Impact of Different Partition Values on Prevalences of Left Ventricular Hypertrophy and Concentric Geometry in a Large Hypertensive Population

The LIFE Study


Abstract—Left ventricular (LV) hypertrophy and concentric remodeling have been defined by using a variety of indexation methods and partition values (PVs) for LV mass and relative wall thickness (RWT). The effects of these methods on the distribution of LV geometric patterns in hypertensive subjects remain unclear. Echocardiograms were obtained in 941 patients with stage I to III hypertension and LV hypertrophy by ECG. LV mass was calculated by using different methods of indexation for body size and different PVs to identify hypertrophy: LV mass/body surface area (g/m²) PV for men/women 116/104, 125/110, or 125/125; LV mass/height (g/m) PV 143/102 or 126/105; and LV mass/height 2.7 (g/m²) PV 51/51 or 49.2/46.7. RWT was calculated by either 2×end-diastolic posterior wall thickness (PWT)/end-diastolic LV internal dimension (LVID) or end-diastolic interventricular septum dimension/end-diastolic PWT/end-diastolic LVID. LV hypertrophy or remodeling was present in 63% to 86% of subjects, and LV hypertrophy was present in 42% to 77%. By any index, eccentric hypertrophy was the common LV geometric pattern. Use of interventricular septum dimension/PWT/LVID to calculate RWT slightly increased the prevalence of normal geometry and eccentric hypertrophy compared with the use of 2×PWT/LVID. Subjects with LV hypertrophy identified by only LV mass/height 2.7 PV 49.2/46.7 were more obese, whereas those identified by only LV mass/body surface area PV 116/104 were taller and thinner than those in the 2 concordant groups with or without LV hypertrophy by both criteria. By either criterion, there were no significant differences between different LV geometric patterns in clinical cardiovascular disease. Hypertensive patients with LV hypertrophy by ECG have a high prevalence of geometric abnormalities, especially eccentric hypertrophy, irrespective of method of indexation or PV, LV mass indexation by body surface area or height 2.7 identifies lean and obese subjects, respectively. We found no difference in prevalent cardiovascular disease in subjects identified by either criterion, suggesting a similar high risk. (Hypertension. 2000;35:6-12.)

Key Words: echocardiography ■ electrocardiography ■ hypertrophy, left ventricular ■ hypertension, essential

Left ventricular (LV) hypertrophy, as determined by echocardiography, has been shown to be a strong predictor of adverse prognosis independent of and, in most instances, stronger than conventional risk factors.1-3 On the basis of distributions of indexed echocardiographic LV mass in normal populations, LV hypertrophy has been identified by calculation of LV mass that has been indexed for body surface area (BSA)1.4,6 or for BSA1.5,7 height, height2.0,8 height2.13,9 height2.7,10 or height3.0.11 The combination of LV mass index (LVMI) and relative wall thickness (RWT) has been used to identify 3 different abnormal LV geometric patterns.2,12 RWT has been calculated either as the ratio of 2×posterior wall thickness/LV internal diameter13 or as the ratio of (interventricular septal+posterior wall thickness)/LV internal diameter.14

The relation between LVMI and RWT seems important in view of the fact that several studies have shown that stratification by different geometric patterns gives valuable information concerning morbidity and mortality. In these studies, subjects with concentric hypertrophy (ie, increased RWT and LVMI) had the highest incidence of cardiovascular events and death, those with eccentric hypertrophy or concentric remodeling had intermediate rates, and those with normal LV geometry had the least complications.2,6,12,15-18 However, studies have used a variety of partition values (PVs) for LV mass and RWT to identify LV hypertrophy and geometric

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remodeling. To date, no study has evaluated the effect of the different indexation methods and PVs for LV hypertrophy and concentric geometry on the distributions of abnormal geometric patterns in a large hypertensive population. The present study examines the impact of various methods of indexing LV mass and calculating RWT on the distributions and correlates of abnormal LV geometric patterns in a large series of subjects with stage I to III hypertension.19

Methods

Subjects

The present study evaluated 964 patients with stage I to III hypertension enrolled in the Losartan Intervention for Endpoint Reduction in Hypertension (LIFE) Study,20,21 constituting ~11% of the overall 9194 LIFE participants. In the LIFE echocardiography study, 2-dimensional and M-mode echocardiograms were obtained at baseline.22 The study was carried out in selected echocardiography centers in Denmark, Finland, Great Britain, Iceland, Norway, Sweden, and the United States. Before enrollment in the study, all subjects had a screening ECG that defined LV hypertrophy, a criterion for enrollment in the LIFE study, by either gender-adjusted Cornell voltage duration criteria, calculated as (SV, + RaVL (+ 6 mm for women))xQRS > 2440 mm m/s, or Sokolow-Lyon voltage criteria, calculated as SV, + RV5/RV6 > 38 mm.20 Patients with an aortic valve pressure gradient >20 mm Hg, symptomatic heart failure, or LV ejection fraction <40% were excluded from the study. The composite ECG criterion used for LIFE recruitment was based on results of previous studies in our laboratory23–25 anticipated to have ~94% sensitivity and 45% to 50% specificity. Pilot data suggested that anatomic LV hypertrophy would be present in ~18% to 22% of hypertensive patients free of the selective exclusion criteria for the LIFE study (lack of severe LV dysfunction, heart failure, or angina requiring therapy with angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, or ß-blockers, myocardial infarction or stroke in the past 6 months, or blood pressure that rose to >200 mm Hg systolic or 115 mm Hg diastolic during a run-in period of placebo treatment). Taken together, these estimates yielded projections indicating that from 62% to 78% of LIFE patients would have anatomic LV hypertrophy detectable by echocardiography.

After a run-in period that documented eligible levels of clinic arterial pressure during 14–19 days of placebo treatment, subjects underwent baseline evaluation. Arterial blood pressure was also measured by arm-cuff sphygmomanometer at the end of the echocardiographic examination, after subjects had been resting supine in a dimly lit room for ~30 minutes.

Echocardiographic Methods and Performance Protocol

Echocardiographic procedures for the present study were designed with regard to the special difficulties of performing objective skill-dependent cardiac tests in a multicenter trial and were based on previously used procedures.26–28 Standardized examinations included 2-dimensional guided M-mode echocardiograms and selected 2-dimensional and Doppler recordings. Study performance and interpretation focused on selected measures of LV mass and geometry, global and regional systolic LV function, and diastolic filling, maximizing the yield of reliable data to answer specific study questions.

Before initiation of the study, formal training sessions were held at The New York Hospital-Cornell Medical Center or Ullevål University Hospital, Oslo, Norway; these training sessions combined didactic teaching of selected relevant aspects of echocardiography, the specific protocol, and hands-on training in performing echocardiograms according to the study protocol. Studies were performed with high-quality commercially available echocardiograms equipped with 3.0- to 3.5-MHz and 2.0- to 2.5-MHz probes and VHS or Super-VHS video recorders. To facilitate performance of standard-ized quantifiable echocardiograms, examining tables with special cutouts were used. Recordings were made from the parasternal window by a standardized protocol to record at least 10 consecutive beats of 2-dimensional and M-mode recordings of the LV internal diameter and wall thicknesses at or just below the tips of the anterior mitral leaflet in both long-axis and short-axis views, with long-axis views of the mitral valve, color Doppler flow recordings to search for mitral and aortic regurgitation, and M-mode and 2-dimensional short-axis and long-axis views of the aortic valve and the left atrium. The apical acoustic window was used to record at least 10 cycles of 2- and 4-chamber images to assess LV wall motion and color flow or pulsed Doppler recording to identify mitral and aortic regurgitation.

Studies were sent to The New York Hospital-Cornell University Medical Center for blinded interpretation by experienced technicians and physicians.

Echocardiographic Measurements

Correct orientation of planes for imaging and Doppler recording was verified as previously described.26,28 Measurements were made blindly by using computerized review stations (Digisons, Inc) equipped with NTSC or PAL standard VCRs and digitizing tablet and monitor screen overlay for calibration and performance of measurements. LV internal dimension and interventricular septal and posterior wall thicknesses were measured at end diastole and end systole for up to 3 cycles by recommendation of the American Society of Echocardiography.29 When optimal orientation of the LV views could not be obtained, as is common in subjects who are overweight or over age 60, correctly oriented 2-dimensional linear dimension measurements were made by the leading-edge convention of the American Society of Echocardiography.30

Calculation of Derived Variables

End-diastolic LV dimensions were used to calculate LV mass by a formula shown to yield LV mass values closely related (r=0.90) to necropsy measurements.31 With excellent intertester reproducibility (r=0.95), in a hypertensive series of 183 hypertensive patients,32 LV mass indexation was performed by a variety of different methods and PVs for men and women: Values of LV mass/BSA were 116 g/m² for men and 104 g/m² for women,6,27 125 g/m² for men and 110 g/m² for women,33 131 g/m² for men and 100 g/m² for women,34 or 125 g/m² for men and women.24 Values of LV mass/height were 143 g/m for men and 102 g/m for women35 or 126 g/m for men and 105 g/m for women.10 Values of LV mass/height were 51 g/m² for men and women10 or 49.2 g/m² for men and 46.7 g/m² for women.10 BSA was calculated by using the Du Bois formula26:0.0071844 [Kg]/[(height [cm]) 0.725]. Overweight was identified by body mass index >27.8 kg/m² in men and 27.3 kg/m² in women.36 RWT was calculated as either 2x/posterior wall thickness in diastole/LV internal diameter (RWT)13 or as (interventricular septal+posterior wall thickness)/LV internal diameter (RWT)3.14 Increased RWT was present when this ratio of RWT exceeded 0.43, which represents the 97.5th percentile in normal subjects13 or when RWT2 exceeded 0.45,14 which represents the 96th percentile in our normal subjects.37 Disproportionate interventricular septal thickening was present if interventricular septal thickness/posterior wall thickness was ≥1.5.38 All normal values were checked in our database of an apparently normal population (n=362) from New York.17

Normal geometry was present when LVMI and RWT were normal, whereas normal LVMI and increased RWT identified concentric remodeling. Increased LVMI but normal RWT identified eccentric LV hypertrophy, and increases of both variables identified concentric LV hypertrophy.12

Statistics

Microsoft Access 97 (Microsoft Corp) and SPSS software version 8.0 (SPSS, Inc) were used for data management and statistical analysis. Data are presented as mean±SD or frequency in percent. Differences between 2 groups were assessed by unpaired Student t test; comparison among multiple groups was performed by ANOVA with the Scheffé post hoc test. Differences in prevalences between subgroups were compared by χ² statistics. Univariate relations
TABLE 1. Descriptive Data of the 941 LIFE Participants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Men/Women</th>
<th>Normal Geometry (1)</th>
<th>Concentric Remodeling (2)</th>
<th>Eccentric Hypertrophy (3)</th>
<th>Concentric Hypertrophy (4)</th>
<th>Abnormal Geometry (2+3+4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>66±7</td>
<td>19</td>
<td>11</td>
<td>47</td>
<td>24</td>
<td>82</td>
</tr>
<tr>
<td>Sex, % women</td>
<td>41</td>
<td>30</td>
<td>16</td>
<td>36</td>
<td>19</td>
<td>73</td>
</tr>
<tr>
<td>Height, cm</td>
<td>169±10</td>
<td>28</td>
<td>16</td>
<td>38</td>
<td>19</td>
<td>73</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>78.3±14</td>
<td>125/102</td>
<td>37</td>
<td>20</td>
<td>42</td>
<td>22</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>68±12</td>
<td>143/102</td>
<td>23</td>
<td>13</td>
<td>42</td>
<td>22</td>
</tr>
<tr>
<td>Clinic systolic blood pressure, mm Hg</td>
<td>173±14</td>
<td>131/100</td>
<td>28</td>
<td>16</td>
<td>38</td>
<td>19</td>
</tr>
<tr>
<td>Clinic diastolic blood pressure, mm Hg</td>
<td>98±9</td>
<td>125/102</td>
<td>37</td>
<td>20</td>
<td>28</td>
<td>14</td>
</tr>
<tr>
<td>End-echo diastolic blood pressure, mm Hg</td>
<td>95±12</td>
<td>143/102</td>
<td>23</td>
<td>13</td>
<td>42</td>
<td>22</td>
</tr>
<tr>
<td>End-echo systolic blood pressure, mm Hg</td>
<td>173±20</td>
<td>125/102</td>
<td>37</td>
<td>20</td>
<td>28</td>
<td>14</td>
</tr>
<tr>
<td>Overweight, %</td>
<td>42</td>
<td>28</td>
<td>16</td>
<td>38</td>
<td>19</td>
<td>73</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.3±4.6</td>
<td>1.89±0.19</td>
<td>8.0</td>
<td>8.8</td>
<td>5.5</td>
<td></td>
</tr>
<tr>
<td>BSA, m²</td>
<td>10.7</td>
<td>8.0</td>
<td>8.8</td>
<td>5.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>10.7</td>
<td>8.0</td>
<td>8.8</td>
<td>5.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular disease, %</td>
<td>8.0</td>
<td>8.8</td>
<td>5.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina pectoris, %</td>
<td>8.0</td>
<td>8.8</td>
<td>5.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior myocardial infarction, %</td>
<td>5.5</td>
<td>5.5</td>
<td>5.5</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean±SD or frequency (in percent).

between variables were assessed by Pearson correlation coefficients. Independent correlates of continuous measures of LV structure and function were identified by linear regression analysis using an enter procedure with assessment of collinearity diagnostics. A 2-tailed value of \( P<0.05 \) was considered statistically significant.

Results

Patient Characteristics

Descriptive data of the whole LIFE population have been reported elsewhere.\(^{20,21}\) Of the 964 patients in the LIFE echocardiography study, 941 had the necessary LV measurements, and thus LV geometric pattern, to be included in the present study. Characteristics of the 941 subjects are reported in Table 1.

LV Measurements

Mean systolic endocardial function was in the normal range with fractional shortening of 33±6%; LV ejection fraction was 61±8%. Compared with data from a large previously studied population of apparently normal adults,\(^{37}\) LIFE subjects had greater interventricular septal thickness (1.16±0.13 versus 0.89±0.12 cm), posterior wall thickness (1.07±0.16 versus 0.82±0.12 cm), LV end-diastolic dimension (5.29±0.57 versus 4.89±0.45 cm), end-systolic dimension (3.55±0.62 versus 3.08±0.38 cm), LV mass (234.7±54.1 versus 146.1±38.1 g), LV mass/BSA (124.0±26.7 versus 78.6±15.9 g/m²), LV mass/height\(^{2.7}\) (56.6±13.8 versus 34.5±7.2 g/m²), and RWT (0.41±0.06 versus 0.35±0.07) (all \( P<0.001 \)). Disproportionate interventricular septal thickening was an uncommon finding, occurring in 0.2% of LIFE subjects and in none of the apparently normal adults (\( P=NS \)).

LV Geometric Patterns

Overall, the prevalence of echocardiographic LV hypertrophy by our primary gender-specific PVs of 104 g/m² in women and 116 g/m² in men was 71%, within the range of 62% to 78% to be expected from previous studies comparing ECG and anatomic evidence of LV hypertrophy. Distributions of abnormal geometric patterns are presented in Table 2 with the use of RWT\(_1\) assessment and Table 3 with the use of RWT\(_2\) assessment. Some form of abnormal geometry was present in 63% to 86% of LIFE subjects when RWT\(_1\) was used (Table 2), depending on LV mass PV criteria. Slightly lower prevalences (61% to 84%) were found when RWT\(_2\) and the same LV mass PVs were used (Table 3).

Depending on the method of LV mass indexation and the PV used, LV hypertrophy was present in 42% to 77% of subjects. With use of RWT\(_1\) (Table 2), the most common geometric abnormality was eccentric hypertrophy (28% to 51%), with concentric hypertrophy present in 19% to 26% and concentric remodeling present in 8% to 20%. For individual PVs using RWT\(_1\), concentric LV hypertrophy was the second most common geometric abnormality with use of an LV mass/BSA PV of 116/104 g/m², an LV mass/height PV of 126/105 g/m, and gender-specific criteria for LV mass/height.\(^{2.7}\) When other PVs were used to detect LV hypertrophy, normal LV geometry and, in one instance, concentric LV...
remodeling were more common than concentric hypertrophy. Comparative data from our reference population revealed that 5% to 8% had abnormal LV geometry with use of various LV mass indexation methods and PVs together with RWT. LV mass/BSA PV of 125/125 g/m² gave the least separation, and LV mass/height PV of 126/105 g/m gave the highest separation of abnormal geometry prevalence between normal adults and LIFE subjects (Table 2). Classification of LV geometry in subjects with RWT calculated as interventricular septal + posterior wall thickness)/LV internal dimension produced slightly lower prevalences of abnormal LV geometric patterns (Table 3).

Comparison of LV geometric patterns by gender (Table 4) produced nearly identical distributions of LV geometric patterns in women and men for LV mass/BSA PVs of 116/104 and 125/110 g/m² and LV mass/height² PV of 51 g/m². LV mass/BSA PV of 131/100 g/m², LV mass/height PV of 143/102 g/m, and LV mass/height² PV of 49.2/46.7 g/m² produced significantly higher prevalences of eccentric and concentric hypertrophy and lower prevalences of normal LV geometry and concentric remodeling in women than in men.

Patients were cross-classified as having LV hypertrophy by use of gender-specific criteria for either LV mass/BSA (≤116 g/m²) or LV mass/height² (≤51 g/m²), by both or by neither, to identify 2 discordant groups (Table 5). LV mass was highest in the group with LV hypertrophy by both sets of criteria, lowest in the group without hypertrophy by either criterion, and intermediate in the groups with LV hypertrophy by only 1 criterion. Subjects included only by LV mass/BSA had significantly higher systolic and diastolic blood pressure than did those included only by LV mass/height². Patients with LV hypertrophy by LV mass/height² were significantly overweight, whereas patients included only by LV mass/BSA had, on average, ideal body weight.

**Discussion**

Although several studies have proposed limits of LV mass and RWT based on confidence intervals derived from reference populations, to our knowledge, no study has evaluated the impact of using different PVs for both these variables on the prevalence of LV geometric patterns in hypertensive patients. Performance of the present analyses in the context of the LIFE trial provided a population in which, as predicted by calculation based on the known performance of the ECG criteria used to screen individuals for LIFE, ≤70% had LV hypertrophy.

### TABLE 3. Distribution of LV Geometric Patterns Using Different Modes of LVMI and PVs With RWT Calculated as (Interventricular Septum + Posterior Wall Thickness)/LV Internal Diameter (RWT₁)

<table>
<thead>
<tr>
<th>Variable (RWT₁)</th>
<th>Men/Women</th>
<th>Normal Geometry (1)</th>
<th>Concentric Remodeling (2)</th>
<th>Eccentric Hypertrophy (3)</th>
<th>Concentric Hypertrophy (4)</th>
<th>Abnormal Geometry (2 + 3 + 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass/BSA 116/104</td>
<td>21/8</td>
<td>47/24</td>
<td>79/47</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 125/110</td>
<td>32/14</td>
<td>36/18</td>
<td>68/36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 131/100</td>
<td>30/14</td>
<td>38/18</td>
<td>70/38</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 125/125</td>
<td>40/18</td>
<td>28/15</td>
<td>61/28</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/height 143/102</td>
<td>25/11</td>
<td>43/21</td>
<td>75/43</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/height 126/105</td>
<td>17/6</td>
<td>51/27</td>
<td>84/51</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/height² 51/51</td>
<td>21/7</td>
<td>49/25</td>
<td>81/49</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass/height² 49.2/46.7</td>
<td>19</td>
<td>7</td>
<td>81</td>
<td>25</td>
<td>49</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 4. Distribution of LV Geometric Patterns by Percentage in Men and Women With Different Modes of LVMI and PVs

<table>
<thead>
<tr>
<th>Variable (RWT₉)</th>
<th>Men/Women</th>
<th>Normal Geometry</th>
<th>Concentric Remodeling</th>
<th>Eccentric Hypertrophy</th>
<th>Concentric Hypertrophy</th>
<th>Abnormal Geometry</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass/BSA 116/104</td>
<td>21/17</td>
<td>10/12</td>
<td>47/46</td>
<td>22/26</td>
<td>79/84</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 125/110</td>
<td>32/26</td>
<td>16/16</td>
<td>36/37</td>
<td>16/22</td>
<td>68/75</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 131/100</td>
<td>40/11</td>
<td>21/8</td>
<td>36/37</td>
<td>16/22</td>
<td>60/89</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>LV mass/BSA 125/125</td>
<td>32/45</td>
<td>16/25</td>
<td>35/18</td>
<td>17/12</td>
<td>68/55</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>LV mass/height 143/102</td>
<td>34/8</td>
<td>18/7</td>
<td>33/55</td>
<td>14/30</td>
<td>65/92</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>LV mass/height 126/105</td>
<td>12/10</td>
<td>9/8</td>
<td>50/53</td>
<td>23/29</td>
<td>82/90</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>LV mass/height² 51/51</td>
<td>25/22</td>
<td>12/14</td>
<td>43/41</td>
<td>20/23</td>
<td>75/78</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>LV mass/height² 49.47</td>
<td>21/12</td>
<td>8/8</td>
<td>47/51</td>
<td>24/29</td>
<td>79/88</td>
<td>&lt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

*Gender differences by ANOVA among the 4 geometric patterns.
hypertrophy by our primary LV mass/BSA criteria and the remainder had LV mass in the normal range.

Echocardiographic measurements of LV wall thickness and mass have facilitated research on the range and determinants of LV mass in normal individuals and on the prognostic implications of LV hypertrophy. Different methods have been used to calculate and index LV mass and to calculate RWT, but only a few studies have systematically assessed the impact of the use of different methods. The present study, in a large population of patients with moderate to severe hypertension as manifested by elevated blood pressure levels, ECG evidence of target organ involvement, and the prevalence of clinical cardiovascular disease reveals wide ranges of the prevalence of LV hypertrophy, ranging from 15% to 40% depending on the choice of criteria (Tables 2 and 3). One important factor in causing this variability, noted in a previous study, is the reciprocal relation between sensitivity and specificity that is shown in Table 2. All approaches to detection of abnormal LV geometry (ie, concentric remodeling and eccentric or concentric hypertrophy) had negative predictive values between 92% and 95% in apparently normal adults and positive predictive values of 63% to 86% in LIFE patients. However, even with criteria that had the highest negative predictive values for LV hypertrophy, the large majority of LIFE patients had LV hypertrophy or concentric remodeling. In addition, eccentric LV hypertrophy was consistently the most common geometric abnormality. In combination with any method of LV mass indexation and the use of any PV to recognize LV hypertrophy, use of RWT caused a shift in the prevalence of LV geometric patterns from concentric remodeling and hypertrophy to normal geometry and eccentric hypertrophy (Table 3). This occurred because the RWT method used a higher PV (0.45 versus 0.43). In addition, the prevalence of abnormal geometry was, on average, slightly lower using RWT than RWT.

One notable result of the present study is that some but not other criteria for abnormal LV geometry resulted in unequal distribution of geometric patterns between men and women. The indices that gave the most similar distribution of LV geometric patterns in men and women were LV mass/BSA PV of 116/104 or 125/110 g/m² and LV mass/height² PV of 143/102 g/m², the first and last of which had been chosen a priori for main analyses of the LIFE echocardiography study. Other criteria, including LV mass/BSA PV of 131/100 g/m² and LV mass/height PV of 143/102 g/m gave substantially higher prevalences of LV hypertrophy in women. A higher prevalence of LV hypertrophy in women by these PVs had already been reported by Levy et al in the Framingham population. Only 1 criterion (LV mass/BSA PV of 125/125 g/m²) resulted in higher prevalence of LV geometric abnormality in men. The finding of concentric LV remodeling in 10% of LIFE patients indicates that an appreciable proportion of hypertensive patients selected on the basis of the ECG features and other criteria used for enrollment in the present study have an LV geometric pattern that constitutes a "false-positive" diagnosis with respect to LV hypertrophy but is itself associated with an adverse prognosis.

By examining the concordance and discordance between the presence of LV hypertrophy by gender-specific criteria based on indexation of LV mass for BSA or height, we found discordant groups, constituting 10% of LIFE patients, that had LV hypertrophy by only 1 criterion. One notable result was that those in whom hypertrophy was identified by the LV mass/height² criterion were significantly more obese.
and had moderate hypertension, whereas those identified only by LV mass/BSA were of approximately ideal body weight and more severely hypertensive than those in the concordant groups with or without LV hypertrophy by both criteria. We found no significant difference in associated abnormalities, including serum creatinine levels, prevalence of diabetes, angina, previous myocardial infarction or cerebral stroke, or the presence of peripheral arterial disease, suggesting no great difference in clinical outcome among these groups. Although the present study will not have results concerning the impact of LV indexatation and PVs on the prediction of morbidity and mortality until the end of the LIFE trial,20,21 the prospective design and large number of subjects in the LIFE echocardiography study will, in due time, give us valuable information concerning clinical outcome.

In conclusion, some form of LV geometric remodeling was present in 62% to 82% of patients, with eccentric LV hypertrophy as the most common geometric abnormality with the use of any method of LV mass indexation and PV (28% to 51%). The mode of calculation of RWT did not substantially affect results. Patients with hypertrophy by only LV mass/height2 PV of 49.2/46.7 g/m² were more obese, whereas those identified by only LV mass/BSA with PV of 116/104 g/m² were less obese and more hypertensive than those in the 2 concordant groups with or without LV hypertrophy by both criteria.

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Impact of Different Partition Values on Prevalences of Left Ventricular Hypertrophy and Concentric Geometry in a Large Hypertensive Population: The LIFE Study
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