Echocardiographic and Electrocardiographic Diagnoses of Left Ventricular Hypertrophy Predict Mortality Independently of Each Other in a Population of Elderly Men

Johan Sundström, MD, PhD; Lars Lind, MD, PhD; Johan Ärnlöv, MD; Björn Zethelius, MD; Bertil Andrén, MD, PhD; Hans O. Lithell, MD, PhD

Background—The increased risk associated with left ventricular hypertrophy (LVH) diagnosed echocardiographically (Echo-LVH) or electrocardiographically (ECG-LVH) is well known, but the clinically relevant question of how much additional prognostic information would be provided by echocardiographically assessing LVH if a subject’s ECG-LVH and hypertension status are known has not been addressed.

Methods and Results—We investigated whether Echo-LVH and ECG-LVH predicted total and cardiovascular mortality and morbidity independently of each other and of other cardiovascular risk factors by using a population-based sample of 475 men investigated at age 70 with a median follow-up time of 5.2 years. Echocardiographic left ventricular mass index (LVMI) predicted total mortality (hazards ratio [HR] 1.44, 95% CI 1.09 to 1.92, for a 1-SD increase in LVMI) and cardiovascular mortality (HR 2.38, 95% CI 1.52 to 3.73) independently of ECG-LVH and other cardiovascular risk factors. ECG-LVH, defined as Cornell product $>244 \text{mV} \cdot \text{s}$, predicted total mortality (HR 2.89, 95% CI 1.41 to 5.96) independently of LVMI and other cardiovascular risk factors. Thus, Echo-LVH and ECG-LVH provided complementary prognostic information, especially in hypertensive subjects.

Conclusions—Echo-LVH and ECG-LVH predict mortality independently of each other and of other cardiovascular risk factors, implying that Echo-LVH and ECG-LVH in part carry different prognostic information. Therefore, to fully assess the increased risk associated with these conditions, both ECG and echocardiography should be performed. (*Circulation*. 2001;103:2346-2351.)

Key Words: hypertrophy ■ diagnosis ■ mortality ■ insulin ■ risk factors

Left ventricular hypertrophy (LVH) imposes a great cardiovascular risk whether it is diagnosed by ECG1–4 or by echocardiography.5–7 However, both these methods for detecting LVH have setbacks. The ECG is rather insensitive in detecting anatomic LVH, but the increased risk associated with electrocardiographically diagnosed LVH (ECG-LVH) has nevertheless been thought to reflect an increased left ventricular mass. Echocardiography is a more expensive and less available method, and it is also sometimes difficult to distinguish physiological from pathological LVH and thereby to grade cardiovascular risk. To our knowledge, no attempt has previously been made to investigate whether subjects with echocardiographically diagnosed LVH (Echo-LVH) who later suffer morbid events or die also have ECG-LVH, or vice versa, or whether the methods provide complementary prognostic information.

Echo-LVH and an increased left ventricular relative wall thickness are related to the insulin resistance syndrome of potent cardiovascular risk factors, including hypertension.8–10 It is not fully known whether the increased cardiovascular risk associated with Echo-LVH or ECG-LVH is independent of the associated metabolic disturbances and hypertension.

The aim of the present study was to investigate, by use of a large cohort of elderly men from the general population, whether Echo-LVH and ECG-LVH predict total and cardiovascular mortality and morbidity independently of each other and of the insulin resistance syndrome. We also investigated how much additional prognostic information was provided by an echocardiographic examination if the subject’s ECG-LVH and hypertension status were known.

Methods

Subjects
From 1970 to 1973, all men born from 1920 to 1924 and residing in the county of Uppsala were invited to a health survey aimed at identifying risk factors for cardiovascular disease. The cohort was
Committee of Uppsala University. Procedures followed were in
written informed consent, and the study was approved by the Ethics

Diastolic blood pressure (45%) were hypertensive (systolic blood pressure
(International Classification of Diseases [ICD]-9 codes 410 to 414)
subjects had been hospitalized because of ischemic heart disease
was defined by using the Swedish national cause-of-death and hospital
6.4 years), contributing to 2415.6 person-years. End points were
The subjects had a median follow-up time of 5.2 years (range 0.7 to
reinvestigated 20 years later, at 70 years of age (the baseline of the
present study, Table 1), which has been extensively described previously. The population of the present study consisted of 475 of
150 g/m², according to data from the Framingham
clamp technique, performed according to the method of DeFronzo et
al.16 with a slight modification: insulin was infused at a constant rate
of 56 mU/(min · m²). The insulin sensitivity index was calculated by
dividing glucose disposal, calculated as milligrams glucose infused/

TABLE 1. Baseline Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Subjects</th>
<th>Subjects With Echo-LVH</th>
<th>Subjects With Cornell Product &gt;244</th>
<th>µV · s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echo-LVH, n/N (%)</td>
<td>134/475 (28.2)</td>
<td>. . .</td>
<td>38/80 (47.5)*</td>
<td></td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>136.4±28.9</td>
<td>172.0±21.6*</td>
<td>151.6±31.4*</td>
<td></td>
</tr>
<tr>
<td>Sokolow-Lyon voltage ≥3.5 mV, n/N (%)</td>
<td>78/475 (16.4)</td>
<td>36/134 (26.9)*</td>
<td>39/80 (48.8)*</td>
<td></td>
</tr>
<tr>
<td>Cornell voltage &gt;2.8 mV, n/N (%)</td>
<td>55/475 (11.6)</td>
<td>23/134 (17.2)†</td>
<td>54/80 (67.5)*</td>
<td></td>
</tr>
<tr>
<td>Cornell product &gt;244 µV · s, n/N (%)</td>
<td>89/475 (16.8)</td>
<td>38/134 (28.4)*</td>
<td>. . .</td>
<td></td>
</tr>
<tr>
<td>Left ventricular strain, n/N (%)</td>
<td>45/404 (11.1)</td>
<td>21/101 (20.8)*</td>
<td>11/33 (33.3)*</td>
<td></td>
</tr>
<tr>
<td>Insulin sensitivity index, mg · min⁻¹ · kg⁻¹/(100 mU/L)</td>
<td>5.2±2.4</td>
<td>5.1±2.6</td>
<td>4.9±2.4</td>
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</tr>
<tr>
<td>Proinsulin, pmol/L</td>
<td>8.0±8.9</td>
<td>9.4±13.1†</td>
<td>10.2±9.5†</td>
<td></td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>3.87±0.87</td>
<td>3.85±0.82</td>
<td>3.84±0.88</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.29±0.35</td>
<td>1.24±0.35†</td>
<td>1.29±0.34</td>
<td></td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.42±0.80</td>
<td>1.56±0.79†</td>
<td>1.49±0.83</td>
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</tr>
<tr>
<td>Waist circumference, cm</td>
<td>94.7±9.2</td>
<td>95.5±9.8</td>
<td>94.1±9.9</td>
<td></td>
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<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>149±19</td>
<td>155±20*</td>
<td>153±20†</td>
<td></td>
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<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>85±9</td>
<td>87±10†</td>
<td>86±10</td>
<td></td>
</tr>
<tr>
<td>Hypertension (≥160/95 mm Hg or treatment), n/N (%)</td>
<td>212/475 (44.6)</td>
<td>87/134 (64.9)*</td>
<td>45/80 (56.3)†</td>
<td></td>
</tr>
<tr>
<td>Smoking, n/N (%)</td>
<td>93/475 (19.6)</td>
<td>31/134 (23.1)</td>
<td>18/80 (22.5)</td>
<td></td>
</tr>
<tr>
<td>Previous ischemic heart disease, n/N (%)</td>
<td>54/475 (11.4)</td>
<td>21/134 (15.7)</td>
<td>14/80 (17.5)</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean±SD or numbers (percentages).
*P<0.001 and †P<0.05 vs subjects without Echo-LVH and Cornell product >244 µV · s, respectively.

Follow-Up
The subjects had a median follow-up time of 5.2 years (range 0.7 to 6.4 years), contributing to 2415.6 person-years. End points were defined by using the Swedish national cause-of-death and hospital discharge registers. During follow-up, 44 subjects died (rate 1.82/100 PYAR); 18 deaths were from cardiovascular disease (ICD codes 390 to 459, rate 0.75/100 PYAR), with 7 deaths from acute myocardial infarction and 4 deaths from stroke. Morbidity was defined as first hospitalization or death from cardiovascular disease or any cause and was evaluated only for subjects who had not previously been hospitalized for cardiovascular disease or any cause, respectively. During follow-up, 48 (39%) of 122 subjects (rate 9.30/100 PYAR) had a morbid event of any cause, and 64 (19%) of 338 subjects (rate 4.09/100 PYAR) had a cardiovascular morbid event.

Electrocardiography
A standard 12-lead ECG was recorded at 50 mm/s and 10 mm/mV. Because different LVH criteria measure different cardiac properties,13 we analyzed several criteria: Sokolow-Lyon voltage, Cornell voltage, and left ventricular strain, which have previously been validated in prospective studies,12,14 and the Cornell product, which has not been previously validated in a prospective study but has been used as inclusion criterion in the Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) study.15 LVH was defined as a Sokolow-Lyon voltage amplitude of (SV1+RV5 or RV6) ≥3.5 mV, a Cornell voltage of (SV1+RaVL) ≥2.8 mV, or a Cornell product of (SV1+RaVL)×QRS duration >244 µV · s. Left ventricular strain was defined as a downsloping ST-segment depression >0.1 mV with T-wave flattening or inversion in leads V5 to V6. Sensitivitiespecificities (%) for detection of echocardiographic LVH were 27/88 for Sokolow-Lyon voltage ≥3.5 mV, 17/91 for Cornell voltage ≥2.8 mV, 28/88 for Cornell product ≥244 µV · s, and 21/92 for left ventricular strain, in the present population.

Echocardiography
Comprehensive 2D and Doppler echocardiography was performed as described previously.14 Left ventricular dimensions (intraventricular septal thickness [IVS], posterior wall thickness [PW], and left ventricular end-diastolic diameter [LVEDD]) were measured at end of diastole with M-mode by using the leading-edge-to-leading-edge convention. Left ventricular mass was determined by using the Troy formula according to the recommendations of the American Society of Echocardiography (ASE):15 left ventricular mass (g) = 1.05[(LVEDD+ IVS+PW)³−LVEDD³]. Left ventricular mass was divided with body surface area to obtain the left ventricular mass index (LVMI). LVH was defined as LVMI ≥150 g/m², according to data from the Framingham Heart Study.13 Examinations and readings of the images were performed by one experienced physician (B.A.) who was unaware of the other data of the subjects. A reproducibility study was made in 22 subjects ~1 month after the original investigations. The intraindividual coefficients of variation were 8.8% for IVS, 6.7% for PW, 3.5% for LVEDD, and 12.5% for LVMI.

Other Cardiovascular Risk Factors
These analyses have been described in detail previously.9 Insulin sensitivity was determined with the hyperinsulinemic euglycemic clamp technique, performed according to the method of DeFronzo et al.16 with a slight modification: insulin was infused at a constant rate of 56 mU/(min · m²). The insulin sensitivity index was calculated by dividing glucose disposal, calculated as milligrams glucose infused/
by the mean plasma insulin concentration \( \times 100 \) (mU/L) during the last 60 minutes of the 2-hour clamp. Blood samples for fasting concentrations were drawn in the morning after an overnight fast. Proinsulin concentrations were measured with a specific 2-site immunoradiometric assay technique.\(^{17}\) Cholesterol and triglyceride concentrations in serum and HDL were assayed by enzymatic techniques, and LDL cholesterol was calculated by using Friedewald's formula. The coding of smoking was based on interview reports. Supine systolic and diastolic blood pressures were measured twice in the right arm after 10 minutes of rest, and the means were calculated.

**Statistical Analysis**

Variables with a skewed distribution (triglycerides and proinsulin) were logarithmically transformed to achieve normal distribution, and these transformed variables were used in all analyses. Two-tailed 95% CIs and significance values were given, with a value of \( P < 0.05 \) regarded as significant. The prognostic value of transfer from one level of a dichotomous variable to another, or a 1-SD increase in a continuous variable, was investigated with Cox proportional hazards ratios (HRs). The proportionality of hazards was confirmed with Kaplan-Meier plots. Adjustments were made for either LVMI, previous ischemic heart disease, or 9 cardiovascular risk factors (clamp insulin sensitivity index, proinsulin, LDL and HDL cholesterol, triglycerides, waist circumference, smoking, hypertension, and previous ischemic heart disease) in multiple Cox proportional hazards analyses. There was no adjustment for age because all subjects were of the same age at baseline, with a narrow age span.

Cutoff levels for LVMI other than 150 g/m\(^2\) were sought by using histograms of quartiles of LVMI for cardiovascular and total mortality and by performing logistic regression and receiver operating characteristic (ROC) curves. Stata 6.0 software (Stata Corp) was used.

**Results**

**Predictive Value of Echo-LVH**

Echo-LVH (as a dichotomous variable) was a significant (HR 1.91 and 3.97) predictor of cardiovascular morbidity and mortality (see Table 2 for details). Also, LVMI as a continuous variable was a significant predictor of morbidity and mortality from cardiovascular disease and total mortality (HR 1.51 to 2.37 for a 1-SD increase, Table 2). Because these results indicated that some predictive information was lost when dichotomizing LVMI, cutoff levels other than the proposed 150 g/m\(^2\) level\(^{15}\) were sought by using quartiles of LVMI and logistic regressions and ROC curves, with total and cardiovascular mortality as dependent variables. Total mortality seemed to increase rather linearly with increasing LVMI (Figure 1), and no obvious cutoff level for prediction of total mortality was found. In contrast, the risk for cardiovascular mortality was markedly increased in only the fourth quartile of LVMI (LVMI 154 to 250 g/m\(^2\), Figure 1). This information combined with the ROC curve for LVMI regarding cardiovascular mortality (Figure 2) indicated that 150 g/m\(^2\) was the cutoff level that provided a reasonable combination of sensitivity and specificity for detection of this increased risk.

Nine cardiovascular risk factors were also evaluated as risk factors for mortality and morbidity. Smoking (HR 3.38, 95% CI 1.33 to 8.59) or a 1-SD increase in proinsulin (HR 1.94, 95% CI 1.36 to 2.77) increased the risk, and a 1-SD increase in HDL cholesterol (HR 0.48, 95% CI 0.26 to 0.88) decreased the risk of mortality from cardiovascular disease. Smoking predicted total mortality (HR 2.00, 95% CI 1.09 to 3.66).
When adjusting for these 9 cardiovascular risk factors, the predictive value of LVMI as a continuous variable for total and cardiovascular mortality remained significant, whereas the impact of dichotomized Echo-LVH on later cardiovascular mortality decreased in significance (Table 2).

### Predictive Value of Electrocardiographic-LVH

LVH that was defined as Sokolow-Lyon voltage \( \geq 3.5 \) mV was a significant predictor of total mortality (HR 2.00; see Table 2 for details). LVH that was defined as Cornell voltage \( \geq 2.8 \) mV was a significant predictor of total mortality (HR 2.89), even after adjustment for LVMI. LVH that was defined as Cornell product \( \geq 244 \) mV \( \cdot \) s was a strong (HR 3.56 and 3.82) predictor of both cardiovascular and total mortality, even after adjustment for 9 other cardiovascular risk factors. Adjustment for LVMI made the prediction of cardiovascular mortality, but not total mortality, decrease in significance. The left ventricular strain pattern predicted morbidity only from cardiovascular disease (HR 2.39).

### Comparison Between the Predictive Capacities of Echo-LVH and ECG-LVH

In multivariate Cox proportional hazards analyses with LVMI, Cornell product \( > 244 \) μV \( \cdot \) s, Sokolow-Lyon voltage \( \geq 3.5 \) mV, and 9 other cardiovascular risk factors as independent variables (Table 3), LVMI and Cornell product \( > 244 \) μV \( \cdot \) s were significant predictors of total mortality. LVMI, previous ischemic heart disease, HDL cholesterol, smoking, and proinsulin were significant predictors of cardiovascular mortality. No interaction terms between LVMI and the ECG-LVH criteria were significant predictors in the analyses.

By assessing Echo-LVH if ECG Cornell product \( > 244 \) μV \( \cdot \) s, another 7 (16%) of the 44 total deaths (rate 1.39/100 PYAR) (another 6 [33%] of the 18 cardiovascular deaths [rate 1.19/100 PYAR]) could be predicted (Figure 3). By assessing the ECG Cornell product if echocardiography showed no LVH, another 8 (18%) of the 44 total deaths (rate 4.03/100 PYAR) (another 2 [11%] of the 18 cardiovascular deaths [rate 1.01/100 PYAR]) could be predicted (Figure 3).

We also stratified for hypertension. In 263 normotensive subjects, no more total or cardiovascular deaths could be predicted by assessing Echo-LVH if ECG Cornell product \( \leq 244 \) μV \( \cdot \) s. In 212 hypertensive subjects, another 7 of the 19 total deaths (another 6 of the 12 cardiovascular deaths) could be predicted by assessing Echo-LVH if ECG Cornell product \( \leq 244 \) μV \( \cdot \) s.

### TABLE 2. Total and Cardiovascular Mortality and Morbidity in Relation to Echocardiographic and ECG Left Ventricular Measurements

| LVMI (1 SD) | Crude | 1.51 (1.17–1.94)* | 2.37 (1.70–3.31)‡ | 1.29 (0.97–1.72) | 1.31 (0.92–1.86) |
| LVMI | 1.60 (0.84–3.26) | 0.95 (0.30–2.96) | 1.50 (0.74–3.03) | 0.56–1.94 |
| LVMI | 2.12 (1.05–4.29) | 1.92 (0.57–6.39) | 1.04 (0.47–2.31) | 0.61–2.20 |
| LVMI | 2.89 (1.46–5.72) | 2.53 (0.83–7.69) | 1.33 (0.60–2.98) | 1.32 (0.65–2.66) |
| LVMI | 2.50 (1.26–4.99) | 1.83 (0.59–5.63) | 1.28 (0.57–2.85) | 1.15 (0.57–2.34) |
| LVMI | 2.53 (1.21–5.29)† | 1.99 (0.56–7.12) | 0.87 (0.36–2.10) | 1.26 (0.58–2.71) |

Values are Cox proportional HRs and 95% CIs, crude, adjusted for LVMI, or adjusted for 9 cardiovascular risk factors. *P < 0.01, †P < 0.05, and ‡P < 0.001 for equal hazards.
Discussion

In the present study, echocardiographic LVMI and the ECG-LVH criterion (Cornell product \( \geq 244 \mu V \cdot s \)) predicted mortality independently of each other and of other cardiovascular risk factors, which indicates that Echo-LVH and ECG-LVH were not identical conditions. It was also shown that many but not all of the subjects whose deaths were predicted by ECG-LVH were also identified by echocardiography, and vice versa.

The main new finding of the present study is that the previously known prognostic value of ECG-LVH to some extent is independent of echocardiographic LVMI, and vice versa.

The main new finding of the present study is that the previously known prognostic value of ECG-LVH to some extent is independent of echocardiographic LVMI, and vice versa. Therefore, we have assessed the clinically relevant question of how much additional prognostic information would be gained by referring a subject to an echocardiographic examination if the subject’s ECG-LVH and hypertension status are known. Our conclusion is that the additional prognostic value of an Echo-LVH assessment if ECG Cornell product \( \geq 244 \mu V \cdot s \) was low in normotensive subjects, whereas in hypertensive subjects or the population as a whole, echocardiography and ECG provided complementary prognostic information. Today, ECG is generally regarded as merely a less sensitive method than echocardiography for detecting anatomic LVH, but in view of the findings of the present study, the LVH information obtained with ECG should rather be regarded as of equal prognostic importance as Echo-LVH information. The implication for the clinician assessing the risk associated with LVH is that the decision of which patient to refer to echocardiography should be based on knowledge of ECG-LVH and hypertension status. A normotensive patient without ECG-LVH may well have Echo-LVH, but the latter information is of low prognostic importance and hardly motivates an echocardiographic examination.

Echo-LVH appeared to be slightly more associated with the prevalence of concomitant cardiovascular risk factors than ECG-LVH defined as Cornell product \( \geq 244 \mu V \cdot s \) (Table 1). This was also reflected in the observation that

![Figure 3. Bars are total (A) and cardiovascular (B) mortality rates in groups with or without LVH as defined by Cornell product \( \geq 244 \mu V \cdot s \) (CP+) or \( \leq 244 \mu V \cdot s \) (CP-) and echocardiographic LVMI \( \geq 150 \) g/m\(^2\) (Echo+) or \(< 150 \) g/m\(^2\) (Echo-) in all possible combinations (open square, CP+/Echo+; solid square, CP-/Echo+; open circle, CP+/Echo-; and solid circle, CP-/Echo+). Kaplan-Meier plots show percentages free from total (A) and cardiovascular (B) mortality in the same groups.](http://circ.ahajournals.org/)

### Table 3. Multivariate Analyses of Risk Factors for Total and Cardiovascular Mortality

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Any Cause</th>
<th>Cardiovascular Disease (ICD 390–459)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI (1 SD)</td>
<td>1.44, 1.09–1.91*</td>
<td>2.37, 1.52–3.71†</td>
</tr>
<tr>
<td>Cornell product ( &gt;244 \mu V \cdot s ) (0/1)</td>
<td>2.89, 1.40–5.95†</td>
<td>2.10, 0.67–6.02</td>
</tr>
<tr>
<td>Sokolow-Lyon voltage ( \geq 3.5 ) mV (0/1)</td>
<td>1.16, 0.53–2.54</td>
<td>1.16, 0.31–4.32</td>
</tr>
<tr>
<td>Insulin sensitivity index (1 SD)</td>
<td>0.69, 0.44–1.08</td>
<td>1.21, 0.56–2.59</td>
</tr>
<tr>
<td>Proinsulin (1 SD)</td>
<td>1.07, 0.74–1.55</td>
<td>2.31, 1.26–4.24†</td>
</tr>
<tr>
<td>LDL cholesterol (1 SD)</td>
<td>1.22, 0.88–1.67</td>
<td>1.20, 0.69–2.09</td>
</tr>
<tr>
<td>HDL cholesterol (1 SD)</td>
<td>0.77, 0.51–1.17</td>
<td>0.40, 0.19–0.87*</td>
</tr>
<tr>
<td>Triglycerides (1 SD)</td>
<td>0.74, 0.50–1.08</td>
<td>0.81, 0.45–1.47</td>
</tr>
<tr>
<td>Waist circumference (1 SD)</td>
<td>0.80, 0.55–1.17</td>
<td>0.62, 0.34–1.14</td>
</tr>
<tr>
<td>Hypertension (0/1)</td>
<td>0.72, 0.37–1.41</td>
<td>2.03, 0.63–6.58</td>
</tr>
<tr>
<td>Smoking (0/1)</td>
<td>1.74, 0.89–3.43</td>
<td>6.07, 1.95–18.88†</td>
</tr>
<tr>
<td>Previous ischemic heart disease (0/1)</td>
<td>1.57, 0.70–3.49</td>
<td>3.37, 1.06–10.78*</td>
</tr>
</tbody>
</table>

Values are Cox proportional HRs and 95% CIs or as indicated.

*P<0.05, †P<0.01, and ‡P<0.001 for equal hazards.
adjusting for cardiovascular risk factors made the impact of Echo-LVH, but not Cornell product $>244 \mu V \cdot s$, on later cardiovascular mortality decrease in significance (Table 2). The relationships between left ventricular geometry and cardiovascular risk factors in this population have been investigated previously.  

Total mortality seemed to increase linearly with increasing LVMI, but the risk of cardiovascular mortality was markedly increased only in the fourth quartile of LVMI. This finding may be due to the limited number of cardiovascular deaths but may also reflect true relationships. In 2 previous studies, the relationships between LVMI and coronary or cardiovascular morbidity were linear, but in 1 larger study, cardiovascular disease and all-cause mortality rates seemed substantially increased in the highest quartile of LVMI only. Defining LVH as LVMI $\geq 150$ g/m$^2$ (a cutoff level originally derived from a healthy, middle-aged sample of men living in Framingham) seems to limit the information carried in the continuous-variable LVMI regarding prediction of all-cause mortality but was relevant for the prediction of cardiovascular mortality and morbidity in the present population. Altogether, a cutoff level of 150 g/m$^2$ seems appropriate for LVMI measured with the leading-edge–to–leading-edge convention and the Troy formula, corresponding to 131 g/m$^2$ measured with the Penn convention and the modified cube formula. Left ventricular mass by both conventions correlates well with left ventricular mass determined at necropsy but slightly overestimates it (6% by the Penn convention and 25% by the ASE convention). Thus, LVMI measured with the leading-edge–to–leading-edge convention and the Troy formula can easily be transformed to reflect anatomic measurements; left ventricular mass = $0.80(\text{ASE mass}) + 0.6$ g. One of the strengths of the present study is that all subjects were the same age at baseline, which overcomes the problem of age differences between quartiles of LVMI found in other studies. The subjects are also of the same sex and ethnicity, which eliminates the need for adjustment for the influence of these important determinants of LVMI and other cardiovascular risk factors but limits the generalizability of the study to women and other ethnic and age groups. Other limitations of the present study include possible misclassification of end points, although the accuracy of the Swedish hospital discharge and cause-of-death registers has been shown to be close to 100% in this population.  

In conclusion, total and cardiovascular mortality risk increased with increasing echocardiographic LVMI, independent of other cardiovascular risk factors, and cardiovascular risk was fairly well assessed by dichotomized Echo-LVH. ECG-LVH also predicted total and cardiovascular mortality, especially the Cornell product criterion, which predicted total mortality independent of LVMI and other risk factors. Thus, Echo-LVH and ECG-LVH are not identical conditions, and to fully assess the considerable risk associated with either condition, both an ECG and an echocardiogram should be performed, especially in hypertensive subjects.  

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References

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