Body Mass Index and Blood Pressure Influences on Left Ventricular Mass and Geometry in African Americans

The Atherosclerotic Risk In Communities (ARIC) Study

Ervin Fox, Herman Taylor, Michael Andrew, Hui Han, Emad Mohamed, Robert Garrison, Thomas Skelton

Abstract—A unique interaction between the influences of body mass index and blood pressure on left ventricular mass index and geometry may contribute to the higher prevalence of left ventricular hypertrophy in African Americans. This cross-sectional study assessed separate and joint influences of body mass index and blood pressure on left ventricular mass index and geometry in 1729 African American participants of the Atherosclerotic Risk in Communities Study. The association between both left ventricular mass index and relative wall thickness and body mass index in each blood pressure category and between these variables and blood pressure in each body mass index category was assessed adjusting for age, diabetes status, hypertension medication, and smoking status. We found that left ventricular hypertrophy and concentric geometry were highly prevalent and that body mass index and blood pressure were independently associated with left ventricular mass index. The adjusted association between blood pressure and left ventricular mass index was stronger with higher body mass index categories; however, there was no significant interaction suggesting merely an additive relationship (not synergistic/multiplicative as tested for in the interaction analysis). Although relative wall thickness was greater with higher categories of body mass index and blood pressure, the mean difference in relative wall thickness between body mass index and blood pressure categories was not statistically significant. The effect on left ventricular geometry as measured by relative wall thickness supports the theory that there is a pathophysiological component in the mechanism of hypertrophy. (Hypertension. 2004;44:55-60.)

Key Words: hypertrophy ■ body mass index ■ blood pressure

Increased left ventricular mass (LVM) as determined by 2-dimensional M-mode echocardiography is a predictor of cardiovascular morbidity and mortality. In the few earlier studies comparing LVM in African Americans to that in whites, results suggest that in African Americans, LVM is increased compared with other ethnic groups at a given blood pressure, representing a potential racial disparity in cardiac adaptation to hypertension. Recent studies have indexed LVM by height and/or body surface area and have not reproduced this racial disparity in LVM severity. Disparities in left ventricular (LV) wall thickness still persisted in a number of these studies, and LV wall thickness in itself has been associated with cardiovascular mortality. Despite the question of racial differences in the prevalence of left ventricular hypertrophy (LVH) and in the patterns of LV remodeling, risk factors contributing to increased LVM and changes in LV geometry have not been extensively investigated in African Americans but mostly in large, predominantly white population-based cohorts such as Framingham Heart Study. Generalizability of these studies to the black population has not been well defined.

This report focuses on an analysis of the independent and combined effects of body mass index (BMI) and blood pressure (BP) on left ventricular mass index (LVMI) and geometry in African Americans. Although there is a high prevalence of individuals with both of these risk factors in this population, their joint influences on LVMI (unlike their independent effects) have not been studied in a population-based middle-aged African American cohort. We are particularly interested in the question of whether the racial disparity in the prevalence of LVH is merely caused by the increased prevalence of risk factors in the African American community or is a component of the ethnic difference caused by a significant interaction between the effects of BMI and BP on LVMI and geometry in African Americans, which has been shown not to be present in white, non-Hispanic populations.

Methods

Study Population
The base population consisted of participants in the Jackson Cohort of the Atherosclerotic Risk in Communities (ARIC) Study. Partici-
Participants were selected by probability sampling of eligible resident adults, aged 45 to 64. The Jackson Cohort was the only site of ARIC receiving echocardiograms and was 100% African American. The echocardiograms were performed between 1993 and 1995. Subjects with significant valvular heart disease (moderate or severe mitral regurgitation, moderate or severe aortic regurgitation, moderate or severe mitral stenosis or any degree of aortic stenosis) and those with missing M-mode measurements were excluded. Detailed study procedures, including study sampling, study design, and examination protocol, for the ARIC study have been reported previously.21

Echocardiographic Methods

Left ventricular internal diastolic diameter (LVIDD), LV posterior wall thickness (PWT), and interventricular septal thickness (IVST) were measured in diastole on 2-dimensional echocardiogram using the American Society of Echocardiography (ASE) criteria. Relative wall thickness (RWT), the ratio of the sum of PWT and IVST to the LVIDD, was derived from these measurements. To calculate LVM, individual dimensions determined by ASE criteria were applied to the formula of Troy.22 The LVM calculated from the formula of Troy was then corrected for overestimation as described by Devereux et al.23

\[
LVM(g) = 0.8 \left[ 1.04 \left( \frac{LVIDD + IVST + PWT}{3} \right)^{0.60} \right] + 0.60
\]

To correct for influences caused by body size, LVM was indexed by height1/3 (g/m²).24 LVIDD, PWT, and IVST were indexed to height (LVIDDI, PWTI, and IVSTI, respectively).

Statistical Analysis

All analyses were sex-specific. Main factors of interest, the BMI and BP, were placed into categories with clinical relevant cut-points. There are 3 levels of BMI groups generated: normal (<25 kg/m²), overweight (25 to <30 kg/m²), and obese (>30 kg/m²). Participants were also categorized into 3 BP groups according to the Joint National Committee VI guidelines: normal (<130 mm Hg systolic and <85 mm Hg diastolic), high normal/borderline (130 to 139 mm Hg systolic or 85 to 89 mm Hg diastolic), and hypertensive (>140 mm Hg systolic or >90 mm Hg diastolic).

Two-way ANCOVA was used to evaluate the strength of association between the aforementioned factors with the LVM and geometry. Possible synergistic (multiplicative) effects between independent variables were first analyzed by putting an interaction term between the BMI group and BP group into the model, in the presence of both factors.

To assess the independent association of each factor to LVM or its geometry, a new subgroup variable was generated by crossing the 3 levels of BMI group and the 3 levels of BP group. Subsequently, the subgroup variable was used in the multivariable ANCOVA analysis in place of the 2 original factors to address joint influence of BMI and BP on the dependent variables with the presence of each other.

The study protocol was approved by the Institutional Review Board at the University of Mississippi Medical Center. The study was performed with the participants’ written informed consent.

Results

Characteristics of Participants

Of the 3728 African American participants, 2445 subjects presented for the third visit or early phase of the fourth visit and received echocardiograms. Of the 2445 receiving echocardiograms, 716 (29.2%) were excluded from the study (687 caused by inadequate echocardiographic studies, 27 caused by valvular heart disease, and 2 caused by missing BMI). The study population therefore consisted of 1729 subjects; 611 (35%) of the participants were men and 1119 (65%) were women. The mean age of the study population was 58.7 ± 5.7 years (range 49 to 75 years).

| TABLE 1. Gender-Specific Demographics for Jackson (All African American) Cohort of ARIC |
|-----------------------------------------------|------------------|
| Demographics                                  | Men (n=611)      | Women (n=1119) |
| Age, y                                       | 58.8 ± 6.0       | 58.7 ± 5.6     |
| Weight, kg                                   | 67.9 ± 16.3      | 83.9 ± 18.1    |
| Height, m                                    | 76.3 ± 6.8       | 163.3 ± 6.1    |
| BMI, kg/m²                                   | 28.2 ± 4.8       | 31.4 ± 6.5     |
| Total cholesterol, mg/dL                    | 197.4 ± 37.5     | 208.5 ± 38.6   |
| LDL cholesterol, mg/dL                      | 124.1 ± 34.1     | 126.6 ± 37.8   |
| HDL cholesterol, mg/dL                      | 49.8 ± 16.7      | 59.5 ± 18.2    |
| LVM, g (ASE-corrected)                       | 226.9 ± 71.1     | 188.2 ± 58.3   |
| LVMI, g/m²                                   | 128.8 ± 40.4     | 115.2 ± 35.4   |
| Posterior wall thickness, cm                 | 1.2 ± 0.23       | 1.1 ± 0.21     |
| Interventricular wall thickness, cm          | 1.2 ± 0.26       | 1.1 ± 0.23     |
| Left ventricular internal diastolic diameter, cm | 4.82 ± 0.60  | 4.54 ± 0.55    |
| LVH, %                                       | 37.3             | 40.9           |
| Current smoker, %                            | 26.6             | 14.7           |
| Diabetes, %                                  | 17.9             | 21.3           |
| Hypertension, %                              | 57.3             | 61.8           |
| Antihypertensive medications, %              | 43.7             | 51.4           |

Values represents mean ± SD.

Sex-specific demographics for the study population are shown in Table 1. Using the criteria of 51 g/m²2, 37.3% of men and 40.9% of women met criteria for LVH.25 Men had slightly larger LV dimensions on average.

Assessment for an Interaction Between BMI and BP Effects on LVM and Geometry

The analysis of the joint influences of BMI and BP on LVM and LV geometry showed there was no significant interaction (multiplicative relationship) between effects of BMI and BP (P interaction for the population=0.69). After stratifying the study population by gender, there was no significant interaction between BMI and BP on LVMI in men (<P=0.3614) or in women (<P=0.7321). The adjusted association between BP and LVMI was stronger with higher BMI categories. These findings suggest that the joint influences of BMI and BP on LVM and LVMI for both genders were merely additive.

Similarly, the joint impact of BMI and BP on RWT and each of the 3 LV dimensions was merely additive. There was no significant interaction between BMI and BP on any of the individual LV dimensions, although the association between BMI and individual dimensions was stronger with higher BMI categories.

Influence of BMI on LVMI

BMI was an independent predictor of LVMI. Horizontal rows in Table 2 show the relationship between BMI and mean LVMI within each category of BP after adjusting for covariates. In both men and women, mean LVMI was greater in higher BMI categories in normotensive, high normal, and hypertensive subjects. For lean normotensive men, mean LVMI was 37.8 g/m²7 compared with 48.1 g/m²7 for obese normotensive men (27% mean difference). For lean men with
Influence of BMI and BP on LVMI

BP was also an independent predictor of LVMI. Vertical columns in Table 2 show the relationship between LVMI and BP within various categories of BMI after adjusting for covariates. In both men and women, mean LVMI was greater for higher BP categories in lean, overweight, and obese subjects. For lean normotensive men, the mean LVMI was 37.8 g/m² compared with 45.4 g/m² in lean hypertensive men (20% mean difference). In overweight normotensive men, the mean adjusted LVMI was 43.2 g/m² versus 54.6 g/m² in overweight hypertensive men (26% mean difference). In obese normotensive men, the mean LVMI was 48.1 g/m² compared with 60.7 g/m² in obese hypertensive (26% mean difference). BP was highly predictive of the LVMI in men for all BMI categories (P=0.007 in lean subjects and P=0.0001 in overweight and obese subjects).

For overweight and obese women the relationship between BP and LVMI was highly significant (P=0.0001 for both). In women, there was no significant relationship between BP and LVMI in lean subjects (P=0.087); however, for overweight and obese subjects, LVMI was greater with higher categories of BP. For lean normotensive women, the mean LVMI was 38.1 g/m² compared with 44.1 g/m² in lean hypertensive women (16% mean difference). In overweight normotensive women, the mean LVMI was 44.5 g/m² compared with 53.8 g/m² in overweight hypertensive women (21% mean difference). In obese normotensive women, the mean LVMI was 51.6 g/m² compared with 61.6 g/m² (19% mean difference) in obese hypertensives. Similar gender-specific results were seen with LVM.

Influence of BMI and BP on LV Geometry

Tables 3 to 6 show the influences of BMI and BP on RWT, LVIDDI, PWTI, and IVSTI. In both men and women, RWT high normal BP, mean LVMI was 44.8 g/m² compared with 51.7 g/m² in obese men with high normal BP (15% mean difference). In lean hypertensive men, mean LVMI was 45.4 g/m² versus 60.7 g/m² in obese hypertensive men (34% mean difference). BMI was highly predictive of LVMI for both normotensive (P=0.0001) and hypertensive men (P=0.0001).

Similarly for women, there was a significant relationship between BMI and LVMI (P=0.0001 in each BP category). For a given BP, mean LVMI was greater in higher BMI categories. In normotensive women, mean LVMI was 38.1 g/m² in lean subjects versus 51.6 g/m² in obese subjects (35% mean difference). In women with high normal BP, mean LVMI was 38.8 g/m² in lean subjects versus 55.2 g/m² in obese subjects (42% mean difference). Finally, for hypertensive women, mean LVMI was 44.1 g/m² in lean subjects versus 61.6 g/m² in obese subjects (40% mean difference). Similar gender-specific results were seen with LVM.

TABLE 2. Effect of BMI and BP on Left Ventricular Mass Index

<table>
<thead>
<tr>
<th>BP Categories</th>
<th>Men, BMI Categories (kg/m²)</th>
<th>Women, BMI Categories (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>37.8</td>
<td>43.2</td>
</tr>
<tr>
<td>High normal</td>
<td>44.8</td>
<td>48.3</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>45.4</td>
<td>54.6</td>
</tr>
<tr>
<td>*P</td>
<td>0.007</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Normal refers to category of subjects with SBP <130 mm Hg and DBP <85 mm Hg. Hypertensive refers to category of subjects with SBP >140 mm Hg or DBP >90 mm Hg. High normal refers to category of subjects with BP between normal and hypertensive.

All means are adjusted for age, diabetes status, hypertension medication, smoking status, and physical activity.

P Interaction=0.69. *P is for the linear trend; †P value is for the linear trend.
was greater for higher BMI and BP categories. However the differences in RWT between the categories of BMI and between the categories of BP were not statistically significant.

Conversely, in both men and women, indexed PWT and IVST were significantly associated with both BMI and BP after adjusting for covariates. The mean PWTI and IVST were greater with higher BMI and BP categories. LVIDDI was significantly associated with BMI in both genders, showing greater values with higher BMI categories. LVIDDI, however, was not strongly associated with the categories of BP for either gender.

Discussion
In this study of a population-based cohort of middle-aged African Americans, there is a high prevalence of LVH and concentric LV geometry. Compared with findings in young African Americans from the Coronary Artery Risk Development In Young Adults (CARDIA) study (in which <5% of African American men and women had LVH and a RWT >4.5), our data suggest there is an apparent shift toward higher LVMI and a more concentric LV geometry as the African American population ages.26,27

Joint Impact of BMI and BP on LVMI
A number of individual studies have shown that both BMI and BP have significant independent effects on LVMI.4–9,11,12 Fewer studies have assessed the joint influences of BMI and BP on LVMI and other variables of LV geometry.13,14,28,29

Gottdiener et al studied a hospital-based population of white American and African American men at 14 VA medical centers nationwide and found a significant association between both obesity and hypertension and cardiac structure and function.20 In this study, obesity was found to be the strongest clinical predictor of LVM and LVH, even in those with mild to moderate hypertension. There was a significant synergistic interaction between systolic blood pressure and obesity, resulting in conclusion that in men the effects of systolic blood pressure on LVM were amplified by obesity. Among the limitations of this study, the randomized trial was multicenter, hospital-based, and did not include women.

Investigators at the Framingham Heart Study looked at the joint influences of BMI and BP on LVMI in their cohort of predominantly white American men and women.19 They found that there was an association between both BMI and BP on LVMI. The effects of obesity and BP on echocardiographic LV variables were additive; however, there was no significant interaction seen. As with the study by Gottdiener, obesity was found to be a stronger predictor of LVMI than BP.

In the current study, both BMI and BP were found to be independent predictors for increased LVMI for African American men and women. Unlike Framingham, BMI and

### TABLE 4. Effect of BMI and BP on Indexed Posterior Wall Thickness

<table>
<thead>
<tr>
<th>BP Categories</th>
<th>Men, BMI Categories (kg/m²)</th>
<th>Women, BMI Categories (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lean</td>
<td>Overweight</td>
</tr>
<tr>
<td>Normal</td>
<td>0.607</td>
<td>0.638</td>
</tr>
<tr>
<td>High normal</td>
<td>0.656</td>
<td>0.679</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>0.676</td>
<td>0.714</td>
</tr>
<tr>
<td>*P</td>
<td>0.0073</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Normal refers to category of subjects with SBP <130 mm Hg and DBP <85 mm Hg. Hypertensive refers to category of subjects with SBP >140 mm Hg or DBP >90 mm Hg. Normal refers to category of subjects with BP between normal and hypertensive.

All means are adjusted for age, diabetes status, hypertension medication, smoking status, and physical activity.

P Interaction = 0.69. *P is for the linear trend; †P value is for the linear trend.

### TABLE 5. Effect of BMI and BP on Indexed Interventricular Septal Thickness

<table>
<thead>
<tr>
<th>BP Categories</th>
<th>Men, BMI Categories (kg/m²)</th>
<th>Women, BMI Categories (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lean</td>
<td>Overweight</td>
</tr>
<tr>
<td>Normal</td>
<td>0.610</td>
<td>0.634</td>
</tr>
<tr>
<td>High normal</td>
<td>0.646</td>
<td>0.670</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>0.667</td>
<td>0.710</td>
</tr>
<tr>
<td>*P</td>
<td>0.0847</td>
<td>0.0013</td>
</tr>
</tbody>
</table>

Normal refers to category of subjects with SBP <130 mm Hg and DBP <85 mm Hg. Hypertensive refers to category of subjects with SBP >140 mm Hg or DBP >90 mm Hg. Normal refers to category of subjects with BP between normal and hypertensive.

All means are adjusted for age, diabetes status, hypertension medication, smoking status, and physical activity.

P Interaction = 0.69. *P is for the linear trend; †P value is for the linear trend.
BP effects on LVMI were similar in both genders. Unlike Gottdiener’s study but similar to the Framingham study, BMI and BP effects on LVMI were merely additive in African American men and women; there was no significant interaction between the influences of these risk factors on LVMI. Similar relationships and trends among known determinants of LVH strengthens the argument that racial disparity in LVH prevalence is related to the higher prevalence of these risk factors in the African American population. These data do not support the notion that the high prevalence of LVH in this population is a result of a synergistic or multiplicative relationship between these risk factors on LVMI in the African American population.

Effect of BMI and BP on LV Geometry

Hypertension creates a state of LV pressure overload. Obesity is associated with volume overload.28 Grossman et al found that RWT tends to increase significantly with pressure overload and remains unchanged with volume overload and hypothesized that hypertrophy occurs to normalize peak systolic wall stress.29

In this study, RWT values were elevated (RWT >0.45 being the standard cutoff value distinguishing normal LV geometry from concentric LV geometry) regardless of gender, BMI, or BP, reflecting a population with mostly concentric hearts. RWT tended to be greater with higher categories of BMI and BP; however, there was not a statistically significant relationship between RWT and BMI or between RWT and BP. The differences in our findings compared with Grossman’s findings suggest that geometric patterns seen in LVH may be caused by reasons other than merely normalizing peak systolic wall stress; there may also be a pathophysiologic component to LV remodeling. Potential mechanisms that have undergone recent investigation include possible neurologic, endocrinologic, and genetic pathways. Although it is if sample size may have contributed to the negative findings with RWT, similar results with LV geometry were found in both the Framingham cohort and Gottdiener’s VA population.

Perspectives

We have shown that LVH and concentric geometry are highly prevalent in the middle-aged African American population. Compared with the CARDIA study, which looked at a young population of African Americans, the findings in ARIC suggests that there is an apparent shift in LVM and geometry in African Americans with aging. BMI and BP are independent risk factors for LVH in this group and their joint influences on LVMI are merely additive (not synergistic or multiplicative as tested for in an analysis for an interaction). The effect of BMI and BP on LV geometry as measured by RWT suggests a potential pathophysiologic component (not merely compensatory hypertrophy as defined by Grossman). These joint influences are similar to those seen in the Framingham cohort. Although comparisons to other ethnic groups are inferential because they are made using previous publications, our findings support the theory that racial differences in the prevalence of LVH are more so caused by the higher prevalence of risk factors such as BMI and BP in the African American population. Future investigations should focus on uncovering pathophysiologic mechanisms that may also contribute to the racial difference in LVH prevalence and explain the increased cardiovascular morbidity and mortality seen in all subjects with LVH.

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