SUMMARY There is a close epidemiological association between obesity and blood pressure for all age groups, although not every obese individual becomes hypertensive. In populations without age-related increases in body weight, an elevation of blood pressure with age is not seen. Treatment of obesity by weight loss decreases blood pressure substantially; however, in a minority of patients blood pressure does not fall with weight loss. Blood pressure generally decreases before normal weight is achieved. Blood pressure after weight loss remains reduced as long as there is no marked regain of body weight. Salt intake reduction does not appear to explain why weight reduction lowers blood pressure. Reduced levels of plasma renin activity, serum aldosterone levels, catecholamine levels, and serum insulin levels may be involved in the blood pressure lowering associated with weight loss. Since the risk to the hypertensive patient is not only determined by the blood pressure, an overall treatment that aims at reduction of all risk factors is advocated. Some risk factors, e.g., glucose intolerance, may be normalized only when desirable weight is achieved. Thus, in any obese hypertensive patient with other risk factors, normalization of excess body weight appears to be the first and most important step of any rational therapeutic strategy. (Hypertension 4 (supp III): III-50-III-55, 1982)

EXTENSIVE studies have demonstrated that there is an association between obesity and hypertension. Much of the evidence for this association has been brought about by actuarial data, including recent results from the 1979 Blood Pressure Study (fig. 1). There is also a relation between hypertensive eye-ground changes and obesity. This finding favors a true connection between obesity and hypertension and refutes a possible artificial relation due to arm-girth-induced false blood pressure readings.

A close relationship of obesity and blood pressure was observed in a study of women with different degrees of overweight and a history of hypertension. It is noteworthy that the association was similar in all age groups investigated. A significant association between relative weight and blood pressure in both sexes was also reported in the Framingham Study. In the same study and in the Evans County Study, it could be demonstrated that overweight subjects who were normotensive at the onset of the study were more likely to develop hypertension later on than subjects without overweight. This finding was confirmed in a study of former college students of the University of Pennsylvania where ponderosity in college years was one of the characteristics that predicted the development of hypertension in middle life. In recent population studies from many different countries, the association between obesity and hypertension was consistently demonstrated. Also, in our Düsseldorf study both systolic and diastolic blood pressures were associated with body mass index (BMI). The highest blood pressures in both sexes were found in the oldest age group and in the highest BMI category.

Black individuals are more prone to have hypertension than white persons. Systolic and diastolic blood pressure levels have been clearly demonstrated to be higher in black males and females than in whites of the same relative weight. When unselected hypertensives were compared with normotensives, they were more often obese and more often had impaired glucose tolerance and a higher fasting insulin. Thus, many hypertensives display the metabolic picture of obesity. This group of patients could include so-called "metabolically obese" normal weight people, i.e., people with enlarged fat cells, hyperinsulinemia, hypertension, and other metabolic
derangements. It has also been shown that lean hypertensives are more likely to gain weight than normotensives.

It should be stressed that the association of overweight and blood pressure also exists in children and adolescents. In the Bogalusa heart study, the height of the children had definitely an influence on blood pressure, but in the different height ranges, body weight was correlated with blood pressure. The correlation of body weight and blood pressure was also observed in 1962 black children. Children of both sexes with elevated blood pressure were three times as likely to be obese than children in the total population.

There is a definite association of blood pressure with age, as demonstrated by the data from the National Health and Nutrition Examination Survey (NHANES) in 17,796 persons 6 to 74 years of age. In both sexes blood pressure increased with increasing age. This increase of blood pressure with age appears to be specific for so-called Western or acculturated Westernized populations. In contrast, rural and tribal nonaffluent populations show no such relationship between age and blood pressure. This may be due to the fact that body weight does not increase with age in such populations. This increase of blood pressure with age appears to be specific for so-called Western or acculturated Westernized populations. In contrast, rural and tribal nonaffluent populations show no such relationship between age and blood pressure. This may be due to the fact that body weight does not increase with age in such populations. This increase of blood pressure with age appears to be specific for so-called Western or acculturated Westernized populations. In contrast, rural and tribal nonaffluent populations show no such relationship between age and blood pressure. This may be due to the fact that body weight does not increase with age in such populations.

Elevated blood pressure is one of the most common and most potent risk factors for coronary heart disease, stroke, intermittent claudication, and congestive heart failure. Hypertension is the most frequent cardiovascular risk factor in obese patients. Therefore, obese patients have an increased risk of developing cardiovascular disease, and the most logical treatment appears to be weight reduction.

Treatment of Hypertension by Weight Reduction

Treatment of hypertension by weight reduction was successfully applied to hypertensive overweight and obese patients 60 years ago by Rose. He concluded that weight reduction through dietetic adjustment substantially lowered blood pressure and relieved symptoms such as shortness of breath, palpitation, edema, albuminuria, and headache.

Subsequently, other authors published reports on successful treatment of hypertension by weight loss in obese patients (fig. 2). Some of the older publications are summarized in table I. It can be seen that a weight loss of about 10 lbs induced a variable decrease of blood pressure. However, not all patients achieved decreased blood pressure with weight reduction. Only 39 of 54 patients responded with a fall of blood pressure after weight reduction. Fifteen patients did not show a blood pressure lowering response, although they lost an average of 23.5 lbs in an 8-month treatment period. There was no obvious difference between the two groups. A long-term evaluation showed that regaining weight was associated with an increase of the blood pressure to preweight-loss levels. Also, Martin showed that one cannot predict whether patients who lose weight will also lower blood pressure. In the
Framingham study\textsuperscript{36} other effects of body weight changes were documented: The strongest effect was seen on serum cholesterol levels, then on blood pressure, uric acid, and blood glucose. For each 10-unit change in relative weight in men there was a change of 11.3 mg/dl cholesterol, 6.6 mm Hg in systolic blood pressure, 2.5 mg/dl in glucose, and 0.33 mg/dl uric acid. For women, the changes were more modest. These results from the Framingham study are not generally applicable to overweight or obese patients, since in the study all patients and not only obese patients were evaluated.

An important intervention study was done by Tyroler et al.\textsuperscript{32} Patients were randomly allocated into either a dietary treatment group or a comparison group. The experiment showed that subjects in a free-living community who are both overweight and hypertensive can be screened and recruited into a weight reduction program. Of 72 subjects randomized into the dietary program, 58 remained in the program, and 62 of 71 in the control group were followed for the same period of time. It became apparent that satisfactory participation, compliance, reduced antihypertensive drug use, and successful, measurable blood pressure reduction were achieved in the obese hypertensives. Similar results were reported by Ramsay et al.\textsuperscript{37} from a blood pressure clinic. Successful weight loss was associated with a substantial improvement of blood pressure control and with less frequent increases in antihypertensive drug treatment. There was a correlation of weight change and a change of systolic and diastolic blood pressure similar to the observations in the Evans County study\textsuperscript{11} and in a recent study by Dershewitz et al.\textsuperscript{38} in obese hypertensive adolescents.

In a recent study of 78 hypertensive obese patients a drop of only 5% of the total body weight induced a marked fall of the blood pressure levels and, in one third of the patients, led to a reduction or withdrawal of the antihypertensive drugs.\textsuperscript{39}

In a Chicago coronary prevention evaluation program 67 men were 15% or more overweight at baseline and were very carefully studied for the first 5 years in the program.\textsuperscript{40} The program consisted of recommendations for improved life-style in regard to eating, exercising, and smoking habits. Results are shown in table 2. There was a considerable decrease of body weight followed by a fall of blood pressure, heart rate, and serum cholesterol levels. Not only men with hypertension but also men with high normal blood pressure decreased blood pressure with weight loss. It could also be shown that achievement of desirable or near-desirable weight is not essential for a sizable, sustained fall in blood pressure, but rather that moderate weight loss (e.g., about 5% to 6% of body weight) influences blood pressure more or less uniformly, largely independently of the degree of initial overweight. The decrease of the pulse rate possibly reflects modest improvement in cardiopulmonary fitness caused by rhythmic (isotonic) exercise. In a recent study, it could also be demonstrated that normal blood pressure was achieved in most overweight hypertensive patients when they had lost only half of their overweight, and were still considerably overweight. Therefore, it did not appear to be necessary to achieve ideal body weight to obtain a meaningful reduction of blood pressure. The blood pressure reduction was not a transient phenomenon, it persisted as long as the decrease in body weight was maintained.\textsuperscript{41,42}

In our own study in Dusseldorf, we evaluated the influence of weight changes over a 4.5-year period in 184 obese hypertensive patients who were not on antihypertensive medication. Patients were allocated to five weight change classes: weight change $-12.00$ kg or more; $-12$ to $-2.1$ kg; $±2$ kg; $+2.01$ to $+12$ kg; $+12.01$ kg or more. It could be demonstrated that blood pressure decrease was greatest in patients who lost more than 12 kg. Still, with an increase of weight after 4.5 years of 2 to 12 kg, there was a slight fall of

\[
\begin{array}{|c|c|c|c|}
\hline
\text{Weight loss (lbs)} & \text{BP decrease (mm Hg)} & \text{Author, ref} \\
\hline
12 & 26 & Terry, 1923\textsuperscript{32} \\
10 & 7.4 & 3.3 & Fletcher, 1954\textsuperscript{35} \\
10 & 7.4 & 3.3 & Adlersberg et al., 1946\textsuperscript{33} \\
10 & 3.5 & Martin, 1953\textsuperscript{34} \\
\hline
\end{array}
\]
Table 2. Mean Changes in the Six Variables of 67 Nondropouts Never Receiving Antihypertensive Medication

<table>
<thead>
<tr>
<th>Year</th>
<th>Δ Weight (lbs)</th>
<th>Δ Relative weight</th>
<th>Δ Pulse rate (beats/min)</th>
<th>Δ Systolic BP (mm Hg)</th>
<th>Δ Diastolic BP (mm Hg)</th>
<th>Δ Serum cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>196.2</td>
<td>130.1</td>
<td>81.2</td>
<td>146.6</td>
<td>95.6</td>
<td>258.3</td>
</tr>
<tr>
<td>1</td>
<td>183.2*</td>
<td>121.4*</td>
<td>77.7*</td>
<td>133.9*</td>
<td>86.2*</td>
<td>226.7*</td>
</tr>
<tr>
<td>2</td>
<td>182.3*</td>
<td>120.7*</td>
<td>77.3*</td>
<td>132.3*</td>
<td>86.8*</td>
<td>232.5*</td>
</tr>
<tr>
<td>3</td>
<td>185.1*</td>
<td>122.7*</td>
<td>77.6*</td>
<td>132.3*</td>
<td>85.8*</td>
<td>235.3*</td>
</tr>
<tr>
<td>4</td>
<td>186.6*</td>
<td>123.0*</td>
<td>76.0*</td>
<td>133.0*</td>
<td>85.4*</td>
<td>233.2*</td>
</tr>
<tr>
<td>5</td>
<td>186.4*</td>
<td>123.6*</td>
<td>75.1*</td>
<td>134.9*</td>
<td>87.0*</td>
<td>233.5*</td>
</tr>
<tr>
<td>1-5</td>
<td>184.5*</td>
<td>122.3*</td>
<td>76.7*</td>
<td>133.3*</td>
<td>85.9*</td>
<td>232.2*</td>
</tr>
<tr>
<td>% Change, 1-5</td>
<td>-6.0</td>
<td>-6.0</td>
<td>-5.5</td>
<td>-9.1</td>
<td>-10.1</td>
<td>-10.1</td>
</tr>
</tbody>
</table>

Last baseline diastolic blood pressure (BP) was 90 mm Hg or greater; relative weight, 115 lbs or greater. Δ indicates change in. (Adapted from Stamler et al., 1980, ref 40.)

*p < 0.01.

Mechanisms Responsible for Decrease in Blood Pressure after Weight Reduction

The mechanism of the blood pressure lowering effect of weight loss has not been clarified. Sodium restriction by restricted food intake has been mentioned as one of the factors possibly operative. However, recent studies have convincingly demonstrated that sodium restriction is not a prerequisite for a fall of the blood pressure during weight loss. Reisin et al. advised 81 obese hypertensive patients to eat fewer calories, with generous use of salty low calorie food. The patients lost about 9.5 kg in weight after more than 2 months on the low calorie diet. This weight reduction was accompanied by a highly significant fall in blood pressure, an average drop of 26 mm Hg systolic and 20 mm Hg diastolic in a group of patients with relatively mild hypertension, who were taking no antihypertensive drug. In another group whose blood pressure was still high, even on antihypertensive therapy, the weight loss resulted in an average drop in pressure of 37 mm Hg systolic and 23 mm Hg diastolic.

In a careful study, Tuck et al. demonstrated that body weight loss by a "modified fast" induced a reduction of blood pressure, plasma renin activity (PRA), and plasma aldosterone levels. These reductions occurred regardless of whether the intake of sodium was 120 or 40 mEq/day (fig. 3). Both hypertensive and normotensive subjects had a comparable decrease in blood pressure, and the former reached normotensive ranges. A 10% to 30% reduction in weight appeared sufficient to accomplish this end. The decline in pressure was not correlated with decrease in PRA and aldosterone. A correlation between blood pressure and PRA decrease was demonstrated after 8 and 12 weeks. Since serum insulin levels declined under these conditions, one may speculate as to whether this may not have been important in the hypotensive effect.

Besides a decrease in insulin, PRA, and aldosterone during weight reduction in obese hypertensives, it was found that in normotensive obese subjects weight loss
induced a significant fall in systolic and diastolic pressures as well as in plasma noradrenaline concentrations.\textsuperscript{45}

**Effect of Weight Reduction on Cardiovascular Risk**

The effect of weight loss on cardiovascular risk is calculated by multiple regression equations in the Framingham study.\textsuperscript{36} For each 10% reduction of weight in men, about a 20% reduction in coronary incidence would be anticipated, and for each 10% increase about a 30% increase in incidence. The effect is slightly less pronounced for women and for older persons. In contrast to the Framingham calculations, a decrease in blood pressure was seen over a 3-year trial in untreated subjects with mild hypertension irrespective of weight changes.\textsuperscript{46} Patients were classified into those with a weight gain of 3 kg or more, those with weight loss of 3 kg or more, and those whose weight remained unchanged within these limits. In all three instances there was a fall in pressure over 3 years but the fall was greatest in those who lost weight and least in those who gained weight. No satisfactory explanation for this pattern is available, but inherent study effects should be taken in account when hypertension studies are evaluated.

It is important to note that the risk of hypertensive patients does not only depend on the blood pressure. There is an enormous variability in the natural history of hypertension that is not predicted by blood pressure alone. The risk varies widely at any blood pressure levels according to HDL and LDL-lipid values and other risk factors, such as glucose intolerance, smoking, and left ventricular hypertrophy (LVH).\textsuperscript{47} Calculations by Madhavan and Alderman\textsuperscript{48} revealed, for instance, that a young man who smokes, has hyperlipidemia, LVH, impaired glucose tolerance, and a systolic blood pressure of 135 mm Hg has 20 times the likelihood of developing cardiovascular disease (CVD) in a 15-year-period as does a woman of the same age and blood pressure free of these risk characteristics. A young man with a systolic pressure of 135 mm Hg together with other risk factors has about 10 times the risk of subsequent premature CVD as does a low-risk young woman with a systolic pressure of 195 mm Hg.

It was shown over 40 years ago that in obese patients with impaired glucose tolerance a substantial weight loss to a normal level was necessary to obtain a normal glucose tolerance.\textsuperscript{49} When normal weight was not achieved, glucose intolerance was not reversible. In a subsequent 10-year study on obesity and glucose tolerance, it was again demonstrated that obese patients who did not achieve normal weight by diet treatment did not achieve a normal glucose tolerance, although they had a considerable weight loss.\textsuperscript{50} Accordingly, the treatment of overweight people should not be terminated early in weight loss when the blood pressure has already fallen. Continuous treatment with the goal of achieving normal weight is required to normalize other important obesity-associated risk factors.\textsuperscript{51}

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