Effect of Irbesartan Versus Atenolol on Left Ventricular Mass and Voltage
Results of the CardioVascular Irbesartan Project
Markus P. Schneider, Arnfried U. Klingbeil, Christian Delles, Malte Ludwig, Rainer E. Kolloch, Michael Krekler, Klaus O. Stumpe, Roland E. Schmieder

Abstract—Regression of hypertensive left ventricular hypertrophy (LVH) is associated with improved prognosis. The aim of this trial was to compare the effects of irbesartan versus atenolol on LVH in subjects with essential hypertension. Because electrocardiographic and echocardiographic parameters of LVH carry disparate prognostic information, both methods were applied in this trial. In the randomized, double-blind, multicenter trial CardioVascular Irbesartan Project, 240 patients with essential hypertension were treated with irbesartan or atenolol for 18 months. Voltage criteria used for LVH were Sokolow index, Cornell index, Cornell voltage x QRS duration product and Lewis index. In parallel, left ventricular mass (LVM) was determined by 2-dimensional guided M-mode echocardiography. After 6 and 18 months, reductions of LVM and voltage criteria for LVH were only found in subjects treated with irbesartan. However, a reduction of LVM was only detectable in subjects within the highest quartile of baseline LVM but not overall. In contrast, reductions of voltage criteria for LVH were detectable after 6 and 18 months even within commonly used normal limits. In conclusion, treatment of hypertension with irbesartan resulted in a significant reduction in the voltage criteria for LVH, although an effect on LVM was only seen in subjects with high baseline LVM. In contrast, atenolol did not lead to reductions in electrocardiographic or echocardiographic parameters of LVH. Because voltage criteria for LVH have been shown to predict cardiovascular outcome independently from LVM, we suggest that both methods should be used to accurately assess the benefits of antihypertensive treatment. (Hypertension. 2004;44:61-66.)

Key Words: hypertension, essential hypertrophy, electrocardiography, echocardiography, drug therapy

In patients with arterial hypertension the diagnosis of left ventricular hypertrophy (LVH) either detected by electrocardiography (ECG)1,2 or by echocardiography3–8 strongly predicts cardiovascular mortality. However, any cutoff value for LVH is defined arbitrarily because cardiovascular risk rises continuously with increasing left ventricular mass (LVM)4 or increasing values of voltage criteria for LVH.2 On the other hand, regression of LVH by antihypertensive therapy detected either by echocardiography7–9 or ECG2,10 is associated with improved cardiovascular prognosis.

The aim of this multicenter trial, the CardioVascular Irbesartan Project (CVIP), was to compare the cardiac effects of antihypertensive therapy with irbesartan versus atenolol in subjects with essential hypertension. Of note, early intervention studies such as the Treatment Of Mild Hypertension Study recognized that changes in voltage criteria for LVH do not correlate well with changes of echocardiographic LVM.11 Therefore, we used both methods to assess the cardiac changes in response to antihypertensive therapy in this trial.

Methods
Design
This randomized, double-blind trial was conducted at 14 university-affiliated centers in Germany. After withdrawal of previous antihypertensive therapy, subjects were randomized 1:1 to receive either 150-mg irbesartan or 50-mg atenolol once daily. After 4 weeks doses were doubled if blood pressure exceeded 150 mm Hg systolic or 90 mm Hg diastolic. Thereafter, hydrochlorothiazide, 12.5 mg and 25 mg, respectively, and amlodipine, 5 mg and 10 mg, respectively, could be added at 4-week intervals.

This trial was conducted according to the Declaration of Helsinki (South Africa, 1996). Informed written consent was obtained from subjects before participation in the trial. The protocol of the trial was approved by each local University Investigation Ethics Committee.

Objectives
The primary objective of the trial was to compare the cardiovascular effects of irbesartan versus atenolol after 6 months and 18 months of double-blind treatment. The secondary objective was to compare changes of echocardiographic versus electrocardiographic parameters.
Trial Population
Males and females between 25 and 65 years of age with a systolic blood pressure (SBP) of 150 to 200 mm Hg or a diastolic blood pressure (DBP) of 95 to 115 mm Hg and mild target organ damage defined as intima media thickness of the common carotid artery on the leading side ≥0.8 mm and ≤1.5 mm were eligible.

Subjects were excluded for known or suspected secondary hypertension; coronary heart disease; cerebrovascular disease; peripheral vascular disease; renovascular disease; insulin-dependent diabetes mellitus; uncontrolled non–insulin-dependent diabetes mellitus; history of intolerance to atenolol, irbesartan, other angiotensin receptor blockers, hydrochlorothiazide, or amlopidine; and pretreatment with an angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker within the last 6 months.

Echocardiographic Measurements
Two-dimensional guided M-mode echocardiography was performed in each center using the same Sigma Iris 880 D Duplex-ultrasound system according to a prefixed protocol. All investigators had been trained and certified by the core center. At the core center, each subject’s videotapes were analyzed blinded to the treatment allocation and to the time point of measurement. ECG indices of LVH, Cornell index, Sokolow-Lyon index were determined as previously described.12 LVM was calculated according to the American Society of Echocardiography, corrected following the suggestions of Devereux and associates,13 and then indexed to body surface area (LVMI).

Electrocardiographic Measurements
Electrocardiograms were read at the core center by 1 single investigator blinded to the patients’ identity, treatment allocation, and timepoint of measurement. ECG indices of LVH, Cornell index, Cornell voltage×QRS duration product, Lewis index, and Sokolow-Lyon index were determined.14-17 All R-wave and S-wave measurements were performed to the nearest 0.1 mV. In addition, the presence of repolarization abnormalities or P sinistro atriale was recorded.2 ECGs with right or left bundle-branch blocks, Wolff-Parkinson-White syndrome, or evidence of prior myocardial infarction were excluded from the ECG analysis.

Statistics
All statistical analyses were carried out using SPSS software (release 10.0, SPSS Inc.). Normal distribution was tested by the Kolmogorov-Smirnov test. ANOVA for repeated measurements was used to compare the changes between treatment groups. The Bonferroni method was applied for subsequent tests. Within treatment groups, paired t tests were used to test changes after therapy. Dichotomous variables were compared by χ² tests, whereas changes were tested by the Wilcoxon test for paired comparisons. Two-tailed P<0.05 was considered statistically significant. Linear regression analysis was performed where indicated. Data are given as mean±SD.

Results
Baseline Characteristics
Of the 428 subjects enrolled in the run-in phase, 188 subjects were not eligible for randomization, mostly because intima media thickness criteria were not met. Thus, 240 hypertensive patients were randomized to receive double-blind therapy (119 irbesartan, 121 atenolol; Table 1). Thirteen subjects (11%) in the irbesartan group and 18 subjects (15%) in the atenolol group discontinued before completion of the 18-month trial duration.

SBP, DBP, and lipid parameters at baseline were similar in both treatment groups (Table 1). Baseline LVM was only moderately elevated, with no difference between the groups (Table 2). Given a cutoff value for LVM of 134 g/m² in men and 110 g/m² in women,13 32% of the men and 43% of the women had LVH by echocardiography. Indices of systolic and diastolic function did not differ between the 2 groups at baseline.

Voltage criteria for LVH were also similar in the 2 groups at baseline. Given a cut-off value of 2.8 mV in men and 2.2 mV in women for the Cornell index,17 5% of the men and 5% of the women had LVH by echocardiography. Indices of systolic and diastolic function did not differ between the 2 groups at baseline.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Irbesartan (n=119)</th>
<th>Atenolol (n=121)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI, g/m²</td>
<td>116.7±26.8</td>
<td>119.3±26.1</td>
<td>n.s.</td>
</tr>
<tr>
<td>PWT, mm</td>
<td>10.8±1.6</td>
<td>11.2±1.6</td>
<td>n.s.</td>
</tr>
<tr>
<td>IVST, mm</td>
<td>12.1±2.3</td>
<td>12.1±2.0</td>
<td>n.s.</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>49.8±5.3</td>
<td>49.9±6.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>RWT</td>
<td>0.44±0.07</td>
<td>0.45±0.08</td>
<td>n.s.</td>
</tr>
<tr>
<td>EF, %</td>
<td>66.3±11.0</td>
<td>66.4±7.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>FFS, %</td>
<td>37.6±9.8</td>
<td>37.1±5.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>Mid-FFS</td>
<td>15.6±3.0</td>
<td>15.7±2.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>A/E ratio</td>
<td>0.99±0.30</td>
<td>1.02±0.51</td>
<td>n.s.</td>
</tr>
<tr>
<td>Sokolow index, mV</td>
<td>2.01±0.68</td>
<td>2.06±0.72</td>
<td>n.s.</td>
</tr>
<tr>
<td>Lewis index, mV</td>
<td>1.19±0.78</td>
<td>1.15±0.61</td>
<td>n.s.</td>
</tr>
<tr>
<td>Cornell index, mV</td>
<td>1.62±0.59</td>
<td>1.51±0.59</td>
<td>n.s.</td>
</tr>
<tr>
<td>QRS-duration, ms</td>
<td>90.19±12.42</td>
<td>89.68±11.71</td>
<td>n.s.</td>
</tr>
<tr>
<td>Cornell×QRS duration product, mV×ms</td>
<td>174±72</td>
<td>158±67</td>
<td>n.s.</td>
</tr>
<tr>
<td>Repolarization abnormalities, yes/no</td>
<td>32/76</td>
<td>42/67</td>
<td>n.s.</td>
</tr>
<tr>
<td>P sinistro atriale, yes/no</td>
<td>39/65</td>
<td>38/68</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

PWT indicates LV posterior wall thickness; IVST, interventricular septum thickness; LVEDD, LV end-diastolic diameter; RWT, relative wall thickness; Mid-FFS, midwall-fractional fiber shortening; n.s., not significant. All other abbreviations are defined in the text.

TABLE 1. Baseline Clinical Characteristics of the Study Population

TABLE 2. Baseline Values of Echo- and Electrocardiographic Parameters
SBP and DBP fell similarly in the 2 groups (after 6 months: 
$-13.9\pm21.6/6.6\pm12.5$ mm Hg in the irbesartan versus 
$-12.4\pm22.5/-6.3\pm15.0$ mm Hg in the atenolol group; 
n and after 18 months: $-14.2\pm21.3/-7.2\pm12.0$ mm Hg versus 
$-14.6\pm21.3/-8.3\pm12.3$ mm Hg; n.s.). Hydrochlorothiazide 
and amlodipine had to be added in similar frequency in the 
irbesartan and the atenolol groups for blood pressure control 
(hydrochlorothiazide 23% versus 27%, n.s., and amlodipine 
4% versus 6%, n.s.). After 6 and 18 months no significant 
change in LVMi could be detected in either of the 2 groups 
(Table 3 and Figure for % changes of LVMi after 18 months). 
We further analyzed the changes of LVMi in subjects within 
the highest quartile of baseline LVMi (LVMi >130 g/m²; 
n=59). Only in this subgroup, LVMi decreased after 18 
months of irbesartan treatment ($-8.4\pm19.8$, $P=0.05$, n=29) 
but not with atenolol treatment ($-3.3\pm23.4$, n.s., n=30). 
However, the difference in the change of LVMi between 
the groups did not reach statistical significance.

With regard to systolic function, a similar increase in 
ejection fraction (EF) and fractional fiber shortening (FFS) 
after 18 months could be observed in the irbesartan and 
atenolol groups. With respect to diastolic function, a decrease 
in the A/E-ratio was noted only in the atenolol group. 
Regression analysis disclosed that the change in heart rate 
was the strongest determinant of the change in A/E ratio 
($r=0.463$, $P<0.001$ after 6 months and $r=0.420$, $P<0.001$ 
after 18 months). In contrast, treatment group did not 
determine changes of A/E ratio.

Changes of Voltage Criteria for LVH After 
6 and 18 Months

After 6 months and 18 months voltage criteria for LVH (Lewis 
index, Cornell index and Cornell voltage×QRS duration product) 
decreased in the irbesartan group but not in the atenolol group 
(Table 4 and Figure for % changes after 18 months). Furthermore, 
Sokolow index decreased in subjects treated with irbesartan after 18 
months. The differences in the changes of Sokolow index and 
Cornell index after 18 months were significant between the 2 
groups ($P=0.006$ and $P=0.05$, respectively) and tended to be 
significant for the Lewis index and Cornell voltage×QRS duration 
product ($P=0.07$ and $P=0.077$, respectively). No change in repolarization 
abnormalities or in the presence of P sinistro atriale were 
detected in either of the 2 groups.

Discussion

In the CVIP, the effects of antihypertensive therapy with 
irbesartan versus atenolol on echocardiographic and 
electrocardiographic parameters of LVH were analyzed. Although 
with irbesartan no overall change of LVM was detected (only 
in the subgroup with initial clearly elevated LVM), consistent 
reductions of voltage criteria for LVH at 6 months and even

### Table 3. Change of Echocardiographic Parameters After 6 and 18 Months

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Irbesartan n=116</th>
<th>Atenolol n=121</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMi, g/m²</td>
<td>116.7±26.8</td>
<td>119.3±26.1</td>
<td></td>
</tr>
<tr>
<td>After 6 months</td>
<td>−1.2±17.9</td>
<td>+3.1±17.3</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+2.4±18.9</td>
<td>+0.2±24.1</td>
<td>n.s.</td>
</tr>
<tr>
<td>PWT, mm</td>
<td>10.8±1.6</td>
<td>11.2±1.6</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+0.21±1.57</td>
<td>−0.08±1.69</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+0.39±1.67</td>
<td>+0.20±1.61</td>
<td>n.s.</td>
</tr>
<tr>
<td>IVST, mm</td>
<td>12.1±2.3</td>
<td>12.1±2.0</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>−0.15±2.07</td>
<td>+0.27±1.77</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+0.20±1.83</td>
<td>+0.08±2.08</td>
<td>n.s.</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>49.8±5.3</td>
<td>49.9±6.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>−0.36±4.43</td>
<td>+0.92±5.91</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>−0.56±3.80</td>
<td>+0.35±6.42</td>
<td>n.s.</td>
</tr>
<tr>
<td>RWT</td>
<td>0.44±0.07</td>
<td>0.45±0.08</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+0.01±0.09</td>
<td>−0.01±0.09</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+0.02±0.08</td>
<td>+0.01±0.09</td>
<td>n.s.</td>
</tr>
<tr>
<td>EF, %</td>
<td>66.3±11.0</td>
<td>66.4±7.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+1.7±10.2</td>
<td>+1.7±7.5*</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+3.3±9.2***</td>
<td>+3.1±9.2**</td>
<td>n.s.</td>
</tr>
<tr>
<td>FFS, %</td>
<td>37.6±9.8</td>
<td>37.1±5.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+0.8±9.7</td>
<td>+1.4±5.9*</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+2.4±6.9**</td>
<td>+3.4±11.4*</td>
<td>n.s.</td>
</tr>
<tr>
<td>Mid-FFS, %</td>
<td>15.6±3.0</td>
<td>15.7±2.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+0.3±3.3</td>
<td>+0.5±2.6*</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 18 months</td>
<td>+0.4±2.5</td>
<td>+0.6±2.9</td>
<td>n.s.</td>
</tr>
<tr>
<td>A/E ratio</td>
<td>0.99±0.30</td>
<td>1.02±0.51</td>
<td>n.s.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>+0.01±0.25</td>
<td>−0.11±0.36***</td>
<td>0.004</td>
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<tr>
<td>After 18 months</td>
<td>+0.05±0.3</td>
<td>−0.08±0.32*</td>
<td>0.005</td>
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</table>

PWT indicates LV posterior wall thickness; IVST, interventricular septum thickness; LVEDD, LV end-diastolic diameter; RWT, relative wall thickness; Mid-wall FFS, midwall-fractional fiber shortening; n.s., not significant. All other abbreviations are defined in the text.

* $P<0.05$; ** $P<0.01$; *** $P<0.001$. 

This table shows changes of echocardiographic parameters after 6 and 18 months in the irbesartan and atenolol groups.
more at 18 months were found in the group treated with irbesartan. In contrast, atenolol did not lead to reductions of LVM or voltage criteria for LVH.

### Effects of Irbesartan Versus Atenolol on Echocardiographic Parameters

Antihypertensive treatment did not affect echocardiographically determined LVM in our trial overall. Only in the subgroup of patients within the greatest quartile of baseline LVM did irbesartan, but not atenolol, significantly decrease LVM over time as expected from the Losartan Intervention For Endpoint reduction in hypertension (LIFE) trial.20

It is well known from a large number of previous echocardiographic studies that baseline LVM influences the degree of regression observed in response to any given antihypertensive treatment (ie, the greater the baseline LVM the greater the degree of regression observed).19 On average, our patients had only mildly elevated LVM. LVH as defined as a LVMi >134 g/m² in men and LVMi >110 g/m² in women13 was observed in 32% and 43%, respectively. Regression was seen only in those patients treated with irbesartan within the highest quartile of LVMi. Thus, the mild degree of LVH in the subjects of our trial may be contributed to the lack of an overall treatment effect on LVM.

### Effects of Irbesartan Versus Atenolol on Electrocardiographic Parameters

Irbesartan consistently decreased ECG indices of LVH over time. In contrast, atenolol did not lead to reductions in any of the ECG indices studied. Interestingly, the reductions achieved with irbesartan were mostly within commonly used normal limits. The validity of these reductions is underscored by the fact that the decreases of voltage criteria for LVH seemed to be duration-dependent (ie, a more marked reduction of voltage criteria for LVH after 18 months as compared with the reduction after 6 months was found). Secondly, the reductions were consistently noted for all 5 voltage criteria for LVH. However, whether these reductions within normal limits translate into a more favorable outcome in terms of cardiovascular mortality cannot be answered by our trial. Most previous studies on cardiovascular prognosis attributed to regression of LVH examined patients with greater baseline values of ECG indices. One previous report by Levy et al from the Framingham Heart Study, however, suggests that a reduction of ECG values even within normal limits as documented in our trial may be of clinical relevance.2

In that study, subjects were divided into voltage quartiles of the Cornell index (R wave aVL+S wave V₅). The ranges for the quartiles were Cornell voltages <1.3, 1.4 to 1.8, 1.9 to 2.4, and >2.4 mV. Decreases from a greater to a lower quartile during follow-up were associated with a 54% relative risk reduction after adjusting for age and baseline voltage quartile. These data from the Framingham Heart Study support the notion that ECG changes even if within the so-called normal range provide prognostic information.

### Disparate Treatment Effects of Angiotensin Receptor Blockade on LVM and on Voltage Criteria for LVH

The strong effects of irbesartan on voltage criteria for LVH in the absence of an overall effect on echocardiographic LVM is surprising. Because LVM determined by echocardiography closely reflects anatomic LVM, it seems that although the anatomic structure of the left ventricle has not been changed, the electric properties of the myocardium have been altered by irbesartan.

Published data from studies in people also support this notion. In the LIFE trial, a detailed analysis of ECG data has become available recently.19 Treatment with losartan led to a greater decrease of ECG indices of LVH than treatment with atenolol as early as the first follow-up examination after 6 months. Interestingly, this advantage of losartan persisted throughout the entire follow-up period of 5 years. In contrast to the marked differences in ECG indices, the advantage in the decrease of LVM with losartan compared with atenolol was numerically small, roughly 4 g/m² (losartan −21.7±21.8 versus atenolol −17.7±19.6 g/m²).20 Because of the large sample size in the LIFE trial, this small difference of 4 g/m² reached statistical significance (P=0.021). Therefore, similarly to our trial, the effect of losartan on voltage criteria for LVH seemed to be much more impressive than on LVM by echocardiography.

### Lack of Consistent Treatment Effects of Angiotensin Receptor Blockade on Echocardiographic Wall Thickness

Because the determination of LVM is strongly dependent on left ventricular end-diastolic diameter, a closer look on
changes of wall thickness per se may provide further insight. The changes of wall thickness in the LIFE study have not been published yet. In other studies comparing the effect of an angiotensin receptor blocker versus a β-blocker on left ventricular structure, however, changes of wall thickness have been published. In a study by Malmqvist et al., irbesartan led to a greater decrease of LVM than atenolol. Interestingly, posterior wall thickness decreased similarly in subjects treated with irbesartan and in subjects treated with atenolol. Furthermore, there was only a tendency to a greater decrease of septal wall thickness in subjects treated with irbesartan ($P=0.054$). Thus, the significantly greater decrease of LVM observed in the irbesartan group versus the atenolol group (26 g/m$^2$ versus 14 g/m$^2$) was partly based on the increase of left ventricular end-diastolic diameter in the atenolol group (+1.2±0.4 mm), whereas left ventricular end-diastolic diameter did not change in the irbesartan group (+0.2±0.5 mm). In another trial by Thürmann et al., LVM was reduced to a greater extent by valsartan than by atenolol but posterior wall thickness and septal wall thickness also decreased similarly in the valsartan and atenolol groups. In the current trial, we did not find any significant effect of irbesartan on posterior wall thickness, septal wall thickness, or left ventricular end-diastolic diameter. Therefore, in response to treatment with angiotensin receptor blockers, a reduction of wall thickness is not a consistent finding.

**Pathophysiological Basis for LVM by Echocardiography and Voltage Criteria for LVH Is Different**

It should be kept in mind that although both methods, echocardiography and ECG, are used for the same purpose, namely to detect LVH, the physiological basis of these 2 methods is quite different. Whereas electrical voltage produced by cardiac myocytes and detected by the ECG reflect electrical properties of the myocardium, cardiac dimensions as detected by echocardiography closely reflect anatomical LVM. Thus, the changes in these parameters do not necessarily have to go in parallel. Specific effects of irbesartan on electrical properties of the myocardium that are not accompanied by changes of cardiac structure may be an underlying pathogenetic mechanism.

Indeed, recent data in humans indicate that angiotensin receptor blockers exert specific antiarrhythmic effects. After electrical conversion of atrial fibrillation, the addition of irbesartan to amiodarone is associated with a lower frequency of recurrence of atrial fibrillation than amiodarone alone. Although the pathogenetic mechanism has not been fully elucidated yet, this effect may be mediated by modulation of ion channels by irbesartan.

**Conclusion**

Treatment of hypertension with irbesartan resulted in significant reductions in voltage criteria for LVH, although an effect on LVM was only seen in the subjects with high baseline LVM. On the other hand, atenolol did not lead to reductions in electrocardiographic or echocardiographic parameters of LVH. The results of this trial therefore add to the evidence that the use of irbesartan is superior to the use of atenolol in the treatment of hypertensive subjects with LVH. Furthermore, because voltage criteria for LVH have been shown to predict cardiovascular outcome independently from LVM by echocardiography, we suggest that both methods should be used for a more accurate detection of the benefits of antihypertensive treatment for the heart.

**Perspectives**

For future studies, it remains to be determined whether the detection of left ventricular changes in response to antihypertensive therapy by the use both methods, ECG and echocardiography, in contrast to the use of 1 method alone will help to more accurately assess the reduction of cardiovascular risk associated with regression of LVH.

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