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Echocardiographic Measures of Left Ventricular Structure and Their Relation With Rest and Ambulatory Blood Pressure in Blacks and Whites in the United Kingdom

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Objectives. **This study attempted to determine whether people of black African descent have more left ventricular hypertrophy than those of white European descent and whether this can be explained by rest or ambulatory blood pressure.**

Background. **Mortality associated with hypertension is higher in black populations than among whites, but differences in morbidity and their associations with blood pressure are inconsistent.**

Methods. **We examined 1,166 black and white men and women 40 to 64 years old in a community survey in London, United Kingdom. Echocardiograms were obtained for all subjects and ambulatory blood pressure recordings for 319.**

Results. **Adjusted for body size, ventricular septal thickness was greater in blacks than whites ($p < 0.05$), and cavity dimension was smaller ($p < 0.05$). In men, ventricular septal thickness was >10 mm for 32% of whites and 53% of blacks; for women these figures were 14% and 38%, respectively. Relative wall thickness was greater in blacks ($p < 0.01$ for men and women), but left ventricular mass index was similar in the two ethnic groups. In men, hypertension resulted in an increase in wall thickness in both ethnic groups, but cavity dimension decreased in blacks and increased in whites. Wall thickness was higher in blacks than in whites for equivalent levels of either rest ($p = 0.05$) or ambulatory ($p = 0.007$) blood pressure.**

Conclusions. **Left ventricular mass index may not be valid for comparisons between ethnic groups because this derived measure does not take into account ethnic differences in ventricular structural response to hypertension. Interventricular wall thickness may be more valid. Using this measure, we demonstrate greater ventricular hypertrophy in blacks than in whites, unexplained by differences in either rest or ambulatory blood pressure. The pattern of ventricular hypertrophy observed in blacks is associated with an increased mortality risk. Conventional blood pressure thresholds for instituting antihypertensive treatment may be too conservative for people of black African descent.**

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Mortality associated with hypertension is higher in people of black African descent than in people of white European descent in the United Kingdom (1), the Caribbean (2) and the United States (3). We show that mean blood pressure is higher in blacks than whites, but this may not be sufficient to account for ethnic differences in stroke mortality (4). One explanation is that blacks may be more susceptible to cardiovascular end organ damage than whites for a given level of rest blood pressure (5), and there is some support for this on the basis of electrocardiographic (ECG) left ventricular hypertrophy (6). Although these ECG changes predict subsequent cardiovascular death (7), ECG measures of left ventricular hypertrophy have poor sensitivity (8) and may have lower specificity for detection of end organ damage in blacks than whites (9).

Echocardiography provides a more precise assessment of left ventricular hypertrophy (10,11), but the results of such studies have been conflicting: some find no ethnic difference in left ventricular mass (5,12–14), whereas others demonstrate significant ethnic differences (15–17). Relative wall thickness has generally been found to be higher in blacks than in whites. But these studies were not based on representative population samples, and their findings cannot be extrapolated to the general population in order to explain ethnic differences in mortality.

In this population-based study, we wished to determine whether there were ethnic differences in hypertensive cardiac damage, assessed by both ECG and echocardiography, and whether these differences could be accounted for by either resting or ambulatory blood pressure.

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Methods

Study population. Full details are described elsewhere (4). We examined 1,166 black and white men and women 40 to 64 years old, stratified by 5-year age groups from family practice lists in London. It is estimated that >98% of the general population in the United Kingdom is registered with a family doctor (18). Blacks are defined as people of black African descent from either the Caribbean or Africa; whites are defined as those of white European descent. The following tests were performed: sitting rest blood pressure, measured twice after 5 min of rest by a trained observer using a random zero sphygmomanometer (19); height, weight and body fat measurements; and a 12-lead ECG. Echocardiographic and Doppler studies were performed according to standardized protocols (20). In subjects not taking medication for diabetes, blood samples were taken in the fasting state and 2 h after a 75-g glucose load for glucose estimation and definition of diabetes (21). A subsample of patients were asked to wear an ambulatory blood pressure monitor for 24 h; this sample was weighted to include a greater proportion of hypertensive subjects than in the original sample (4).

Electrocardiographic measurements. All ECGs were Minnesota coded (22) by two experienced coders who had no knowledge of any other data for the patient. Any disagreements between the two coders were arbitrated by the senior coder. Tall R waves on the ECG (Minnesota code 3-1, i.e., R amplitude >26 mm in lead V_5 or V_6; >20 mm in lead I, II, III or aVF or >12 mm in lead aVL) were used as an indicator of left ventricular hypertrophy. The ECGs were classified as normal if they did not have tall R waves or any of the Minnesota codes 1-1 to 1-3, 4-1 to 4-3, 5-1 to 5-3 or 7-1. Tall R waves formed part of the definition of ECG left ventricular hypertrophy in the Framingham study; these criteria generally compared well with accepted criteria and, further, were strong predictors of subsequent heart disease (7). Probable coronary heart disease was defined as either a previous physician diagnosis of coronary heart disease, a positive response to the Rose angina questionnaire or major Q waves on the ECG (Minnesota codes 1-1 and 1-2) (23).

Echocardiographic studies. All patients underwent two-dimensional M-mode echocardiography in the left lateral decubitus position according to recommended procedures (24). A 3.5-MHz transducer was used for cardiac imaging, and a 2.5-MHz transducer for Doppler measurements. All echocardiography was performed by one of three trained observers and recorded onto videotape and checked by the senior observer (P.N.), who was uninformed of all other data available for each subject. Both within and between-observer variability was <5%. Left ventricular dimensions and measures of function were taken from at least three cycles according to the American Society of Echocardiography criteria (25). These included left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), interventricular septal thickness (IVST) and posterior wall thickness (PWT). Measures of left ventricular structure and function were derived from the corrected American Society of Echocardiography formulas for left ventricular mass index (LVMI) (26),

$$\text{LVMI} = 1.04 \times [(\text{IVST} + \text{PWT} + \text{LVEDD})^3 - (\text{LVEDD})^3] \times 0.8 \div 0.6,$$

and relative wall thickness (RWT) (27),

$$\text{RWT} = 2 \times \text{PWT}/\text{LVEDD}.$$

Left ventricular mass was indexed to body surface area using the Dubois formula (28). Indexing of left ventricular mass to height was also performed because there are doubts about the adequacy of body surface area as an accurate assessment of body size (29).

Ambulatory studies. Three blood pressure categories were defined: normotensive (systolic ≤140 mm Hg and diastolic ≤90 mm Hg), untreated hypertensive (systolic >140 mm Hg or diastolic >90 mm Hg) and treated hypertension (30). The Tokeda TM2420 (Tokyo, Japan) ambulatory blood pressure monitor was fitted for the 24-h period after screening in all subjects who agreed to participate. This monitor has been extensively validated and judged to be reliable (31).

Complete ambulatory blood pressure data were obtained from 319 subjects not receiving treatment for hypertension. Mean 24-h ambulatory blood pressure, mean daytime blood pressure (between 9 and 11 AM), mean evening blood pressure (between 5 and 7 PM) and mean nighttime blood pressure (between 3 and 5 AM) were calculated for each subject.

Statistical analyses. The mean of two rest blood pressures for each subject was used in all analyses. Echocardiographic indexes, measures of body size and blood pressure were all significantly associated with age, and there was no significant ethnic difference in the relation between age and echocardiographic indexes as assessed by an interaction test. The main purpose of this study was to examine the relation between blood pressure and echocardiographic variables. Thus, age-adjusted mean values are presented, calculated by least-squares regression models, to examine ethnic differences in these variables once age was accounted for. Further adjustments were made for body size in Table 4 to assess whether ethnic differences in ventricular structure could be attributable to ethnic differences in body size alone. Analysis of variance was used to calculate the proportion of variance in interventricular septal thickness accounted for by the different measures of blood pressure. Prevalence rates were directly standardized to the age distribution of the combined study group. All analyses were performed using the SAS statistical package (32).

Analysis of echocardiographic findings excludes six subjects for whom echocardiographic recordings were judged to be of poor quality. Tables 2 and 4 exclude the 18 white and 52 black men and 27 white and 101 black women currently taking medication for hypertension.

Table 1. Age-Adjusted Mean Values of Echocardiographic Measures and Blood Pressure and Prevalence of Tall R Waves (±SD) for All Subjects

	Men			Women		
	Whites (n = 270)	Blacks (n = 245)	p Value*	Whites (n = 311)	Blacks (n = 334)	p Value*
Tall R waves	7%	20%	< 0.001	0%	11%	< 0.001
Systolic BP	124 ± 14	128 ± 17	< 0.001	118 ± 15	131 ± 17	< 0.001
IVST (mm)	10.0 ± 1.3	10.5 ± 1.7	< 0.01	9.1 ± 1.4	9.9 ± 1.8	< 0.001
PWT (mm)	9.7 ± 1.3	9.9 ± 1.6		8.9 ± 1.3	9.5 ± 1.6	< 0.001
LVEDD (mm)	48.2 ± 5.0	46.5 ± 4.7	< 0.001	43.5 ± 4.6	43.5 ± 4.7	
LVESD (mm)	33.4 ± 5.4	31.1 ± 4.4	< 0.001	29.0 ± 4.2	28.2 ± 4.2	< 0.05
LVMI (g/m^2)	87.1 ± 19.1	87.7 ± 19.2		74.3 ± 18.4	78.4 ± 19.5	< 0.01
LVM height (g/m)	98.7 ± 21.9	98.3 ± 22.6		81.7 ± 21.4	90.2 ± 23.4	< 0.001
RWT	0.41 ± 0.07	0.43 ± 0.10	< 0.001	0.41 ± 0.08	0.45 ± 0.10	< 0.001

*Blacks versus whites; p values > 0.05 are not shown. BP = blood pressure; IVST = interventricular septal wall thickness; LVEDD = left ventricular end-diastolic diameter; LVESD = left ventricular end-systolic diameter; LVM height = left ventricular mass indexed to height; LVMI = left ventricular mass index (indexed to body surface area); PWT = posterior wall thickness; RWT = relative wall thickness.

Results

Ethnic differences in ECG measures of left ventricular hypertrophy. The prevalence of tall R waves was significantly higher in blacks than in whites (Table 1). These ethnic differences persisted when normotensive and hypertensive subjects were examined separately but were significant for normotensive subjects only (Table 2). The prevalence of tall R waves within each ethnic group was higher, but not significantly so, in hypertensive than in normotensive subjects.

Ethnic differences in echocardiographic variables. Both measures of wall thickness were greater in blacks than in whites (Table 1). In contrast, mean left ventricular end-diastolic dimension was similar in black and white women and significantly lower in black than in white men. These ethnic differences persisted when normotensive and untreated hypertensive subjects were examined separately (Table 2). Wall thicknesses for all four gender/ethnic groups were generally higher in hypertensive than in normotensive subjects. In men, left ventricular end-diastolic dimension was higher in white hypertensive than in normotensive subjects (47.9 vs. 47.7 mm) but lower in black hypertensive than in normotensive subjects (44.5 vs. 46.6 mm). This ethnic difference in cavity dimension change with hypertension was statistically significant ($p < 0.05$ for the interaction). The ethnic difference in left ventricular mass index was significant in women only but did not persist when normotensive and hypertensive women were compared separately. Indexing left ventricular mass for height did not qualitatively alter these findings. Relative wall thickness was significantly higher in blacks than in whites. The increase in relative wall thickness from normotensive to hypertensive status was also greater in blacks than in whites.

The cumulative frequency plot (Fig. 1) shows the ethnic difference in the distribution of interventricular septal thickness. Only 32% of white men but 53% of black men had an interventricular septal thickness >10 mm. These ethnic differences are even more marked in women: 14% of white women and 38% of black women had an interventricular septal thickness >10 mm.

Age-adjusted means for echocardiographic variables for the whole sample (excluding treated hypertensive subjects) were recalculated after exclusion of people with known and newly diagnosed diabetes and those with evidence of probable coronary heart disease and adjusted for exercise and alcohol intake.

Table 2. Age-Standardized Prevalence of Electrocardiographic Tall R Waves and Mean Blood Pressure and Echocardiographic Measures Comparing Normotensive and Untreated Hypertensive Subjects

	Normotensive Subjects						Untreated Hypertensive Subjects				
	Men			Women			Men			Women	
	White (n = 203)	Black (n = 147)	p Value*	White (n = 260)	Black (n = 169)	p Value*	White (n = 49)	Black (n = 46)	p Value*	White (n = 24)	Black (n = 64)
Tall R waves (%)	5	14	< 0.01	0	7	< 0.001	9	19		0	8
Systolic BP (mm Hg)	120	121		117	123	< 0.001	146	148		148	144
IVST (mm)	9.8	10.1		9.3	9.8	< 0.001	10.5	10.7		9.7	10.2
PWT (mm)	9.5	9.5		9.0	9.4	< 0.001	10.1	10.3		9.1	9.4
LVEDD (mm)	47.7	46.6	< 0.001	43.2	43.5		47.9	44.5	< 0.001	43.0	42.3
LVESD (mm)	33.0	31.4	< 0.001	29.0	28.1	< 0.001	33.8	30.5	< 0.001	28.0	27.6
LVMI (g/m^2)	84.8	84.6		72.3	74.2		88.3	83.8		79.0	77.6
RWT	0.40	0.41		0.41	0.42	< 0.05	0.41	0.45		0.43	0.46

*Blacks versus whites; p values > 0.05 are not shown. Abbreviations as in Table 1.

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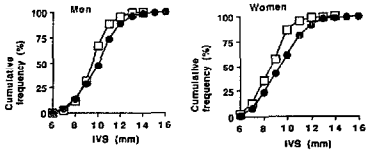

Figure 1. Age-adjusted cumulative frequency plots of interventricular septal thickness (IVS) by ethnic group. **Squares** = whites; **circles** = blacks.

This did not appreciably alter the ethnic differences in any of these variables.

Relation between echocardiographic and ECG measures of left ventricular hypertrophy. For both white and black men, interventricular septal thickness and left ventricular mass index were significantly higher in those with tall R waves on the ECG compared with subjects who had normal findings on the ECG (Table 3). This difference was not observed for cavity dimension.

Relation between body size and echocardiographic variables. In both genders, body size-adjusted interventricular septal thickness was consistently and significantly higher in blacks than whites ($p < 0.05$), and cavity dimension was significantly lower in blacks than in whites (apart from the body mass index and height adjustment in women) (Table 4).

Relation with rest and ambulatory blood pressure. Rest blood pressure was positively correlated with interventricular septal thickness, posterior wall thickness, left ventricular mass index and relative wall thickness in all gender and ethnic groups. The strongest correlations were generally with measures of wall thickness (Table 5). The high correlation between blood pressure and left ventricular mass index in women presumably reflects the strong correlation between blood pressure and both interventricular and posterior wall thickness. Nighttime ambulatory blood pressures correlated significantly with echocardiographic variables in women but not in men. Left ventricular end-diastolic dimension did not correlate significantly with any measure of rest or ambulatory blood pressure in any of the four gender/ethnic groups. These correlations were reduced, but not significantly, when body size was accounted for.

Interventricular septal thickness was significantly higher in blacks than in whites for each blood pressure category ($p = 0.05$) (Fig. 2). This ethnic difference persisted and was enhanced for categories of daytime ambulatory blood pressure ($p = 0.007$). However, the slope of the relation between blood pressure and wall thickness was not different in the two ethnic groups ($p = 0.7$ for interaction test). Rest systolic blood pressure accounted for 4% of the variance in interventricular septal thickness in hypertensive men ($p = 0.2$) and 8% of the variance in hypertensive women. Mean ambulatory systolic blood pressure accounted for a further 7% of the variance in both men and women ($p = 0.09$). The relations between blood pressure and interventricular septal thickness were much weaker in normotensive subjects.

Discussion

Explaining mortality differences—the population approach. We believe that this is the first community-based study to assess the relation between blood pressure and related cardiac disease in black and white populations. Other surveys have studied either clinic (12–15,17,33) or employed (5,16,34) populations or have yet to report (35). A clear understanding of why black people of African descent have a higher mortality from conditions associated with increased blood pressure compared with white populations can only be gained by examining representative samples of these populations, not just those perceived to be at high risk (36).

Ethnic differences in directly measured echocardiographic variables. Wall thickness measures are generally greater in blacks than in whites, and cavity measures are either smaller (in men) or similar (in women). Similar to other reports (14), we show that the ethnic difference in interventricular septal wall thickness is greater than that for posterior wall thickness. Although these mean values are within the clinical normal ranges, and the ethnic difference in mean values are relatively small, we show that this small ethnic difference in mean values reflects a substantial population shift in the distribution of wall

Table 3. Relation Between Mean Age-Adjusted Echocardiographic Variables and Electrocardiographic Tall R Waves by Ethnic Group (men only)

	Whites			Blacks		
	Normal ECG (n = 232)	Tall R Waves (n = 19)	p Value*	Normal ECG (n = 179)	Tall R Waves (n = 49)	p Value*
IVST (mm)	10.1	10.9	< 0.01	10.3	10.9	< 0.05
PWT (mm)	9.7	10.3		9.7	10.4	< 0.01
LVEDD (mm)	47.3	48.8		46.3	46.9	
RWT	0.42	0.43		0.43	0.45	
LVMI (g/m^2)	85.5	99.9	< 0.001	86.2	97.2	< 0.001

*Subjects with normal findings on the electrocardiogram (ECG) versus those with tall R waves within ethnic group; p values > 0.05 are not shown. Normal ECG excludes subjects with Minnesota codes 1-1 to 1-3, 4-1 to 4-3, 5-1 to 5-3 and 7-1; other abbreviations as in Table 1.

Table 4. Age-Adjusted Mean Indexes of Body Size and Interventricular Septal Wall Thickness and Left Ventricular End-Diastolic Diameter Adjusted for Indexes of Weight, Body Surface Area, Body Mass Index and Height*

	Men			Women		
	Whites (n = 252)	Blacks (n = 193)	p Value†	Whites (n = 284)	Blacks (n = 233)	p Value†
Weight (kg)	78.9	75.4	< 0.01	67.5	75.3	< 0.001
Height (m)	1.73	1.71	< 0.001	1.60	1.60	
BSA (m^2)	1.93	1.87	< 0.001	1.70	1.78	< 0.001
BMI (kg/m^2)	26.3	25.8		26.5	29.5	< 0.001
IVST adjusted for						
Weight (mm)	10.0	10.3	< 0.05	9.5	9.8	< 0.05
BSA (mm)	10.0	10.3	< 0.05	9.5	9.9	< 0.01
BMI (mm)	10.0	10.2	< 0.05	9.5	9.8	< 0.05
Height (mm)	10.0	10.3	< 0.05	9.4	9.9	< 0.001
LVEDD adjusted for						
Weight (mm)	47.7	46.5	< 0.01	43.5	42.7	< 0.05
BSA (mm)	47.7	46.5	< 0.01	43.6	42.8	< 0.05
BMI (mm)	47.7	46.3	< 0.001	43.4	42.7	
Height (mm)	47.7	46.3	< 0.01	43.3	43.3	

*Excludes treated hypertensive subjects. †Blacks versus whites; p values > 0.05 are not shown. BMI = body mass index; BSA = body surface index; other abbreviations as in Table 1.

thickness, so that a far greater proportion of black people will have greater than normal values compared with whites. It is precisely those proportions of people in the high normal ranges of risk that account for population differences in morbidity and mortality (36). In general, the closest relation of rest and ambulatory blood pressure with echocardiographic measures was with wall thickness in all four gender/ethnic groups. This suggests that wall thickness is the best measure of left ventricular response to changes in blood pressure when two different populations are compared.

Body size is a major determinant of left ventricular dimension (37,38), and this is acknowledged when calculated left ventricular mass and directly measured cavity dimension are indexed for body surface area (14). There are differences in measures of mean body size in the two ethnic groups, and it may be body size that governs ethnic differences in echocardiographic structure. To test this, we further adjusted wall thickness and cavity dimension for body size. This adjustment resulted in consistent ethnic differences in interventricular septal thickness and left ventricular end-diastolic dimension in both genders. Interventricular septal thickness was greater in blacks than in whites, and left ventricular end-diastolic dimension was generally smaller. Therefore, the observed ethnic difference in wall thickness cannot be accounted for by ethnic differences in body size alone; in fact, adjusting for body size enhanced ethnic differences in ventricular structure.

Unlike other echocardiographic variables, left ventricular end-diastolic dimension did not correlate with any measure of

Table 5. Correlations Between Echocardiographic Measures and Rest and Ambulatory Systolic Blood Pressures (excludes treated hypertensive subjects)

	Ambulatory BP in Whites				Ambulatory BP in Blacks			
	Rest BP in Whites	24-h	Day (9–11 AM)	Night (3–5 AM)	Rest BP in Blacks	24-h	Day (9–11 AM)	Night (3–5 AM)
Men								
IVST	0.30*	0.24†	0.30†	0.06	0.09	0.20‡	0.23	−0.01
PWT	0.18	0.22	0.32*	0.05	0.14	0.22	0.28†	−0.06
LVEDD	−0.04	0.05	−0.04	0.06	0.09	−0.01	−0.04	−0.09
LVMI	0.14	0.23†	0.21†	0.07	0.07	0.20	0.23†	−0.12
RWT	0.15	0.12	0.29†	0.00	0.15	0.16	0.24†	0.00
Women								
IVST	0.38‡	0.42‡	0.37‡	0.33*	0.35†	0.31*	0.33*	0.26†
PWT	0.37‡	0.34*	0.32*	0.31*	0.27	0.32*	0.31†	0.26†
LVEDD	0.13	0.04	0.03	0.10	−0.06	−0.17	−0.07	0.11
LVMI	0.32*	0.28†	0.24†	0.32*	0.27†	0.38‡	0.34*	0.37‡
RWT	0.24†	0.27†	0.26†	0.22	0.25‡	0.22†	0.23†	0.16

*$p < 0.01$, †$p < 0.05$, ‡$p < 0.001$. Data for men are based on 73 whites and 78 blacks; those for women on 79 whites and 89 blacks. Abbreviations as in Table 1.

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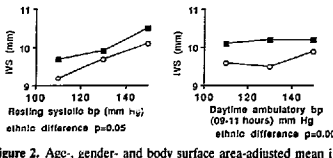

Figure 2. Age-, gender- and body surface area-adjusted mean interventricular septal thickness (IVS) by rest and daytime ambulatory systolic blood pressure (bp) (excludes treated hypertensive subjects). **Circles** = whites; **squares** = blacks.

rest or ambulatory blood pressure in any of the four gender/ethnic subgroups. Although cavity dimension is smaller in black hypertensive than in normotensive subjects, in whites, this cavity dimension is greater in hypertensive subjects. This suggests a possible ethnic difference in the structural responses to hypertension (34). This "concentric remodeling," (i.e., increased wall thickness accompanied by a reduction in cavity dimension) has been demonstrated in a smaller study of hypertensive black Americans (17,39) and is associated with a higher risk of death from heart disease.

Ethnic differences in derived echocardiographic measures of left ventricular hypertrophy. We confirm that altho h relative wall thickness is greater in blacks than in whites, there is no ethnic difference in left ventricular mass index (5,12,13,34). Left ventricular end-diastolic dimension is algebraically the major determinant in the calculation of left ventricular mass index, so that a given percent change in left ventricular end-diastolic dimension will have a substantially greater effect on calculated left ventricular mass index than a similar change in wall thickness. Hence, in men, calculated left ventricular mass is greater in whites than blacks because, even in healthy normotensive subjects, left ventricular end-diastolic dimension is much greater in whites. Indexing left ventricular mass to body surface area, which is also greater in whites, results in a calculated left ventricular mass index that is no different in the two ethnic groups. In contrast, black women have thicker ventricular walls than white women, with no difference in left ventricular end-diastolic dimension. Calculated left ventricular mass is therefore greater in black women, but when indexed for body surface area, left ventricular mass index is again no different in the two ethnic groups.

Derived left ventricular mass index measures have been validated against necropsy findings (10,26), but the ethnic breakdown of the original study populations has not been published. Although calculated left ventricular mass index may be positively associated with blood pressure within ethnic groups, the use of this calculated index in comparisons between ethnic groups may be less helpful. Proportionate changes in wall thickness and chamber dimension do not have the same effect on calculated left ventricular mass, and this may be a problem in comparing blacks, where chamber dimension is reduced in the presence of hypertension, with whites, where this dimension is increased.

Relative wall thickness was generally greater in hypertensive than in normotensive subjects, but this difference was greater for blacks than whites. Again, examination of directly measured indexes will demonstrate why this is so.

Reasons for persistent ethnic differences in echocardiographic measures of left ventricular hypertrophy. We show that ethnic differences in wall thickness persist when age, body size and even blood pressure are taken into account, but rest blood pressure may not adequately characterize the burden of blood pressure that the heart is exposed to. However, we demonstrate that wall thickness is greater at similar levels of both rest and ambulatory blood pressure in blacks than in whites. Although ambulatory blood pressures generally correlate more closely with echocardiographic variables, they are little better than rest measures at explaining the variation in left ventricular hypertrophy. The proportion of the variance in wall thickness explained by ambulatory blood pressure was stronger when hypertensive subjects were examined but not as high as in other studies (33), perhaps because others have only examined selected hypertensive populations. It is therefore unlikely that better characterization of blood pressure would help to account for any more of the variance in wall thickness. Genetic differences may also account for our findings. Wall thickness is greater in normotensive blacks than in whites, and changes in cavity dimension differ by ethnic group.

Conclusions and clinical implications. We conclude that wall thickness measures, adjusted for body size, may be a better marker for comparing left ventricular changes associated with blood pressure in these two ethnic groups than derived measures, which may not take into account population differences in left ventricular structure and response to increased blood pressure. We suggest that directly measured echocardiographic variables should be reported as well as derived measures when different ethnic groups are compared. Wall thicknesses were higher in blacks than in whites in both men and women, and this difference could not be accounted for by ethnic differences in rest or ambulatory blood pressure. This pattern of structural change in response to hypertension (i.e., increased wall thickness and diminished cavity dimension) is associated with a particularly poor prognosis with regard to heart disease (17,39). These findings have important clinical implications when assessing the hypertensive patient of black African descent and deciding when to institute antihypertensive therapy. In view of the increased risk of mortality, it may be beneficial to institute antihypertensive treatment at a lower blood pressure threshold in blacks than in whites. The reasons for the differences in structural response to hypertension in the two ethnic groups remain to be determined.

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