Background—Sudden cardiac death (SCD) occurs more often in patients with ECG left ventricular (LV) hypertrophy. However, whether LV hypertrophy regression is associated with a reduced risk of SCD remains unclear.

Methods and Results—The Losartan Intervention for End Point Reduction in Hypertension (LIFE) study included 9193 patients 55 to 80 years of age with essential hypertension and ECG LV hypertrophy by gender-adjusted Cornell product (CP) (RaVL + SV1 [+6 mm in women]) · QRS duration >2440 mm · ms) and/or Sokolow-Lyon voltage (SLV) (SV1 + RV5/6 >38 mm). During follow-up (mean, 4.8 years), 190 patients (2%) experienced SCD. In time-dependent Cox analyses, absence of in-treatment LV hypertrophy was associated with a decreased risk of SCD: every 1-SD-lower in-treatment CP (1050 mm · ms) was associated with a 28% lower risk of SCD (hazard ratio [HR], 0.72; 95% CI, 0.66 to 0.79) and 1-SD-lower SLV (10.5 mm) with a 26% lower risk (HR, 0.74; 95% CI, 0.65 to 0.84). After adjustment for time-varying systolic and diastolic blood pressures, treatment allocation, age, gender, baseline Framingham risk score, ECG strain, heart rate, urine albumin/creatinine ratio, smoking, diabetes, congestive heart failure, coronary heart disease, atrial fibrillation, and occurrence of myocardial infarction, atrial fibrillation, heart failure, and noncardiovascular death, both in-treatment CP and SLV remained predictive of SCD: each 1-SD-lower CP was associated with a 19% lower risk of SCD (HR, 0.81; 95% CI, 0.73 to 0.90) and 1-SD-lower SLV with an 18% lower risk (HR, 0.82; 95% CI, 0.70 to 0.98). Absence of in-treatment LV hypertrophy by both SLV and CP was associated with a 30% lower risk of SCD (HR, 0.70; 95% CI, 0.54 to 0.92).

Conclusions—Absence of in-treatment ECG LV hypertrophy is associated with reduced risk of SCD independently of treatment modality, blood pressure reduction, prevalent coronary heart disease, and other cardiovascular risk factors in hypertensive patients with LV hypertrophy.

Key Words: death, sudden ▪ electrocardiography ▪ hypertension ▪ hypertrophy ▪ mortality

Left ventricular (LV) hypertrophy detected by ECG has been shown to be associated with higher prevalences of premature ventricular contractions and complex ventricular arrhythmias in members of the general population,1 in a case-control series of hypertensive patients,2 and in never-treated hypertensive patients from a defined population.3 Studies have consistently shown that ECG LV hypertrophy is strongly associated with increased risk for sudden cardiac death (SCD).4-7 In a study from Cornell in patients with uncomplicated hypertension,8 the risk of fatal or nonfatal complications was increased 2- to 4-fold by the presence of LV hypertrophy independently of age, gender, and other risk factors. Furthermore, Schillaci and co-

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workers9 demonstrated that echocardiographic LV hypertrophy predicted complex ventricular arrhythmias in never-treated hypertensive subjects independently of age and high nocturnal blood pressure. The Framingham Heart Study reported that increased echocardiographic LV hypertrophy was associated with an increased risk of SCD in a community-based cohort.10 However, to the best of our knowledge, no previous studies have investigated whether a reduction in LV hypertrophy during antihypertensive treatment is associated with a lower risk of SCD. Hence, the present study was undertaken to evaluate whether regression of LV hypertrophy predicted lower risk of SCD.
Methods

Study Design and Results

The Losartan Intervention for End Point Reduction in Hypertension (LIFE) study was a prospective, randomized, double-blind, parallel-group study (n=9193) using the double-dummy technique that evaluated the long-term effects of losartan- and atenolol-based antihypertensive therapy in patients with hypertension and ECG LV hypertrophy. The main outcome\(^\text{11}\) and the complete study protocol, including study design, organization, clinical measures, exclusion criteria, basis for choice of comparative agents, statistical considerations, and baseline characteristics,\(^\text{12,13}\) have been published.

As previously described, patients 55 to 80 year of age with previously treated or untreated hypertension and ECG signs of LV hypertrophy\(^\text{13}\) were randomized to initial therapy with losartan or atenolol and treated to a target blood pressure of <140/90 mm Hg. All patients were followed up for ≥4 years (mean, 4.8 years).

Electrocardiography

ECGs were obtained at study baseline, at 6 months, and at yearly follow-up intervals until study termination or patient death. ECGs were interpreted at the Core Laboratory at Sahlgrenska University Hospital/Ostra (Gothenburg, Sweden) as previously reported in detail.\(^\text{12,14-16}\) The product of QRS duration times Cornell voltage (RaVL+/SV1, with 6 mm added in women\(^\text{14,17}\) >2440 mm · ms or Sokolow-Lyon voltage (SV1+RV5 or RV6) >38 mm\(^\text{18}\) was used to identify LV hypertrophy.\(^\text{15,16}\)

Clinical Classification of Death

Sudden unexpected death was defined as death that was sudden and unexpected, including observed arrhythmic deaths and those not attributable to myocardial infarction, intractable heart failure, or other identifiable cause. These deaths were classified as witnessed or unwitnessed and, if unwitnessed, by the time interval between death and the last time another individual saw the patient alive: <1, 1 to 24, or >24 hours. Patients with sudden loss of consciousness who were successfully resuscitated but ultimately died of sequelae such as pneumonia also were classified as SCD and formed the “>24 hours” group. If an autopsy was performed in a patient who died suddenly and evidence of a recent myocardial infarction was found, the death was classified as secondary to myocardial infarction.

Statistical Analysis

SPSS version 12.0 (SPSS Inc, Chicago, Ill) was used for statistical analysis. Results are given as mean±SD or frequencies expressed as percentages.

Differences between groups were analyzed with Student t test for continuous and the χ² test for categorical variables with log transformation, if needed, to satisfy normal distribution.

All end points were analyzed with the intention-to-treat approach; all randomized patients were included in their randomized treatment group, and all available follow-up data were included from randomization until study termination date. To determine whether in-treatment LV hypertrophy predicted SCD, hazard ratios (HRs) for in-treatment time-varying Sokolow-Lyon voltage and/or Cornell voltage-duration product were calculated using Cox models.\(^\text{19,20}\) In-treatment time-varying Sokolow-Lyon voltage and/or Cornell voltage-duration product predicted SCD, hazard ratios (HRs) for in-treatment time-varying Sokolow-Lyon voltage and/or Cornell voltage-duration product were calculated using Cox models.

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unadjusted (HR, 0.88; 95% CI, 0.66 to 1.17; \( P = 0.36 \)) or adjusted for differences in systolic and diastolic blood pressures and Framingham risk score (Figure 1).

Because patients who suffered SCD differed significantly from those who did not with respect to demographic and clinical variables that could affect outcome (Table 1), the independent relations of SCD to baseline Cornell product, and Sokolow-Lyon voltage, a 1-SD-higher Cornell product remained associated with a 27% higher risk of SCD (95% CI, 1.14 to 1.42; \( P < 0.001 \)); in a parallel analysis, 1-SD-higher baseline Sokolow-Lyon voltage was associated with an 8% higher risk of SCD (95% CI, 0.93 to 1.26), which did not remain predictive after controlling for the above-mentioned risk factors (\( P = 0.29 \)).

### In-Treatment LV Hypertrophy and Different Causes of Death

In-treatment reduction by 1-SD Cornell product significantly reduced risk of SCD, death resulting from heart failure, cardiovascular death after 24 hours, and death resulting from other causes but not noncardiovascular death (Table 3). Sokolow-Lyon voltage reduction by 1 SD was associated with a lower risk of death resulting from cardiac and noncardiac causes (Table 3).

### In-Treatment LV Hypertrophy and SCD

The relations of in-treatment Cornell product and Sokolow-Lyon voltage criteria for LV hypertrophy to incident SCD are examined in Table 4 and Figures 2 and 3. In Cox analyses, controlling for treatment effects, less in-treatment ECG LV hypertrophy by Sokolow-Lyon voltage and Cornell voltage-duration product was associated with a decreased risk of SCD. Lower in-treatment Cornell voltage-duration product by 1 SD was associated with a 28% lower risk of SCD (Table 3). Similarly, lower Sokolow-Lyon voltage by 1 SD was associated with a 26% lower risk of SCD. When both ECG measures of LV hypertrophy were included simultaneously in the same Cox model, 1-SD-lower values of both Cornell voltage and Sokolow-Lyon voltage were associated with a 43% lower risk of SCD (95% CI, 28 to 54; \( P < 0.001 \)). With adjustment for risk factors, lower time-varying Cornell voltage-duration product and Sokolow-Lyon voltage each remained predictive of a lower risk of SCD by 19% (\( P < 0.001 \)) and 18% (\( P < 0.05 \)), respectively (Table 4). With adjustment for the above-mentioned risk factors, 1-SD-lower values of both Cornell voltage-duration product and Sokolow-Lyon voltage were associated with a 30% lower risk of SCD.

Modified Kaplan-Meier curves comparing incident SCD according to 4 different levels of Cornell product LV hypertrophy (Figure 2) or Sokolow-Lyon voltage (Figure 3) on ECGs over the time course of the study demonstrated that lower in-treatment LV hypertrophy was associated with a lower risk of developing SCD compared with higher in-treatment ECG LV hypertrophy. In-treatment Cornell product <2000 compared with values >3000 was associated with an estimated 66% lower risk of SCD after 4 years of follow-up (Figure 2). Hazard curves for patients in varying strata of Cornell product or Sokolow-Lyon voltage over the course of the study further illustrate that stratification of SCD risk by ECG Cornell product and Sokolow-Lyon voltage was not dependent on use of the standard 2440 mm · ms and 38 mm thresholds for LV hypertrophy, respectively (Figures 2 and 3); higher in-treatment levels of Cornell product and Sokolow-Lyon voltage across the spectrum of observed values were associated with step-wise greater risks of SCD.

The association between in-treatment Cornell product and SCD was similar in men and women; in black and nonblack patients; in patients >67 and <67 years of age; among patients with and without diabetes; in those with a history of ischemic heart disease, cerebral vascular disease, atrial fibrillation, or albuminuria; in smokers and nonsmokers; and among patients with and without LV hypertrophy or ECG strain at baseline, with nonsignificant interaction terms for these variables (\( P = 0.10 \)), but did differ significantly between

### TABLE 2. Change in Blood Pressure and ECG LV Hypertrophy in Patients With and Without SCD

<table>
<thead>
<tr>
<th>End Point</th>
<th>No SCD</th>
<th>SCD</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>9003</td>
<td>190</td>
</tr>
<tr>
<td>Change in systolic blood pressure, mm Hg</td>
<td>-28.0 (19.1)</td>
<td>-25.5 (21.7)</td>
</tr>
<tr>
<td>Change in diastolic blood pressure, mm Hg</td>
<td>-15.9 (10.1)</td>
<td>-13.5 (11.7)</td>
</tr>
<tr>
<td>Change in Cornell voltage-duration product, mm · ms</td>
<td>-216 (826)</td>
<td>-63 (1040)</td>
</tr>
<tr>
<td>Change in Sokolow-Lyon, mm</td>
<td>-4.0 (7.1)</td>
<td>-2.9 (7.5)*</td>
</tr>
</tbody>
</table>

Data are mean (SD).

*\( P < 0.05 \), †\( P < 0.01 \), groups with vs without SCD.

### TABLE 3. Single-Variable Cox Regression Analyses to Assess the Predictive Value of In-Treatment Cornell Voltage Duration Product and Sokolow-Lyon Voltage for the Development of SCD and Other Causes of Death

<table>
<thead>
<tr>
<th>End Point</th>
<th>Cornell Voltage-Duration Product*</th>
<th>Sokolow-Lyon Voltage*</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD</td>
<td>0.72 (0.66 to 0.79)</td>
<td>0.74 (0.65 to 0.84)</td>
</tr>
<tr>
<td>Cardiovascular death after 24 h</td>
<td>0.78 (0.67 to 0.92)</td>
<td>0.82 (0.65 to 1.03)</td>
</tr>
<tr>
<td>Death caused by heart failure</td>
<td>0.71 (0.57 to 0.87)</td>
<td>0.54 (0.41 to 0.71)</td>
</tr>
<tr>
<td>Death caused by stroke</td>
<td>0.87 (0.74 to 1.02)</td>
<td>0.70 (0.59 to 0.84)</td>
</tr>
<tr>
<td>Other cardiovascular death</td>
<td>0.80 (0.66 to 0.97)</td>
<td>0.66 (0.52 to 0.84)</td>
</tr>
<tr>
<td>Noncardiovascular death</td>
<td>0.98 (0.89 to 1.07)</td>
<td>0.78 (0.71 to 0.86)</td>
</tr>
</tbody>
</table>

Data are expressed as HR (95% CI).

*Time varying.
patients with and without prior myocardial infarction ($P=0.04$; data not shown). The association between intreatment Sokolow-Lyon voltage and SCD was similar in patients $>67$ and $<67$ years of age; among patients with and without diabetes or a history of ischemic heart disease; in those with and without prior myocardial infarction, cerebral vascular disease, or atrial fibrillation; in patients with albuminuria; in smokers and nonsmokers; and among patients with and without LV hypertrophy or ECG strain at baseline, with nonsignificant interaction terms for these variables ($P>0.18$). The association did differ significantly between men and women ($P<0.05$) and between black and nonblack patients ($P=0.03$, data not shown).

### Discussion

The present study is, to the best of our knowledge, the first in treated hypertensive patients with ECG LV hypertrophy to evaluate the association of LV hypertrophy regression with SCD. Not surprisingly, patients who experience SCD are older and have more comorbid disease and higher ECG measures of LV hypertrophy at baseline. Of interest was the fact that losartan seemed to protect similarly against SCD compared with atenolol, a pharmacological treatment generally thought to reduce SCD by its presumed antiarrhythmic properties and demonstrated value in primary and secondary prophylaxis in acute myocardial infarction, known to cause SCD. Baseline measures of ECG LV hypertrophy were independent of other known risk factors and markers associated with SCD, confirming smaller previous studies.\(^4\)\(^-\)\(^6\)

Most important, our analysis of the effects of in-treatment ECG LV hypertrophy suggests that LV mass regression reduces the risk of SCD. The finding that less ECG LV hypertrophy during antihypertensive therapy is associated with a lower likelihood of SCD independently of blood pressure lowering and other known predictors of SCD supports the value of serial measurement of ECG LV hypertrophy criteria for assessing SCD risk in hypertensive patients and suggests that antihypertensive therapy targeted at regression or prevention of ECG LV hypertrophy may reduce the risk of SCD.

One further observation suggests that a reduction in time-varying Cornell voltage-duration product is robust in detecting the risk of death from cardiac causes, including SCD but not stroke, whereas a reduction in time-varying Sokolow-Lyon voltage seems to predict risk of death resulting from heart failure, other cardiovascular death (including peripheral

<p>| TABLE 4. Single-Variable and Multivariable Cox Regression Analyses to Assess the Predictive Value of In-Treatment Cornell Voltage-Duration Product, Sokolow-Lyon Voltage, or Both for the Development of SCD |
|--------------------------------------------------|--------|-------|</p>
<table>
<thead>
<tr>
<th></th>
<th>HR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivariable model 1*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time-varying Cornell voltage-duration product (1050 mm \cdot ms decrease)</td>
<td>0.81</td>
<td>0.73 to 0.90</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Multivariable model 2*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time-varying Sokolow-Lyon voltage (10.5 mm decrease)</td>
<td>0.82</td>
<td>0.70 to 0.98</td>
<td>0.027</td>
</tr>
<tr>
<td>Multivariable model 3*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time-varying Cornell product (1050 mm \cdot ms) and Sokolow-Lyon voltage (10.5 mm decrease)</td>
<td>0.70</td>
<td>0.54 to 0.92</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Adjusted for time-varying systolic and diastolic blood pressures, treatment allocation, age, gender, baseline Framingham risk score, ECG strain, heart rate, and urine albumin creatinine ratio; prevalence of smoking, diabetes, congestive heart failure, coronary heart disease, and atrial fibrillation; and occurrence of myocardial infarction, atrial fibrillation, heart failure, and noncardiovascular death.
mass reduction with losartan compared with atenolol.16,20

This hypothesis because we have previously shown better LV oxygen and nutrients and hence diminished “demand-side” reduction in myocardial mass requiring delivery of oxygen and nutrients23 caused by a frank hypertrophy but is graded throughout the whole spectrum of ECG LV mass. Whether this is due to a simultaneous reduction in myocardial oxygen consumption caused by a reduction in the myocardial mass requiring delivery of oxygen and nutrients and hence diminished “demand-side” ischemia remains unknown. Our finding that losartan is at least as effective in preventing SCD as atenolol could support the hypothesis that the reductions in risk for SCD associated with lower in-treatment Cornell voltage-duration product and Sokolow-Lyon voltage for SCD (Figures 2 and 3) suggest that the association of lower risk of SCD with less LV hypertrophy not only occurs at partition values for ECG LV hypertrophy according to gender and race. These differences may well be reflective of the recruitment effects of the respective ECG criteria in different populations, in addition to a play of chance.22

Of note, the predictive value of in-treatment Cornell voltage-duration product and Sokolow-Lyon voltage for SCD was robust across most subsets of the population. However, there were significant differences in the association between in-treatment ECG LV hypertrophy observed for the Cornell product between patients with versus without myocardial infarction and for Sokolow-Lyon voltage between patients grouped according to gender and race. These differences may well be reflective of the recruitment effects of the respective ECG criteria in different populations, in addition to a play of chance.22

Of further note, our stratified analyses of the severity of in-treatment LV hypertrophy in relation to SCD (Figures 2 and 3) suggest that the association of lower risk of SCD with less LV hypertrophy not only occurs at partition values for frank hypertrophy but is graded throughout the whole spectrum of ECG LV mass. Whether this is due to a simultaneous reduction in myocardial oxygen consumption caused by a reduction in the myocardial mass requiring delivery of oxygen and nutrients and hence diminished “demand-side” ischemia remains unknown. Our finding that losartan is at least as effective in preventing SCD as atenolol could support this hypothesis because we have previously shown better LV mass reduction with losartan compared with atenolol.16,20

However, we have previously reported that LV mass reduction to the normal range per se reduces incident myocardial infarction independently of the blood pressure reduction and thereby could reduce triggering SCD by undiagnosed myocardial infarcts in hypertensive individuals.

**Study Limitations**

The present study has several limitations. The Cornell product and/or Sokolow-Lyon voltage ECG criteria used to select patients for LIFE increased the baseline risk of the study population, whereas exclusion of patients with active heart failure, known ejection fraction <40%, or recent myocardial infarction may have reduced it; consequently, the present findings may not be representative of hypertensive populations with less or more severe disease. Second, the statistical phenomenon of regression to the mean may affect the present findings, particularly in light of the use of values of Cornell product and/or Sokolow-Lyon voltage above threshold levels to select patients for LIFE, despite our attempt to minimize this problem by using separate screening and baseline ECGs. As a consequence of this selection process and the intrinsic variability of ECG measurements, it is likely that both the degree of ECG LV hypertrophy at baseline and the subsequent decrease in ECG LV hypertrophy during therapy were overestimated in some patients. However, decreased risk of SCD was associated with lower ECG indexes of LV hypertrophy on multiple recordings (up to 7 ECG recordings per patient) throughout the study, which would not be affected by regression to the mean.

Furthermore, correlated causation is possible (ie, that some uncontrolled factor related to cardiac health results in both better outcomes and a reduction in LV mass). Despite advanced statistics, the design is correlational in nature because the LIFE study was not designed to manipulate ECG measures of LV hypertrophy to different targets.

**Therapeutic Implications**

Our findings have important therapeutic implications. The fact that SCD may be reduced by reducing LV mass indicates that reversing LV structural remodeling might be important as a treatment goal, in addition to electric stabilization and heart rate reduction for prevention of SCD.

**Conclusions**

LV hypertrophy at baseline predicted SCD independently of other cardiovascular risk factors and remained predictive in the subpopulation without signs of atherosclerosis. Less in-treatment...
LV hypertrophy strongly predicted lower risk of SCD independently of multiple risk factors. Furthermore, losartan versus atenolol treatment appeared to benefit SCD similarly.

Acknowledgment
We are indebted to Sigrid Helle Berg for her dedicated work with the LIFE study.

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Disclosures
Dr Wachtell, Okin, Olsen, Dahlöf, Devereux, Ibsen, Kjeldsen, and Lindholm receive occasional speaker honoraria from Merck & Co, Inc. The other authors report no conflicts.

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