Ethnic Differences in the Hypertensive Heart and 24-Hour Blood Pressure Profile


Abstract—Black hypertensive persons have been observed to have a greater degree of left ventricular hypertrophy than white hypertensives. However, previous studies have matched groups for blood pressure (BP) measured in the clinic, and it has been demonstrated that black hypertensives have an attenuated nocturnal BP dip. Clinic BPs may thus underestimate mean 24-hour BP in this group. To investigate whether the differences in left ventricular hypertrophy can be accounted for by the greater mean 24-hour BP in black hypertensives, 92 previously untreated hypertensives were studied with 24-hour ambulatory BP monitoring and echocardiography. The 46 black hypertensives (24 men and 22 women) were matched with the 46 white hypertensives for age, gender, and mean 24-hour BP. Despite similar mean 24-hour BPs (blacks, 142/93 mm Hg; whites, 145/92 mm Hg; P = .53/.66), the black group had a smaller mean nocturnal dip than the white group (blacks, 8/8 mm Hg; whites, 16/13 mm Hg; P < .01). In addition, mean left ventricular mass index (LVMI) was greater (blacks, 130 g/m²; whites, 107 g/m²; P < .001). Mean 24-hour systolic BP was significantly related to LVMI in both groups (blacks, r = .45, P < .01; whites, r = .56, P < .01). However, systolic BP dip correlated inversely with LVMI only in the black group (blacks, r = -.30, P < .04; whites, r = .05, P = .76). In a multiple regression model, LVMI was independently related to both mean daytime BP and mean nocturnal BP dip in black subjects but only to mean daytime BP in white subjects. In conclusion, the increased left ventricular hypertrophy observed in black hypertensives compared with white hypertensives is not accounted for by differences in mean 24-hour BP. However, LVMI in black hypertensives appears to be more dependent on nocturnal BP than that in white hypertensives; this, coupled with the attenuated BP dip in black hypertensives, suggests that the BP profile rather than 24-hour BP may be important in determining the differences in left ventricular hypertrophy. (Hypertension. 1998;31:1190-1194.)

Key Words: hypertrophy, left ventricular ♦ ethnicity ♦ race ♦ blood pressure monitoring, ambulatory

lectric ventricular hypertrophy has been shown to be a powerful predictor of increased morbidity and mortality, an effect that is independent of other classic risk factors for cardiovascular disease, including high BP. Studies from both the United Kingdom and the United States have shown that the prevalence of hypertension is higher in black than in white populations, as is the prevalence of LVH. This may in part explain why the mortality rate of black hypertensives exceeds that of white hypertensives, even when the greater prevalence of hypertension is taken into account. The observation of an increased left ventricular mass in black hypertensives has been confirmed in studies in which both black and white patients have been carefully matched for clinic BP. However, mean 24-hour BP correlates better with left ventricular mass than clinic BP, and it is possible that the racial differences in left ventricular mass are a reflection of greater mean 24-hour BP in black hypertensives. It has been demonstrated that black hypertensives have a blunted nocturnal BP dip compared with white hypertensives, and hence, even though matched for clinic (daytime) BPs in previous studies, the black groups may have had higher average 24-hour BP levels than the white groups. Although a greater mean 24-hour BP has been suggested as a likely cause for the excess of LVH in black hypertensives, no previous study has directly addressed this issue. This study was designed to evaluate this possibility by comparing LVH levels in black and white hypertensives matched for 24-hour BP.

Methods
Forty-six consecutive white patients with previously untreated essential hypertension who had been referred to the hypertension clinic at our institution and who underwent routine echocardiography and 24-hour ambulatory BP monitoring were compared with 46 previously untreated black hypertensives who had undergone similar investigations. The black patients were selected retrospectively from computerized clinic records on the basis of being matched with the white patients for gender and having an age within 3 years and a mean 24-hour ambulatory BP level within 5 mm Hg of the corresponding subject. Our black study population consisted of West African immigrants and both Caribbean immigrants and their British-born sons and daughters. Our white population was European, largely Anglo-Saxon and Celtic.
All patients had normal systolic function determined with two-dimensional echocardiography and no clinical or Doppler evidence of valvular stenosis or regurgitation. Patients were excluded if they had a history of ischemic heart disease, congestive cardiac failure, peripheral vascular disease, diabetes mellitus, or alcohol abuse or an abnormal hematological or biochemical profile.

Echocardiography
Each patient underwent two-dimensional and Doppler echocardiography (Pass II 3.3-MHz transducer; General Electric). IVS, PWT, and LVID were measured at end diastole with the use of the M-mode strip in each of three separate frames in accordance with the Penn convention. Results of the three measurements for each structure were averaged. Left ventricular mass was calculated from Penn measurements with the cubed formula: Left Ventricular Mass = 1.04[(IVS + PWT)3] − (LVID)2 − 14 g. This figure was then divided by body surface area to give a value for LVMI.

24-Hour Ambulatory BP Monitoring
In addition, each patient underwent 24-hour ambulatory BP monitoring with the use of SpaceLabs 90207 machines, the accuracy of which has been validated. Measurements were made every 30 minutes throughout the day and hourly at night. Ambulatory monitoring was deemed acceptable if >90% of readings were recorded. Mean BP was calculated from the readings during the entire 24-hour period. Daytime was considered to be between 6:00 AM and 12:00 midnight, and nighttime was from 12:00 midnight to 6:00 AM. BP dip was calculated by subtracting mean nighttime BP from mean daytime BP.

Statistical Analysis
Student’s unpaired two-tailed t-test was used for statistical analysis. Simple regression analysis was used to examine whether correlations existed. Values of \( P < .05 \) were taken to be statistically significant.

### Results
Characteristics of black and white subjects are given in the Table. Although the black group had a smaller mean nocturnal dip compared with the white group, the average 24-hour systolic, diastolic, and mean arterial BPs were by design similar for the two groups, as were the mean ages and proportion of men. In addition, mean body mass index and reported duration of hypertension were similar in the two groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Black Hypertensives</th>
<th>White Hypertensives</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>43 (1.9)</td>
<td>44 (2.0)</td>
<td>.5</td>
</tr>
<tr>
<td>Gender, M/F</td>
<td>24/22</td>
<td>24/22</td>
<td>. . .</td>
</tr>
<tr>
<td>Reported duration of hypertension, mo</td>
<td>35 (7.9)</td>
<td>40 (9.7)</td>
<td>.68</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.6 (0.68)</td>
<td>26.2 (0.78)</td>
<td>.2</td>
</tr>
<tr>
<td>Clinic BP, mm Hg</td>
<td>158 (3.0)/96 (1.6)</td>
<td>164 (3.3)/96 (1.8)</td>
<td>.15/.83</td>
</tr>
<tr>
<td>Mean 24-hour BP, mm Hg</td>
<td>142 (2.2)/93 (1.6)</td>
<td>145 (2.7)/92 (1.5)</td>
<td>.53/.66</td>
</tr>
<tr>
<td>Mean daytime BP, mm Hg</td>
<td>145 (2.2)/95 (1.7)</td>
<td>149 (2.7)/95 (1.5)</td>
<td>.22/.9</td>
</tr>
<tr>
<td>Mean nighttime BP, mm Hg</td>
<td>136 (2.6)/86 (1.7)</td>
<td>132 (3.2)/81 (2.0)</td>
<td>.32/.06</td>
</tr>
<tr>
<td>Mean nocturnal BP dip, mm Hg</td>
<td>8 (1.4)/8 (1.2)</td>
<td>16 (1.8)/13 (1.3)</td>
<td>&lt;.01/=.01</td>
</tr>
<tr>
<td>IVS, mm</td>
<td>11.0 (0.3)</td>
<td>9.9 (0.3)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>LVID, mm</td>
<td>49.0 (0.8)</td>
<td>47.4 (0.8)</td>
<td>.18</td>
</tr>
<tr>
<td>PWT, mm</td>
<td>11.2 (0.3)</td>
<td>9.9 (0.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>130 (4.4)</td>
<td>107 (4.9)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

BP indicates blood pressure. Values are mean ± SEM.

Selected Abbreviations and Acronyms
BP = blood pressure
IVS = interventricular septal wall thickness
LVH = left ventricular hypertrophy
LVID = left ventricular internal diameter
LVMI = left ventricular mass index
PWT = posterior wall thickness

2 \( r \) values were taken to be statistically significant.

BP: blacks, \( r = .45, P < .01 \); whites, \( r = .56, P < .001 \), as did mean nighttime BPs (systolic BP: blacks, \( r = .52, P < .01 \); whites, \( r = .51, P < .001 \)). Mean 24-hour BP was significantly related to LVMI in both black and white groups (systolic BP: blacks, \( r = .45, P < .01 \); whites, \( r = .56, P < .01 \)) (Fig 1). The results were similar when men and women were analyzed separately. When both the intercepts and slopes of the relationship between mean 24-hour BP and LVMI are considered, the equation for blacks was \( y = 0.92x - 0.81 \) compared with \( y = x - 36.81 \) for whites. The slopes were not significantly different (\( P = .83 \)), but the intercepts were (\( P < .001 \)).

Systolic BP dip correlated inversely with LVMI only in the black group (blacks, \( r = - .30, P = .04 \); whites, \( r = .05, P = .76 \)) (Figs 2). When both intercepts and slopes for this relation were examined, the equation for blacks was \( y = -0.93x + 138.02 \) compared with \( y = -0.13x + 109.39 \) for whites. The difference between the slopes was not statistically significant (\( P = .19 \)), but the difference between the intercepts was (\( P = .007 \)). In a multiple regression model containing LVMI as the dependent variable and mean daytime BP and mean nighttime BP as independent variables, LVMI was independently related in black subjects only to nighttime blood pressure (daytime BP, \( t = - .03, P = .77 \); nighttime BP, \( t = 2.5, P = .02 \)) in white subjects, the opposite was true (daytime BP, \( t = 2.0, P = .05 \); nighttime BP, \( t = 0.6, \)
When nocturnal systolic BP dip replaced mean nighttime BP in the model, LVMI in black subjects was independently related to both mean daytime BP and systolic nocturnal BP dip (daytime BP, \( t = 3.2, P < .01 \); nocturnal systolic BP dip, \( t = -2.5, P = .02 \)); in white subjects, LVMI was independently related only to mean daytime BP (daytime BP, \( t = 4.5, P < .01 \); nocturnal systolic BP dip, \( t = -0.7, P = .50 \)).

The percentage of variance of LVMI accounted for by mean 24-hour BP was 19.3%; 6.9% was accounted for by nocturnal systolic BP dip, and 10% was accounted for by race.

**Discussion**

This is the first study to directly address ethnic cardiac structural differences in relation to 24-hour BP profile differences. Although the black group had an attenuated nocturnal BP dip compared with the white group, mean 24-hour BPs were similar (mean 24-hour arterial pressure, 110 mm Hg in the white group and 109 mm Hg in the black group). Despite this close matching for mean 24-hour BP, black hypertensives still had significantly greater IVS, PWT, and LVMI than white hypertensives. Therefore, the previous suggestion that ethnic cardiac structural differences may be explained by differences in mean 24-hour BP (differences that are masked when subjects are matched for clinic BP)\(^{12,13} \) is refuted. However, more careful analysis suggests that nocturnal BP is more important in determining LVMI in black hypertensives than in white hypertensives. This statement is supported by that fact that the relation between LVMI and mean daytime BP in black subjects was dependent on mean nighttime BP; the opposite was true in white subjects. In addition, mean nocturnal systolic BP dip was inversely related to LVMI independent of mean daytime BP in black subjects. This was not the case with white subjects. It is possible that the importance of nocturnal BP in black subjects coupled with the attenuated nighttime BP dip may, at least in part, explain the ethnic variations observed in cardiac structure. When the relation between LVMI and mean 24-hour BP was examined, it was apparent that the difference between black and white subjects was wholly related to the intercept rather than the slope of the relation, suggesting that for every level of BP, LVMI is a constant amount greater in black than in white...
subjects. When the relation between LVM1 and mean nocturnal systolic BP dip was considered, there was a large difference in the slopes between black and white subjects; although this did not reach statistical significance, the present study may not have had sufficient power to confirm a significant difference. We interpret the results as suggesting that factors other than BP probably mediate a large proportion of the ethnic LVM1 differences observed (as indicated by the difference in origins of the relations between LVM1 and mean 24-hour BP). However, it is possible that nocturnal systolic BP dip differences do provide a link between hemodynamic and LVM1 variations.

There are many ways that LVH may be mediated by factors other than hemodynamic ones, and part of the apparent ethnic differences in LVH might be due to one or more of these. There are marked ethnic sociocultural differences, and neurohumoral variations have long been recognized. Black hypertensives have lower renin levels than their white counterparts\(^{19,20}\); there is, however, little evidence that renin levels are correlated with LVH.\(^{21}\) More recent interest has focused on possible genetic mediators of LVH with the hypothesis that ACE genotype may influence LVH and the recognition of racial variations in the frequency of the different ACE genotypes. However, after an initial indication that deletion homozygotes may have an increased LVM1,\(^{22-24}\) more recent studies, including from the large Framingham population,\(^{25,26}\) have not demonstrated any relation between ACE genotype and LVH. Further candidate genes continue to be proposed and require evaluation. Variations in sympathetic activity might also account for some of the differences observed. This

**Figure 2.** Relation between mean nocturnal systolic BP dip and LVM1 for blacks (A) and whites (B).
might be particularly relevant if there were differing 24-hour profiles of sympathetic activity. For instance, a higher nocturnal sympathetic drive in black subjects might be responsible for an increased LVMI directly and indirectly by increasing nocturnal BP. This area requires further investigation.

In conclusion, the increased LVMI observed in black hypertensives compared with white hypertensives cannot be explained by differences in mean 24-hour BP. However, nocturnal BP in black hypertensives is a more important determinant of LVH than in white hypertensives, and the differing BP profiles, with an attenuated nocturnal BP dip apparent in the black subjects, may at least in part explain the increased LVMI.

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References
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