years, mean age 52±9) completed the follow-up with final echocardiogram and clinical evaluation. At baseline, patients were never treated (n=247) or had withdrawn for at least 4 weeks before antihypertensive treatment (n=275). The period between the pretreatment and the last follow-up echocardiogram was 72±38 months (range 12 to 168 months), whereas the total duration of clinical follow-up after the last echocardiogram was 42±16 months (range 4 to 96 months). We have assessed pharmacological treatment of patients: β-blockers, diuretics, calcium-channel blockers, and angiotensin-converting enzyme (ACE) inhibitors were used alone in 14%, 4%, 15%, and 12% of patients, respectively, and in various combination in 43% of patients (we considered the drug class used for the longest period of treatment during follow-up). Antihypertensive treatment was changed several times and temporarily withdrawn in 12% of patients.

Cardiovascular Morbidity and Mortality
Seventy-one patients had a documented cardiovascular event occurring after the follow-up echocardiographic examination. Sixty-six morbid events were reported (stroke or transient ischemic attack in 21, myocardial infarction in 16, angina [plus ventricular fibrillation in 1 patient] requiring hospitalization in 10, angioplasty or coronary bypass in 6, heart failure requiring hospitalization [plus atrial fibrillation in 2 patients] in 12, and end-stage renal disease in 1). Five patients died of a cardiovascular event (stroke in 2, myocardial infarction in 2, and end-stage renal disease in 1).

Age, systolic BP, RWT, and LVMI were greater at baseline and at follow-up in patients who experienced cardiovascular events than in those without events (Table 1).

When cardiac and cerebrovascular events were analyzed separately, cardiac events occurred more frequently in men than in women (OR: 3.06; 95% CI: 1.01 to 8.96); no other significant differences in demographic characteristics, LVMI, or LV geometry were observed between patients who had a cardiac or a vascular event (see Table available online at http://www.hypertensionaha.org).

LVH and Cardiovascular Risk
According to the presence of LVH at baseline and at the end of follow-up, patients were divided into 3 groups: with normal LVMI at both examinations, with LVH at baseline and regression of hypertrophy, and with LVH at baseline and at follow-up. Few patients with normal LVMI at baseline who had cardiac hypertrophy during follow-up (n=26, in whom 6 cardiovascular events were documented) were included in the latter group (Table 2). On average, a significant decrease of RWT was observed in patients with and without LVH regression (Table 3). The incidence of cardiovascular events, as analyzed according to LVH status, was significantly different among these 3 groups of patients (Table 2). Outcome was not related to the class of drugs used during follow-up. In the group of patients with persistence of LVH, a greater percentage of patients receiving association treatment was observed (Table 2), while no differences among monotherapy regimens were noted.

LV Geometry and Cardiovascular Risk
At baseline, 209 patients had a normal LVMI and geometry, 59 had concentric remodeling, 90 had eccentric LVH, and 78 had concentric LVH. At follow-up, a normal LVMI and geometry was found in 247 patients and concentric remodeling was found in 74 patients, whereas eccentric and concentric LVH were observed in 79 and 36 patients, respectively. In the whole group of patients, the change in LVMI was related to changes in diastolic and systolic BP (r=0.19, P=0.01 and r=0.13, P=0.05, respectively).

Cardiovascular events occurred in fewer patients with an eccentric geometry at baseline and at follow-up (n=233) than those observed in patients with a persistence of concentric geometry (n=46) (10% versus 53%, χ², P=0.001).

However, in the latter group, LVMI at follow-up was also significantly higher (129±41 versus 106±28 g/m², P=0.001). To clarify whether the degree of LV mass at follow-up, rather than the geometric pattern, could have influenced the clinical outcome, we evaluated the association
of events to changes in LV geometry, taking into account LVMI. Step-wise multiple logistic regression showed that the occurrence of cardiovascular events was independently associated to increasing age, male gender, persistence of LVH (as compared with persistently normal LV mass), and persistence of concentric geometry (as compared with persistently normal LV geometry), whereas BP (considered as a categorical variable, ie, BP $< 140/90$ mm Hg during treatment) did not enter into the equation (Table 4). When the analysis was restricted to patients with LVH at baseline, persistence of concentric geometry was the most potent predictor of cardiovascular events.

Patients were divided into tertiles of LVMI, measured at follow-up, and the rate of cardiovascular events was considered in relation to the presence of concentric or eccentric geometry; LVMI was not different between eccentric and concentric geometry in each tertile. The number of cardiovascular events was greater in patients with concentric geometry than those with eccentric geometry in the first tertile and reached statistical significance in the second and third tertiles (Figures 1 and 2).

The classification of patients into tertiles of LVMI and geometric pattern measured at follow-up was used in a Cox proportional hazard model, and a statistically significant excess risk was evident in the second and third tertiles, progressively increasing from subjects with eccentric to those with concentric geometry, after adjustment for covariates (adjusted HR [95% CI]=1 in the first tertile with eccentric geometry; 2.9 [95% CI: 0.64 to 13.7] in the first tertile with concentric geometry, NS; 1.9 [95% CI: 0.60 to 6.1] in the second tertile with eccentric geometry, NS; 6.66 [95% CI: 1.68 to 26.4] in the second tertile with concentric geometry, $P=0.007$; 3.69 [95% CI: 1.04 to 13.94] in the third tertile with eccentric geometry, $P=0.05$; and 7.96 [95% CI: 1.7 to 36.5] in the third tertile with concentric geometry, $P=0.008$). Other covariates that attained statistical significance to enter the model were age (adjusted HR: 1.07 [95% CI: 1.03 to 1.11]) and male gender, (adjusted HR: 1.99 [95% CI: 1.05 to 3.8]).

### Discussion

The main result of this study is that not only changes in LV mass but also changes in the pattern of LV geometric adaptation from baseline to follow-up have a prognostic value, because they may predict the risk for a subsequent cardiovascular event in uncomplicated essential hypertensive patients. The difference in cardiovascular morbidity and mortality remained significant after adjustment for several covariates.

LV geometric adaptation to increased cardiac load may be different among patients being associated to different hemodynamic characteristics and to a different risk for cardiovascular events. Several studies have confirmed this finding, although the apparent unfavorable effect of LV concentric geometry in terms of prediction of cardiovascular events did not always result independent from LV mass.

The closer association between concentric geometry and cardiovascular events may be explained by decreased myocardial contractility, severe diastolic filling abnormalities, increased oxygen requirement of myocardium, greater risk for arrhythmias, and sudden death. In addition the extent of
arterial disease, including early endothelial dysfunction, subclinical atherosclerotic disease and structural changes in large and small vessels have been proposed as a possible mechanism implicated in the occurrence of cardiovascular events. In patients with mild to moderate hypertension, carotid arterial structure is most abnormal when concentric geometry is present, possibly explaining the association with cerebrovascular events. Few studies have demonstrated an increase in the prevalence of retinopathy grades III and IV or in the forearm minimal vascular resistance in patients with concentric geometry. We have recently provided additional explanation by demonstrating the presence of concomitant vascular damage in small resistance arteries of patients with concentric hypertrophy and remodeling that could justify a more severe impairment of coronary vasodilator reserve and possibly of cerebral perfusion. In addition, it has been shown that the increase in LV mass and geometry is related to other traditional risk factors for coronary and cerebrovascular atherosclerotic disease, including hypercholesterolemia, insulin resistance, and a prothrombotic state. The mechanisms by which persistence of LVH and of concentric remodeling may predispose to the occurrence of events cannot be assumed from our study. Besides office BP and traditional risk factors assessment, we have observed that systolic performance after treatment remained significantly lower in patients with persistence of LVH and higher relative wall thickness. The possible influence of concomitant renal disease on geometric adaptation in patients with LVH seems not to be relevant; only 2 patients, 1 with concentric LVH at baseline and follow-up and 1 with concentric LVH and concentric remodelling at follow-up, had end-stage renal disease.

### TABLE 2. Patient Characteristics According to LVMI Changes

<table>
<thead>
<tr>
<th>Variable</th>
<th>No LVH → No LVH</th>
<th>LVH → No LVH (LVH regression)</th>
<th>LVH → LVH or No LVH → LVH (LVH persistence or development)</th>
<th>P Value ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, n</td>
<td>244</td>
<td>77</td>
<td>115</td>
<td></td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>103/141†</td>
<td>50/27</td>
<td>96/19</td>
<td>0.001</td>
</tr>
<tr>
<td>Age, y</td>
<td>53±6‡</td>
<td>49±9</td>
<td>53±8‡</td>
<td>0.003</td>
</tr>
<tr>
<td>Follow-up, months</td>
<td>68±37</td>
<td>78±40</td>
<td>76±37</td>
<td>0.06</td>
</tr>
<tr>
<td>Antihypertensive treatment (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temporarily withdrawn</td>
<td>16</td>
<td>11</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>ACE inhibitors or ARBS</td>
<td>12</td>
<td>14</td>
<td>10</td>
<td>NS</td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td>18</td>
<td>17</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>β-blockers</td>
<td>16</td>
<td>14</td>
<td>12</td>
<td>NS</td>
</tr>
<tr>
<td>Diuretics</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>Association</td>
<td>33</td>
<td>41</td>
<td>54</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>26.4±3.7†</td>
<td>27±3.4</td>
<td>27.3±3</td>
<td>0.03</td>
</tr>
<tr>
<td>Follow-up</td>
<td>26.5±3.8</td>
<td>27.5±3.6</td>
<td>27.6±3.2</td>
<td>0.01</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>5.3±0.8†</td>
<td>5.4±1.2</td>
<td>5.5±0.7</td>
<td>0.04</td>
</tr>
<tr>
<td>Follow-up</td>
<td>5.8±1.1</td>
<td>5.8±0.8</td>
<td>5.7±1.01</td>
<td>NS</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>5.9±1.1</td>
<td>6.02±1.2</td>
<td>5.9±1.2</td>
<td>NS</td>
</tr>
<tr>
<td>Follow-up</td>
<td>5.9±1.01</td>
<td>6.1±1.24†</td>
<td>5.9±1.01</td>
<td>0.05</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>156±18†</td>
<td>159±20†</td>
<td>162±23</td>
<td>0.04</td>
</tr>
<tr>
<td>Follow-up</td>
<td>145±15†</td>
<td>146±14*</td>
<td>151±17*</td>
<td>0.006</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>97±10 †</td>
<td>101±11</td>
<td>103±12</td>
<td>0.003</td>
</tr>
<tr>
<td>Follow-up</td>
<td>90±9†</td>
<td>92±9*</td>
<td>92±10*</td>
<td>0.028</td>
</tr>
<tr>
<td>CV events, n</td>
<td>24</td>
<td>7</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Relative risk (95% IC)</td>
<td>1(reference)</td>
<td>0.92</td>
<td>4.89</td>
<td></td>
</tr>
</tbody>
</table>

LVMI indicates left ventricular mass index; LVH, left ventricular hypertrophy; ACE, angiotensin-converting enzyme; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; CV, cardiovascular.

ANOVA P value refers to differences among the three groups.
The results of this study are in keeping with previous reports\(^8,9\) demonstrating that regression of echocardiographically determined LVH is associated with an improvement in prognosis. In this study, we followed a larger number of patients with serial LV mass measurements, and this has allowed us to evaluate the prognostic value of changes in LV geometry. Echocardiographic studies evaluating LVH regression by antihypertensive treatment have sometimes shown a

<table>
<thead>
<tr>
<th>Variable</th>
<th>No LVH (\rightarrow) No LVH</th>
<th>LVH (\rightarrow) No LVH (LVH regression)</th>
<th>LVH (\rightarrow) LVH or No LVH (\rightarrow) LVH (LVH persistence or development)</th>
<th>(P) Value ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI (g/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(95 \pm 18)†‡</td>
<td>(148 \pm 24)</td>
<td>(152 \pm 38)</td>
<td>0.001</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(91 \pm 18)†§</td>
<td>(102 \pm 14)§</td>
<td>(150 \pm 24)</td>
<td>0.001</td>
</tr>
<tr>
<td>RWT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(0.38 \pm 0.1)†‡</td>
<td>(0.43 \pm 0.1)</td>
<td>(0.44 \pm 0.1)</td>
<td>0.001</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(0.39 \pm 0.1)†‡</td>
<td>(0.37 \pm 0.1)§</td>
<td>(0.42 \pm 0.1)§</td>
<td>0.001</td>
</tr>
<tr>
<td>Endocardial FS %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(40 \pm 8)†</td>
<td>(38 \pm 8)</td>
<td>(35 \pm 9)</td>
<td>0.001</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(41 \pm 9)†</td>
<td>(41 \pm 7)§</td>
<td>(37 \pm 9)§</td>
<td>0.001</td>
</tr>
<tr>
<td>Midwall FS %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(17.2 \pm 3.7)†‡</td>
<td>(15.3 \pm 3.2)</td>
<td>(14.6 \pm 3.8)</td>
<td>0.001</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(17.6 \pm 3.2)†‡</td>
<td>(17.7 \pm 3.2)§</td>
<td>(15.5 \pm 3.2)§</td>
<td>0.001</td>
</tr>
<tr>
<td>Endocardial FS % of predicted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(101 \pm 23)†</td>
<td>(108 \pm 18)</td>
<td>(101 \pm 21) NS</td>
<td>0.01</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(115 \pm 17)§</td>
<td>(117 \pm 17)</td>
<td>(112 \pm 18) NS</td>
<td>0.001</td>
</tr>
<tr>
<td>Midwall FS % of predicted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>(99 \pm 21)†</td>
<td>(97 \pm 23)</td>
<td>(91 \pm 26)</td>
<td>0.01</td>
</tr>
<tr>
<td>Follow-up</td>
<td>(112 \pm 19)†</td>
<td>(116 \pm 21)§</td>
<td>(102 \pm 21)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

LVH indicates left ventricular hypertrophy; LVMI, left ventricular mass index; RWT, relative wall thickness; FS, fractional shortening.

ANOVA \(P\) value refers to differences among the 3 groups.

*\(P<0.05\) vs baseline; † vs LVH persistence or development; ‡ vs LVH regression; § \(P<0.01\) vs baseline; ¶ \(P<0.001\) vs baseline.

The results of this study are in keeping with previous reports\(^8,9\) demonstrating that regression of echocardiographically determined LVH is associated with an improvement in prognosis. In this study, we followed a larger number of patients with serial LV mass measurements, and this has allowed us to evaluate the prognostic value of changes in LV geometry. Echocardiographic studies evaluating LVH regression by antihypertensive treatment have sometimes shown a
progressive decrease in relative wall thickness, although the prognostic significance of changes in LV geometric adaptation has not been evaluated.\textsuperscript{39}

Echocardiography is a sensitive and specific method for the identification of LVH.\textsuperscript{40} Other techniques, including cardiac magnetic resonance, can provide a more accurate measurement of LV mass, although they are of limited availability, expensive, and time consuming.\textsuperscript{41} The method of calculation of LV mass by echocardiography might carry the possibility of a technical error, which has been repeatedly assessed.\textsuperscript{42,43} To this regard, the reproducibility of relative wall thickness values is higher than that of LV mass.\textsuperscript{44}

After adjustment for other covariates, the presence of concentric geometry was associated with an increased risk of subsequent cardiovascular complications, even in patients with follow-up LVMI values within a “normal” range.

Some limitations deserve considerations. BP values at baseline and follow-up or their changes during the follow-up did not result in association with cardiovascular risk in these patients; however, it cannot be excluded that the observed changes of LV mass may reflect, at least in part, pressure control.\textsuperscript{45} LVH regression is more strictly related to the reduction in 24 hours rather than in clinic BP values,\textsuperscript{46} and 24-hour ambulatory BP monitoring could have given additional information, although in a previous study\textsuperscript{14} showing the favorable prognostic impact of LV mass reduction, neither clinic nor ambulatory BP attained statistical significance to enter a Cox model. In this study, no clear assessment of those disturbances of cardiac rhythm (such as atrial fibrillation) that may impact on cardiovascular outcome in patients with LVH was made. We could not properly assess the specific influence of antihypertensive treatment prescribed in a general practice setting, and it was not possible to relate outcome to the class of antihypertensive drugs used. Echocardiographic studies evaluating reversal of LVH with the use of different antihypertensive drugs have been performed and have indicated that ACE inhibitors, angiotensin II receptor blockers, and calcium antagonists may be more effective than β-blockers and diuretics in reducing LV mass for similar BP reduction.\textsuperscript{\textdagger,39} It should be kept in mind that BP control may require the use of antihypertensive agents in combination; long-term antihypertensive treatment, associated with a progressive control of BP and decrease of LV mass, might reduce possible differences among classes of drugs, as shown in this study.

Because our study was conducted in white subjects, results cannot be extended to other ethnic groups. Another limitation is the relatively small number of fatal cardiovascular events, probably due to the inclusion of many uncomplicated patients.

**Perspectives**

The results of this study support the concept that measurement of LV mass and relative wall thickness after several years of treatment can help identify subsequent level of risk in hypertensive patients.
Persistence of concentric geometry, even in the presence of normal LV mass, should raise concern about appropriate BP control, compliance to treatment, or concomitant vascular disease in hypertensive patients, implying a more strict clinical control.

References


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Hypertension. published online March 8, 2004;
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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