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Body Weight, Blood Pressure, and Mortality in a Cohort of Obese Patients

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The associations between body weight, raised blood pressure, and mortality remain controversial. The authors examined these relations by considering all degrees of obesity in the Düsseldorf Obesity Mortality Study (1961–1994). Among 6,193 obese German patients aged 18–75 years and having a body mass index (BMI) of ≥25 kg/m^2, 1,059 deaths were observed after a median follow-up of 14.8 years. The entire cohort was grouped into quartiles according to BMI (25–<32, 32–<36, 36–<40, ≥40 kg/m^2) and systolic blood pressure (SBP) (<140, 140–<160, 160–<180, ≥180 mmHg). Cox proportional hazards analyses were performed to adjust for age. For women, the mortality risk curves for the four BMI groups in relation to SBP were flat without crossing, whereas the risk curve for moderately obese men (BMI 25–<32 kg/m^2) crossed the risk curves for the higher BMI groups. In the group of patients with very high blood pressure (SBP ≥ 180 mmHg), moderately obese subjects (BMI 25–<32 kg/m^2) had a higher mortality risk for men when compared with the BMI group 32–<36 kg/m^2 (hazard ratio =1.62, 95% confidence interval: 1.0, 2.7) but not for women (hazard ratio = 0.71, 95% confidence interval: 0.4, 1.2). These findings support previous observations that the risk of death is lower for hypertensive men in high compared with low BMI groups. *Am J Epidemiol* 2002;156:239–45.

blood pressure; body mass index; cohort studies; hypertension; mortality; obesity; obesity, morbid

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure.

The associations between body weight, raised blood pressure, and mortality are controversial. Although hypertension and obesity have been identified as risk factors for cardiovascular and total mortality, a number of studies have found that lean, hypertensive subjects are at greater risk than obese hypertensives (1–9). Most of these studies were based on observations among men; only a few studies have also investigated women (6, 7, 9, 10). Some authors have suggested that the high mortality rate among lean people with hypertension may be due to unhealthful lifestyles such as smoking and excess alcohol intake (5, 6, 11). Others have hypothesized a protective effect of obesity with regard to the cardiovascular risks associated with hypertension (1–4, 7). Some reasons for discrepancies between study results may be their different age distributions, different follow-up times, and different choices of cutoff points for blood pressure and body mass index (BMI) (5, 10, 12). Only scant information exists about the relations between blood pressure, body weight, and mortality in subjects with higher degrees of obesity.

In the present study, the associations between body weight, blood pressure, and mortality were investigated by using data from the Düsseldorf Obesity Mortality Study (13, 14). In this study, a large cohort of obese patients, including a considerable number of those who were grossly (BMI 32–<40 kg/m^2) and morbidly (BMI ≥ 40 kg/m^2) obese, was recruited over a period of 33 years and was followed for a median of 14.8 years. Excess mortality associated with several degrees of obesity was assessed by comparing the study cohort with the general population living in the same geographic area (13). It has been shown that the excess mortality associated with obesity declines with age (14).

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Here, we present an analysis of the blood pressure data to investigate the relation of body weight and blood pressure to mortality for all degrees of obesity.

MATERIALS AND METHODS

Subjects and data

The Düsseldorf Obesity Mortality Study is a prospective cohort study of 6,193 obese patients (1,591 men, 4,602 women) who were referred to the obesity clinic of the Heinrich-Heine-University of Düsseldorf, Germany, between 1961 and 1994. In general, these overweight patients were referred to the obesity clinic by their general practitioners to subject the patients to a 1,000-calorie dietary treatment, including elements of group therapy and behavior modification (15). Some patients were also referred by the endocrine clinic of this medical school or by surgical departments so they could lose weight before elective surgery. All obesity clinic services are provided free of charge to patients. The initial medical examination includes a case history, physical examination, and clinical chemistry.

A detailed description of the study design and data collection has already been published (13). In short, the following baseline data were collected for each patient at the initial medical examination: date of examination, name, current address, sex, date of birth, height, weight, blood pressure, glucose tolerance, and cholesterol. Since 1977, additional information on smoking habits has been collected systematically. Inclusion criteria were age at entry 18–75 years and BMI ≥ 25 kg /m^2. Height and weight were measured with the patient wearing light clothes (shirts and trousers or skirts) and no shoes. Blood pressure was measured by means of a mercury sphygmomanometer with an 18×42-cm extra-large cuff (16). To determine serum cholesterol and blood glucose levels, routine clinical chemistry methodology was used, as described previously (16). Glucose tolerance was assessed by measuring the 2-hour capillary blood glucose level following a 10-g oral glucose load given after an overnight fast. A patient was classified as having diabetes if the diagnosis of diabetes was already known or if the fasting blood glucose value was ≥6.7 mmol/liter (120 mg/dl) or the glucose tolerance test yielded a capillary blood glucose value of ≥11.1 mmol/liter (200 mg/dl). A patient was classified as having impaired glucose tolerance if the glucose tolerance test yielded 2-hour capillary blood glucose values of ≥7.8 mmol/liter (140 mg/dl) and <11.1 mmol/liter (200 mg/dl). After 1972, all patients have had to sign an agreement that their anonymous data could be used in the context of scientific studies.

Mortality follow-up

Vital status was ascertained from municipal residents' registries. We were able to determine vital status up to 1994–1995 for 5,775 patients (93.3 percent). Additionally, the vital status of 278 patients for any time earlier than 1994 was obtained from former follow-up investigations or from removal dates. Thus, we could calculate patient-year data based on 6,053 patients (97.7 percent). Only 140 patients (2.3 percent) contributed no survival information. All available survival data were used as either an event or a censored observation. Patients' survival time was calculated principally on a daily basis.

Statistical analysis

To investigate the associations between body weight, blood pressure, and mortality, we grouped the study population approximately into quartiles according to BMI (group 1: 25–<32, group 2: 32–<36, group 3: 36–<40, group 4: ≥40 kg/m^2) and systolic blood pressure (SBP) (group 1: <140, group 2: 140–<160, group 3: 160–<180, group 4: ≥180 mmHg). Additionally, four combined blood pressure groups were defined by using the following categories: normal blood pressure (SBP < 140 and diastolic blood pressure (DBP) < 90 mmHg); mild hypertension (SBP 140–<160 or DBP 90–<100 mmHg); moderate hypertension (SBP 160–<180 or DBP 100–<110 mmHg); and severe hypertension (SBP ≥ 180 or DBP ≥ 110 mmHg) (17). A BMI of 25–32 kg/m^2 (group 1) was defined as moderate obesity, a BMI of 32–40 kg/m^2 (groups 2 and 3) as gross obesity, and a BMI of ≥40 kg/m^2 (group 4) as morbid obesity. Bivariate associations were analyzed by using Pearson (continuous variables) and Spearman correlation coefficients (ordinal and binary variables). Crude mortality rates were calculated for the cross-classified BMI × SBP and the BMI × combined blood pressure groups by means of the number of deaths per 1,000 patient-years. Hazard ratios for increasing blood pressure adjusted for age were calculated by means of the Cox proportional hazards model (18). The proportional hazards assumption was checked by plotting the log of the negative log of the estimated survival functions against log time. Interactions were investigated by testing the first-order cross-product terms. Because of the exploratory character of the study, no adjustments for multiple hypotheses testing were performed (19). For computations, the SAS software procedures TABULATE (20), CORR (20), FREQ (21), and PHREG (22) were used.

RESULTS

Our study patients were recruited between 1961 and 1994 and were followed for a median of 14.8 years (range, 0–33; interquartile range, 7.3–20.2; mean, 14.3; standard deviation, 8.2 years). The range of BMI was 25.0–74.4 kg/m^2 (mean, 36.6; standard deviation, 6.1 kg/m^2); for age, it was 18–74 years (mean, 40.4; standard deviation, 12.9 years). As of 1994–1995, 1,059 (17.1 percent) patients had died (379 men, 680 women). The total number of observed patient-years was 88,646 (men: 22,257, women: 66,389). A descriptive analysis of the baseline data for the four SBP groups is shown in table 1. The correlation coefficients (Rs) for BMI with age, blood pressure, and glucose tolerance are given in table 2. It is conspicuous that the correlation of BMI with age was negative for men ($R = -0.107$, $p < 0.0001$) but positive for women ($R = 0.076$, $p < 0.0001$). Furthermore, diabetes prevalence increased with blood pressure for women only (table 1), leading to a positive correlation between BMI and

TABLE 1. Descriptive analysis of the baseline data* for four systolic blood pressure groups of patients in the Düsseldorf Obesity Mortality Study, 1961–1994

	Total (n = 6,193)	Systolic blood pressure (mmHg)			
		<140 (n = 1,298)	140–<160 (n = 1,676)	160–<180 (n = 1,450)	≥180 (n = 1,513)
Men	(n = 1,591)	(n = 270)	(n = 423)	(n = 389)	(n = 416)
Age (years)	39.7 (13.2)	39.5 (12.7)	38.9 (13.1)	39.6 (13.2)	40.5 (14.0)
Body mass index (kg/m^2)	36.6 (5.9)	35.0 (4.7)	35.4 (5.0)	37.1 (5.8)	38.4 (6.6)
Weight (kg)	112.6 (20.3)	108.5 (18.0)	109.7 (18.4)	113.5 (20.4)	117.6 (21.7)
Height (cm)	175.3 (7.1)	175.9 (7.6)	175.8 (7.5)	174.7 (6.7)	174.9 (6.9)
Systolic blood pressure (mmHg)	161.4 (26.9)	126.6 (7.9)	146.2 (5.2)	165.3 (5.2)	195.9 (17.2)
Diastolic blood pressure (mmHg)	100.8 (17.3)	83.8 (9.1)	93.8 (10.1)	103.6 (11.3)	116.5 (17.5)
Cholesterol (mmol/liter)	6.21 (1.51)	6.23 (1.49)	6.20 (1.75)	6.13 (1.35)	6.27 (1.41)
Diabetes	304 (21.6%)	55 (22%)	86 (23%)	66 (19%)	74 (19%)
Impaired glucose tolerance	217 (15.4%)	31 (13%)	52 (14%)	54 (15%)	79 (20%)
Ever smoking	341 (65.5%)	123 (43%)	100 (65%)	73 (68%)	44 (47%)
Women	(n = 4,602)	(n = 1,028)	(n = 1,253)	(n = 1,061)	(n = 1,097)
Age (years)	40.7 (12.8)	35.9 (11.5)	39.7 (12.6)	41.5 (12.7)	45.3 (12.6)
Body mass index (kg/m^2)	36.6 (6.2)	34.8 (5.5)	35.6 (5.5)	37.0 (6.2)	39.0 (6.7)
Weight (kg)	97.2 (17.4)	93.6 (15.8)	94.6 (15.9)	97.8 (17.3)	102.8 (18.5)
Height (cm)	162.8 (6.7)	164.0 (6.7)	163.1 (6.4)	162.5 (6.9)	161.9 (6.7)
Systolic blood pressure (mmHg)	158.4 (28.3)	125.3 (8.6)	145.9 (5.4)	165.2 (5.3)	197.1 (19.0)
Diastolic blood pressure (mmHg)	97.8 (15.9)	83.2 (9.1)	92.6 (8.9)	101.3 (10.7)	114.0 (15.3)
Cholesterol (mmol/liter)	5.95 (1.29)	5.77 (1.22)	5.95 (1.28)	6.00 (1.31)	6.08 (1.33)
Diabetes	602 (14.7%)	75 (8%)	117 (10%)	152 (16%)	228 (23%)
Impaired glucose tolerance	696 (16.9%)	119 (12%)	158 (14%)	194 (20%)	219 (22%)
Ever smoking	639 (36.8%)	290 (48%)	191 (39%)	112 (34%)	46 (15%)

* Values are expressed as mean (standard deviation) or number (%). Data on body mass index were missing for 12 patients, blood pressure for 260 patients, glucose tolerance for 678 patients, cholesterol for 1,724 patients, and smoking for 3,937 patients.

diabetes for women ($R = 0.121$, $p < 0.0001$); this correlation was quite low for men ($R = –0.017$, $p = 0.4567$).

The crude mortality rate was 11.95 deaths per 1,000 patient-years (men: 17.03, women: 10.24). Overall, the mortality rates increased with BMI and SBP (table 3). However, for men, the highest mortality rate was observed in the group with moderate obesity (BMI 25–<32 kg/m^2) and very high SBP (SBP ≥ 180 mmHg). On the other hand, for women, the highest mortality rate was found in the group with the highest BMI (BMI ≥ 40 kg/m^2) and highest blood pressure (SBP ≥ 180 mmHg). Similar results were obtained when the combined blood pressure groups were used (table 4).

For moderately obese subjects (BMI 25–<32 kg/m^2), the age-adjusted hazard ratio for SBP per 10 mmHg, calculated by means of the Cox proportional hazards model, was highest for men but lowest for women compared with the hazard ratios for those in the other BMI groups (table 5). The corresponding mortality risk curves in relation to SBP in the four BMI groups calculated for a person aged 40 years after 15 years of follow-up increased slightly for women, without substantial crossing. However, for men, moderately obese patients had the steepest risk curve, which crossed the curves of the other BMI groups as SBP increased (figure 1). In the group of patients with very high blood pressure (SBP ≥ 180 mmHg), moderately obese subjects (BMI 25–<32 kg/m^2) had an almost significantly higher mortality risk for men when compared with BMI group 2 (32–<36 kg/m^2) (hazard ratio = 1.62, 95 percent confidence interval: 1.0, 2.7; p = 0.067) but not for women (hazard ratio = 0.71, 95 percent confidence interval: 0.4, 1.2; p = 0.166). In Cox models in which SBP and BMI were used as continuous covariates, the corresponding interaction term was not significant for women (p = 0.681) or for men (p = 0.452).

DISCUSSION

This study represents by far the largest known mortality follow-up of a cohort of obese patients, which includes a considerable number of grossly (BMI 32–40 kg/m^2) and morbidly obese (BMI > 40 kg/m^2) subjects. We investigated the relation and interaction of BMI and blood pressure with mortality in a group of more than 6,000 obese patients. The entire cohort was grouped into quartiles according to BMI

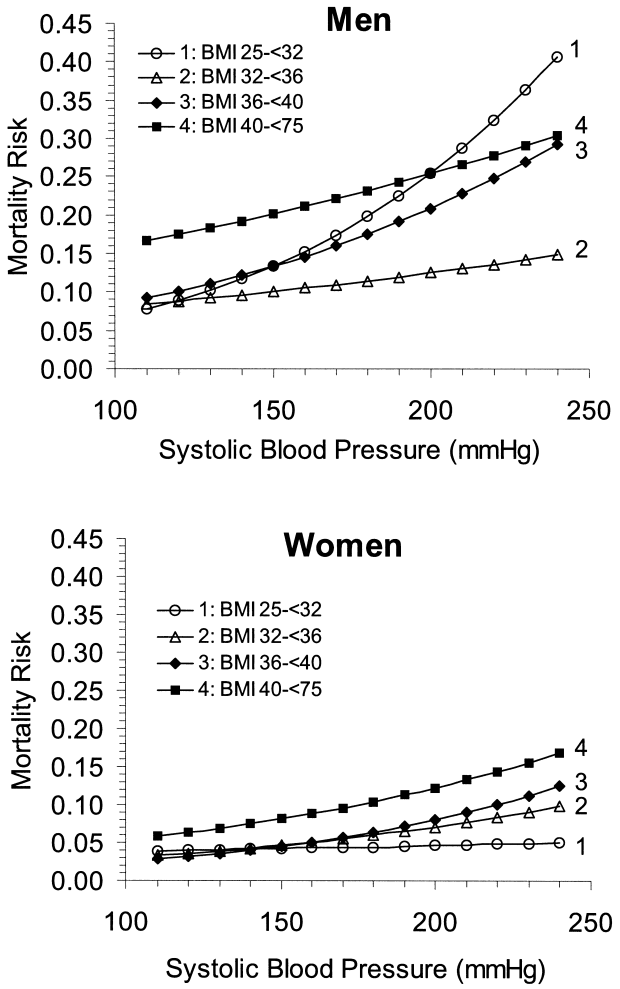

FIGURE 1. Age-adjusted mortality risk after 15 years for obese men and women in relation to systolic blood pressure and body mass index (BMI; kg/m^2) groups, as derived from the Cox proportional hazards model, Düsseldorf Obesity Mortality Study, 1961–1994.

and SBP. For women, mortality increased as BMI and SBP increased. The female group with the highest mortality (20 deaths per 1,000 patient-years, age-adjusted mortality risk = 0.10–0.17) was that with morbid obesity (BMI ≥ 40 kg/m^2) and very high blood pressure (SBP ≥ 180 mmHg); for men, the highest mortality rate (32 deaths per 1,000 patient-years, age-adjusted mortality risk = 0.20–0.41) was observed in the group with moderate obesity (BMI 25–<32 kg/m^2) and very high blood pressure (SBP ≥ 180 mmHg).

These findings support the hypothesis that, for obese men, the hypertension-associated risk of death is increased for lean versus more-overweight patients. Several studies have shown similar tendencies when comparing lean with normal-weight or moderately overweight people with hypertension (1–9). Thus, Barrett-Connor and Khaw (1) and Carman et al. (7) divided their study population of hypertensives into those subjects whose BMI was more than or less than 27 kg/m^2, and Menotti (5) used a cutoff point of 24.9 kg/m^2. In other studies, the subjects were stratified by means of BMI tertiles or quintiles (2–4, 8, 9), with nearly all obese people stratified in one BMI group with an upper limit of 27–30 kg/m^2. In the Hypertension Detection and Follow-up Program, a highly increased mortality risk was found for only very lean, hypertensive patients (BMI < 22 kg/m^2), whereas, in the obese groups, no significant differences could be demonstrated, probably because of small numbers of subjects (6).

TABLE 2. Correlation coefficients for body mass index with age, blood pressure, and glucose tolerance for patients in the Düsseldorf Obesity Mortality Study, 1961–1994

	Correlation coefficient (*R*)	
	Men	Women
Age	–0.107	0.076
Systolic blood pressure	0.243	0.286
Diastolic blood pressure	0.243	0.293
Glucose tolerance*	0.007	0.152
Diabetes	–0.017	0.121
Impaired glucose tolerance	0.041	0.068

* 0 = normal, 1 = impaired glucose tolerance, 2 = diabetes.

Two studies did not report an inverse relation between obesity and cardiovascular disease in hypertensive patients (12, 23), but both studies did not investigate mortality risk due to all causes. Moreover, in the Honolulu Heart Program, a specific population of Japanese Americans was studied (12). In the Framingham Study, it was suggested that lean compared with overweight hypertensives may be at increased risk of death due to cardiovascular disease after 8 years, but no evidence for this hypothesis was found after 26 years of follow-up (23). However, no specific data were provided in the results section of their paper, and a relation of cardiovascular death to degree of obesity, after adjusting for major cardiovascular risk factors including SBP, was found for women only. The data from our study reveal that, for men, not only are lean hypertensives at greater risk than obese hypertensives, but moderately obese hypertensives with very high blood pressure are also at higher risk than grossly or even morbidly obese hypertensive patients.

Four of the above-mentioned studies also investigated females; they showed that leaner female hypertensives were at higher risk (6, 7, 9, 10). The comparison was between either 1) lean and normal-weight (6) or lean and higher-weight quintiles (normal weight and moderate obesity) (10) or 2) normal-weight and obese females (7, 9). In our large, obese female population, an apparent protective effect of overweight in hypertensive women does not seem to be operative in those who are grossly or morbidly obese. The reasons that we were not able to observe a protective effect of overweight in hypertensive women are unclear. However, well-known differences in the effects of obesity on atherogenic risk factors and mortality that exist between sexes (13, 24–26) and also races (24, 26) may be responsible.

The following limitations of our study have to be considered. Although more than 6,000 obese patients were followed for a median period of 15 years, our capacity to

TABLE 3. Crude mortality rates per 1,000 patient-years for four body mass index and systolic blood pressure groups of patients in the Düsseldorf Obesity Mortality Study, 1961–1994

Body mass index (kg/m^2)	Total	Systolic blood pressure (mmHg)			
		<140	140–<160	160–<180	≥180
Men					
<32	16.01	8.69	9.17	16.23	32.25
32–<36	15.08	9.75	12.53	12.74	22.92
36–<40	17.27	10.41	14.06	19.46	19.05
≥40	20.91	5.78	13.08	19.07	27.86
Total	17.03	9.36	12.06	16.66	24.62
Women					
<32	6.06	3.94	4.66	7.18	9.84
32–<36	9.01	3.34	5.44	10.18	16.30
36–<40	10.20	3.77	5.26	9.65	17.19
≥40	15.51	5.24	9.31	14.99	20.37
Total	10.24	4.21	5.89	10.70	17.32

TABLE 4. Crude mortality rates per 1,000 patient-years for four body mass index and combined blood pressure groups of patients in the Düsseldorf Obesity Mortality Study, 1961–1994

Body mass index (kg/m^2)	Total	Combined blood pressure group*			
		Normal blood pressure	Mild hypertension	Moderate hypertension	Severe hypertension
Men					
<32	16.01	9.37	10.54	11.20	26.76
32–<36	15.08	7.91	10.05	12.93	20.96
36–<40	17.27	4.40	15.44	16.61	19.63
≥40	20.91	8.57	9.72	16.86	25.78
Total	17.03	7.48	11.10	14.07	22.73
Women					
<32	6.06	3.41	4.60	6.03	9.36
32–<36	9.01	3.60	5.46	8.62	13.86
36–<40	10.20	3.42	5.04	8.56	14.57
≥40	15.51	6.89	7.72	12.69	19.18
Total	10.24	3.91	5.44	8.92	15.41

* Normal blood pressure: systolic blood pressure (SBP) <140 and diastolic blood pressure (DBP) <90 mmHg; mild hypertension: SBP 140–<160 or DBP 90–<100 mmHg; moderate hypertension: SBP 160–<180 or DBP 100–<110 mmHg; severe hypertension: SBP ≥180 or DBP ≥110 mmHg.

determine mortality risks in terms of dependence on sex, BMI, and blood pressure was limited. When both sexes, four BMI groups, and four SBP groups are considered, the entire cohort was subdivided into 32 groups of limited size. Thus, the sample size was insufficient to reach statistical significance for the interaction term between BMI and blood pressure, which should be negative if the hypothesis of a benign effect of overweight in hypertensive patients is true.

The cohort cannot be considered a random sample. The correlation of BMI with age in our cohort was positive for women but negative for men. The higher age of moderately obese men may explain in part the high mortality rate for moderately obese men with very high blood pressure. However, the negative correlation of BMI with age was present in all male blood pressure groups, not only in the group with very high SBP. Moreover, after we adjusted for age, moderately obese men still had the steepest risk curve, and the curve crossed the curves of the other BMI groups as blood pressure increased.

It is unusual that diabetes prevalence increases with blood pressure in women but not in men. Because diabetes is positively correlated with overweight and hypertension (27), an increasing prevalence of diabetes with increasing BMI and blood pressure is to be expected. Different (self-)referral

TABLE 5. Age-adjusted hazard ratios of systolic blood pressure per 10 mmHg as derived by the Cox proportional hazards model, Düsseldorf Obesity Mortality Study, 1961–1994

Body mass index (kg/m^2)	Men			Women		
	Hazard ratio	95% confidence interval	*p* value	Hazard ratio	95% confidence interval	*p* value
<32	1.154	1.05, 1.27	0.004	1.020	0.93, 1.12	0.675
32–<36	1.047	0.97, 1.13	0.240	1.091	1.03, 1.16	0.005
36–<40	1.103	1.01, 1.21	0.032	1.126	1.06, 1.20	<0.001
≥40	1.055	0.98, 1.13	0.149	1.090	1.05, 1.14	<0.001

patterns between men and women may have masked existing associations of diabetes with obesity and blood pressure in men. However, it is unlikely that the absent association between diabetes prevalence and hypertension produced the strong relation observed between blood pressure and mortality in moderately obese men. On the contrary, if there is any effect of the missing trend of increasing diabetes prevalence with blood pressure, it would be a diminution of the relation between blood pressure and mortality in all obesity groups.

Information on putative confounding factors, such as smoking, alcohol intake, medication, and body fat distribution, as well as patients' social status and activity level, was insufficient to be included in the analysis of this cohort recruited during a period of 33 years. It has clearly been shown that excess alcohol intake and smoking are important risk factors contributing to the excess risk for lean hypertensives (6, 9). A high percentage of underweight people are heavy smokers (6). However, even after adjustment for these risk factors in most of the former studies, the lean hypertensives were at higher risk (1, 3, 4, 7–9). In our study, information about smoking was available for only one third of the patients. Among men, the percentage of smokers and former smokers (40 percent) was lowest in the group of moderately obese hypertensives with the highest mortality risk. Among women, there was a trend for less smoking with increasing blood pressure.

No systematic information was available regarding the course of obesity after patients were recruited. Thus, the possible effects of weight change could not be investigated. Earlier analyses of subgroups of patients indicate that the overall long-term effect on weight reduction as a result of our obesity clinic intervention was almost negligible. As in other comparable reports, approximately 50 percent of patients did not attend the therapeutic program after the initial sessions; for the remaining patients, a mean weight loss of about 9 kg was achieved after a mean duration of 6 months. However, a relevant long-term weight reduction was demonstrable in less than 5 percent of patients (15).

Despite these limitations, our data complement previous results indicating that the relative risk associated with high blood pressure declines with increasing adiposity in hypertensive men. There is speculation that the differences in outcome for lean and obese hypertensive subjects may reflect different entities of hypertension. If men become hypertensive despite being lean, the underlying causal factors may add to their overall mortality risk. Lean hypertensive subjects may carry stronger genetic determinants for hypertension that may also be linked to other yet-unknown risk factors. Epidemiologic studies have shown that an increase in blood pressure is correlated with weight change (28). Therefore, it may be possible that the duration of hypertension was shorter in the obese, especially for those persons who gained weight later in their adult life. In addition, Messerli has suggested that lower vascular resistance associated with an increase in adiposity could serve as a mechanism by which obesity exerts a protective effect on certain vascular beds (29).

In summary, our findings support previous observations that the risk of death is lower in high compared with low BMI groups of hypertensive men. An apparent protective effect of overweight in hypertensive patients could not be demonstrated in an obese female population.

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