Left Ventricular Hypertrophy, Subclinical Atherosclerosis, and Inflammation

Sameer K. Mehta, J. Eduardo Rame, Amit Khera, Sabina A. Murphy, Russell M. Canham, Ronald M. Peshock, James A. de Lemos, Mark H. Drazner

Abstract—To elucidate mechanisms by which left ventricular (LV) hypertrophy (LVH) increases the risk of atherosclerotic heart disease, we sought to determine whether LVH is independently associated with coronary artery calcium (CAC) and serum C-reactive protein (CRP) levels in the general population. The Dallas Heart Study is a population-based sample in which 2633 individuals underwent cardiac MRI to measure LV structure, electron beam CT to measure CAC, and measurement of plasma CRP. We used univariate and multivariable analyses to determine whether LV mass and markers of concentric LV hypertrophy or dilation were associated with CAC and CRP. Increasing quartiles of LV mass indexed to fat-free mass, LV wall thickness, and concentricity, but not LV volume, were associated with CAC in both men and women (P<0.001). After adjustment for traditional cardiovascular risk factors and statin use, LV wall thickness and concentricity remained associated with CAC in linear regression (P<0.001 for each). These associations were particularly robust in blacks. LV wall thickness and concentricity were also associated with elevated CRP levels (P=0.001 for both) in gender-stratified univariate analyses, although these associations did not persist in multivariable analysis. In conclusion, concentric LVH is an independent risk factor for subclinical atherosclerosis. LVH is also associated with an inflammatory state as reflected in elevated CRP levels, although this relationship appears to be mediated by comorbid conditions. These data likely explain in part why individuals with LVH are at increased risk for myocardial infarction. (Hypertension. 2007;49:1385-1391.)

Key Words: left ventricular hypertrophy • atherosclerosis • inflammation • myocardial infarction • coronary artery disease

Left ventricular (LV) hypertrophy (LVH), whether determined by the ECG1–9 or echocardiogram,6–8.10–18 has been associated with various adverse cardiovascular outcomes, including mortality, myocardial infarction, and heart failure. Although there has been considerable speculation as to why LVH is such an important marker of risk,19,20 the basic mechanisms that predispose patients with LVH to develop atherosclerotic heart disease (ASHD) are not known. Previous hypotheses have included abnormalities in the coronary vasculature21–25 or platelets,26 increased blood viscosity,27 and a prothrombotic state.28 In addition to these factors, which may contribute to reduced myocardial oxygen supply, myocardial oxygen demand is also increased in patients with LVH.20 Another simple explanation for the association of LVH and ASHD is that LVH reflects target organ damage from concomitant risk factors, such as hypertension, thus providing a noninvasive barometer of the extent of ASHD. The association of LVH with atherosclerosis in other vascular territories, for example, the carotid artery,29 supports this hypothesis. There are scant data as to whether LVH is associated with the burden of coronary atherosclerosis as estimated by coronary artery calcium (CAC).30–32 Yet another emerging hypothesis is that LVH itself is a low-level inflammatory state, as has been suggested recently from animal33 and human studies.34,35 If this is the case, one could postulate that the proinflammatory state associated with LVH may increase the risk of ASHD and incident myocardial infarction.

The Dallas Heart Study is a large, ethnically diverse, population-based sample of Dallas County, Tex, in which subjects underwent cardiac MRI and coronary electron beam CT (EBCT) and had serum C-reactive protein (CRP) measured, affording an ideal opportunity to determine the mechanisms by which LVH increases the risk of ASHD. In this study we tested the following 2 hypotheses: LVH is independently associated with extent of subclinical atherosclerosis as measured by CAC, and LVH is independently associated with an elevation in CRP. Measures of LVH included elevated LV mass, wall thickness, and concentricity (the ratio of LV mass:volume).

Methods

Study Population

The Dallas Heart Study is a multiethnic, population-based, probability-based sample of 6101 residents of Dallas County designed to...
study cardiovascular disease. A description of the study design, sample description, and variable definitions has been described previously.36 The initial visit for all 6101 participants included an interview for demographic and health-related data, as well as 5 blood pressure measurements. Subjects between the ages of 30 and 65 years were invited to participate in 2 additional visits, which included blood and urine sampling, as well as imaging procedures. Of these, a total of 2744 participants underwent cardiac MRI and EBCT, as measurement of CRP from stored plasma specimens. Participants with a previous history of myocardial infarction were excluded from the present analysis, leaving 2633 individuals in the final study cohort.

**Blood Pressure**

Five sets of blood pressure measurements were obtained using an automatic oscillometric device (Welch Allyn, series 52 000) with an appropriately sized blood pressure cuff at each of 3 visits. This device has been validated against catheter measurement of arterial pressure.37 The blood pressure was considered the average of measurements 3 through 5 at each visit (total 9 readings: n=2596; total 6 readings: n=36; total 3 readings: n=1).

**EBCT Protocol and CAC Classification**

Gated EBCT image acquisition was performed using an Imatron C-150 XP EBCT scanner. Details of image acquisition and scanner properties have been described previously.38 A focus was defined as a region of ≥3 continuous voxels with a computed tomography number >130. The voxel size was 0.586×0.586×3 mm, so that 3 voxels would be a volume of 3.08 mm. Scans were read blindly by a single individual at 2 different settings, and only foci within the coronary arteries were scored. Results were reported in Agatston units,39,40 and the mean of the 2 scores was used as the final CAC score. CAC-positive individuals were defined as individuals with a mean CAC score ≥10 Agatston units.

**Cardiac MRI**

Multiple breath-hold electrocardiographic-gated cine magnetic resonance images were obtained from 2 similar 1.5-T MRI systems. The details of this protocol have been described previously.8,41 Mean LV wall thickness was determined via short axis slices. The wall thickness was computed for each slice excluding the apical slice and then averaged to determine the mean LV wall thickness. Concentricity was defined as the ratio of LV mass/LV end-diastolic volume.

**TABLE 1. Baseline Characteristics of the Study Subjects Stratified by Gender-Specific Quartiles of Concentricity**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Quartile of LV Concentricity</th>
<th>Quartile 1</th>
<th>Quartile 2</th>
<th>Quartile 3</th>
<th>Quartile 4</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td></td>
<td>42</td>
<td>43</td>
<td>45</td>
<td>49</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>35 to 48</td>
<td>35 to 49</td>
<td>37 to 52</td>
<td>42 to 56</td>
<td></td>
</tr>
<tr>
<td>Race, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Non-Hispanic black</td>
<td></td>
<td>35</td>
<td>44</td>
<td>52</td>
<td>64</td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic white</td>
<td></td>
<td>43</td>
<td>34</td>
<td>29</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td></td>
<td>19</td>
<td>19</td>
<td>17</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td></td>
<td>28</td>
<td>29</td>
<td>30</td>
<td>31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>23 to 30</td>
<td>24 to 32</td>
<td>25 to 34</td>
<td>26 to 35</td>
<td></td>
</tr>
<tr>
<td>Smoker, %</td>
<td></td>
<td>24</td>
<td>27</td>
<td>27</td>
<td>33</td>
<td>0.003</td>
</tr>
<tr>
<td>Family history of MI, %</td>
<td></td>
<td>27</td>
<td>30</td>
<td>32</td>
<td>38</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td></td>
<td>5</td>
<td>6</td>
<td>11</td>
<td>21</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td></td>
<td>15</td>
<td>17</td>
<td>28</td>
<td>55</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>On statin, %</td>
<td></td>
<td>4</td>
<td>4</td>
<td>7</td>
<td>9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td></td>
<td>119</td>
<td>122</td>
<td>126</td>
<td>138</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>111 to 126</td>
<td>113 to 129</td>
<td>117 to 134</td>
<td>124 to 149</td>
<td></td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td></td>
<td>75</td>
<td>77</td>
<td>79</td>
<td>84</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>70 to 79</td>
<td>72 to 82</td>
<td>74 to 84</td>
<td>78 to 90</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td></td>
<td>175</td>
<td>180</td>
<td>182</td>
<td>187</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>148 to 197</td>
<td>157 to 202</td>
<td>156 to 204</td>
<td>159 to 211</td>
<td></td>
</tr>
<tr>
<td>LDL cholesterol, mg/dL</td>
<td></td>
<td>101</td>
<td>106</td>
<td>108</td>
<td>113</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>79 to 121</td>
<td>82 to 127</td>
<td>86 to 129</td>
<td>85 to 135</td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td></td>
<td>53</td>
<td>50</td>
<td>50</td>
<td>49</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>42 to 60</td>
<td>40 to 58</td>
<td>40 to 57</td>
<td>40 to 58</td>
<td></td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td></td>
<td>113</td>
<td>108</td>
<td>114</td>
<td>102</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>63 to 127</td>
<td>67 to 139</td>
<td>69 to 150</td>
<td>72 to 164</td>
<td></td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td></td>
<td>53.4</td>
<td>54.7</td>
<td>55.7</td>
<td>57.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>43.6 to 62.1</td>
<td>45.5 to 63.0</td>
<td>46.7 to 63.8</td>
<td>47.2 to 65.7</td>
<td></td>
</tr>
<tr>
<td>Fat mass, kg</td>
<td></td>
<td>24.6</td>
<td>25.9</td>
<td>28.1</td>
<td>29.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>16.5 to 29.8</td>
<td>17.7 to 32.1</td>
<td>19.4 to 34.4</td>
<td>21.3 to 36.6</td>
<td></td>
</tr>
</tbody>
</table>

IQ indicates interquartile range; MI, myocardial infarction; LDL, low-density lipoprotein; HDL, high-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure. Quartiles of LV concentricity (g/mL) were 0.8 to 1.5, 1.5 to 1.69, 1.69 to 1.89, and ≥1.89 (men) and 0.7 to 1.31, 1.31 to 1.48, 1.48 to 1.69, and ≥1.69 (women).
High-Sensitivity CRP Assay

Blood samples were obtained after an overnight fast in ethylenediamine tetra-acetic acid, centrifuged at 1000g for 15 minutes at 4°C and stored at −80°C before the assay was performed. CRP measurements were performed on thawed samples using the Roche/Hitachi 912 System, Tina-quant assay, as described previously. The minimal detectable range of this assay is 0.1 mg/L, and the upper limit is 20 mg/L.

Statistical Analysis

Categorical data are reported as proportions and continuous data as median values and interquartile ranges. Baseline demographic variables, including coronary artery disease risk factors, were compared among gender-specific quartiles of LV concentricity. The χ² test was used for categorical variables and the Wilcoxon rank sum test for continuous variables, because many were nonparametric. Univariate analyses between variables and CAC were performed by analyzing

the log of CAC+1 to transform CAC to a continuous variable, as well as by analyzing CAC as a categorical variable (presence or absence of CAC). A test for trend across ordered groups was performed for the association between quartiles of LV structure and CAC and CRP. Multivariable logistic regression models were constructed in which prevalent CAC was the dependent variable. Linear regression models were constructed using log CAC+1 as the dependent variable. In addition to a measure of LV structure, these models included adjustment for traditional risk factors for the development of coronary artery disease, including diabetes, hypertension, systolic blood pressure, smoking, cholesterol levels, and age, as well as fat mass, fat-free mass, the use of statin medications, and the use of oral estrogens. These analyses were repeated in the following subgroups: blacks, whites, and in patients not on antihypertensive treatment (n=1973). We also performed formal analyses testing the interaction between race (black or white) and LV mass, wall thickness, and concentricity on CAC with multiplicative interaction terms using multivariable linear (outcome: log CAC+1) and logistic (outcome:

Figure 1. Association between gender-specific quartiles of LV structure and CAC. Note that increasing LV wall thickness (A), concentricity (B), and LVM mass indexed to fat-free mass (C) were associated with increasing CAC, whereas LV end-diastolic volume indexed to body surface area (D) was not. P values are for trend across interquartile ranges.
TABLE 2. Association of LV Structure and CAC

<table>
<thead>
<tr>
<th>LV Parameter</th>
<th>Logistic (CAC10) Unadjusted</th>
<th>Logistic (CAC10) Adjusted</th>
<th>Linear (log CAC+1) Unadjusted</th>
<th>Linear (log CAC+1) Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (CI)*</td>
<td>P</td>
<td>β</td>
<td>P</td>
</tr>
<tr>
<td>LV mass</td>
<td>1.79 (1.58 to 2.04)</td>
<td>&lt;0.001</td>
<td>1.18 (0.91 to 1.5)</td>
<td>0.21</td>
</tr>
<tr>
<td>LV end-diastolic volume</td>
<td>1.00 (0.88 to 1.13)</td>
<td>0.98</td>
<td>1.03 (0.85 to 1.23)</td>
<td>0.8</td>
</tr>
<tr>
<td>LV wall thickness</td>
<td>2.43 (2.10 to 2.80)</td>
<td>&lt;0.001</td>
<td>1.23 (0.97 to 1.55)</td>
<td>0.09</td>
</tr>
<tr>
<td>Concentricity†</td>
<td>1.91 (1.71 to 2.13)</td>
<td>&lt;0.001</td>
<td>1.14 (0.99 to 1.32)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Odds ratios are for interquartile ranges: LV mass, 57 g; LV volume, 31 mL; LV wall thickness, 2.4 cm; and concentricity, 0.4 g/mL. Models adjusted for age, sex, family history of early myocardial infarction, systolic blood pressure, fat mass, fat-free mass, total cholesterol, high-density lipoprotein, diabetes, smoking, antihypertensive therapy, use of statin medications, and oral estrogen use.

†LV mass/end-diastolic volume.

Results

Baseline characteristics of the cohort are shown, stratified by gender-specific quartiles of LV concentricity (mass/volume). Increasing concentricity was associated with increasing age, body mass index, blood pressure, cholesterol, fat mass, and fat-free mass (Table 1). Higher concentricity was also associated with black ethnicity, diabetes, smoking history, and use of statins.

Measures of concentric LVH, including wall thickness (Figure 1a), concentricity (Figure 1b), or LV mass indexed to fat-free mass (Figure 1c), were associated with an increased prevalence of CAC in both men and women (P<0.001 for each). In contrast, there was no significant association between LV end-diastolic volume indexed to body surface area and CAC for either gender (Figure 1d). After adjusting for important potential confounders, LV mass, wall thickness, and concentricity each remained associated with the quantity of CAC in multivariable linear regression analyses (Table 2). There was a strong trend for an independent association of concentricity (P=0.06) and wall thickness (P=0.09) with the presence of CAC in multivariable logistic regression analyses as well (Table 2). LV end-diastolic volume was not associated with CAC in similar multivariable models. When restricting the multivariable linear regression analysis to the subgroup of participants who were not on antihypertensive treatment, the significant relationships between LV mass (P=0.008), wall thickness (P=0.006), concentricity (P=0.01), and CAC persisted.

We sought to determine whether the association of concentric LVH and CAC differed between blacks and whites. In subgroup analyses (Table 3), the association of concentric remodeling and subclinical atherosclerosis appeared particularly robust in blacks. In formal interaction testing in multivariable linear regression using the same covariates as in Table 2, however, the multiplicative interaction term between race (black or white) and measure of LVH (LV mass, LV wall thickness, or LV concentricity) on the outcome of log CAC+1 was not significant (P=0.8, P=0.2, and P=0.16, respectively). Similar results were seen in interaction testing in multivariable logistic regression (P=0.2, P=0.8, and P=0.3, respectively).

In gender-specific analyses,42 measures of concentric hypertrophy, including increasing wall thickness (Figure 2a) and concentricity (Figure 2b), were associated with increased CRP, whereas LV mass indexed to fat-free mass was not (P not significant). In contrast to the findings with CAC, multivariable analysis demonstrated that measures of concentric hypertrophy were not independently associated with CRP after adjusting for important confounders (Table 4).

Discussion

Although LVH is an important risk factor for incident adverse cardiovascular events, the mechanisms behind this associa-

TABLE 3. Association of Measures of LV Hypertrophy and CAC in Blacks and Whites in Multivariable Analysis

<table>
<thead>
<tr>
<th>LV Parameter</th>
<th>Blacks (N=1162) Logistic (CAC10)</th>
<th>Whites (N=808) Logistic (CAC10)</th>
<th>Linear (log CAC+1) Blacks (N=1162)</th>
<th>Linear (log CAC+1) Whites (N=808)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (CI)*</td>
<td>P</td>
<td>OR (CI)*</td>
<td>P</td>
</tr>
<tr>
<td>LV mass</td>
<td>1.32 (0.95 to 1.8)</td>
<td>0.10</td>
<td>0.96 (0.60 to 1.6)</td>
<td>0.9</td>
</tr>
<tr>
<td>LV wall thickness</td>
<td>1.37 (1.0 to 1.9)</td>
<td>0.05</td>
<td>1.21 (0.8 to 1.9)</td>
<td>0.4</td>
</tr>
<tr>
<td>Concentricity†</td>
<td>1.18 (0.98 to 1.42)</td>
<td>0.08</td>
<td>1.2 (0.92 to 1.5)</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Odds ratios are for interquartile ranges: LV mass, 57 g; LV wall thickness, 2.4 cm; and concentricity, 0.4 g/mL. Models adjusted for age, sex, family history of early myocardial infarction, systolic blood pressure, fat mass, fat-free mass, total cholesterol, high-density lipoprotein, diabetes, smoking, antihypertensive therapy, use of statin medications, and oral estrogen use.

†LV mass/end-diastolic volume.
tion remain ill defined. In this large, population-based cohort, we have demonstrated that markers of concentric ventricular hypertrophy (LV wall thickness, concentricity, and indexed LV mass) but not ventricular dilation were associated with the burden of atherosclerosis as assessed by CAC. These associations were particularly robust in blacks. Furthermore, we have demonstrated that concentric hypertrophy (wall thickness and concentricity) was associated with elevated CRP, although this association appears to be mediated via the risk factors for LVH rather than by LVH itself. Thus, patients with concentric LVH may be at increased risk for incident atherothrombotic events because of the synergistic combination of increased atherosclerotic burden in the setting of coexisting risk factors associated with a proinflammatory milieu.

Previous data have suggested that LVH may be associated with CAC. The initial description of such an association was a convenience sample of 249 hypertensive patients in which LVH was found to be associated with CAC in those over the age of 60 years but not in younger patients. Subsequently, LVH was found to be associated with CAC in 159 young-to-middle-aged blacks recruited from a longitudinal study assessing HIV infection, cocaine use, and atherosclerosis. Because 55% of the subjects were HIV positive and 74% were cocaine users, the generalizability of these findings was uncertain. The Coronary Artery Risk Development In young Adults (CARDIA) Study reported recently that echocardiographic LVH was associated with the presence of CAC, but this study was limited by a 10-year difference between the index echocardiogram and the subsequent EBCT. Similarly, recent data from the National Heart, Lung, and Blood Institute Family Heart Study and HyperGen Study also reported that echocardiographic LVH was also associated with the presence of CAC, particularly in black patients, but this study was also limited by an average of >5 years between the index echocardiogram and the evaluation for CAC. Thus, although previous studies suggest that LVH was associated with CAC, the applicability to the general population was previously unknown. In addition, because of the much larger sample size of our study and detailed phenotypic characterization of our study cohort, we have extended previous studies by demonstrating that the association of LVH and CAC is present in both men and women and is independent of a number of important potential confounders, including systolic blood pressure, hypertensive status, lipid levels, statin use, and age. We also have demonstrated that the association of LVH and CAC is particularly robust in blacks, an important observation given the marked increase in the prevalence of LVH in black as compared with white subjects. Furthermore, we have been able to demonstrate that it is concentric remodeling and not ventricular dilation that drives the association of LVH and subclinical atherosclerosis, an important distinction when assessing the underlying risk associated with LVH.

Recently, it has been hypothesized that LVH may itself be an inflammatory state as assessed by elevations in CRP.

**TABLE 4. Association of LV Structure and (log) CRP**

<table>
<thead>
<tr>
<th>LV Parameter</th>
<th>Unadjusted β</th>
<th>P</th>
<th>Adjusted* β</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass</td>
<td>0.037</td>
<td>0.06</td>
<td>-0.02</td>
<td>0.5</td>
</tr>
<tr>
<td>LV end-diastolic volume</td>
<td>-0.031</td>
<td>0.09</td>
<td>-0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>LV wall thickness</td>
<td>0.092</td>
<td>&lt;0.001</td>
<td>0.025</td>
<td>0.38</td>
</tr>
<tr>
<td>Concentricity</td>
<td>0.074</td>
<td>&lt;0.001</td>
<td>0.025</td>
<td>0.15</td>
</tr>
</tbody>
</table>

*β-Coefficients are for interquartile ranges, shown in Table 2. Models adjusted for race, age, sex, family history of early myocardial infarction, systolic blood pressure, fat mass, fat-free mass, total cholesterol, high-density lipoprotein, diabetes, smoking, antihypertensive therapy, use of statin medications, and oral estrogen use.
levels. This possibility was first raised in a study of patients with end-stage renal disease undergoing hemodialysis. Subsequently, 2 studies of hypertensive patients determined that increased LV wall thickness (concentric remodeling) or echocardiographic LVH were associated with elevations of CRP. Herein, we found that increasing quartiles of LV wall thickness and concentricity were associated with elevated CRP in stepwise fashion in both men and women. However, the association of LVH with CRP did not persist in multivariable analysis after adjusting for body composition, lipid levels, age, and other confounders, suggesting that CRP was elevated in patients with LVH because of risk factors common to the development of LVH and CRP elevation. Thus, our data do not support the hypothesis that LVH itself is a primary proinflammatory state.

There are important limitations of our study that should be recognized. First, our study is cross-sectional. Thus, despite a large sample size and diverse population, assigning causality between LVH and CAC, or vice versa, is not possible. Second, because 24-hour blood pressure recordings were not performed, there may be residual confounding by an unmeasured effect of elevated blood pressure in the association between concentric LVH and CAC despite multivariable analysis. However, particular attention was paid to careful blood pressure measurement in the Dallas Heart Study, including the use of an automatic oscillometric device, which has been validated against catheter measurement of arterial pressure. Furthermore, we had 3 independent measurements of blood pressure with 2 sets obtained in the home environment and 1 in an office setting. The inclusion of a home and office measurement has been shown to provide additive information compared with office measurement alone. Finally, we performed only a single measurement of CRP, and variability in CRP measurements over time could influence the association between LVH and CRP.

**Perspectives**

Individuals in the general population with concentric LVH have both increased coronary atherosclerotic burden as assessed by CAC and evidence for an inflammatory state as measured by CRP. Although the latter appears to be secondary to comorbid conditions, an increased coronary atherosclerotic burden coupled with an inflammatory state, as suggested by elevated CRP levels, may explain in part why LVH is a risk factor for incident ASHD events, including myocardial infarction.

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**Disclosures**

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**References**


12. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Am Intern Med*. 1991;114:345–352.


23. Houghton JL, Strogatz DS, Tosoff MT, Smith VE, Fein SA, Kuhner PA, Philbin EF, Carr AA. African Americans with LVH demonstrate...


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