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Obesity and Hypertension in Blacks

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Summary. In the United States, obesity and hypertension are more common in blacks than in whites, but that general statement hides some important sex differences. Thus, in black women the prevalences of both obesity and hypertension are greater than in white women, whereas in men, although there is no racial difference in obesity, in blacks hypertension is more common and more severe than in whites. For white people, there is a well-documented causal relationship between obesity and hypertension, however, results from the second National Health and Nutrition Examination (NHANES II) suggest that this relationship is not so strong for blacks. Obesity is also an important risk factor for diabetes, which in itelf is associated with hypertension.

The mechanism of obesity-associated hypertension appears to be an inadequate vasodilation in the face of the increased blood volume and cardiac output, which are the natural consequences of an increased body mass. This defect in control of vascular resistance has been attributed to increased activity of the sympathetic nervous system, abnormal renin-angiotensin-aldosterone relationships, and insulin resistance. However, none of these attributes has been found to be the exclusive characteristic of obese hypertensive as compared with normotensive obese subjects.

Key Words. hypertension, blacks, Indians, renin, aldosterone, adrenergic.

The importance of obesity to the hypertension of blacks is difficult to assess. Although there is considerable information available about obesity hypertension in general, there is little about it in blacks. Therefore, this discussion will focus on the general problem, but it will include some race-specific information, much of which has come from studies in the United States.

Racial Differences in Prevalence

Although we do not know whether the hypertension associated with obesity in blacks has mechanisms similar to that in whites, we do have a great deal of information about racial differences in the prevalences of obesity and hypertension. These seem worthwhile to present because of the insights that they provide into mechanisms of various types of hypertension. Both obesity and hypertension are common in the United States, as they are in all industrialized societies; both are more common in blacks than in whites.

The most recent U.S. data concerning the prevalences of hypertension and obesity come from results of the Second National Health and Nutrition Examination Survey (NHANES II), which surveyed 16,204 persons between the ages of 6 and 74 over a period of 4 years from 1976 to 1980 [1].

Obesity

Analyzing the NHANES II data, obesity was related to body mass index (BMI, body weight in kg/height in m^2) rather than percent overweight because this calculation makes a systematic allowance for height [2]. Men were considered overweight at a BMI of more than 27.8 kg/m^2 and women at 27.3 kg/m^2. Overweight was calculated for persons 20–75 years of age, and of these 26% were found to be overweight. This single figure, however, obscures striking age-sex-race differences, and these have been detailed by Van Itallie [2]. The sex-race differences are presented in Table 1. Thus, it is seen that obesity is more common in blacks than in whites, and among blacks is more common in women than in men. The prevalence of obesity was practically equal in men of the two races but was markedly disparate in the women. For this group only a quarter of the whites (25.2%) were overweight versus about one half of the blacks (43.8%). Age affects this prevalence because in industrialized societies body weight increases with age, in contrast with primitive societies, in which weight and age are inversely related [3]. The influence of age on the racial prevalences of obesity appear in Figures 1A and 1B. For white women, the prevalence of obesity increased with each decade, while in black women the maximum weight was found between 45 and 54 years and was stable therafter. Among white men, the prevalence of obesity did not change much after 45 years of age,

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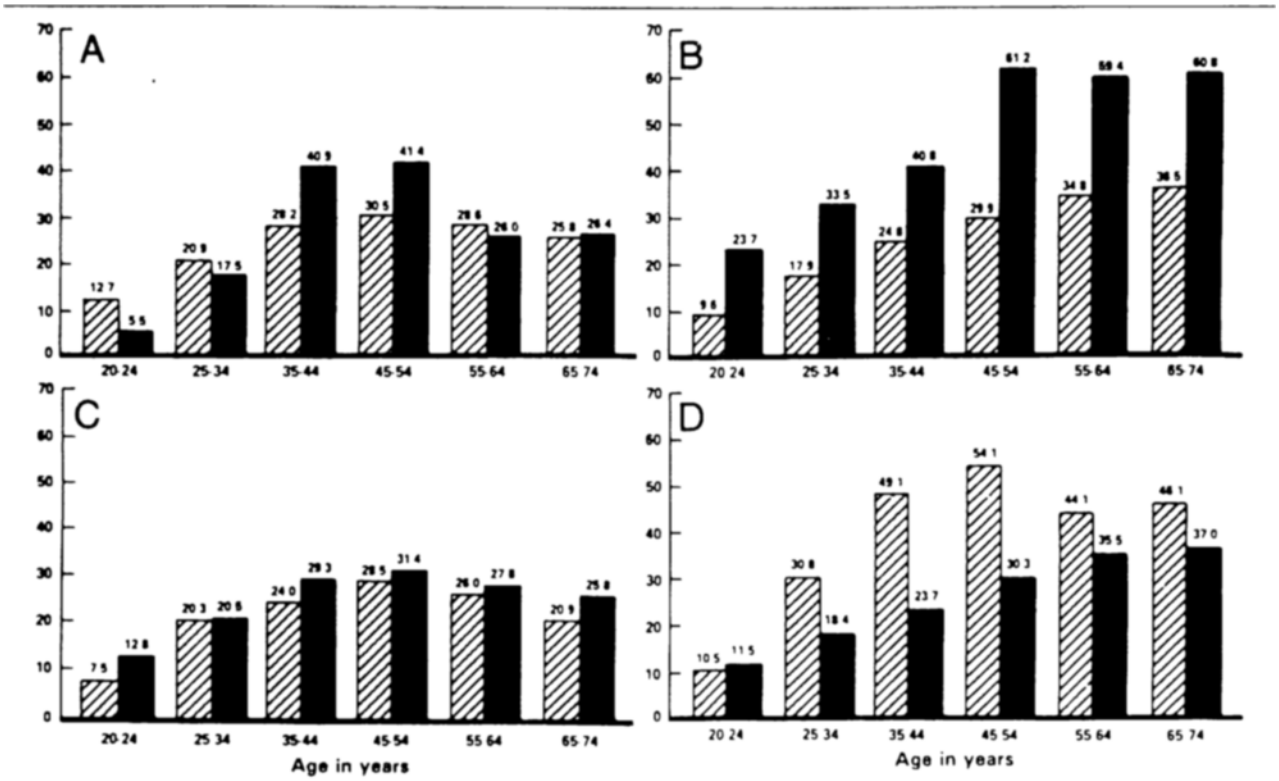

Fig. 1. *A: Percentage of males overweight by race and age. Cross-hatched bars indicate whites and solid bars indicate blacks. B: Percentage of nonpregnant females overweight by race and age. Cross-hatched bars indicate whites and solid bars indicate blacks. C: Percentage of males overweight by poverty status and age. Cross-hatched bars indicate poverty and solid bars indicate nonpoverty. D: Percentage of nonpregnant females overweight by poverty status and age. Cross-hatched bars indicate poverty and solid bars indicate nonpoverty. Reproduced with permission from* Ann Int Med *1985;103(Suppl Part 2):984.*

Table 1. *Prevalence of overweight[a] in NHANES II[b]*

Subjects	%
Black men	25.7
women	43.8
White men	24.4
women	25.2

[a]Overweight is defined as a body mass index (kg/m^2) ≥ 27.8 for men and ≥ 27.3 for women.
[b]National Health and Nutrition Examination Survey 1976–1980, Vital and Health Statistics Series II, Number 238, National Center for Health Statistics.
All subjects were 20–75 years.

while in black men it peaked somewhat later and was stable after 55 years of age.

Socioeconomic factors are important in obesity, particularly in women (Figures 1C and 1D), and the relationship is an inverse one, with those of lower socioeconomic status being more often obese than the more well-to-do. This influence is more striking in black women than it is in white women.

Hypertension

The prevalence of hypertension is quite different than that of obesity, being greater in black men than in black women. In white men, obesity was less common than hypertension, and in white women the prevalence of the two was about the same [4].

Table 2 shows the distribution by blood pressure level as developed from the NHANES II data by the Subcommittee on Definition and Prevalence of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure, 1984 [4]. This table gives data about the severity of hypertension through percentages of prevalence among the various diastolic blood pressure categories: high normal pressure—85–89 mmHg; mild hypertension—90–104 mmHg; moderate hypertension—105–14 mmHg; and severe hypertension—≥ 115 mmHg. Included also are percentages for two categories of isolated systolic hypertension—with systolic pressure ≥ 140 mmHg when diastolic pressure is < 90 mmHg. It is obvious that diastolic hypertension is more frequent in blacks than it is in whites and it is more severe. For mild hypertension the black/white ratio is 1.3; for moderate

Table 2. *Frequency distribution by blood pressure level regardless of medication status civilian, noninstitutionalized population (ages 18–74 years) 1976–80*

Blood pressure level	Males			Females			Total		
	Whites (%)	Blacks (%)	All races (%)	Whites (%)	Blacks (%)	All races (%)	Whites (%)	Blacks (%)	All races (%)
Normal blood pressure*	60.5	54.8	60.0	73.3	62.9	72.1	67.1	59.3	66.3
High normal blood pressure	8.9	9.5	9.0	5.6	5.9	5.6	7.2	7.6	7.2
Mild high blood pressure	18.4	20.6	18.7	10.8	17.9	11.7	14.5	19.1	15.0
Moderate high blood pressure	1.9	3.0	2.0	1.1	3.1	1.3	1.5	3.1	1.6
Severe high blood pressure	0.5	2.2	0.7	0.4	0.8	0.5	0.5	1.4	0.6
Borderline isolated systolic hypertension	8.5	8.8	8.4	7.2	6.9	7.1	7.8	7.7	7.7
Isolated systolic hypertension	1.3	1.2	1.3	1.7	2.4	1.8	1.5	1.9	1.5
Totals	100	100	100	100	100	100	100	100	100

From NHANES II.

*Includes those hypertensives who are controlling their blood pressures.

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hypertension, it is about 2, and for severe hypertension, it is almost 3. The table gives two other interesting items of information. Whereas they may not have relevance for obesity-associated hypertension, they have great relevance for mechanisms of hypertension. One is that the race difference in regard to the severity of hypertension is most marked for men, with blacks having 4.4 times the prevalence of whites. In contrast, among women, black females have severe hypertension only twice as often as white females. The other item concerns isolated systolic hypertension for which no racial differences were found, and whatever may be the mechanisms for this type, it does not seem to have racial determinants.

As for obesity, socioeconomic status (as judged from years of education) accounts for some of the racial differences in hypertension. The influence of educational level on blood pressure was analyzed from the data of the Hypertension, Detection and Follow-up Program (HDFP) with regard to its interactions with race, age, and socioeconomic status [5]. This analysis showed an inverse relationship between years of schooling and prevalence, regardless of race, and a positive relationship to obesity. When age, level of education, and obesity were controlled for in a multivariate analysis, hypertension was found to be about twice as common in blacks as it was in whites.

Obesity as a Risk Factor

Figure 2 shows a relationship between obesity and hypertension. In NHANES II, the relative risk of hypertension of being overweight among all the people surveyed was three times that for those who were not overweight [2]. The age most vulnerable appeared to be young adulthood, because for those people the relative risk of being hypertensive was 5.6 times that of the nonobese. For the decades between 45 to 75 years, that risk fell to 1.9.

Considering the fact that black women tend to be obese more often than white women, one would expect that they would have more obesity-related hypertension. Apparently, this is not the case, because from the NHANES II data one sees that obesity increases the risk of hypertension 2.6 times in white females and only 1.5 times in black females.

Obesity and/or weight gain during young adult life have been shown to be potent risk factors for the later development of hypertension [6–9]. Some of this information comes from the Framingham experience, showing that weight gain during young adult years greatly increased the incidence of hypertension in later life [6]. Paffenbarger, Thorne, and Wing's study of 25-year alumni of the University of Pennsylvania found that the development of hypertension with aging was in part related to greater weight during their college years [7]. Perhaps not relevant to this discussion was the 1000 Aviator Study of young white men who reported weight gain as a factor in the subsequent development of hypertension [8]. However, in Evans County, Georgia, a 7-year resurvey of young people initially examined at ages 15–29 years found that weight gain during the interval was associated with the onset and/or worsening of hypertension in

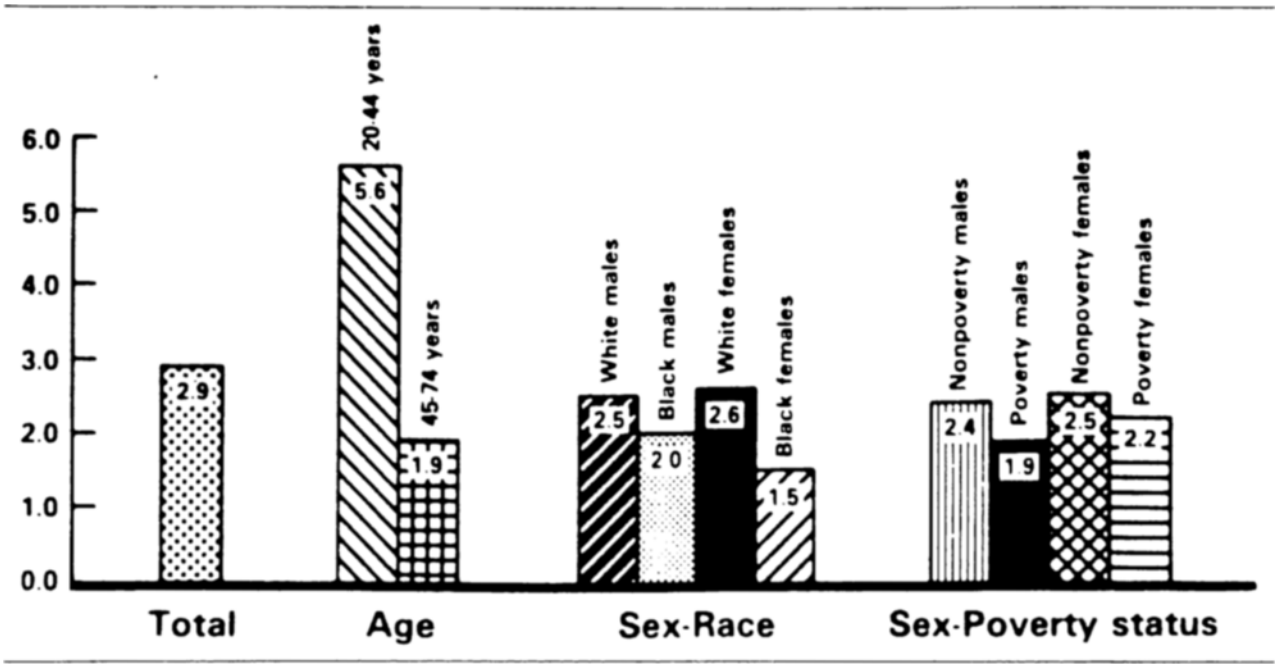

Fig. 2. Risk of hypertension by being overweight according to age, sex, race, and poverty status. Data are from NHANES II. Reproduced with permission from Ann Int Med *1985;103(Suppl Part 2):985.*

white men, white women, and black women but not in black men [9]. Thus the 1000 Aviator Study may not be relevant to this discussion, which concerned only white men.

From the foregoing, it is tempting to conclude that obesity is not so great a risk factor for hypertension in black women as it is in white. This possibility is strengthened from other work coming from the Evans County studies [10]. Tyroler et al. have predicted from these data that, "By controlling both baseline weight and weight gain, there is a potential for prevention of 27.5% of the new cases of high blood pressure in blacks and a potential prevention of almost one half of the incidence in white cases (47.7%)."

There is another aspect of obesity that may be important for hypertension and that is its function as a risk factor for the development of non-insulin-dependent diabetes mellitus (NIDDM), which, of itself, carries a risk for development of hypertension. The prevalence of hypertension among diabetics is said to be between 40% and 50%, and this average persisted in population studies, even after correlation for age, sex, and degree of obesity [11].

Diabetes is more common in blacks than it is in whites [12], and this may explain part of the difference in the prevalence of hypertension between blacks and whites, particularly that of the mild to moderate degree of severity. Although not necessarily relevant to this discussion, race differences in mortality from selected diseases, including diabetes, are of interest [13]. As seen from Table 3, mortality among blacks is greater than among whites for all of the listed diseases, except ischemic heart disease, in white men. For the purpose of this discussion, it would be of interest to focus on diabetes mellitus for which death rates of black women are highest of all. This may relate to the increased prevalence of obesity among black women. Additionally, the increased prevalence of hypertension may intensify the predisposition of such patients to develop renal disease [14].

As will be detailed later in the discussion of the mechanisms of obesity-associated hypertension, hyperinsulinemia and insulin resistance may turn out to

Table 3. *Race sex differences in mortality from cardiovascular (CV) diseases: 1985*

	Blacks		Whites	
	Women	Men	Women	Men
All causes	589.1	1024.0	390.6	688.7
Major CV diseases	250.5	379.5	157.4	292.1
Ischemic heart disease	100.8	164.9	82.9	180.8
Stroke	50.3	60.8	27.9	32.8
Hypertension	5.2	6.0	1.2	1.7
Renal failure	9.7	12.8	3.1	4.9
Diabetes mellitus	21.1	17.7	8.1	9.2

The figures are age-adjusted deaths/100,000.

be the explanation for the hypertension, since it is present both with obesity and with diabetes. For example, Christlieb and associates reported plasma insulin concentrations to be closely correlated with blood pressure levels in patients with glucose intolerance and suggested that insulin might be the link between hypertension and obesity [15]. However, the study of Weinsier et al. [16] of 204 obese subjects did not support this view. In this study fasting serum insulin, aterial pressure, body composition, and fat distribution were related to blood pressure. Although insulin and mean arterial pressure were positively correlated ($p < .0001$), this association was eliminated by controlling for overweight, percentage body fat, fat mass, lean body mass, body build, and upper body fat pattern. When they controlled for the insulin level, the correlations between the mean arterial pressure and measures of body composition and fat distribution continued to be significant. This suggested that the relationship between serum insulin and blood pressure is indirect and is probably a function of body composition.

Physiologic Characteristics of Obesity Hypertension

Hemodynamic studies in the last two decades have greatly refined the questions that must be answered concerning the relationship of obesity to hypertension. These have described the pressure-flow relationships and have shown that these relationships depend on both the degree of obesity and the severity of hypertension. Alexander found an increased cardiac output in extremely obese people and associated with this was an expanded plasma volume [17]. Some of the obese patients were mildly hypertensive and they had a normal total peripheral resistance (TPR), whereas the normotensive obese had a reduced TPR in comparison with normotensive, nonobese control subjects. Messerli et al. studied both lean and obese hypertensives, and found that the obese had higher cardiac output, larger blood volume, and lower vascular resistance [18]. In this study they focused on the differences in cardiac output and not on cardiac index, which is cardiac output corrected for body surface area.

It is logical that as body mass increases with increasing obesity, cardiac output and blood volume also increase. This raises the question of how to express these functions, because traditionally both are expressed in relationship to surface area. Mujais et al. recognized this problem by using both values—the raw data and values expressed in relationship to body surface area [19]. They found that obese hypertensives, when compared with lean hypertensives, had increased cardiac output and expanded blood volume, but when these values were related to body surface area, obese hypertensives did not differ from their lean counterparts.

Another matter of importance is the severity of hypertension. The patients studied by Alexander and Messerli had mild hypertension and "normal" TPR. However, with moderate to severe hypertension, vascular resistance must elevate. Such a situation was found by Mujais et al., whether expressing TPR in arbitrary units or in relationship to body surface area [19]. In later publications also, Messerli et al. reported that obese hypertensives with moderately severe hypertension (MAP 126 mmHg) had elevated TPR [20].

Hypervolemia is clearly one of the important characteristics of the hypertension associated with obesity. Messerli et al. found by echo-cardiography that normotensive obese patients had greater diameters of the left atrium, left ventricle, and aortic root; thicker ventricular posterior and septal walls; and increased ventricular mass, cardiac output, and stroke work than did normotensive nonobese individuals [21]. When hypertension was added, there was a further increase in posterior wall thickness, diastolic wall stress, and stroke work, and a lower radius to posterior wall thickness ratio, indicating concentric hypertrophy. The cardiac adaption to obesity alone consisted of left ventricular dilation and eccentric hypertrophy irrespective of arterial pressure; when hypertension was superimposed, concentric hypertrophy occurred.

The results just discussed indicate that there are at least two types of obese hypertensive individuals: one in which the hypertension is the result of the obesity and the other in which hypertension and obesity are not causally related. The former are those patients described by Alexander and Messerli et al. [17,18] who have hypervolemia, elevated cardiac output, and normal vascular resistance; their hypertension results from an inability to vasodilate appropriately in response to the elevated cardiac output. The second type, as described by Mujais et al. and Messerli et al. [19,20], has elevated vascular resistance as its hemodynamic hallmark.

This classification of hypertension in obese people was suggested because of the hemodynamic consequences of weight reduction. For example, Alexander, who studied extremely obese patients, found that substantial weight loss was associated with a fall in pressure, decreased blood volume and cardiac output, and unchanged vascular resistance [22]. Reisin et al. compared two groups of obese hypertensives, one who lost weight and the other who did not [23]. Those that lost weight had a fall in pressure and cardiac output

with unchanged vascular resistance, in contrast with those in whom weight was stable who had no change in pressure, vascular resistance, or cardiac output. It seems likely that obese patients with elevated vascular resistance may not have blood pressure as responsive to weight reduction as those with "normal" resistance.

Mechanisms of Obesity-Associated Hypertension

Not all obese patients are hypertensive yet they too have hypervolemia and increased cardiac output [17,18], as would be expected as a logical consequence of an increased body mass. Thus, the hypertension that is the result of obesity is brought about by the failure of normal vasodilatory mechanisms. Three possible mechanisms have been suggested: hyperinsulinemia, abnormal aldosterone-renin relationships, and increased noradrenergic activity due to overfeeding.

Hyperinsulinemia

Insulin resistance and hyperinsulinemia are hallmarks of obesity and, as indicated above, they have been linked to hypertension [15]. Other reports have also postulated such a role [24–26]. However, since hyperinsulinemia is characteristic of obesity regardless of blood pressure, this possible relationship requires further investigation. The subject is of considerable current interest because Ferrannini et al. found insulin resistance in young hypertensives who were not overweight [23]. For the purpose of this discussion it should also be noted that Falkner et al. recently reported finding insulin resistance in lean, normoglycemic young black males with borderline hypertension [37]. At present there is no explanation for this and the situation is made more complicated by a report of insulin resistance in patients with thalassemia major treated by hypertransfusion who apparently were normotensive [27].

Abnormal aldosterone-renin relationships

This possibility was suggested by Hiramatsu et al., who measured the plasma renin activity and aldosterone concentration in 85 patients whose ideal weight ranged from −10% to +49% and found that as body weight increased the aldosterone/renin activity ratio rose as plasma renin activity fell progressively [28]. Furthermore, combination therapy with hydrochlorothiazide-triamterene given along with a low-sodium and low-calorie diet normalized the ratio and reduced arterial pressure. In this regard, it should be noted that Dahl, Silver, and Christie considered the hypertension of obesity to be salt dependent [29], and we have recently described a role for aldosterone as a factor in salt-induced changes in arterial pressure [30].

Increased noradrenergic activity

Studies in rats have shown that fasting and refeeding influence catecholamine metabolism. In one study, Landsburg and Young found a reduced cardiac norepinephrine turnover in rats fasted for 2 days and a substantial increase in turnover after 3 days of glucose refeeding [31]. These and other laboratory studies raise the possibility that the noradrenergic division of the autonomic nervous system plays a role in the hypertension of obesity. Both Reisin et al. and Sowers et al. found that weight reduction of obese patients was associated with a fall in plasma norepinephrine [20,32]. However, this apparently is not a universal effect because Amatruda et al. found a reduction in only 2 of 5 obese patients after 40 days of a very low calorie (420 kcal) diet, which resulted in a 10% weight loss [33]. James and associates found other evidence of changes in noradrenergic activity during weight loss by measuring the urinary excretion rate of the catecholamine metabolite, 4-hydroxy-3-methoxy-mandelic acid (HMMA) [34]. They found that when normotensive obese women lost weight there was not only a fall in blood pressure and heart rate, but also a decrease in the excretion of HMMA, and that carbohydrate refeeding promptly increased it. However, when they studied obese hypertensives, they found that the excretion of HMMA did not decrease as much during weight loss as it had in the normotensive obese subjects.

This is a fruitful area of investigation and it would be important to know whether obese black hypertensives in any way differ from white obese hypertensives in regard to estimates of noradrenergic activity at baseline and during weight reduction.

Effects of Weight Loss

There is very little information about the effects of weight loss in obese black patients. Tyroler and colleagues enrolled 63 Evans County, Georgia overweight hypertensives in a treatment program of weight reduction and moderate sodium restriction [10]. Of these, 28 were black females. This group was contrasted with results obtained in a control group of 64, of which 25 were black women. After a year of treatment, the experimental group had lost an average of 18 lbs. and had a decrease in arterial pressure. Although the control group also had a decrease in pressure, it was less than that found in the treated group and, in the latter, fewer of the treatment group

were taking antihypertensive drugs than in the control group. This study is not as "clean" as one would want it to be in regard to the effect of weight reduction alone, because moderate sodium restriction was used, although no information was given as to the adequacy of dietary sodium control, nor was any specific mention made as to the responses of the black patients.

As indicated above, there has been a suggestion that some fraction of hypertension associated with obesity is salt sensitive [29]. In contrast, Reisin et al. found that two thirds of their patients achieved normal blood pressure prior to achieving normal weight during several months of calorie restriction during which sodium intake was not reduced [35]. Also, Tuck et al. studied the effects of weight reduction in two groups of patients, one of which was given a 40 mEq sodium intake and the other 120 mEq [37]. They found no difference in the blood pressure fall with the two diets.

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