

# LEFT VENTRICULAR STRUCTURE AND SYSTOLIC FUNCTION IN AFRICAN AMERICANS: THE ATHEROSCLEROSIS RISK IN COMMUNITIES (ARIC) STUDY

**Objectives:** To estimate prevalence of left ventricular (LV) hypertrophy and its relation to systolic function in a population-based sample of African Americans.

**Design:** A baseline 2D guided M-mode echocardiogram was conducted as part of a longitudinal cohort study to assess prevalence and cross-sectional relationships between echocardiographic and clinical parameters.

**Setting:** Data were collected as part of the Atherosclerosis Risk in Communities study.

**Participants:** Analysis is limited to 1543 African Americans, aged 51–70 years, without clinically apparent cardiovascular or echocardiographically determined valvular disease.

**Main Outcome Measures:** LV hypertrophy prevalence was defined as LV mass/height<sup>2.7</sup> ≥ 51 g/m<sup>2.7</sup>. LV systolic chamber function was assessed at the midwall using the ratio of observed midwall fractional shortening (MWS%) to the value predicted from circumferential end-systolic stress.

**Results:** The prevalence of LV hypertrophy was 33% in men, 38% in women. The prevalence of concentric hypertrophy (LV hypertrophy with relative wall thickness ≥ 0.45) was greater than that of eccentric hypertrophy (men: 24% vs 9%; women: 27% vs 11% women). Observed/predicted (O/P) MWS% was strongly and inversely related to LV mass/height<sup>2.7</sup> ( $P < .001$ ) and LV hypertrophy ( $P < .001$ ). The O/P MWS% was inversely related to LV mass/height<sup>2.7</sup> quartile: O/P MWS% was 106% and 99% in the first and 97% and 89% in the fourth quartile of LV mass/height<sup>2.7</sup> for men and women, respectively. Adjusting for age, adiposity, diabetes, blood pressure, antihypertensive medication use, and smoking did not remove association between O/P MWS% and LV mass/height<sup>2.7</sup>.

**Conclusions:** LV hypertrophy was highly prevalent in this population-based middle-aged sample of African Americans and was associated with poorer LV systolic chamber function. (*Ethn Dis.* 2004;14:483–488)

**Key Words:** African Americans, Cohort Studies, Echocardiography, Left Ventricular Mass

From the University of Minnesota, School of Public Health, Division of Epidemiology, Minneapolis, Minnesota.

Vuyisile T. Nkomo, MD; Donna K. Arnett, PhD;  
Emelia J. Benjamin, MD, ScM;  
Philip R. Liebson, MD; Richard G. Hutchinson, MD;  
Thomas N. Skelton, MD

## INTRODUCTION

Left ventricular (LV) hypertrophy is a strong risk factor for cardiovascular morbidity and mortality.<sup>1–8</sup> The higher prevalence of LV hypertrophy in African Americans may partially explain the greater incidence of cardiovascular disease in this group compared with Caucasians.<sup>9</sup> However, there is limited population-based information on the prevalence of echocardiographic LV hypertrophy or the range and correlates of LV mass in African Americans.

An opportunity to evaluate a large African-American population was provided by the ARIC study, which is a prospective cohort study designed to evaluate the etiology and natural history of atherosclerosis. Cohort participants, aged 45–64 years at the time of enrollment between 1987 and 1989, were se-

lected from Forsyth County, North Carolina; Jackson, Mississippi (an all African-American center); Minneapolis, Minnesota; and Washington County, Maryland.<sup>10</sup> Echocardiography was performed at the Jackson Center during the second follow-up examination. These data were used to define the population distribution of LV structural and functional indices. This report describes the prevalence and patterns of LV hypertrophy and the association of LV mass with LV function in this large African-American cohort. Connections between LV mass and obesity, hypertension, and other cardiovascular risk factors will also be discussed.

## METHODS

### Study Population

At the first examination, eligible participants were identified by probability sampling and were invited to participate. The clinic response rate for the baseline examination in Jackson was 46%; 70% of participants who had a baseline examination returned for the follow-up visit. Echocardiography<sup>11</sup> was conducted during this visit for 2445 volunteers, of which 1823 (75%) had analyzable M-mode echocardiograms. Given the prevalence of obesity and the age distribution of the sample, this success rate is favorably comparable to similar studies in participants of similar age.<sup>12</sup> An additional 280 participants were excluded for significant echocardiographically detected valvular disease, severe LV dysfunction, wall motion abnormalities, and/or self-reported history of myocardial infarction or coronary ar-

Author affiliations: Division of Cardiovascular Disease and Internal Medicine, Mayo Clinic, Rochester (VTN); University of Minnesota, School of Public Health, Division of Epidemiology, Minneapolis (DKA), Minnesota; Boston University School of Medicine, Cardiology Section, Boston, Massachusetts (EJB); Section of Cardiology, Department of Medicine and Department of Preventive Medicine, Rush Medical College, Rush University, Chicago, Illinois (PRL); Preventive Cardiology (RGH), Division of Cardiovascular Diseases (TNS), University of Mississippi Medical Center, Department of Medicine, Jackson, Mississippi.

Address correspondence and reprint requests to Donna K. Arnett, PhD; University of Minnesota; School of Public Health, Division of Epidemiology; 1300 South Second Street, Suite 300; Minneapolis, MN 55454-1015; 612-626-8863; 612-624-0315 (fax); arnett@epi.umn.edu

*The higher prevalence of LV hypertrophy in African Americans may partially explain the greater incidence of cardiovascular disease in this group compared with Caucasians.<sup>9</sup>*

tery revascularization procedure. The final analysis included 992 women and 551 men. Body mass index (BMI) and blood pressure did not differ between the final analysis sample and those who underwent echocardiography but did not have analyzable echocardiograms; however, those subjects with unusable echocardiography data were older (60.0 vs 58.7 years,  $P < .001$ ) compared with those who had analyzable echocardiograms. The research was approved by the local Institutional Review Board, and informed consent was collected from all participants.

Interviews and standardized measurements were conducted per the ARIC study protocol. These measurements included seated blood pressure using a random zero sphygmomanometer, anthropometrics (weight and height), and plasma glucose. The last 2 of 3 blood pressure measurements were averaged and used in this analysis. Use of pharmacologic agents for treatment of hypertension (defined as taking medication during the prior 2 weeks) was ascertained via self-reporting. Body mass index (BMI) was calculated based on weight and height ( $\text{kg}/\text{m}^2$ ) measurements. Diabetes mellitus was identified either by self-reported medical history or fasting plasma glucose ( $>126 \text{ mg}/\text{dL}$ ). Tobacco smoking status was classified as current, former, or not ever.

### Echocardiographic Methods

The echocardiography protocol followed guidelines used in other epide-

miologic cohorts.<sup>11</sup> Echocardiography was performed using the Acuson 128XP/10c (Siemens, Malvern, Pa) system and images were digitized for off-line analysis using Freeland Systems' CineView (Freeland Systems, Westfield, Ind). Scans were performed with the head of the table inclined at an angle of 15 degrees and participants rotated 30 to 45 degrees in the left lateral decubitus position at end-expiration using a table with a special cut out to enhance visualization. The parasternal acoustic window was used to record LV internal diameter and wall thicknesses at or just below the tips of the anterior leaflets of the mitral valve in both short and long axis views. The apical window was used to record 2 and 4 chamber images and the long axis view to assess LV wall motion and valvular function.

### Echocardiographic Measurements

Left ventricular (LV) measurements were taken according to the American Society of Echocardiography (ASE) recommendations.<sup>13</sup> The ASE convention uses a leading edge boundary at the end of diastole, which is identified by the beginning of the QRS complex of the simultaneously recorded ECG. Left ventricular mass (LVM) was calculated according to Devereux-Reichek anatomically validated Penn Convention formula with modification for ASE measurement convention<sup>14,15</sup>:

$$\text{LVM (g)} = (0.8[1.04((\text{IVSd} + \text{LVIDd} + \text{PWTd})^3 - \text{LVIDd}^3)] + 0.6),$$

where IVSd was interventricular septum end-diastole, LVIDd was left ventricular internal diameter end-diastole, and PWTd was posterior wall thickness end-diastole. As recommended,<sup>16</sup> LVM values were normalized by height<sup>2,7</sup> (hereafter LV mass/height<sup>2,7</sup>) to correct for the effect of overweight and to eliminate potential differences in gender due to body size differences. Relative wall thickness (RWT) was defined as

(2PWTd)/LVIDd. LV hypertrophy was defined as LV mass/height<sup>2,7</sup>  $\geq 51 \text{ g}/\text{m}^2$ . Geometric patterns were defined as follows:

- Normal: No LV hypertrophy and  $\text{RWT} < 0.45$
- Concentric hypertrophy: LV hypertrophy and  $\text{RWT} \geq 0.45$
- Eccentric hypertrophy: LV hypertrophy and  $\text{RWT} < 0.45$
- Concentric remodeling:  $\text{RWT} \geq 0.45$  in the absence of hypertrophy

Myocardial contractility was assessed using formulae which correlated LV systolic shortening with end-systolic stress measured at the level of the LV minor axis. Midwall fractional shortening (MWS%), which accounts for the epicardial migration of the midwall during systole, was calculated from the elliptic model used by Shimizu et al.<sup>17</sup> Volumes were derived from M-mode readings and blood pressure measurements determined at the time of echocardiograph. Circumferential end-systolic stress (cESS) was estimated at the midwall for M-mode readings using a cylindrical model.<sup>18</sup> To evaluate LV performance taking cESS into account, shortening was calculated as a percentage of the value predicted from end-systolic stress [Observed/Predicted (O/P) MWS%] derived from a group of normal subjects.<sup>18</sup> These measures have been shown to be reliable indicators of LV systolic performance.<sup>19–21</sup>

Echocardiograms were read by one cardiologist reader (TS). Intra- and inter-sonographer and intra-reader variability were assessed on a randomly selected 2% sample of participants. Of 102 participants scanned twice by either the same or different sonographer, paired measurements were obtained in 73 subjects for M-mode LV mass. Intra- and inter-sonographer correlation of M-mode LV mass between the first and second scan was 0.94 and 0.82, respectively. Intra-reader correlation of co-variability for M-mode LV mass was 0.98.

**Table 1. Gender-specific baseline demographic, anthropometric, and clinical characteristics. *P* value reflects test of gender differences\***

	Men	Women	All	<i>P</i> Value
N	551	992	1543	
Age (y)	59 ± 6.0	59 ± 5.6	59 ± 5.7	.72
Height (m)	1.8 ± 6.8	1.6 ± 6.1	1.7 ± 9.0	<.001
Weight (kg)	87.3 ± 15.8	82.8 ± 17.4	84.4 ± 17.0	<.001
Body mass index (kg/m <sup>2</sup> )	28.0 ± 4.7	31.1 ± 6.3	30.0 ± 5.9	<.001
Overweight (%)†	46	34	38	<.001
Obese (%)‡	29	34	38	<.001
Diabetes (%)	18	22	20	.042
Hypertension (%)	54	58	56	.103
Antihypertensive medication (%)	38	48	45	<.001
Systolic BP (mm Hg)	131 ± 20	130 ± 20	130 ± 20	.67
Diastolic BP (mm Hg)	79 ± 11	76 ± 10	77 ± 10	<.001
Current smoking (%)	26	13	18	<.001

\* Data are presented as mean ± SD or %.

† Overweight defined as 25 ≤ BMI < 30 kg/m<sup>2</sup>.‡ Obese defined as BMI ≥ 30 kg/m<sup>2</sup>.

In addition, an external panel of echocardiographers (EB, PL) conducted annual site visits to oversee quality control and conducted readings on a random subsample of echocardiograms; excellent comparability in LV mass was observed ( $r=0.82-0.96$  between readers). Between-reader differences in LV mass were  $-4.2$  and  $24.9$  g, with the primary reader's (TS) mean LV mass falling between values of the 2 external readers (EB, PL).

### Statistical Analysis

Gender-specific analyses were performed using the Statistical Analysis System (SAS Institute, Inc., Cary, NC).<sup>22</sup> Quartiles of LV mass/height<sup>2.7</sup> were calculated for the overall population. Multiple regression models for LV mass/height<sup>2.7</sup> were calculated separately for men and women, and included variables that were significantly associated with LV mass/height<sup>2.7</sup> and/or O/P MWS% in this study population (age, BMI, diabetes, systolic blood pressure, antihypertensive medication use, and smoking).

## RESULTS

Table 1 summarizes participant characteristics at the echocardiography

visit. The mean age was 59 years and there was a high prevalence of obesity and central adiposity among the participants. The mean ± SD BMI was  $28.0 \pm 4.7$  kg/m<sup>2</sup> in men and  $31.1 \pm 6.3$  kg/m<sup>2</sup> in women, and more than half of the women were obese (defined as BMI ≥ 30 kg/m<sup>2</sup>). About one fifth of the participants were diabetic. Many participants in the cohort were receiving pharmacologic treatment for hypertension (38% of men and 48% of women). The mean systolic blood pressure was within the high-normal range ( $131 \pm 20$  mm Hg in men and  $130 \pm 20$  mm Hg in women).

### LV Mass, Geometry, and Functional Indices

Mean values ± SD for LV chamber size, wall thicknesses, LV mass, RWT, LV hypertrophy prevalence, and functional indices are listed in Table 2. There was a high prevalence LV hypertrophy (33% in men and 38% in women). In general, mean values of posterior wall thickness, LV chamber size, and RWT indicated a concentric pattern of geometric remodeling. The predominant geometric pattern was concentric remodeling (40% in men and 39% in women), followed by concentric LV hypertrophy (24% in men and 28% in

women). Eccentric LV hypertrophy was found in 9% of men and 11% of women. The average MWS% was approximately that expected, with O/P MWS% 96% in men and 101% in women.

### Association of LV Mass/Height<sup>2.7</sup> and Hypertrophy to LV Midwall Function

Both men and women with LV hypertrophy had poorer O/P MWS% than those without hypertrophy (men: 92% vs 98%; women: 98% vs 103%). O/P MWS% fell consistently across increasing quartiles of LV mass/height<sup>2.7</sup> (g/m<sup>2.7</sup>) in both men and women (Figure 1). In multiple linear models adjusted for factors that were associated with LV mass and/or O/P MWS% in men or women, O/P MWS% was associated strongly and negatively associated with LV mass (overall model  $R^2=0.51$  in men and 0.52 in women). The standardized beta coefficients for O/P MWS% from the multiple regression models were  $-0.53$  in men and  $-0.45$  in women.

## DISCUSSION

The results from this analysis of the community-based ARIC study cohort

Table 2. Echocardiographic measures of left ventricular structure and function. P value reflects test of gender differences\*

	Men	Women	All	P Value
	551	992	1543	
Posterior wall thickness (mm)	1.18 ± 0.19	1.11 ± 0.18	1.14 ± 0.19	<.001
Septal wall thickness (mm)	1.18 ± 0.22	1.11 ± 0.20	1.14 ± 0.21	<.001
LV internal dimension (mm)	4.80 ± 0.56	4.51 ± 0.53	4.62 ± 0.56	<.001
Relative wall thickness (ratio)	0.50 ± 0.11	0.50 ± 0.11	0.50 ± 0.11	.803
LV mass (g)	217.5 ± 59.0	181.6 ± 51.5	194.6 ± 57.0	<.001
LV mass/height <sup>2.7</sup> (g/m <sup>2.7</sup> )	47.2 ± 13.3	48.6 ± 14.2	48.1 ± 13.9	.0558
LV mass BSA (g/m <sup>2</sup> )	106.8 ± 27.2	96.7 ± 25.0	100.3 ± 26.2	<.001
LV hypertrophy (%)	33	38	36	<.001
LV geometric pattern (%)				.125
Normal	27	22	25	
Eccentric hypertrophy	9	11	10	
Concentric remodeling	40	39	39	
Concentric hypertrophy	24	28	26	
MWS (%)	14.8 ± 2.7	15.8 ± 2.7	15.5 ± 2.7	<.001
cESS (kdyn/cm <sup>2</sup> )	110 ± 35.7	101 ± 34.3	104 ± 35.1	<.001
O/P MWS (%)	96 ± 17.3	101 ± 17.2	99 ± 17.4	<.001

\* Data are presented as mean ± SD or %.  
† LV hypertrophy defined as LV mass/height<sup>2.7</sup> ≥ 51 g/m<sup>2.7</sup>.

suggest that LV hypertrophy is a common condition in middle-aged African Americans, affecting up to 33% of men and 38% of women using criteria based on LV mass/height.<sup>2,7</sup> In addition, LV hypertrophy and increasing levels of LV mass were associated with decreasing LV systolic chamber function. Since LV hypertrophy and impaired LV systolic chamber function are strong predictors of subsequent cardiovascular morbidity and mortality in other populations,<sup>23</sup>

these findings may partially explain the excess cardiovascular risk in Jackson compared to the other ARIC communities.<sup>24</sup> The high prevalence of LV hypertrophy in Jackson may be attributable to the high prevalence of other risk factors among this population, particularly hypertension, diabetes, and obesity. Approximately 50% of cohort participants reported medication use for hypertension, one-fifth was diabetic, and mean

BMI was markedly higher than published population means reported for Whites. Diabetes is emerging as an important determinant of LV hypertrophy and appears to magnify alterations of the cardiovascular system induced by the increased afterload associated with systemic hypertension.<sup>25</sup> This elevated risk associated with diabetes is independent of blood pressure and obesity, which are both strongly correlated with diabetes and LV hypertrophy. Since hy-

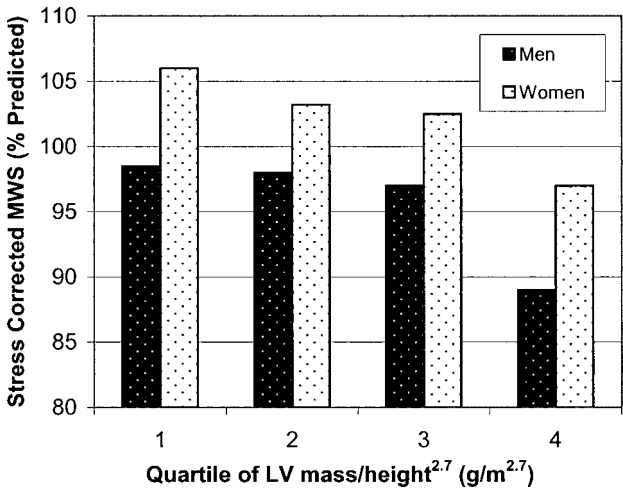


Fig 1. Lower stress-corrected midwall shorting (% predicted) with increasing quartiles of LV mass/height<sup>2.7</sup> (g/m<sup>2.7</sup>) in men and women

*The results from this analysis of the community-based ARIC study cohort suggest that LV hypertrophy is a common condition in middle-aged African Americans, affecting up to 33% of men and 38% of women using criteria based on LV mass/height.<sup>2,7</sup>*



pertension, diabetes, and obesity are strongly related with LV hypertrophy, it is not unexpected that the prevalence of LV hypertrophy is greater in African Americans in this sample than other ethnic groups.

Similar to other published reports in African Americans,<sup>26,27</sup> concentric hypertrophy was the most common pattern, accounting for 71% of all patterns of LV hypertrophy. In the general population, the predominant pattern of LV hypertrophy is eccentric.<sup>28</sup> Left ventricular (LV) geometric patterns may differ across ethnic groups because the causes of LV hypertrophy may differ correspondingly. The concentric pattern of LV hypertrophy observed in this analysis may reflect the integrative effects of blood pressure and obesity over time. African Americans have an earlier age of onset of hypertension, more severe hypertension, and more obesity compared to other ethnic groups.<sup>9</sup> Therefore, the concentric LV hypertrophy pattern may result from the natural history of hypertension and not ethnicity per se. African Americans may also have a genetic predisposition to LV hypertrophy—the heritability of LV mass is greater in hypertensive African Americans (70%) than Caucasians (26%).<sup>29</sup> The concentric LV hypertrophy pattern may also reflect the interaction of an underlying genetic susceptibility and the superimposed hypertensive environment. Longitudinal studies specifically of the kind that follow multiple ethnic groups simultaneously may help delineate the unique roles of blood pressure, ethnicity, and genetics in the development of the various patterns of LV hypertrophy.

In this analysis, the criteria used to define LV hypertrophy were derived from a healthy subgroup of non-obese Caucasians without hypertension.<sup>15</sup> Whether the criteria for LV hypertrophy derived in Caucasians are appropriate for African Americans is debatable. African Americans have more lean body mass compared with Whites,<sup>30</sup> and lean body mass is the strongest predictor of

LV mass among the anthropomorphic variables. It is plausible that the distribution of “normal” LV mass is shifted toward higher levels in African Americans compared with Whites. If so, the prevalence of LV hypertrophy reported herein may be artificially high.

This analysis has a number of strengths and limitations. The study was conducted in a large, population-based cohort of African Americans. Participants with cardiovascular disease or valvular abnormalities, conditions that are associated with LV hypertrophy and poor LV midwall function, were excluded from analysis; therefore, the findings are generalizable to healthy individuals. The measurements were collected using methods comparable to other population-based studies done exclusively in Caucasians, so the results may be compared to prior research. Finally, extensive quality control measures indicate excellent reliability of the echocardiographic measurements and excellent comparability of measurements across echocardiographer readers from other studies.

However, the present study has several limitations. First, there was not complete participation at the baseline visit (46%) and about 30% did not return for the second follow-up visit, which may indicate that the prevalence estimates are potentially biased. Prior reports, however, indicate that non-participants were older and had higher blood pressure than respondents. Since both age and blood pressure are strong determinants of LV hypertrophy in the ARIC cohort, it is likely that the reported prevalences are under-, not over-reported. Second, because the analysis is cross sectional, we were unable to define the temporal relationship between any of the variables studied and LV mass. Nonetheless, our findings are consistent with other studies demonstrating a relationship between systolic function and LV mass. Third, as noted above, approximately 25% of the echocardiograms were not useable due to technical

inadequacy of the M-mode measurement of LV mass. While no systematic differences were noted in blood pressure or BMI between those participants excluded and those included in the final analysis, those excluded for missing data were older. Given the positive association of age with LV mass/height<sup>2,7</sup> in the present analysis, it is plausible that the prevalence estimates are negatively biased by the absence of echocardiographic data.

In summary, in the participants from the Jackson cohort of the ARIC study, we found a high prevalence of LV hypertrophy (33% in men and 38% in women) based on LV mass/height<sup>2,7</sup> criteria, and a high prevalence of concentric remodeling and concentric hypertrophy. Left ventricular (LV) systolic chamber function, similar to other studies reported in Caucasians, was strongly and negatively associated with LV mass and hypertrophy. This high prevalence of LV hypertrophy and the adverse associations with systolic dysfunction suggest that LV hypertrophy is an important public health issue in African Americans that warrants longitudinal investigation.

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# AUTHOR CONTRIBUTIONS

*Design and concept of study:* Arnett, Benjamin, Skelton  
*Acquisition of data:* Arnett, Benjamin, Hutchinson, Skelton  
*Data analysis and interpretation:* Nkomo, Arnett, Benjamin, Liebson, Hutchinson  
*Manuscript draft:* Nkomo, Arnett, Benjamin, Liebson  
*Statistical expertise:* Nkomo  
*Acquisition of funding:* Hutchinson  
*Administrative, technical, or material assistance:* Hutchinson, Skelton  
*Assurance:* Benjamin