A Study of 1 000 South African Non-White Hypertensive Patients

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SUMMARY

This study consisted of the first 1 000 non-White hypertensive patients (500 Blacks and 500 Indians) who attended the hypertension clinic at King Edward VIII Hospital over a period of 12 years. Essential hypertension in the Blacks occurs in a younger age group in females, with death occurring in cerebrovascular episodes, uraemia or congestive cardiac failure. Accelerated hypertension was seen in 7% of the Black hypertensives, with death occurring frequently in uraemia.

Ischaemic heart disease occurred in 12% of the Indian hypertensive patients, whereas it did not occur in the Blacks, although they developed congestive cardiac failure. The absence of ischaemic heart disease in Black hypertensive patients may suggest that hypertension is not an important predisposing event to myocardial infarction. Predisposing factors in the aetiology of hypertension in the Black and Indian patients are discussed. In the Indian hypertensive patient the incidence of hypertension rose with age, and complications were those of cerebrovascular episodes and congestive cardiac failure, including ischaemic heart disease and uraemia.

A close relationship between diabetes mellitus and gouty arthritis was present in the Indian. It appears that hypertension is a common disease in the urbanised Black, and it is increasing in incidence.

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Hypertension is recognised as being extremely common in the Black population in South Africa.[1-4] There is no published work on the clinical pattern of hypertension in the Indian population in South Africa. This study compares and contrasts the clinical pattern of hypertension as seen in Blacks and Indians who attended the hypertension clinic at King Edward VIII Hospital, Durban from 1960 to 1972. For convenience of study the first 1 000 patients (500 Black and 500 Indian) were selected and analysed, although many more patients were seen. Both groups were seen over a similar period of time and treated on an outpatient basis, although many of the patients were initially investigated and treated in the ward. Thus the data do not accurately reflect the hypertensive inpatient.

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METHODS

The blood pressure (BP) of every patient was taken on 3 separate occasions; a diastolic level of at least 90 mmHg and a systolic of at least 150 mmHg was regarded as abnormal.[5] All the patients had their urine examined for albumin, sugar, and for granular casts, red and white blood cells on microscopy. Blood urea, serum albumin, serum uric acid, chest X-ray film and electrocardiogram (ECG) were done. In patients under the age of 40 years an intravenous pyelogram (IVP) and renal angiogram were done to exclude renal hypertension. In those over the age of 40 years an IVP was done only if a renal cause for the hypertension was suspected clinically. Patients were assessed as regards obesity by correlating the mass and height according to the Metropolitan Table.

RESULTS

Table I shows the age and sex distribution of the patients. A family history of hypertension was obtained in 40% of the Indians and 9% of the Blacks. This deduction was made if there was a history of either high blood pressure, strokes or renal failure in any blood relative of the patient. Our population was largely urbanised in that 90% of the Blacks and 92% of the Indians had resided in Durban for at least 5 years.

Table II shows the symptoms of our hypertensive patients in percentages obtained on direct questioning. The definition of a good diet was when a patient took meat twice a week, fresh vegetables 2 - 3 times, and milk and eggs at least once a week. Using this definition 86% of the Blacks and 99% of the Indians stated that they had a good diet. A high salt intake was present when a patient added extra salt to his food in addition to that which had been added during cooking. Many of our patients had an average salt intake of at least 300 mEq of sodium per day.

Using the Metropolitan Table it was found that 34% of the Blacks (female 65%, male 35%) and 24% of the Indians (female 60%, male 40%) were obese in that they were 20% above their expected normal weight. The occupations of hypertensive patients are shown in Table III. The initial diastolic BP is shown in Table IV. Fundal gradings are shown in Fig. 1; other eye changes consisted of central vein thrombosis in 5 Blacks and 2 Indians; optic atrophy in 1 Black and 3 Indians; choroiditis in 4 Blacks and 4 Indians. Table V shows the albuminuria pattern, blood urea and serum uric acid.

TABLE I. AGE AND SEX DISTRIBUTION OF THE PATIENTS

Age group (years)	No.	%	Age (years)	Male No.	Male %	Female No.	Female %
Blacks							
20 - 30	75	15	20 - 40	84	16,8	141	83,2
30 - 40	150	30	40 - 60	112	44	138	56
40 - 50	155	31	>60	5	20	20	80
50 - 60	95	19		—	—	—	—
>60	25	5	Total	201	40	299	60
Indians							
20 - 30	10	2	20 - 40	25	10	75	26
30 - 40	90	18	40 - 60	168	78	173	64
40 - 50	170	34	60 - 80	22	12	37	10
50 - 60	180	36		—	—	—	—
>60	50	10	Total	215	43	285	57

TABLE II. SYMPTOMS OF HYPERTENSIVE PATIENTS

	Blacks (%)	Indians (%)
Headache	60	55
Dyspnoea	40	24
Palpitations	45	15
Blurring of vision	26	29
Nocturia	23	20
Swelling of body	21	9
Weakness	10	6
Epistaxis	6	2
Loin pain	4	1
Blackout	—	3
Stroke	3	—
Insomnia	2	3
Duration of symptoms		
<6 months	50	32
6 months - 1 year	20	33
> 1 year	30	35

TABLE III. OCCUPATION OF HYPERTENSIVE PATIENTS

Professional	(%)	Non-professional	(%)
Blacks		Housewives	36
Nurses	4	Domestic workers	14
Teachers	4	Labourers	5
Businessmen	1	Chefs	3
Clerks	1	Drivers	1
		Unclassified: casual labourers	31
Indians			
Businessmen	5	Housewives	50
Clerks	4	Factory workers	10
Teachers	3	Labourers	5
		Drivers	3
		Domestic workers	1
		Chefs	1
		Farmers	1
		Unclassified: casual labourers	13

TABLE IV. INITIAL DIASTOLIC BP (mmHg)

	Males (%)	Females (%)
Blacks		
90 - 100	4,0	10,4
101 - 120	16,2	23,8
121 - 140	15,0	19,0
141 - 160	4,6	5,0
> 160	1,0	1,0
Indians		
90 - 100	6,0	10,0
101 - 120	16,0	21,0
121 - 140	16,0	21,0
141 - 160	5,0	3,0
> 160	1,0	1,0

TABLE V. EXAMINATION OF URINE

Albuminuria in urine on first examination	Blacks (%)	Indians (%)
Normal	60	38
Trace	14	26
+ 1	10	20
+ 2	10	9
+ 3	4	5
+ 4	1	2

Initial blood urea of hypertensive patients				
< 20 mg/100 ml	14		10	
21 - 40 mg/100 ml	66		67	
41 - 100 mg/100 ml	19		21	
>100 mg/100 ml	1		2	
Initial serum uric acid of hypertensive patients				
Normal	72	In 76 cases	56	In 112 cases
Abnormal	28		44	

Elevated level: Males more than 7 mg/100 ml; females more than 6.5 mg/100 ml.

The complications of hypertension are shown in Table VI. Hypertension associated with diabetes mellitus occurred in 6% of the Blacks and 25% of the Indian patients. Renal investigations are shown in Table VII and cardiac findings in Table VIII.

TABLE VI. COMPLICATIONS OF HYPERTENSION

	Blacks (%)	Indians (%)
Congestive heart failure	16	8
Cerebrovascular episode	5	10
Pre-eclamptic toxaemia	8	5
Associated disease:		
Diabetes mellitus	6	25

1 case hypertensive encephalopathy.

TABLE VII. RENAL CAUSES FOR HYPERTENSION

	Blacks	Indians
Aortogram		
Renal artery stenosis	1	2
Giant cell arteritis	—	1
Intravenous pyelogram		
Pyelonephritis	13	6
Obstruction due to bilharzia, leading to hydronephrosis and pyelonephritis	1	—
Renal calculi	—	3
Obstruction due to renal calculi	—	1
Congenital polycystic kidney	—	1
Horse-shoe kidney	—	1

TABLE VIII. CARDIAC CHANGES OF HYPERTENSION

	Blacks (%)	Indians (%)
ECG LV hypertrophy	23	22
Clinical cardiomegaly	29	26
Ischaemic heart disease	—	12
Chest X-ray LV hypertrophy	30	35
Normal chest X-ray film	70	65

DISCUSSION

In 1971 Durban had a population of 730 000, of which there were 209 000 Blacks (mainly Zulus) and 289 000 Indians. King Edward VIII Hospital is one of the largest hospitals in Southern Africa, consisting of 2 000 beds. It serves a population of 2 million; there are approximately 800 000 patients attending the outpatient department, and about 99 000 patients are admitted to the hospital per year (Black 83%, Indian 17%). No figures of male and female ratios of outpatient and inpatient admissions are available.

The majority of the Indian population in South Africa are urbanised; the Blacks normally reside in rural areas, but recently increasing numbers have moved into cities

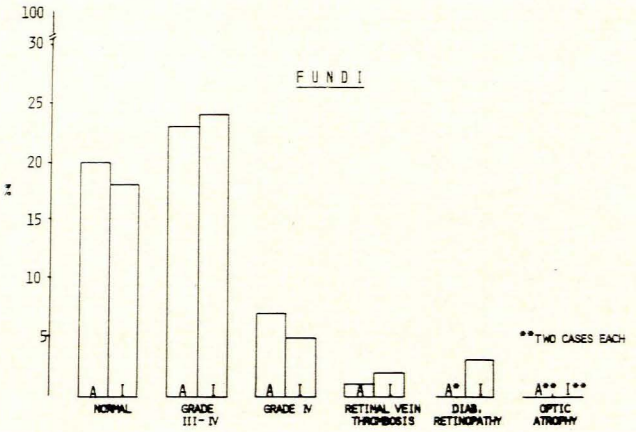

Fig. 1. Fundal gradings of hypertensive patients. Grade 3—exudates or haemorrhages; grade 4—papilloedema.

for employment. Ninety per cent of the Black patients in this study suffering from hypertension were from the urban areas in that they had resided in the cities for at least 5 years. Scotch *et al.*[6] found that hypertension was more common in the urban Zulu than the rural Zulu. Gampel *et al.*[4] suggested that the urban Zulu is exposed to considerably more emotional stress. Similar work by Kaminer and Lutz[7] among nomadic Bushmen in the Kalahari Desert has shown that hypertension is less common compared to the Bushmen who became prisoners and labourers. Edginton *et al.*[8] found that hypertension was uncommon at the Jane Furse Memorial Mission Hospital, a rural hospital at Sekhukhuneland, Transvaal, whereas essential hypertension was common in the Johannesburg Blacks.

As seen in Table I, hypertension occurred earlier in Blacks than Indians (45% of the <40 years and 76% <50 years). The majority of Indians are economically better off than the Blacks and thus the figure merely indicates that the younger Indians attend private practitioners rather than a clinic. However, it must be emphasised that since the hypertension clinic was established in 1960 many local general practitioners have been referring their patients for investigation and advice, including many affluent Indians. The urban Blacks are of the younger age group. Autopsy studies by Becker[9] have shown that the peak incidence of essential hypertension was at 50 years of age, and that 54% of cases occur below the age of 40 years. Schrire[2] found that hypertension occurred at an earlier age in the Cape Coloureds and Blacks compared with Whites. Weiss and Prusmack[10] found that hypertension occurred a decade earlier in the American Negro compared with Whites. We found that in contrast to the Blacks the Indian hypertensive patient showed an increase in the incidence of hypertension with age up to a maximum incidence at 60 years. This pattern is similar to that found by Schrire among White patients in South Africa.[2]

In this study, and in Table I, it is seen that 80% of the hypertension seen in Indian patients occurred after the age of 40 years. In both Black and Indian patients hypertension occurred more commonly in females. A

striking feature was that in Black females under the age of 40 years the incidence was 5 times greater than in males. This earlier rise in BP in females compared with males is consistent with the other findings among Zulus,[6] Cape Coloureds[2] and Coloured and Black patients in Johannesburg.[3]

The commonest symptom (Table II) was headache, mainly occipital, which occurred in 60% of the Blacks and 55% of the Indians. This symptom was elicited after direct questioning, which may have influenced the high figures. Nocturia occurred in 23% Blacks and 20% Indians, and we found this symptom to be a valuable index to determine the duration of hypertension. Nocturia in hypertension usually denotes some impairment of renal function and may disappear once the BP is controlled. Epistaxis was the presenting clinical feature in 6% of the Black patients, a really high figure.

A family history of hypertension was obtained in only 9% of the Blacks and 40% of the Indians. There did not appear to be a difference in the family size between the two races. This difference in family history suggests that hypertension is an emerging disease among urbanised Blacks; the Black is becoming more aware of hypertension; or the Indian was always urbanised and traditionally medically orientated. With the increasing stress in an urbanised environment it is likely that hypertension will increase still further. Many studies indicate a correlation between obesity and hypertension, especially systolic hypertension.[11] Zulu residents in Durban tend to weigh considerably more than those in rural areas where a lower prevalence of hypertension has been reported by Slome *et al.*[12] who found that relative mass and skinfold thickness had no direct relationship to hypertension or to rurality of background. Although they found that females had a markedly higher prevalence of overweight, relatively little sex variation has been shown in the BP levels. Obesity was common in both Blacks (34%) and Indians (24%) in our study. There was also a higher incidence of hypertension in females, unlike that found by Scotch *et al.*,[5] and this may be partly due to female obesity. While obesity may be a correlate of hypertension, we agree with Gampel *et al.*[4] that this difference does not account for the difference in hypertension between the urban and rural groups.

The staple diet of the Blacks is mealie meal and in order to make it palatable they add a large amount of salt; 45% of the Blacks and 40% of the Indians admitted to taking excessive amounts of salt. There is a divergence of opinion on the salt intake of the Blacks. Walker[13] states that the intake is much the same as among Whites, namely 8 - 12 g/day. We found that many of our urban patients have an intake of at least 300 mEq of sodium per day. Certainly the high salt intake may be responsible for the high incidence of hypertension, a feature similar to that of the northern part of Japan where, because of the high salt intake, there is a high incidence of hypertension and strokes are responsible for one-third of all deaths.

The incidence of accelerated hypertension in this study was 7% in Blacks and 5% in Indians. This figure is slightly higher than that seen in the Royal Postgraduate Medical School.[14] The higher incidence of accelerated hypertension in Blacks is probably due to the large numbers of untreated hypertensive patients seen at a late stage. In a detailed study of accelerated hypertension previously reported,[15] it was found that in patients whose blood urea was over 50 mg/100 ml, 77% died during their first admission to hospital. Most of the patients in our study suffering from accelerated hypertension had a raised blood urea. Uys[16] found an incidence of malignant nephrosclerosis in Black hypertensive vascular disease to be 30,3% from autopsy studies.

A feature among Indian patients was that 10% had evidence of a past or recent stroke. The complications of hypertension are shown in Table VI. Although congestive cardiac failure occurred in 16% of the Black hypertensive patients and there was ECG evidence of left ventricular hypertrophy in 23%, none of the Blacks evidenced ischaemic heart disease on ECG examination. The Framingham study[17] found that a high BP was synonymous with an increased risk for the development of coronary heart disease. However, when hypertension and hypercholesterolaemia coexist the risk is increased still further. The rarity of myocardial infarction in Blacks could be because the mean serum lipid levels are low in both urban and rural Blacks. In contrast, ischaemic heart disease either in the form of angina pectoris or myocardial infarction, and confirmed with ECG, occurred in 12% of the Indian patients. These findings suggest that hypertension alone is not an important cause in the aetiology of myocardial infarction. However, hypertension is not uncommonly associated with cerebral atheroma and atheroma in the aorta.[18] In this study cerebrovascular episodes in the form of cerebral thrombosis occurred in 5% of Blacks and 10% of Indians. However, from a clinical observation of ward admissions one is amazed at the frequency of cerebral haemorrhage in the Blacks who have no preceding history of hypertension, whereas in Indians cerebral thrombosis occurs more commonly. Unlike the observation in Nigeria[19] of the rarity of severe hypertensive retinopathy, we observed vascular changes consistent with hypertension in 80% of 500 Black hypertensives. In 23% of them severe fundal changes consisting of exudates, haemorrhages (grade 3), or papilloedema (grade 4), were present, and these fundal changes were not invariably associated with a raised blood urea.

The Blacks are mainly labourers who work in mines, factories or as domestics. It is thus surprising that 10% of the Black hypertensives in this study belonged to the professional class, an indication that the urbanisation may be responsible for the high incidence of hypertension. In South Africa there is a very small (under 0,5%) but emerging Black population who are becoming professionals, and it is likely that myocardial infarction will become more common. The incidence of the professional Indian class in this study was 12% (Table III).

The Black domestic's diet is very similar to that of Whites and it is not surprising that the incidence of diabetes mellitus is increasing. Goldberg *et al.*[20] have found diabetes mellitus as common in the urban Black in Cape Town as in the White population. In this study 6% of our Black hypertensive patients had overt diabetes mellitus. The high incidence of diabetes mellitus in the Natal Indian[21] accounts for the fact that 25% of our hypertensive

patients had diabetes mellitus. In a previous study,[22] an abnormal glucose tolerance test was reported in 78% of the Indian and 49% of the Black hypertensive patients. Serum uric acid levels were abnormal in 28% of the Black and 44% of the Indian patients (Table V). Serum uric acid levels were tested before the patients were put on thiazide diuretics. In spite of this finding there was no case of gouty arthritis among the Blacks (as is generally recognised), whereas gouty arthritis was frequently seen in the Indian. It appears that the aetiology of hypertension, hypercholesterolaemia and diabetes mellitus in the Natal Indian rests upon a fundamental common biochemical abnormality.

As seen in Table V, gross albuminuria occurred in 1% of the Black and 2% of the Indian patients. As previously recorded[15] gross albuminuria occurred commonly in accelerated hypertension. There was a good correlation between the level of albumin in the urine and the BP, and on control of the BP the albuminuria usually disappeared. All the patients under 40 years had an intravenous pyelogram, and where relevant a renal aortogram was done to exclude a renal cause for the hypertension. As shown in Table VII pyelonephritis was the commonest renal cause of hypertension. Renal calculi are extremely uncommon in Blacks, whereas they are common in the Indian population,[23] and hypertension was proved due to renal calculi in 4 Indian patients.

This work illustrates the pattern of hypertension as seen in the hypertension clinic of our hospital. Hypertension is very common in Blacks and occurs at a younger age; it behaves in an explosive manner, with death occurring frequently from cerebral haemorrhage, uraemia or congestive cardiac failure. The Indian hypertensive patient resembles the White hypertensive patient in age distribution, and close correlation with diabetes mellitus and gout, and this suggested a common biochemical factor. The complications in the Indian hypertensive patient were mainly due to cerebral thrombosis, and congestive heart failure, including ischaemic heart disease and uraemia. It would appear that with increasing urbanisation and associated stress the incidence of hypertension will increase even further in the Blacks. This would be in agreement with the belief that in Johannesburg the second most common cause of death in the adult Black (after violence) is hypertension and its complications.[24]

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