had urinary flow rates of 15 ml/s or more despite our aiming at excluding them: the outcome in this group was satisfactory, in agreement with previous studies.[26-28] On the other hand, Jensen *et al* recently reported that urinary flow rates above 15 ml/s were associated with a poor outcome (5/17) compared with the remainder (10/117),[12] but the numbers were small and most men with high flow rates did well.

Most men contemplating the prospect of prostatectomy should be reassured by our study. There is, however, a clear need to identify beforehand that quarter of men who do not benefit greatly from the operation. Though the performance of a speculative prostatectomy as a diagnostic test is unsatisfactory, urodynamic studies, as presently carried out, do not seem to hold the key to this problem.

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Independent effects of weight change and attained body weight on prevalence of arterial hypertension in obese and non-obese men

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Abstract

***Objective*—To assess the relations among prevalence of arterial hypertension, history of weight change, and current body weight in the range from normal weight to severe obesity.**

***Design*—Retrospective analysis of medical records of men registered with Danish military authorities from 1943 to 1977 and followed up four to 40 years later.**

***Setting*—Draft board of Copenhagen and surrounding counties and the rest of Sjaelland and surrounding islands.**

***Subjects*—964 Men who were severely obese (body mass index ≥31 kg/m^2 at the first examination) and 1134 random controls.**

***Main outcome measures*—Blood pressure and weight.**

***Results*—Hypertension was more prevalent in subjects with an unchanged body mass index as that index increased over the range studied. At any body mass index hypertension was more prevalent in subjects who had increased to this index and less common in those who had decreased to it than in those who had stayed the same weight since the first examination. Hypertension among controls was most common in those subjects who had become obese during adulthood.**

***Conclusions*—Changes in body weight have a great influence on arterial hypertension independent of the effect of attained weight, particularly in obese subjects.**

Introduction

Several prospective clinical studies have shown a correlation between change in weight and blood pressure in severely obese patients treated with diet or a bariatric operation.[1-3] This effect is so pronounced that a reducing diet is considered a rational treatment of moderate hypertension in obese subjects.[4,5] The mechanism of the close relation between body weight and blood pressure is not fully clarified.[6] One major unsolved question is whether the blood pressure after changes in body weight corresponds to the predicted blood pressure for the attained body weight. If it does the body composition as such rather than the effects of the changes in body weight is essential to the blood pressure. This is particularly important in the understanding of the association between obesity and hypertension.

We investigated the relations among current blood pressure, current body mass index, and body mass index assessed four to 40 years earlier of men who at the first examination had been either severely obese or representative of the underlying population.

Subjects and methods

STUDY POPULATION

All Danish men are registered with the military authorities at the age of 18 and are examined by the medical board within the next few years. The records of volunteers are not available, but, according to the authorities, this group comprised only about 2% of the men during the period of this study.

The study population comprised the 400 975 men processed by the board in the metropolitan area of Copenhagen and surrounding counties from 1943 to 1977 and in the remaining part of Sjaelland and surrounding islands from 1964 to 1977. All underwent systematic examination, including measurement of height and weight, except for 18 000 who were unfit for service and did not appear before the board. This group did not include subjects with severe obesity or known arterial hypertension, according to a random survey of 529 records.[7] Records were unavailable for those examined in the metropolitan area and found fit for service from 1958 to 1968 who were living in another region in 1969. Thus 362 200 were available for the study. The population has been described elsewhere.[8-10]

We defined severe obesity as a body mass index ≥ 31 kg/m^2, which is about 45% or more above the old insurance standard.[11] We made a complete search of the files and found 1940 men who fulfilled the criterion for severe obesity at the first examination. A control group was derived from a random sample of the study population (4010). After we had excluded those for whom height and weight measurements were not available and 21 severely obese subjects already included in the obese group the control group comprised 3601 men.

FOLLOW UP STUDY

All subjects in the obese group and half of the controls, a total of 3707 men, were selected for the follow up study. Every third month during 1 November 1981 to 15 August 1983 information was obtained from the Central Person Register regarding the current address of subjects scheduled for examination during the ensuing three months. Only subjects living in the eastern part of Denmark at the time of examination were invited—that is, 1651 (85·1%) obese subjects and 1504 (83·5%) controls. The others had moved to another region (395), emigrated (79), disappeared (6), or died (65) or could not be traced (7). Those selected for the follow up examination were invited by letter to participate in a health examination performed by the Copenhagen city heart study at the Rigshospital.[12] The response rate was 58·4% (964/1651) for the obese subjects and 75·4% (1134/1504) for the controls. The median duration of the interval between the first examination and follow up was 14·5 years (range 4-40 years).

At follow up the subjects were weighed and measured while wearing light indoor clothes and no shoes. Mechanical balances were used for subjects weighing up to 150 kg. Subjects weighing over 150 kg were weighed with two spring balances. The subjects were weighed between 8 am and 4 pm and had not fasted. We used a London School of Hygiene sphygmomanometer[13] to measure the blood pressure in the left arm after at least five minutes' rest in the sitting position. The diastolic pressure was measured at the disappearance of the Korotkoff sound (phase V). Normally a cuff measuring 12×22 cm was used, but for subjects with a circumference of the upper arm exceeding 46 cm a cuff measuring 15×38 cm was used. The blood pressure was classified according to the recommendations of the World Health Organisation.[14] Normal blood pressure was defined as a systolic pressure ≤ 140 mm Hg with a diastolic pressure ≤ 90 mm Hg. Hypertension was defined as a systolic pressure ≥ 160 mm Hg or a diastolic pressure ≥ 95 mm Hg, or both. Borderline hypertension was defined as blood pressure values between the normal and hypertensive ranges.

The response rate was related to the body mass index within the different weight groups as well as to several psychosocial and demographic factors, as described elsewhere.[15] To evaluate a possible selection bias we examined the relations between the psychosocial and demographic factors and the prevalence of hypertension.

STATISTICAL METHODS

The statistical analyses were carried out with χ^2 test, Goodman-Kruskal's gamma test (for evaluating trends in two or three dimensional contingency tables), Spearman's rank correlation coefficient, and logistic regression models (for multivariate assessment of determinants of prevalence of hypertension).

TABLE I—*Prevalence of hypertension and age at follow up among obese subjects and controls*

Age at follow up (years)	Obese subjects		Controls	
	No of subjects	No (%) with hypertension	No of subjects	No (%) with hypertension
22-	142	34 (24)	93	5 (5)
27-	189	51 (27)	121	13 (11)
30-	113	28 (25)	87	7 (8)
32-	112	32 (29)	118	10 (9)
34-	136	50 (37)	106	10 (9)
36-	97	43 (44)	109	17 (16)
38-	86	35 (41)	125	19 (15)
41-	41	20 (49)	129	31 (24)
46-65	47	13 (28)	244	68 (28)
Total	963*	306 (32)	1132*	180 (16)

*In one obese and two control subjects Korotkoff sound phase V could not be identified.

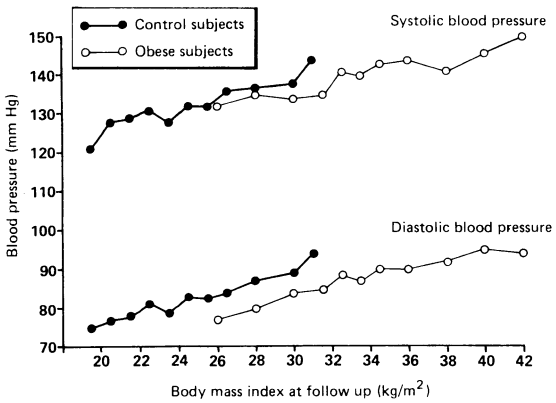

FIG 1—*Median systolic and diastolic blood pressures and body mass index at follow up for obese subjects and controls*

Results

Among the obese subjects and controls body mass index at follow up and systolic and diastolic blood pressure were positively correlated (for obese subjects $r=0·32$ for systolic blood pressure and 0·34 for diastolic blood pressure and for controls $r=0·31$ and 0·35 respectively, all $p<0·001$) (fig 1). The systolic blood pressure correlated significantly with age in the controls ($r=0·197$) but not in the obese subjects ($r=0·055$). The diastolic blood pressure correlated significantly with age for both obese subjects and

controls (r=0·166 and 0·301 respectively). Within each age group, with the exception of 46-65 years, hypertension was consistently more prevalent in obese subjects (table I).

To estimate the impact of previous changes in weight on current blood pressure the subjects were divided into four weight groups according to their body mass index at both the first examination and follow up, making a total of 16 classes (table II and fig 2). Five of the weight classes contained fewer than 10 subjects and are therefore not depicted. The diagonal values—representing those who remained in the same weight class—show rising blood pressure and prevalence of hypertension with increasing body mass index. Within each class of body mass index at the first examination the subjects with the highest body mass index at follow up showed the highest blood pressure and the highest prevalence of hypertension, and those who had lost weight had the lowest blood pressure and prevalence of hypertension. Within each class of body mass index at follow up subjects who had gained weight showed the highest prevalence of hypertension and those who had lost weight the lowest prevalence (fig 2). The highest prevalence (61%) was observed among those whose body mass index had been 21-30 kg/m^2 and whose current body mass index was above 33 kg/m^2.

The relative risk (odds ratio) of hypertension was estimated with logistic regression models on the basis

TABLE II—*Mean systolic and diastolic blood pressure (mm Hg) and body mass index at first and follow up investigations**

Body mass index (kg/m^2) at first examination		Body mass index (kg/m^2) at follow up				
		<21	21-30	31-32	≥33	Total
<21	Systolic	125·7	134·3			132·4
	Diastolic	75·2	83·0			81·2
	No in group	117	384	2	0	503
21-30	Systolic		133·6	139·8	150·6	134·2
	Diastolic		82·4	89·4	94·2	82·9
	No in group	8	583	22	18	631
31-32	Systolic		134·6	139·3	144·9	140·4
	Diastolic		82·4	86·9	91·6	87·6
	No in group	1	192	105	280	578
≥33	Systolic		132·9	140·2	144·1	141·5
	Diastolic		82·0	86·7	89·6	87·8
	No in group	1	67	60	257	385
Total	Systolic	125·8	133·9	139·8	144·7	
	Diastolic	74·7	83·3	87·2	90·6	
	No in group	127	1226	189	555	2097

*In groups with <10 subjects no values are presented.

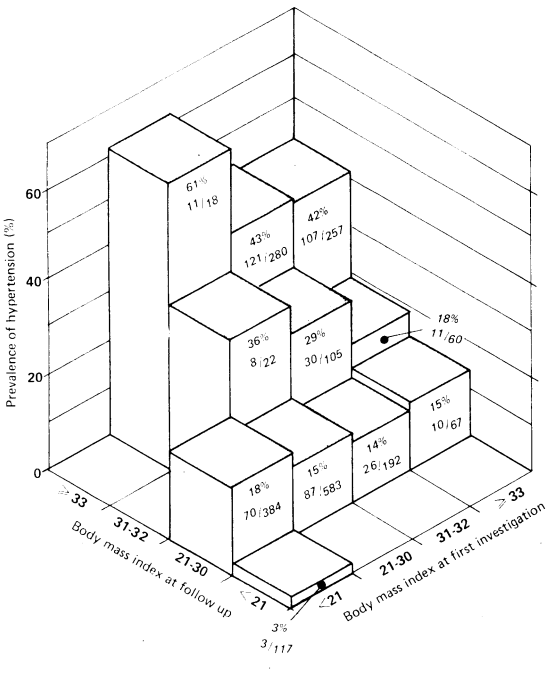

FIG 2—*Prevalence of hypertension and body mass index at first investigation and follow up. (Prevalence not shown for groups when n<10)*

TABLE III—*Coefficients (standard errors) obtained in logistic regression analyses of occurrence of hypertension**

	Obese subjects (n=963)	Controls (n=1132)
Constant	−4·411 (1·170)	−7·002 (0·995)
Body mass index at follow up	0·074 (0·032)	0·121 (0·039)
Change in body mass index	0·069 (0·004)	0·141 (0·050)
Age	0·030 (0·012)	0·044 (0·010)

*Odds ratio may be obtained as exponential function of coefficients.

of current body mass index, changes in body mass index, and age for the obese subjects and controls separately (table III). In both obese subjects and controls the model indicated that at any current body mass index the change in body mass index had an independent effect on the risk of hypertension of the same magnitude on a unit (kg/m^2) basis as the current body mass index itself. Thus in the obese subjects the odds ratio for a difference of 1·0 kg/m^2 at follow up was 1·08 (95% confidence interval 1·01 to 1·15) and for a change of 1·0 kg/m^2 it was 1·07 (1·06 to 1·08). In the controls the corresponding odds ratio for current body mass index was 1·13 (1·05 to 1·22) and for change in body mass index 1·15 (1·04 to 1·27).

There were no significant relations among intelligence test score, education, social state, and prevalence of hypertension (Goodman-Kruskal and χ^2 tests, all $p>0·2$).

Discussion

The main result obtained in our study was that for any given current body mass index subjects who had previously lost weight had a lower prevalence of hypertension than subjects who had remained at that weight. Subjects who had previously gained weight had a higher risk of hypertension than subjects who had stayed the same weight.

Both systolic and diastolic blood pressures of those obese subjects who had lost weight—those with a body mass index <31 kg/m^2 at follow up—were lower than those in controls with the same current body mass index. The results obtained for the controls were concordant with those obtained in cross sectional studies. It is therefore impossible to evaluate the effect of weight loss on blood pressure in severely obese subjects by extrapolation from the results in the controls. The effect of weight loss on blood pressure thus seems to be greater than expected from the relation between body weight and blood pressure in cross sectional studies. Similar considerations apply to weight gain.

In our study the rate of response among the controls (75%) was similar to that in other population studies.[15] Among the obese subjects the response rate was lower (58%), which was partly attributable to various psychosocial and demographic factors[15] that among the responders was unrelated to the subjects' blood pressure. Furthermore, morbidity among responders and non-responders among the obese subjects and the controls, as evaluated on the basis of admissions to hospital, showed no significant differences.[15] Another possible selection bias was that obese subjects with hypertension may have died before follow up. Increased mortality among severely obese people[7] probably could not have produced a bias because of the small number of deaths. It seems reasonable therefore to disregard early death among obese subjects as an important selection bias.

The relation between obesity and hypertension has been questioned by some investigators, who claim that the circumference of the upper arm may influence the blood pressure measured by a cuff, the measurements tending to be higher than intra-arterial pressure

measurements.[16-19] The relation between relative weight and intra-arterial blood pressure, however, as well as clinical manifestations of hypertension show that the correlation is not an artefact.[20,21] On the other hand, the strength of the relation may be exaggerated by the measurement bias.

The results in our controls cannot be compared directly with those of previous prospective population studies. In the Evans County study, comprising subjects under the age of 30 followed up for seven years, Johnson *et al* found that body mass index at both the first and second examinations was closely related to the blood pressure at the second examination.[22] In a multiple regression analysis including such risk factors as age, cholesterol concentration, blood pressure, and body mass index at the first examination they found body mass index to be the second most important factor, but neither the body mass index at the second examination nor the change in body mass index was taken into account.

Abraham *et al* studied the importance of childhood relative weight and adult relative weight in relation to the prevalence of hypertensive vascular disease and cardiovascular renal disease in a population of men.[23] The highest risk for disease was found in men who became overweight as adults. The higher prevalence of these diseases among overweight adults was largely attributable to adults who had changed from below average weight in childhood to being overweight as adults. Moderately or extremely overweight adults who had been similarly classified in childhood did not seem to be at greater risk than adults of average weight who had been of average weight as children. Our study shows that the effects of weight change in adult life on the prevalence of hypertension are similar to the effect of weight changes from childhood to adulthood on cardiovascular diseases later in life.

In the Evans County study Heyden *et al* found excessive weight gain and being overweight after the age of 20 to be definite risk factors for cerebrovascular disease.[24] The 2530 subjects were asked about their weight at the age of 20 and their subsequent maximum weight gain. At the examination neither the systolic nor the diastolic blood pressure showed a significant correlation to the weight at 20. There was, however, a significant correlation between weight gain and systolic and diastolic blood pressure. These results are consistent with the results from our control group.

Several hypotheses have been put forward to explain the mechanism between hypertension and obesity.[6] Irrespective of the mechanisms the relation between blood pressure and weight change at all levels of body mass index strongly suggests shared environmental causal factors. Possibly sodium intake in food or sodium retention (dependent on degree of insulinaemia), or both, influence the risk of hypertension.[6] If we assume that the weight changes are caused by corresponding changes in food consumption there are at least two possible mechanisms to be considered. Firstly, subjects of the same current body weight may differ in their preceding food consumption according to their preceding weight changes. Secondly, dietary changes may lead to changes in blood pressure earlier than changes in body weight. The findings suggest that obesity shares causal factors with arterial hypertension rather than leading to the disease.

In population studies hypertension has been singled out as one of the major risk factors for the development of cardiovascular diseases.[25-28] Some investigators, however, have found that hypertension constitutes less of a risk when associated with obesity.[29-31] A recent investigation from the Honolulu heart study, however, found after 12 years of observation an adverse effect of hypertension independent of body mass index.[32]

Our results have therapeutic implications for severely obese people. Despite the hopelessness of attaining normal weight, it may be worth while encouraging them to even minor weight losses as the effect on blood pressure may be greater than expected from the attained body weight

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