Effects of Drug Therapy on Cardiac Arrhythmias and Ischemia in Hypertensives With LVH

Salvatore Novo, Maurizio G. Abrignani, Giuseppina Novo, Emilio Nardi, Ligia J. Dominguez, Antonio Strano, and Mario Barbagallo

Left ventricular hypertrophy (LVH) in hypertensive subjects is associated with an increased prevalence of ventricular arrhythmias. To evaluate the effect of antihypertensive treatment on cardiac arrhythmias (CA) and transient episodes of myocardial ischemia (TEMI), we studied 46 hypertensive patients with LVH, divided into four groups randomly treated with enalapril, hydrochlorothiazide (HCTZ), atenolol, or verapamil (SR-V) for 6 months. Office blood pressure and office heart rate values were recorded, in basal conditions, after 1 and 6 months of treatment, and all patients underwent echocardiography, electrocardiographic Holter monitoring, and stress testing. All drugs significantly lowered blood pressure, whereas left ventricular mass index was reduced by atenolol, enalapril, and SR-V, but not by HCTZ. Treatment induced a significant reduction in the number of patients with supraventricular arrhythmias (35 v 15, P < .034, and 28 v 8, excluding patients treated with HCTZ, P < .008). The number of patients with ventricular arrhythmias was also reduced (32 v 16 considering all groups, P < .08, and 24 v 9, excluding patients treated with HCTZ, P < .04). The number of TEMI during Holter monitoring significantly decreased from 47 to 23 (P = .043) in all patients, and from 39 to 14 (P = .013) excluding patients treated with HCTZ. In all groups, irrespective of treatment, a reduction of blood pressure, heart rate, and systolic blood pressure/heart rate product measured by exercise stress test was observed. The present study shows that in hypertensive patients with LVH, antihypertensive treatment with atenolol, enalapril and SR-V reduces LVH and decreases the prevalence of CA and TEMI. Treatment with HCTZ during the 6-month study did not alter LVH and did not appear to reduce CA and TEMI. Am J Hypertens 2001; 14:637– 643 © 2001 American Journal of Hypertension, Ltd.

Key Words: Hypertension, left ventricular hypertrophy, cardiac arrhythmias, ambulatory electrocardiographic monitoring, antihypertensive drugs.

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Inhibitors, 5 with were already on treatment at screening time (11 with ACE before the beginning of the study. Thirty-three patients LVH. The diagnosis of hypertension was made according to the WHO/ISH recommendations, at least 6 months before the beginning of the study. Thirty-three patients were already on treatment at screening time (11 with ACE inhibitors, 5 with β-blockers, 10 with calcium antagonists, and 7 with diuretics).

Secondary forms of hypertension were excluded by history, physical examination, and routine biochemistry. Patients with history of angina, acute myocardial infarction, cardiac palpitations or syncope, valve disease, and hypertrophic cardiomyopathy were excluded from the study; patients with an echocardiographically measured ejection fraction (EF) <50% were also excluded. Patients with diabetes mellitus were also excluded from the study. Informed consent was obtained before the study started. All patients included in the study had normal resting electrocardiograms and normal plasma levels of sodium, potassium, and magnesium.

At basal condition, after screening and a 4-week placebo run-in period, all patients underwent clinical examinations, evaluation of serum parameters, standard 12-lead electrocardiograph, Holter monitoring, M-mode and two-dimensional echocardiography, and exercise stress testing. Although we did not perform an angiography, major epicardial coronary artery lesions were excluded by the absence of clinical and ECG (in basal and exercise conditions) signs of CAD.

Patients were subsequently divided into four groups, randomly assigned to enalapril 20 mg/day (n = 10), hydrochlorothiazide 25 mg/day (n = 10), atenolol 100 mg/day (n = 13), or verapamil SR 240 mg/day (n = 13). During the study patients followed a standard hyposodium diet. An echocardiographic examination and a 24-h ECG recording and a cycloergometer stress test were also performed after 1 and 6 months of treatment. Office systolic and diastolic blood pressures (SBP, DBP) and office heart rate (HR) were also evaluated at each time point. Tablet count was performed at each visit to monitor the compliance of the patients.

Echocardiographic Examination
All patients underwent M-mode and two-dimensional echocardiography by standard parasternal and apical windows. To decrease interobserver variability, all the examinations were carried out by the same investigator, not aware of the aim of the study, using an Irex III Kontron system with a 2.5-MHz transducer and an aperture size of 16 mm.

M-mode recordings were made on 15-cm paper at a speed of 50 mm/sec. The analysis was performed with a digitalizing Kontron table and a microcomputer Cardio 80. Posterior wall and interventricular septum thickness were measured off-line at R peak on the electrocardiogram and the mean of the values of three cardiac beats was used. Left ventricular mass (LVM) was calculated according to the method described by Devereux and Reichek. It was considered LVH an LVM normalized for body size (LVMI) >134 g/m² in men and >110 g/m² in women. We only enrolled patients with concentric LVH.

We also evaluated end-diastolic diameter (EDD), end-systolic diameter (ESD), fractional shortening (FS), end-systolic stress (ESS), and LVM. FS of the left ventricle (LV) was calculated by the following formula:

(diastolic LV internal diameter – systolic LV internal diameter)/diastolic LV internal diameter.

Twenty-Four-Hour ECG Holter Monitoring
Ambulatory 24-h ECG recording was performed by using a 445 B Del Mar Avionics Cassette obtaining two traces through the use of CM2 and CM5 leads. Recordings were analyzed blindly and the following parameters were evaluated: 1) number of TEMI, defined as an ST segment depression of >1 mm, 80 msec after the J point, with a duration of 1 min or more, with or without HR increase, as detected during 24-h Holter monitoring; 2) number and percentage of patients with supraventricular ectopic beats (SVEB) >30/h, with supraventricular couplets (SVC) and with supraventricular paroxysmal tachycardia (SVT) runs; 3) number and percentage of patients with ventricular ectopic beats (VEB) >30/h, with ventricular couplets (VC) and ventricular tachycardia (VT) runs.

Exercise Stress Testing
Stress tests was performed by using a Bosch cycloergometer with an electromagnetic brake, and a computerized electrocardiograph Meta III Hittman, with steps of 25 W increased every 3 min, evaluating SBP and DBP, HR, SBP × HR product at the maximal common work (MCW), and the number of ventricular ectopic beats during the last minute of the stress test.

Statistical Analysis
All data are expressed as means ± SD. Student’s t test for paired and unpaired data was used to calculate statistical significance between means. Differences between frequencies of CA and TEMI were calculated using the χ² test.

Results
Systolic and DBP values, HR, and echocardiographic, Holter, and ergometer parameters at baseline are shown in Table 1.

At the end of the study period, all of the antihyperten-
\[\text{Echo-ESS (10}^3\text{ dyne }\times \text{cm}^2)\]
\[\text{Echo-FS (%)}\]
\[\text{Echo-ESD (mm)}\]
\[\text{Echo-EDD (mm)}\]
\[\text{HCTZ (mg/dL)}\]
\[\text{Office HR (beats/min)}\]
\[\text{Office SBP (mm/Hg)}\]
\[\text{Echo-LVMI (g/m}^2\text{)}\]
\[\text{SVC number and % of patients with supraventricular ectopic beats}\]
\[\text{patients with ventricular ectopic beats}\]
\[\text{Modification of SBP and DBP and HR after 1 and 6 months of treatment}\]
\[\text{Table 2.}\]
\[\text{Asymptomatic TEMI (HR increase)}\]
\[\text{Table 1.}\]
\[\text{SBP} - \text{systolic blood pressure}; \text{DBP} - \text{diastolic blood pressure}; \text{HR} - \text{heart rate}; \text{EDD} - \text{end-diastolic diameter}; \text{EDS} - \text{end-systolic diameter}; \text{LVM} - \text{normalized for body size left ventricular mass}; \text{FS} - \text{fractional shortening of the left ventricle}; \text{ESS} - \text{end-systolic stress}; \text{TEMI} - \text{transient episodes of myocardial ischemia}; \text{SVEB} > 30/h - \text{number and % of patients with supraventricular ectopic beats >30/h}; \text{SVC} - \text{no. and % of patients with SV couplets}; \text{SVT runs} - \text{no. and % of patients with SV tachycardia runs}; \text{VEB} > 30/h - \text{no. and % of patients with ventricular ectopic beats >30/h}; \text{VC} - \text{no. and % of patients with ventricular couplets}; \text{VT runs} - \text{no. and % of patients with runs of ventricular tachycardia}; \text{SBP, DBP, HR, SBP } \times \text{HR, at MCW} - \text{stress test parameters at maximal common work.}\]

\[\text{Table 2.}\]
\[\text{Modification of SBP and DBP and HR after 1 and 6 months of treatment}\]
\[\text{Drug} \quad \text{Basal} \quad \text{1 Month} \quad \text{6 Months}\]
\[\text{Enalapril (n = 10)}\]
\[\text{SBP} \quad 160 \pm 10 \quad 143 \pm 11^* \quad 130 \pm 12^†\]
\[\text{DBP} \quad 106 \pm 7 \quad 94 \pm 7^* \quad 85 \pm 8^†\]
\[\text{HR} \quad 74 \pm 8 \quad 72 \pm 7 \quad 71 \pm 7\]
\[\text{HCTZ (n = 10)}\]
\[\text{SBP} \quad 153 \pm 10 \quad 139 \pm 10^* \quad 135 \pm 12^*\]
\[\text{DBP} \quad 104 \pm 8 \quad 95 \pm 7^* \quad 88 \pm 8^*\]
\[\text{HR} \quad 74 \pm 8 \quad 78 \pm 8 \quad 75 \pm 7\]
\[\text{Atenolol (n = 13)}\]
\[\text{SBP} \quad 158 \pm 11 \quad 141 \pm 10^* \quad 131 \pm 12^†\]
\[\text{DBP} \quad 104 \pm 5 \quad 90 \pm 6^† \quad 84 \pm 8^†\]
\[\text{HR} \quad 78 \pm 8 \quad 62 \pm 7^* \quad 60 \pm 7^*\]
\[\text{Verapamil (n = 13)}\]
\[\text{SBP} \quad 158 \pm 10 \quad 143 \pm 11^* \quad 139 \pm 12^*\]
\[\text{DBP} \quad 102 \pm 5 \quad 91 \pm 6^* \quad 88 \pm 6^*\]
\[\text{HR} \quad 74 \pm 8 \quad 70 \pm 6 \quad 71 \pm 7\]

\[\text{HCTZ} - \text{hydrochlorothiazide}; \text{other abbreviations as in Table 1.}\]

\[^* P < .01;\]
\[^† P < .001.\]
>30/h diminished from 14 to 8, with VC from 15 to 7, with VT runs from 3 to 1. Total reduction of ventricular arrhythmias was moderately significant (from 32 to 16 patients, 250%, P = .08) and it was more evident when excluding patients taking HCTZ (from 24 to 9 patients, 262.5%, P = .04). In all the groups, exercise stress testing showed a significant and similar decrease in SBP, DBP, HR, and SBP × HR product at MCW after treatment (Table 5).

### Discussion

The present study shows that in hypertensive patients with LVH, antihypertensive treatment with atenolol, enalapril, and SR-V reduces LVH and decreases the prevalence of CA and TEMI. Treatment with HCTZ during the 6-month study did not alter LVH and did not appear to reduce CA or TEMI.

The LVH in hypertensive patients is associated with an increased prevalence of supraventricular and ventricular arrhythmias. The presence of many confounding factors (sympathetic tone, hormones, circadian rhythm of blood pressure, effects of antihypertensive drugs, etc.) and the variability of premature beats prevents easily establishing a cause–effect relationship between LVH and ventricular arrhythmias. Several mechanisms have been suggested to explain this association.

Arrhythmogenic cofactors, such as activation of the sympathoadrenergic system, drug-induced electrolyte disturbances, proarrhythmic treatments, circadian BP changes, and BP peaks, may aggravate the occurrence of ventricular arrhythmias.

It is still unclear whether ventricular ectopic activity can be a marker of sudden cardiac death, because silent myocardial ischemia, as a consequence of either CHD or LVH, may be present in hypertensive patients. As ventricular arrhythmias and silent ischemia are risk factors for cardiac events in hypertension, their control and the prevention of TEMI is a desirable goal of antihypertensive treatment.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Drugs</th>
<th>Basal</th>
<th>1 Month</th>
<th>6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDD (mm)</td>
<td>Enalapril</td>
<td>50 ± 5</td>
<td>49 ± 4</td>
<td>48 ± 4*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>48 ± 5</td>
<td>47 ± 4</td>
<td>46 ± 5*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>49 ± 4</td>
<td>49 ± 3</td>
<td>51 ± 4</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>50 ± 3</td>
<td>50 ± 3</td>
<td>50 ± 4</td>
</tr>
<tr>
<td>ESD (mm)</td>
<td>Enalapril</td>
<td>33 ± 4</td>
<td>32 ± 4</td>
<td>31 ± 4*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>32 ± 4</td>
<td>32 ± 4</td>
<td>31 ± 4</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>33 ± 3</td>
<td>31 ± 3</td>
<td>32 ± 4</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>32 ± 2</td>
<td>31 ± 3</td>
<td>32 ± 2</td>
</tr>
<tr>
<td>FS (%)</td>
<td>Enalapril</td>
<td>34 ± 4</td>
<td>34 ± 3</td>
<td>35 ± 4</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>33 ± 4</td>
<td>32 ± 3</td>
<td>32 ± 4</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>33 ± 4</td>
<td>34 ± 3</td>
<td>33 ± 4</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>36 ± 3</td>
<td>36 ± 3</td>
<td>36 ± 4</td>
</tr>
<tr>
<td>ESS (dyne/cm²)</td>
<td>Enalapril</td>
<td>87 ± 7</td>
<td>77 ± 7*</td>
<td>75 ± 5*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>83 ± 7</td>
<td>76 ± 8*</td>
<td>73 ± 5*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>89 ± 7</td>
<td>78 ± 6*</td>
<td>75 ± 5*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>88 ± 6</td>
<td>80 ± 5*</td>
<td>80 ± 5*</td>
</tr>
<tr>
<td>LVM (g/m²)</td>
<td>Enalapril</td>
<td>140 ± 11</td>
<td>139 ± 12</td>
<td>118 ± 11*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>143 ± 15</td>
<td>139 ± 16</td>
<td>133 ± 15</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>142 ± 10</td>
<td>143 ± 12</td>
<td>115 ± 10*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>136 ± 10</td>
<td>133 ± 12</td>
<td>118 ± 10*</td>
</tr>
</tbody>
</table>

LVM = left ventricular mass; other abbreviations as in Tables 1 and 2.

*P < .01.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Basal</th>
<th>1 month</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enalapril</td>
<td>9</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>HCTZ</td>
<td>8</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Atenolol</td>
<td>14</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Verapamil</td>
<td>16</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>All drugs</td>
<td>47</td>
<td>28 (−40.43%)</td>
<td>23 (−51.07%)*</td>
</tr>
<tr>
<td>All drugs except HCTZ</td>
<td>39</td>
<td>20 (−48.72%)</td>
<td>14 (−64.11%)†</td>
</tr>
</tbody>
</table>

Abbreviation as in Tables 1–3.

* P = .043 v basal.
† P = .013 v basal.
therapy. There is evidence that this control may be achieved by the reversal of LVH induced from some antihypertensive drug regimens for high blood pressure. On the other hand, supraventricular arrhythmic control, which likely occurs by the reduction in left atrial size, may also be important.

Our study confirms a comparable antihypertensive efficacy of all tested drugs in hypertensive patients with LVH. No significant changes were observed on HR except for atenolol, and only enalapril was able to decrease ventricular diameters.

The decrease in EDD observed after HCTZ chronic treatment is attributable to its ability to reduce the preload. LV systolic function, as assessed by FS, was normal at baseline and after 1 and 6 months of treatment with all the studied drugs. After 6 months of treatment with atenolol, enalapril, and verapamil, LVM was significantly reduced, if compared with baseline, confirming the action of these drugs on LVH. Our study is also in agreement with previous reports in which no reduction in LVM was observed after treatment with HCTZ. However, the lack of substantial changes in FS and the significant decrease of ESS may support the hypothesis that the only finding of a slight compensatory LVH in mild hypertensives may not necessarily be detrimental.

Our study clearly indicates a reduction in ventricular and supraventricular arrhythmias after chronic treatment with antihypertensive drugs, and particularly in those patients treated with atenolol, enalapril, and SR-V. We cannot determine with the present data whether this action is drug specific or a nonspecific effect of LVM diminution, or of other unknown mechanisms. The reduction in CA may also be consequent to the control of BP values, although patients treated with HCTZ did not confirm this favorable trend. The ability of β-blockers and ACE inhibitors of reducing CA arrhythmias have been studied extensively after myocardial infarction and in patients with stable congestive heart failure. Efficacy of verapamil in reducing the all-cause mortality in patients who had experienced a non-Q wave acute myocardial infarction has also been extensively investigated, but not in hypertensive patients.

Because the reduction in CA was similar with the three different drug classes, it is unlikely that the underlying mechanism is an antiarrhythmic-specific effect of one drug on the myocardium. It is more likely the effect, common to all three drugs, to lower BP and to reduce LVM, which may be implicated in the decrease of CA. LVM inducing myocardial remodeling may ultimately impair ventricular perfusion and make the myocardium susceptible to the development of arrhythmogenic foci. The LVM regression, restoring the ventricular anatomy, perfusion balance, and wall stress may indirectly reduce the occurrence of ventricular arrhythmias.

Hydrochlorothiazide treatment was able to reduce wall stress but not CA. Changes in left ventricular internal diameter was not associated with a reduction of CA. Diuretic-induced hypokalemia and hypomagnesemia may be responsible for increasing CA. Hypokalemia has a clear effect on the electrophysiologic properties of the heart. It could be responsible for generating reentrant arrhythmias by increasing the resting membrane potential, the duration of action potential and the refractory period, by increasing threshold potential, automaticity, and reducing conductivity. A role for electrolyte disturbance in ventricular arrhythmias in hypertensives treated with HCTZ was excluded because their levels were within the normal range in this study. However, we cannot rule out the possibility of an ionic alteration at the intracellular level, as we did not measure intracellular electrolyte lev-

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**Table 5.** Modifications of SBP, DBP, HR, SBP × HR product at maximal common work during stress testing before and after treatment

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Drugs</th>
<th>Basal</th>
<th>1 Month</th>
<th>6 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td>Enalapril</td>
<td>218 ± 23</td>
<td>198 ± 19*</td>
<td>185 ± 12*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>213 ± 22</td>
<td>196 ± 16*</td>
<td>189 ± 15*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>219 ± 16</td>
<td>198 ± 18*</td>
<td>183 ± 12*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>209 ± 16</td>
<td>193 ± 17*</td>
<td>189 ± 12*</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>Enalapril</td>
<td>127 ± 13</td>
<td>109 ± 14*</td>
<td>107 ± 13*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>137 ± 13</td>
<td>121 ± 13*</td>
<td>115 ± 15*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>133 ± 13</td>
<td>108 ± 12*</td>
<td>107 ± 12*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>128 ± 14</td>
<td>117 ± 11*</td>
<td>113 ± 12*</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>Enalapril</td>
<td>150 ± 16</td>
<td>134 ± 16*</td>
<td>133 ± 18*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>140 ± 16</td>
<td>133 ± 16*</td>
<td>132 ± 14*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>144 ± 19</td>
<td>120 ± 16*</td>
<td>118 ± 19*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>144 ± 18</td>
<td>122 ± 14*</td>
<td>121 ± 17*</td>
</tr>
<tr>
<td>SBP × HR</td>
<td>Enalapril</td>
<td>32,700 ± 4876</td>
<td>26,540 ± 5112*</td>
<td>24,598 ± 3990*</td>
</tr>
<tr>
<td></td>
<td>HCTZ</td>
<td>29,818 ± 4667</td>
<td>26,040 ± 4356*</td>
<td>24,968 ± 4221*</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>31,550 ± 4678</td>
<td>23,740 ± 5258*</td>
<td>21,566 ± 3666*</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>30,050 ± 4370</td>
<td>23,540 ± 4118*</td>
<td>22,865 ± 3987*</td>
</tr>
</tbody>
</table>

Abbreviations as in Tables 1–4.

* p < .01.
els. Previous findings of a reduction in CA with both short- and long-term antihypertensive therapy using calcium antagonists, β-blockers, and ACE inhibitors, but not thiazide diuretics, is also in agreement with our present data.

Published studies showed conflicting results on the behavior of CA after antihypertensive long-term treatment, probably because of the large variability in the parameters and the relatively small sample size. It is possible that our present result of a less satisfactory effect of HCTZ on CA may be related to its minimal effect on LVM, at least during the studied period. It is also possible that diuretics may need a longer period of time to achieve the same goal on LVM and CA as other antihypertensive treatments, and this should be assessed by future studies.

Our study also demonstrates a clear reduction in the number of TEMI, irrespective of the HR changes, which was more evident with treatments that were able to reduce LVH in the observed time (A, E, and SR-V). Thus, ST-T depression frequently observed during the 24-h ECG Holter monitoring may be related to an imbalance between increased oxygen demand and insufficient blood supply in a hypertrophic heart in the absence of main coronary vessel obstructive disease. This pathophysiological mechanism may also explain the reduction of TEMI occurring after LVH regression (Table 4).

All drugs reduced BP and pressure rate product (PRP), at MCW during stress testing. The diminution of PRP appeared to be more evident in the atenolol and verapamil groups, which may be due to the influence of these drugs, especially atenolol, in reducing HR. The decrease of PRP during exercise, by reducing myocardial oxygen consumption, may be useful in hypertensive patients with LVH and in part, could explain the favorable effect of antihypertensive drugs on TEMI and CA.

As a possible caveat to the present study, we should mention that for ethical reasons we did not include a control group without antihypertensive treatment, and we could not perform angiography in our population of asymptomatic hypertensives. The influence of a preclinical CHD on our findings was thus ruled out by performing an exercise stress test. A further limitation of the study is the relatively small sample size that prevented us from performing an analysis of the effects of each drug class, which needs to be addressed by future studies. The low baseline prevalence of arrhythmias also reduced the power of the study in evaluating CA variation in response to each antihypertensive therapy. Furthermore, the exclusion of HCTZ-treated patients, which improved the statistical significance of our results (after pooling all patients that had a borderline reduction in CA and TEMI), was not a predefined analysis.

In conclusion, our study shows that antihypertensive treatment reduces LVH and decreases the prevalence of CA and TEMI. We believe that our findings may have clinical relevance, although future studies are needed to follow more patients for longer periods of time to detect major arrhythmic events and a possible relationship with cardiac death, and to further confirm that the goal of antihypertensive treatment should not be only to lower blood pressure, but also to obtain a regression of LVH, a reduction of ventricular arrhythmias, major arrhythmic events, TEMI, and cardiac death.

References


