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Mild hypertension in patients with suspected dilated cardiomyopathy: cause or consequence?

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Summary

This study was undertaken to clarify the relationship between mild transient hypertension and dilated cardiomyopathy. Fifty-five patients were studied:

group 1 —controls (12 patients),
group 2 —hypertensives without clinical evidence of heart failure (14 patients),
group 3 —patients with hypertensive heart failure and diastolic blood pressure above 100 mmHg (10 patients),
group 4 —patients with possible dilated cardiomyopathy with mild hypertension, i.e. diastolic blood pressure of 90–100 mmHg (8 patients),
group 5 —patients with dilated cardiomyopathy and normal blood pressure (11 patients).

The haemodynamic status and cardiac contractility indices were measured in each patient on admission, using M-mode echocardiography. Serum sodium and potassium as well as the urinary sodium, potassium and vanillyl mandelic acid excretions were also measured. The stroke volume, cardiac output and cardiac index fell with heart failure, but much more remarkably in group 4. The peripheral vascular resistance was higher in groups 2, 3 and 4 than in groups 1 and 5; so also were the aortic diameter, left posterior wall thickness and left ventricular mass. The plasma volume, aldosterone and cortisol levels were higher and the urinary sodium and potassium excretion lower in patients with heart failure (groups 3, 4 and 5). It is concluded that the raised blood pressure found in some patients suspected to have dilated cardiomyopathy is not due to the haemodynamic and biochemical changes that occur in heart failure. Such patients are 'chronic' hypertensives with hypertensive heart failure. Their presenting blood pressure is low because of their markedly reduced cardiac output.

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Résumé

Cette étude a été entreprise dans le but de clarifier les rapports entre l'hypertension peu sévère et transitoire d'une part et la cardiomyopathie dilatée d'autre part. Cinquante-cinq patients ont été étudiés:

1er groupe —contrôle (12 patients),
2e groupe — des cas sûrs d'hypertension avec arrêt cardiaque (14 patients),
3e groupe — patients avec arrêt cardiaque hypertensif et tension artérielle diastolique au-dessus de 100 mmHg (10 patients),
4e groupe — patients atteints possiblement d'une cardiomyopathie dilatée avec tension artérielle entre 90 et 100 mmHg (8 patients),
5e groupe — patients avec cardiomyopathie dilatée et tension artérielle normale,

A son admission à l'hôpital, le statut hémodynamique et les indices de contractilité cardiaque de chaque patient ont été mesurés utilisant un échocardiographie M-mode. On a également mesuré les excrétions de sérum de sodium, du sérum de potassium, du sodium et potassium urinaire et de l'acide vanillyl mandélique. Surtout dans le 4e groupe on remarque une chute dans le volume d'apoplexie, de production cardiaque et des indices cardiaques en cas d'arrêt cardiaque. La résistance périphérale vasculaire était plus élevée dans les 2e, 3e et 4e groupes dans les 1er et 5e

groupes. On a fait la même constatation en ce qui concernait le diamètre aortique, l'épaisseur de la paroi postérieure de gauche et la masse ventriculaire de gauche. Le volume du plasma, les niveaux de l'aldosterone et du cortisol étaient plus élevés tandis que les excretions du sodium urinaire et de potassium étaient moins élevés dans les patients souffrant d'arrêt cardiaque (3, 4 et 5e groupes). La conclusion en était que la tension artérielle élevée qu'on retrouve chez certains patients soupçonnés d'être atteints de la cardiomyopathie dilatée, ne résulte pas des changements hémodynamiques et biochimiques dûs à l'arrêt cardiaque. De tels patients sont plutôt des hypertensifs 'chroniques' — avec arrêt du coeur hypertensif. Leur tension artérielle est peu élevée à cause de la production cardiaque réduite.

Introduction

Dilated cardiomyopathy (DCM) is a common cause of heart failure in most parts of the tropical and sub-tropical areas of the world [1, 2]. In Nigeria, for example, clinical and autopsy studies have shown that DCM is next to hypertension as the most common cause of heart failure [3–5].

One of the clinical features that has been associated with DCM in several published reports is the finding of an elevated blood pressure in some patients during the period of heart failure [4]. The hypertension is usually mild and transient, reverting to normal with treatment of heart failure and remaining normal after the condition has improved. Potain in 1889 [6] described it as the high blood pressure of failure and it was called 'hypertension of decompensation' by Gallavardin and Hirschfelder, both in 1910 [7, 8]. Recently, it was labelled as 'reactive hypertension' because it was thought to be due to intense peripheral vasoconstriction, increased cathecholamine secretion, sodium retention and other biochemical disturbances that accompany heart failure [9, 10].

Conversely, there are suggestions that the hypertension seen in these patients is not due to those factors just listed, but that the patients are primarily hypertensives whose blood pressures fell to lower levels on development of heart failure because of poor myocardial function [11–15]. These authors contend that a failing left ventricle cannot achieve an increase in mean blood pressure and that there are well-documented hypertensives who lost the need for hypotensive drugs as their hearts got larger [12]. Normalization of blood pressure during treatment for heart failure is thought to be due to a combination of bed rest, low salt diet and diuretics, measures that are known to lower blood pressure [14].

In several African countries, hypertension is very common and heart failure is its commonest complication [10]. Mild hypertension also occurs in many of the patients considered to have DCM. Difficulties, therefore, often rise concerning what level of diastolic blood pressure should be used to separate hypertensive heart failure (HHF) from DCM with mild hypertension, and various publications in the literature have not been helpful. For instance, some publications have quoted figures as low as 100 mmHg [16] and others as high as 130 mmHg [1], as the cut-off level of diastolic blood pressure for separating these two clinical entities.

We, therefore, studied the haemodynamic and biochemical features of a group of Nigerian hypertensives (with and without heart failure) and compared them with patients with DCM (with and without mild hypertension) in order to have a clearer understanding of the role of hypertension in the latter patients. It was hoped that these measurements would clarify whether the haemodynamic and biochemical changes that occur with heart failure are indeed responsible for the rise in blood pressure in patients who have features of DCM but present with mild hypertension, or whether such patients are truly hypertensives.

Patients and methods

Patients

A total of 72 adult Nigerians were initially selected for study but 17 were excluded because of the presence of paradoxical septal motion on M-mode echocardiography (which precluded haemodynamic measurements) and technical reasons. The remaining 55 patients were divided into five groups:

group 1 —12 normal subjects on no medication who served as controls;

group 2 —14 hypertensives (diastolic blood

pressure above 100 mmHg), without clinical evidence of heart failure;

group 3 —10 patients with HHF (diastolic blood pressure above 100 mmHg);

group 4 —8 patients with features of DCM but with mild hypertension (diastolic blood pressure of 90–100 mmHg);

group 5 —11 patients with features of DCM but no hypertension (i.e. diastolic blood pressure below 90 mmHg).

The patients were obtained from three sources, the Medical Out-patient, General Out-patient and the Casualty Departments of the University College Hospital, Ibadan, Nigeria. A diagnosis of DCM was made if a patient had congestive cardiac failure of unknown cause, cardiac enlargement confirmed by postero-anterior plain chest radiograph and typical echocardiographic features described by Adesanya and Sanderson [17]. A written or thumb-printed consent was obtained from each patient before investigations.

Methods

Haemodynamic measurements. Haemodynamic measurements were made using M-mode echocardiography. This method was chosen because it is harmless, reasonably accurate and provides reproducible data. Echocardiography was performed using a Smith-Kline Ekoline 20 Echocardiograph with a 2.25 MHz acoustically focussed transducer (S.K.I. Model C-10) of 1.25 cm diameter. The output of the ultrasonoscope was displayed oscilloscopically and recorded on the stripchart of the Cambridge multipurpose recorder. The recordings were made at a speed of 50 mm per second; simultaneous ECG tracings were included for timing. The patients were examined in the left semi-lateral position at 40° incline to the horizontal as this position was most convenient for all the subjects, especially those with heart failure.

Echocardiographic patterns of the mitral valve, inter-ventricular septum, left ventricular cavity size and posterior left ventricular wall were recorded by the technique described by Feigenbaum [18]. Measurement of the left ventricular (LV) minor axis at end-diastole (LVIDd) was taken between the endocardial surface of the posterior LV wall and the septum at the peak of the R wave of a simultaneously recorded electrocardiogram. The short axis at end-systole (LVIDs) was taken at the point where the endocardial surface of the septum and posterior wall were nearest to each other. The LV wall thickness was measured at end-diastole from the endocardial echo to the edge of the epicardial echo. Left ventricular wall mass was calculated from the equation of Bennet and Evans [19]. Ejection fraction (EF) was calculated using the formula:

$$EF = \frac{\text{LV diastolic volume} - \text{LV systolic volume}}{\text{LV diastolic volume}},$$

where LV systolic volume = stroke volume.

The ventricular volumes were estimated in normal and hypertensive patients by the cube method [20] and in patients with DCM by the regression equation of Fortuin *et al.* [21]. The latter was considered more accurate than the cube method for estimating left ventricular volumes in dilated hearts. The mean velocity of circumferential fibre shortening (mVcF) was calculated from the equation:

$$mVcF = \frac{LVIDd - LVIDs}{LVIDd \times ET},$$

where

LVIDs = LV internal dimension in end-systole,

LVIDd = LV internal dimension in end-diastole, and

ET = ejection time.

The cardiac output (CO) was calculated by multiplying the heart rate (HR) by the stroke volume (SV). Heart rate was determined from the electrocardiographic tracing obtained at the beginning of the procedure.

The mean arterial blood pressure (BPm) and systemic vascular resistance (SVR) were calculated by the non-invasive method described by Stefadouros *et al.* [22].

Systolic time intervals were measured in each patient according to the method of Weissler *et al.* [23].

Each patient had a plain radiograph of the chest taken in the postero-anterior position. The cardiothoracic ratio was calculated by the method of Lusted and Keats [24], while the aortic diameter was measured by the method of Brockington and Bohrer [25].

The body surface area was derived from the weight and height of each patient using the Dubois and Dubois [26] height–weight equation:

$$BSA = 71.84W^{0.425} \times H^{0.725},$$

where BSA = body surface area (cm^2), W = weight (kg), and H = height (cm).

Biochemical measurements. The following biochemical measurements were made on each patient: serum electrolytes and urea, plasma renin activity, aldosterone, and cortisol. Plasma volume, 24-h urinary sodium and potassium excretion, and 24-h VMA excretion were also measured. 24-h urine was collected after 5 days on the hospital diet, which contains 150 mEq of sodium (Na^+) per day (Dietetics Department of the University College Hospital, Ibadan) in the control and hypertensive groups, while this was done on the first day of admission in patients with heart failure. Plasma volume was measured by the radio-iodinated serum albumin dilution technique as optimized by Tarazi *et al.* [27]. Plasma aldosterone and plasma renin activity were determined by radioimmunoassay (Seronodiagnostic, London).

Statistical analysis

The haemodynamic and biochemical data were analysed with the aid of an IBM system 370 computer of the University of Ibadan computing centre. The software available was the statistical package for the social sciences (SPSS) and this was used to calculate the summary indices of the data. Since there are more than two disease groups to be compared for each variable, analysis of variance technique was first used to detect whether there were differences between, and within, the groups. The results were referred to the F-distribution table for decision. A result was regarded as statistically significant if it reached the 5% error. Comparisons between the mean values of the variables were then made using Student's *t*-test.

Results

The clinical characteristics of the patients are shown in Table 1. Four of the five groups had similar mean ages, but group 4 had a higher mean age than the rest. As expected, patients in heart failure had higher heart rates.

Cardiac size parameters

This is shown in Table 2. Table 3 shows the *t* values and statistical significance of the various contrasts. The LVIDd of groups 2, 3 and 4 did not defer at the probability level of 5%. Group

Table 1. Clinical characteristics of subjects

	Normal subjects	Hypertensive subjects		Suspected DCM with mild hypertension	DCM
			Heart failure patients		
	Group 1	Group 2	Group 3	Group 4	Group 5
Number	12	14	10	8	11
Sex ratio (men/women)	10:2	7:7	7:3	7:1	5:6
Age (years) mean and range	45 (30–60)	45 (24–63)	45 (35–60)	56 (33–65)	42 (23–60)
Body surface area (m^2) (mean ± s.d.)	1.69 ± 0.06	1.71 ± 0.08	1.70 ± 0.08	1.72 ± 0.10	1.68 ± 0.08
Heart rate (beats/min) (mean and range)	68 (64–74)	75 (60–100)	101 (80–112)	103 (100–108)	104 (96–114)
Mean arterial pressure (mmHg)(mean ± s.d.)	83 ± 4	131 ± 40	131 ± 5	112 ± 9	88 ± 7

Table 2. Cardiac size parameters of subjects

	Normal subjects	Hypertensive subjects		Suspected DCM with mild hypertension	DCM
			Heart failure patients		
	Group 1	Group 2	Group 3	Group 4	Group 5
End-diastolic diameter (cm)	4.8 ± 0.11	5.1 ± 0.53	5.1 ± 0.25	5.2 ± 0.13	5.9 ± 0.33
End-systolic diameter (cm)	3.4 ± 0.14	4.0 ± 0.53	4.5 ± 0.25	4.6 ± 0.10	5.5 ± 0.16
Left ventricular posterior wall thickness (cm)	0.6 ± 0.08	0.9 ± 0.23	1.3 ± 0.08	1.1 ± 0.12	0.6 ± 0.10
Left ventricular mass (gm)	191 ± 18	294 ± 100	382 ± 34	309 ± 47	221 ± 52
Left atrial diameter (cm)	2.5 ± 0.06	2.9 ± 0.38	4.0 ± 0.65	3.7 ± 0.67	3.4 ± 0.48
Aortic diameter (echo) (cm)	2.4 ± 0.11	2.8 ± 0.41	3.1 ± 0.34	2.8 ± 0.46	2.5 ± 0.25
Aortic diameter (CXR) (cm)	5.4 ± 0.47	6.9 ± 0.65	7.5 ± 0.47	7.1 ± 0.64	6.3 ± 0.48
Cardiothoracic ratio	0.43 ± 0.04	0.52 ± 0.04	0.62 ± 0.05	0.70 ± 0.07	0.68 ± 0.06

Values are means ± s.d.

5 had the largest end-diastolic diameter and this was significantly larger than those of the other groups. Group 5 patients also had the largest LVIDs. Patients in heart failure had significantly larger hearts at end-systole than those without. This is expected, as LV dimensions increase with heart failure. Although the end-diastolic and end-systolic dimensions of group 5 were significantly larger than those of groups 3 and 4, there was no significant difference between the latter two groups, who, like group 5 patients, were in heart failure. It was interesting, however, to note that the LV dimensions in systole of patients with hypertension, but without clinical evidence of heart failure (group 2), were also significantly increased compared with controls, although not to the same extent as those with heart failure. Similar trends were also found in the values obtained for left atrial dimensions and cardiothoracic ratios. As expected, patients in heart failure (groups 3, 4 and 5) had larger left atria and cardiothoracic ratios than controls.

Table 2 also shows that the left ventricular wall was thicker in the hypertensives (groups 2 and 3). The LV wall of patients in group 4 (DCM with mild hypertension) was also thick and comparable with those with hypertension (groups 2 and 3), whereas those with DCM and no hypertension (group 5) had similar wall thicknesses as controls. A similar trend was also found with the left ventricular mass.

The results of aortic diameters measured on plain chest radiograph and echocardiography showed that those with hypertension (groups 2 and 3) had significantly higher values than controls. The values for group 4 patients (DCM with mild hypertension) were similar to groups 2 and 3 (hypertensives), and significantly higher than group 5 patients (DCM without hypertension), on plain chest radiograph. Group 5 patients had comparable values with controls on echocardiography but slightly higher values on chest X-ray.

Haemodynamic measurements

This is shown in Table 4. The *t* values and statistical significance of the various contrasts are shown in Table 5.

Stroke volume and index were significantly reduced in all the patients with heart failure, compared with controls and hypertensives without heart failure (group 2). The lowest values were obtained in group 4 patients (DCM with mild hypertension). Group 4 patients also had the smallest cardiac output and index and these were significantly decreased compared with controls. The cardiac index of group 4 patients

Table 3. Student's *t*-test values and statistical significance of important contrasts

	1 *v.* 2 (d.f. = 24)	1 *v.* 3 (d.f. = 20)	1 *v.* 4 (d.f. = 18)	1 *v.* 5 (d.f. = 21)	2 *v.* 3 (d.f. = 22)	2 *v.* 4 (d.f. = 20)	2 *v.* 5 (d.f. = 23)	3 *v.* 4 (d.f. = 16)	3 *v.* 5 (d.f. = 19)	4 *v.* 5 (d.f. = 17)
End-diastolic diameter	2.07	3.52*	7.16*	10.54*	0.00	0.67	4.62*	1.09	6.29*	6.39*
End-systolic diameter	4.07*	12.39*	22.35*	33.37*	3.08*	4.11*	10.03*	1.15	10.80*	15.05*
Left ventricular posterior wall thickness	4.57*	20.50*	10.35*	0.00	6.02*	2.68*	4.38*	4.06*	17.78*	9.62*
Left ventricular mass (g)	3.78*	15.99*	6.78*	1.82	3.06*	0.48	2.36*	3.69*	8.47*	3.85*
Left atrial diameter (echo)	3.88*	7.27*	5.05*	6.18*	4.80*	3.10*	2.83*	0.96	2.39*	1.08
Aortic diameter (echo)	3.51*	6.24*	2.41*	1.22	1.95	0.00	2.26*	1.54	4.56*	1.67
Aortic diameter (CXR)	6.81*	10.44*	6.44*	4.54*	2.62*	0.70	2.65*	1.48	5.78*	2.98*
Cardiothoracic ratio	5.72*	9.70*	9.89*	11.65*	5.24*	6.68*	7.62*	2.72*	2.49*	0.65

*Statistically significant values, $P < 0.05$.

Table 4. Left ventricular function parameters of the subjects

	Normal subjects	Hypertensive subjects		Suspected DCM with mild hypertension	DCM
				Heart failure patients	
	Group 1	Group 2	Group 3	Group 4	Group 5
Stroke volume (ml/beat)	71 ± 3.8	66 ± 17.0	49 ± 10.5	42 ± 6.3	45 ± 8.2
Stroke index (ml/beat/m^2)	42 ± 6.9	39 ± 4.8	29 ± 4.2	24 ± 5.1	27 ± 5.4
Cardiac output (l/min)	4.83 ± 0.16	4.96 ± 1.27	4.92 ± 0.75	4.31 ± 0.60	4.63 ± 0.77
Cardiac index (l/min/m^2)	2.78 ± 0.15	2.82 ± 0.69	2.86 ± 0.33	2.25 ± 0.23	2.74 ± 0.43
Systemic vascular resistance	1384 ± 58	2183 ± 561	2266 ± 237	2091 ± 237	1541 ± 181
Ejection fraction (%)	65 ± 2.3	50 ± 5.2	35 ± 2.6	30 ± 3.0	20.9 ± 3.9
Mean velocity of circumferential titre shortening (circ/sec)	1.3 ± 0.08	0.7 ± 0.10	0.6 ± 0.10	0.4 ± 0.08	0.3 ± 0.06
Pre-ejection period (msec) (PEPI)	120 ± 5.7	146 ± 14.4	171 ± 12.9	174 ± 12.0	182 ± 13.2
Left ventricular ejection time (msec) (LVETI)	403 ± 9.3	399 ± 3.3	374 ± 13.2	364 ± 6.6	358 ± 5.1
PEP/LVET	0.30 ± 0.09	0.37 ± 0.15	0.46 ± 0.21	0.48 ± 0.12	0.51 ± 0.21
Left ventricular ejection rate	104.2 ± 5.0	96.7 ± 6.2	77.2 ± 4.5	67.0 ± 3.0	74.9 ± 5.1

Values are means ± s.d.

was significantly less than those of patients in groups 3 and 5, the other groups with heart failure.

The SVR, as expected, was increased in hypertensives. The value in group 4 patients (DCM with hypertension) was comparable with those of hypertensives (groups 2 and 3). While the SVR in group 5 patients (DCM without hypertension) was significantly higher than controls, it was significantly less than those of groups 2, 3 and 4.

Evidence for reduced myocardial function in all the patients, including hypertensives without heart failure, is also shown in the values obtained for the following parameters: left ventricular ejection rate, ejection fraction, mVcF, pre-ejection period and PEP/LVET ratio. As expected, those with heart failure had worse values.

Biochemical measurements

The results of the various biochemical parameters measured are shown in Table 6; the *t* values and statistical significance of the various contrasts are shown in Table 7. Plasma aldosterone was raised compared with controls in hypertensives without heart failure (group 2) but much more significantly in all patients who had heart failure (groups 3, 4 and 5). Plasma cortisol was similarly raised in group 2 patients, compared with controls, although it did not reach a level of significance. Significantly higher levels were obtained in groups 3, 4 and 5 patients who had heart failure. Plasma volume corrected for height was contracted in group 2 patients, but significantly elevated in others. Plasma renin activity, however, showed no change, except in patients with HHF who had elevated values. Normal values of plasma renin activity in Nigerians vary from 0.15 to 2.5 ng/ml/h [28].

Serum sodium showed a significant decrease in group 3 patients, compared with controls, and a significant elevation in group 5 patients, compared with groups 3 and 4. Serum potassium in group 2 patients was significantly less than the values obtained in groups 1 and 4.

The 24-h urinary sodium excretion was increased in hypertension without heart failure but significantly reduced in all the groups who

Table 5. Student's *t*-test values and statistical significance of important contrasts

	1 *v.* 2 (d.f. = 24)	1 *v.* 3 (d.f. = 20)	1 *v.* 4 (d.f. = 18)	1 *v.* 5 (d.f. = 21)	2 *v.* 3 (d.f. = 22)	2 *v.* 4 (d.f. = 20)	2 *v.* 5 (d.f. = 23)	3 *v.* 4 (d.f. = 16)	3 *v.* 5 (d.f. = 19)	4 *v.* 5 (d.f. = 17)
Stroke volume	1.07	3.50*	11.69*	9.63*	3.02*	4.74*	4.06*	3.99*	4.14*	0.90
Stroke index	1.27	5.44*	6.69*	5.84*	5.41*	6.79*	5.80*	2.23*	0.95	1.23
Cardiac output	0.38	0.12	2.39*	0.85	0.97	1.63	0.80	1.92	0.87	1.01
Cardiac index	2.11*	0.71	5.75*	0.29	0.19	0.89	0.35	4.62*	0.72	3.20*
Systemic vascular resistance	5.29*	10.88*	8.27*	2.75*	0.49	0.54	4.02*	1.52	7.53*	5.51*
Ejection fraction	9.74*	28.38*	28.00*	32.67*	9.32*	11.44*	15.99*	3.73*	9.86*	5.74*
Mean velocity of circumferential fibre shortening (mVCF)	6.85*	17.88*	24.66*	34.09*	1.11	3.37*	4.63*	4.72*	8.24*	2.99*
Pre-ejection period (PEPI)	6.21*	11.59*	11.87*	14.39*	4.46*	4.89*	6.49*	0.51	1.93	1.37
Left ventricular ejection time (LVETI)	1.42	5.84*	10.96*	14.54*	5.86	14.06*	23.12*	2.09*	3.59*	2.15*
PEP/LVET	1.47	2.24*	3.62*	3.07*	11.59*	1.88	1.87	0.25	0.54	0.39
Left ventricular ejection rate	3.64*	13.32*	20.78*	13.89*	8.92*	15.09*	9.65*	5.73*	1.10	4.23*

*Statistically significant values, $P < 0.05$.

Table 6. Biochemical parameters of subjects

	Normal subjects	Hypertensive subjects		Suspected DCM with mild hypertension	DCM
			Heart failure patients		
	Group 1	Group 2	Group 3	Group 4	Group 5
Serum sodium (mEq/l)	136.9 ± 4.6	135.1 ± 3.2	132.9 ± 3.9	133.9 ± 2.5	137.5 ± 2.8
Serum potassium (mEq/l)	3.9 ± 0.45	3.8 ± 0.50	3.5 ± 0.33	4.0 ± 0.37	3.7 ± 0.36
Plasma aldosterone (pg/ml)	118 ± 25	224 ± 117	318 ± 85	309 ± 69	311 ± 69
Plasma renin activity (ng/ml/h)	0.42 ± 0.41	0.33 ± 0.36	3.51 ± 2.24	0.49 ± 0.49	1.26 ± 0.28
Plasma cortisol (mg/100 ml)	5.3 ± 2.1	7.0 ± 2.4	13.7 ± 5.4	12.7 ± 2.9	14.0 ± 4.0
Plasma volume (l)	1.94 ± 0.34	1.62 ± 0.67	3.16 ± 0.47	3.64 ± 0.88	3.97 ± 0.83
Plasma volume per cm height	11.8 ± 1.7	9.7 ± 4.2	18.9 ± 2.7	21.1 ± 5.3	24.1 ± 4.5
24-h urinary sodium excretion (mEq/day)	67 ± 25	95 ± 33	25 ± 11	31 ± 10	25 ± 10
24-h urinary potassium excretion (mEq/day)	43 ± 25	33 ± 17	16 ± 12	18 ± 8	14 ± 6
24-h urinary VMA excretion (mg/day)	7.0 ± 0	12.8 ± 3.0	7.0 ± 0	8.7 ± 4.8	7.0 ± 0

Values are means ± s.d.

had heart failure. The 24-h urinary potassium excretion was similary reduced in all the groups with heart failure. The values for 24-h urinary VMA excretion showed that there was no significant difference between the control value and those of the other groups, except group 2. Also, there was no significant difference between the values obtained in the groups with heart failure (groups 3, 4 and 5).

Discussion

The results showed the changes expected with heart failure: depressed stroke volume, ejection fraction and cardiac output; increased heart rate and SVR; elevated plasma cortisol and aldosterone; increased plasma volume; sodium and potassium retention. These are the factors suspected to be responsible for the elevation of blood pressure in patients with DCM. However, a close look at our findings shows that this could not be so, for these changes occurred in DCM regardless of whether blood pressure was raised. They also occurred in patients with HHF. Thus, these changes are the normal response to heart failure.

Although SVR was significantly raised in patients with DCM without hypertension (group 5), compared with controls, the elevation was considerably less than those of hypertensives (groups 2 and 3). Conversely, the rise of SVR in patients with DCM and mild hypertension (group 4) was substantial and comparable with those of groups 2 and 3. It was also significantly higher than those obtained in group 5 patients, suggesting that the elevation is not due to heart failure alone. Increased SVR is considered the haemodynamic hallmark of diastolic hypertension [29, 30]. The SVR is also raised in patients with heart failure, irrespective of cause, as shown in this study. However, the phenomenal rise of the SVR in group 4 patients, i.e. DCM with mild hypertension, with values similar to those with definite hypertension (groups 2 and 3), suggests to us that group 4 patients are, in fact, hypertensives, and that the marked increase in their SVR is not due solely to the normal response of the body to heart failure.

Table 7. Student's *t*-test values and statistical significance of important contrasts

	1 *v.* 2 (d.f. = 24)	1 *v.* 3 (d.f. = 20)	1 *v.* 4 (d.f. = 18)	1 *v.* 5 (d.f. = 21)	2 *v.* 3 (d.f. = 22)	2 *v.* 4 (d.f. = 20)	2 *v.* 5 (d.f. = 23)	3 *v.* 4 (d.f. = 16)	3 *v.* 5 (d.f. = 19)	4 *v.* 5 (d.f. = 17)
Serum sodium	1.14	2.20*	1.91	0.40	1.43	0.98	1.71	0.64	3.06*	3.00*
Serum potassium	0.49	2.47*	0.54	1.47	1.90	1.02	0.93	3.05*	1.06	2.05
Plasma aldosterone	3.32*	7.19*	7.48*	8.60*	2.27*	2.13*	2.30*	0.23	0.20	0.05
Plasma renin activity	0.55	4.31*	0.36	5.78*	4.44*	0.80	7.25*	4.13*	3.15*	3.98*
Plasma cortisol	1.99	4.68*	6.20*	6.50*	3.70*	4.69*	5.14*	0.49	0.14	0.80
Plasma volume	1.60	6.85*	5.21*	7.57*	6.63*	5.62*	7.66*	1.39	2.79*	0.84
Plasma volume per cm height	1.75	7.13*	4.75*	8.49*	6.52*	5.20*	8.20*	1.08	3.29*	1.31
24-h urinary sodium excretion		5.11*	4.29*	5.23*	7.32*	6.59*	7.44*	1.15	0.09	1.28
24 h urinary potassium excretion	2.43*	3.32*	3.17*	3.94*	2.98*	2.83*	4.08*	0.48	0.57	1.39
24-h urinary VMA excretion	1.09	0.00	1.0	0.00	7.23*	2.18	7.23*	1.00	0.00	1.0
	7.23*									

*Statistically significant values, $P < 0.05$.

There are other pieces of evidence that reinforce this suggestion. The left ventricular wall thickness and LV mass of group 4 patients were markedly increased, compared with controls and patients with DCM without mild hypertension (group 5), and were comparable with, or sometimes higher than, those of hypertensives. The implication of this finding is that the LV muscle of group 4 patients is hypertrophied, as in groups 2 and 3, most likely in response to a chronic pressure load. Furthermore, the aortic diameter of group 4 patients was markedly increased and again comparable with those of definite hypertensives (groups 2 and 3). Dilatation of the aorta is a useful confirmatory sign of hypertension in Africans [25]. This finding, therefore, not only suggests that these patients are long-standing hypertensives, but also confirms previous reports, which showed that enlargement of aortic shadow is very useful in identifying those patients with suspected DCM who have background hypertension [31].

If patients with DCM and mild hypertension are long-standing hypertensives, why then do they present with relatively low blood pressures compared with other hypertensives such as patients in group 3? When the left ventricular function and contractility indices of groups 3 and 4 are compared, group 4 patients consistently had lower stroke volume, stroke index, cardiac output and cardiac index, and all these values except the cardiac output reached a level of significance. Group 4 patients also had slightly worse systolic time interval indices, mVcF and significantly lower ejection fraction. It is well known that blood pressure is a product of cardiac output and SVR. Since the contractility indices of the left ventricle of patients in group 4 are markedly reduced, although they still had a high SVR, it follows that their blood pressure must have dropped as a result of reduced cardiac output. The marked reduction in both the stroke index and the cardiac contractility in group 4 patients, despite a significant compensatory tachycardia, clearly explains why they could neither maintain a higher cardiac output nor a higher blood pressure, unlike group 3 patients.

From this study, it seems that most of our patients with hypertension have low plasma renin activity, even when they are in heart failure. Group 3 patients, however, had high renin levels, probably because of a decreased hepatic renin clearance [32]. Another explanation may be that they had a more pronounced sympathetic nervous system activity. Their 24-h urinary VMA excretion was, however, not raised, but this is an insensitive index of increased adrenergic nervous activity. It is nevertheless clear from our results that the raised aldosterone levels in our patients with heart failure is not due to increased renin–angiotensin activity, since renin levels were low in most cases. The high plasma aldosterone values could be due to decreased rate of parenchymal extraction and inactivation of aldosterone by the liver cells [32].

Another observation in this study is the contracted plasma volume corrected for height in group 2 patients (hypertensives without clinical evidence of heart failure), although it was expanded, as expected, in those with heart failure. These findings are similar to those of Tarazi *et al.* [27], who found that patients with essential hypertension tended to have a contracted plasma volume.

Group 2 patients excreted more sodium in their urine, compared with controls. This phenomenon is also well-known and is called hypertensive exaggerated natriuresis [33]. The reason for this is, however, not clear.

In conclusion, this study disproves the concept of 'reactive hypertension' in our patients with suspected DCM and mild hypertension. There is definite haemodynamic evidence of hypertensive cardiovascular disease in the patients. They, therefore, constitute a group of HHF with poorer myocardial function and should be classified and treated as such.

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