Racial differences in cardiac structure and function in essential hypertension

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Abstract

***Objective*—To assess racial differences in cardiac structure and function in patients presenting with previously untreated hypertension.**

***Design*—Untreated black patients with hypertension were compared with untreated white patients matched for age and sex. Both groups had similar body mass indices, blood pressures, and reported duration of hypertension.**

***Setting*—Cardiovascular risk factor clinic for outpatients.**

***Subjects*—36 men and 22 women with untreated essential hypertension.**

***Main outcome measures*—Variables of heart structure and function on cross sectional and Doppler echocardiography.**

***Results*—The black patients had a significantly greater interventricular septal thickness (mean 1·23 (95% confidence interval 1·14 to 1·33) *v* 1·09 (1·02 to 1·16) cm; P=0·02) and posterior wall thickness (mean 1·14 (1·07 to 1·22) *v* 0·96 (0·88 to 1·03) cm; P=0·001) than the white patients, although left ventricular internal diameter was not significantly different (mean 4·90 (4·68 to 5·12) *v* 4·82 (4·64 to 5·01) cm; P=0·59). This resulted in a significantly greater left ventricular mass index (mean 151 (137 to 164) *v* 120 (107 to 133) g/m²; P=0·001) and relative wall thickness (mean 0·47 (0·43 to 0·51) *v* 0·40 (0·37 to 0·42) cm; P=0·004) in the black patients. Comparison of Doppler measures of left ventricular diastolic function showed a significantly longer isovolumic relaxation time in black patients (mean 107 (98 to 116) *v* 92 (83 to 101) ms; P=0·02) compared with white patients, although peak early to atrial filling ratios were similar in both groups (mean 1·14 (0·95 to 1·32) *v* 1·04 (0·94 to 1·15); P=0·37).**

***Conclusion*—Among previously untreated hypertensive patients, black subjects compared with white subjects have significantly higher left ventricular mass index and relative wall thickness, as well as more impairment of left ventricular function during diastole.**

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Introduction

Studies from the United States and the United Kingdom have shown that the prevalence of hypertension is higher in black than in white populations.[1-4] Furthermore, the prevalence of left ventricular hypertrophy, a powerful independent predictor of sudden death, cardiovascular disease, and cardiac failure, is much higher in black hypertensive patients.[3,5] The prevalence of electrocardiographically determined left ventricular hypertrophy in black patients with hypertension ranges from two to six times that present in white patients, depending on the criteria used. In contrast, the results of echocardiographically based comparisons of the left ventricle in black and white hypertensives have been less impressive. In a biracial group of patients who exhibited similar differences in electrocardiographically determined left ventricular hypertrophy to those described in previous studies (more among black subjects) there was no difference in left ventricular mass index determined echocardiographically, although the relative wall thickness was greater in black subjects.[5] Similar changes in cardiac structure were seen in one study,[6] though two others have observed increases in both relative wall thickness and left ventricular mass index in black subjects.[7,8] A further study has found no structural differences between black and white subjects with hypertension.[9] One possible reason for the differences in echocardiographic studies of black subjects with hypertension to date is the effect of drug treatment. Although most studies were done after withdrawal of treatment for three to four weeks, the effects of previous treatment are uncertain.

In addition to changes in cardiac structure, change in cardiac function—for instance, altered left ventricular diastolic function—are known to occur as an early manifestation of hypertension. This has been observed in a range of patients, from those with left ventricular hypertrophy to those with mild and borderline hypertension with normal ventricular size.[10,11] Not all groups of patients with left ventricular hypertrophy have associated diastolic dysfunction: athletes with left ventricular hypertrophy as a result of intensive physical training have a normal pattern of ventricular filling.[12,13] Changes in the left ventricle due to hypertension consist of both myocyte hypertrophy and an increase in the collagen matrix. Impaired diastolic function probably reflects qualitative changes in the ventricle, and increases in the collagenous matrix which result in a stiffer, less compliant ventricle may be more important determinants of diastolic function than muscular hypertrophy. Thus alterations in left ventricular diastolic function in hypertension provide additional information about the consequences of structural changes visualised by cross sectional echocardiography.

We assessed racial differences in cardiac structure and function at presentation in patients with previously untreated hypertension.

Subjects and methods

PATIENTS

Twenty nine consecutive black patients with previously untreated essential hypertension (diastolic blood pressure >90 mm Hg or systolic >160 mm Hg, or both) who had been referred to the hypertension clinic at this hospital and who underwent routine echocardiography were compared with 29 previously untreated white patients with hypertension who had undergone echocardiography. The white patients were selected retrospectively from computerised clinic records on the basis of being matched with the black patients for sex and having an age within five years of the corresponding subject. Both groups had a similar mean body mass index and mean arterial pressure and a similar reported duration of hypertension (based on when patients had first been told that their blood pressure was high). All patients had normal systolic function determined by cross sectional echocardiography and no clinical or Doppler evidence of valvar stenosis or regurgitation. Patients were excluded if

they had a history of ischaemic heart disease, congestive cardiac failure, peripheral vascular disease, diabetes mellitus, or alcohol abuse or if they had an abnormal haematological or biochemical profile.

ECHOCARDIOGRAPHY

Each patient underwent cross sectional and Doppler echocardiography with a phased array sector scanner (General Electric Pass II, 3·5 MHz transducer). Interventricular septal wall thickness, posterior wall thickness, and left ventricular internal diameter were measured from the left ventricular short axis view with cross sectional guided echocardiography. Special attention was paid to obtaining a precise cross sectional "on axis" view of the left ventricle at the level of the papillary muscle tip. The papillary muscles were then bisected by the M mode beam and simultaneous cross sectional and M mode images were obtained. Leading edge to leading edge measurements were made at the end of diastole in each of three separate frames in accordance with the guidelines of the American Society of Echocardiography and the results averaged.[14] Left ventricular mass was calculated by using the cubed formula: mass = 1·04 ((interventricular septal wall thickness + left ventricular internal diameter + posterior wall thickness)^3 − (left ventricular internal diameter)^3). This figure was then adjusted for body surface area to give a value for left ventricular mass index. Relative wall thickness was taken as the ratio of 2 × (posterior wall thickness/left ventricular internal diameter).

DOPPLER STUDIES

Doppler echocardiography was used to measure both left ventricular filling and isovolumic relaxation as indices of diastolic function.[15] Pulsed Doppler examination of transmitral flow for assessment of left ventricular filling was done with reference to the cross sectional echocardiographic image from the apical view of the four chambers. The sample volume was located between the mitral annulus and the tips of the mitral leaflets and the position adjusted to maintain the sample volume at an angle as near parallel to transmitral flow as possible by means of an audible signal and spectral velocity display. When the maximum transmitral velocity for the early filling wave was detected the velocity profile was recorded with the patient in passive end expiration. The peak flow velocities of the early and atrial waves were measured from three consecutive cardiac cycles displaying the highest measurable velocity profiles and an average value used for subsequent analysis.

Continuous wave Doppler was used to examine an area between the mitral and aortic valves on the apical view of the four chambers so that both mitral and aortic flow patterns were visualised. The velocity profiles were recorded with the patient in passive end expiration. Isovolumic relaxation time was taken to be the time interval between the end of the aortic velocity envelope and the onset of the early filling wave. Measurements were made from three consecutive cycles and the results averaged.

All measurements and analyses were carried out by one of two observers (JM or MS).

BLOOD PRESSURE

Blood pressure was measured with an automated blood pressure monitor (Sentron) with the patient in a supine position after each echocardiographic examination.

STATISTICS

Data are expressed as means with 95% confidence intervals. Student's unpaired two tailed t test was used for statistical analysis. Means are presented with 95% confidence intervals and P values for the differences. Simple regression analysis was used to examine whether correlations existed, and an analysis of covariance was performed to assess the relation of isovolumic relaxation time. P values of $< 0·05$ were taken to be significant.

Results

The two groups of patients were similar with regard to age, sex, body mass index, reported duration of hypertension, and blood pressure. The white patients had a faster mean heart rate (table I).

ECHOCARDIOGRAPHIC FINDINGS

The black patients had a significantly greater interventricular septal wall thickness and posterior wall thickness than white patients, although the left ventricular internal diameter was not significantly different. This resulted in a significantly greater left ventricular mass index and relative wall thickness in black patients (table II). Mean arterial pressure correlated with left ventricular mass index in the black and white groups individually as well as the group as a whole. (Black subjects $r = 0·36$, P = 0·05; white subjects $r = 0·38$, P = 0·04; whole group $r = 0·32$, P = 0·006.)

DOPPLER FINDINGS

Comparison between the patients showed that the isovolumic relaxation time was significantly longer in black patients with hypertension, but there was no difference in the peak early filling velocities, the peak atrial filling velocities, or the peak early to atrial velocity ratio (E/A ratio) between the two groups (table III). When compared with data from normal subjects[16] 18 of the 29 black hypertensive patients (62%) had an abnormal diastolic function for their age, as characterised by a reduced peak early to atrial velocity ratio and a prolonged isovolumic relaxation time compared with six of the 29 white hypertensive patients (21%).

There was no significant correlation between the E/A ratio or the isovolumic relaxation time and left ventricular mass index in either black or white subjects or in the group as a whole. (Black subjects E/A ratio and left ventricular mass index $r = -0·09$, P = 0·66; white subjects $r = -0·25$, P = 0·19 and $r = 0·09$, P = 0·64; whole group $r = 0·13$, P = 0·28 and $r = 0·1$, P = 0·4). The isovolumic relaxation time was significantly related to black race, and this relation was found to be independent of left ventricular mass index on analysis of covariance (F ratio = 4·69, P = 0·03).

TABLE I—*Clinical findings in black and white patients with hypertension. Figures are mean values (95% confidence intervals)*

Variable	Black patients (n = 29)	White patients (n = 29)	Mean difference	P value
Men/women	18/11	18/11	—	—
Age (years)	45 (40 to 49)	47 (43 to 52)	2 (−3·6 to 8·5)	0·42
Body mass index (kg/m^2)	28·2 (25·7 to 30·6)	26·9 (24·6 to 29·1)	1·3 (−1·9 to 4·5)	0·60
Mean arterial pressure (mm Hg)	124 (120 to 128)	125 (121 to 129)	1 (−5 to 7)	0·71
Heart rate (beats/minute)	64 (60 to 68)	71 (67 to 76)	7 (1 to 13)	0·03
Reported duration of hypertension (months)	42 (22 to 62)	40 (15 to 65)	2 (−29 to 33)	0·89

TABLE II—*Left ventricular anatomy in black and white patients with hypertension. Figures are mean values (95% confidence intervals)*

Measure	Black patients (n = 29)	White patients (n = 29)	Mean difference	P value
Interventricular septal wall thickness (cm)	1·23 (1·14 to 1·33)	1·09 (1·02 to 1·16)	0·10 (0·02 to 0·26)	0·02
Left ventricular internal diameter (cm)	4·90 (4·68 to 5·12)	4·82 (4·64 to 5·01)	0·1 (−0·20 to 0·35)	0·59
Posterior wall thickness (cm)	1·14 (1·07 to 1·22)	0·96 (0·88 to 1·03)	0·1 (0·08 to 0·29)	0·001
Left ventricular mass index (g/m^2)	151 (137 to 164)	120 (107 to 133)	31 (12·1 to 49·1)	0·001
Relative wall thickness (cm)	0·47 (0·43 to 0·51)	0·40 (0·37 to 0·42)	0·07 (0·03 to 0·12)	0·004

TABLE III—*Doppler indices in black and white patients with hypertension. Figures are mean values (95% confidence intervals)*

Measure	Black patients (n=29)	White patients (n=29)	Mean difference	P value
Peak velocity early filling wave (E) (cm/s)	63·9 (58·2 to 69·5)	62·3 (57·1 to 67·5)	1·6 (−6·0 to 9·1)	0·68
Peak velocity atrial filling wave (A) (cm/s)	60·8 (55·3 to 66·4)	61·2 (57·0 to 65·5)	0·4 (−6·4 to 7·2)	0·12
E/A ratio	1·14 (0·95 to 1·32)	1·04 (0·94 to 1·15)	0·1 (−0·11 to 0·30)	0·37
Isovolumic relaxation time (ms)	107 (98 to 116)	92 (83 to 101)	15·0 (2·5 to 27·1)	0·02

Discussion

Our major findings are that in previously untreated hypertensive patients black subjects have a significantly higher left ventricular mass index and relative wall thickness and more impairment of left ventricular diastolic function than white subjects matched for sex, age, body mass index, and duration and degree of hypertension.

The observations on racial differences in cardiac structure are compatible with those from some studies[7,8] but not others.[5,6,9] They would seem to support the widely held belief that black patients with hypertension suffer a greater degree of left ventricular hypertrophy than white patients for a given blood pressure.

Differences between this and other studies may be explained in part by inadequate matching of the groups compared or be due to the differential effects of previous drug treatment. Black and white patients are likely to have been treated with different classes of drugs as it is recognised that black patients respond less well to β blockers and angiotensin converting enzyme inhibitors. Since there is a variable effect on left ventricular regression depending on the class of agent used, the treatment regimens supplied to the two groups may have caused a differing response in their left ventricles, perhaps masking any differences that were originally present. Another possible explanation is that black subjects in the United Kingdom and United States differ, the latter probably having more "white" genes. Most previous studies have been done in the United States, and this might account for the less clear differences detected.

REASONS FOR VARIATION

There are several possible reasons for the racial variation that we have observed.

Firstly, black patients with hypertension tend to show a smaller nocturnal dip in blood pressure than white patients[17-19] so daytime measurements in a clinic may underestimate the total 24 hour blood pressure load in this group. The 24 hour load has been shown to have a stronger correlation than blood pressure measured in a clinic with left ventricular hypertrophy; this increased total 24 hour blood pressure load for a similar blood pressure measured in a clinic in the black patients might explain their greater left ventricular mass index. A complicating factor is the observation that even in normotensive populations, black subjects show a smaller nocturnal dip in blood pressure than white subjects, once again leading to a greater total 24 hour blood pressure load in black subjects.[17] To gain a proper insight into hypertensive cardiac adaptation it is important to have good data on the structure of the normal heart. Unfortunately there is little information on racial variations in normal hearts. One study has found no differences in cardiac structure in normal black and white subjects,[6] while another has found a greater relative wall thickness in the black subjects with a similar left ventricular mass index.[20]

Secondly, a higher total peripheral resistance and lower cardiac output have been noted in black hypertensives[6]; these together with our findings on cardiac structure may be considered as evidence of differing remodelling of both heart and blood vessels in response to hypertension in this group. Increased wall thickness in the resistance arteries would be compatible with a raised total peripheral resistance if the lumen was simultaneously narrowed and would result in a lower cardiac output. Alternatively, there may be primary cardiovascular differences between the races, and it may be an increase in peripheral resistance or left ventricular mass that makes black subjects more likely to develop hypertension. Not all studies, however, have shown racial variations in haemodynamics,[7,21] and there is a lack of information on normal populations.

A third possibility is that the racial differences observed may have a neurohumoral basis. Black patients with hypertension have lower renin concentrations than similar white patients[22,23]; there is, however, little evidence that concentrations are correlated with left ventricular hypertrophy.[24] Alternatively while sympathetic tone might be expected to influence the size of the left ventricle the slower pulse rate among black subjects undermines any importance of this mechanism.

LEFT VENTRICULAR DIASTOLIC FUNCTION

Much attention has recently been focused on left ventricular diastolic function in hypertension. Abnormal diastolic function has been shown to be the earliest cardiac manifestation of hypertension[10] and while it is usually asymptomatic it may cause cardiac failure even in the presence of normal systolic function.[25] In addition, diastolic dysfunction is probably a precursor of systolic impairment, an important cause of increased morbidity and mortality in hypertension. At present the prognostic importance of diastolic dysfunction is not known. Important determinants of diastolic function are age,[16] haemodynamic factors such as blood pressure[11] and heart rate,[26] and structural variables of the heart.[27] In particular, left ventricular mass has been found to be related to diastolic impairment in some[27,28] though not all studies.[29]

Doppler echocardiography is an accepted technique for assessing diastolic function and Doppler derived measurements of isovolumic relaxation time and peak E/A ratio are accurate and reproducible.[30] We found that isovolumic relaxation time was greater in black patients, though left ventricular filling as assessed by measuring the E/A ratio was similar in both sets of subjects. In addition, isovolumic relaxation time was significantly related to black race. These findings and the fact that a greater proportion of black patients had a considerable impairment of diastolic function as characterised by both an abnormal E/A ratio and isovolumic relaxation time lead us to conclude that black subjects with hypertension have more severe diastolic dysfunction.

Heart rate is a possible confounding factor since it was different in the two groups. The difference in heart rate between the groups, however, was small and is unlikely to have had a large impact on the variables of diastolic function. It should be noted that there is little information on racial variations in normal hearts and that previous normal data are likely to have been derived from a predominantly white population.[16] Thus it is difficult to know for certain whether the differences we have observed are related to hypertension or are normal racial variants.

Diastolic dysfunction is related to left ventricular hypertrophy,[27,28] and so these results may be taken to be a reflection of the increased left ventricular mass index in the black subjects. The ventricular response to high blood pressure, however, results in both cardiac myocyte hypertrophy and an increase in the collagen

Clinical implications

- Hypertension is more common in black subjects than white subjects
- Left ventricular hypertrophy is a powerful predictor of cardiovascular complications in patients with hypertension
- At presentation black patients with hypertension have more severe cardiac abnormalities than white patients, as manifest by a greater degree of left ventricular hypertrophy and a greater impairment of diastolic function
- Prospective studies are required to determine whether these racial differences are important

interstitial matrix. Diastolic dysfunction is probably a result of an increase in the collagen element of the ventricle making it stiffer and impairing relaxation. Thus our findings would imply that the greater left ventricular mass in the black subjects is due to an increase in both cardiac myocyte size and also collagen matrix.

QUESTIONS RAISED

Limitations of this study include the possible confounding nature of social class. These data were not available for our patients. Since black subjects are liable to come from a lower social class it is difficult to separate an effect due to this from true racial differences. In addition, although the black and white groups had a similar reported duration of hypertension, the black patients may have presented at a later stage and the changes observed may relate to a delayed diagnosis of hypertension in this group. Whatever the cause of the more pronounced cardiac changes in the black patients, the obvious question is whether these racial cardiac differences are important.

Left ventricular hypertrophy is well documented as a strong risk factor for cardiovascular morbidity and mortality in hypertension,[31] and the increased hypertrophy in black patients may be regarded as ominous. This would provide a link with earlier studies that found black patients with hypertension to be at higher risk of cardiovascular death than white subjects.[32 33] Not all studies agree on this point, however,[34 35] and it is still contentious whether black patients with hypertension still fare worse when other risk factors for cardiovascular disease are taken into account and when they are given equal access to medical care.[32]

As many questions have been raised as answered by these data. It is important that further observations are made, both in racial variations in hypertension and equally important in normal subjects. This will help the management of hypertension in black patients and also give valuable clues to the underlying pathophysiology in essential hypertension.

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