REVIEWS ARTICLE

Overweight and Hypertension

A Review

By Benjamin N. Chiang, M.D., Lawrence V. Perlman, M.D., and Frederick H. Epstein, M.D.

SUMMARY

The interrelationships between hypertension and obesity, two common and major health hazards, are reviewed. Comparisons of simultaneous intra-arterial and cuff blood pressure measurements indicate in general that the association between blood pressure and body weight is real and independent of arm circumference. Hypertension is more common among the obese than among the nonobese and, conversely, a significant proportion of hypertensive persons in the population are overweight. Obese hypertensive subjects experience a greater risk of coronary heart disease than the nonobese, and mortality rates for obese hypertensive persons are higher than for those with obesity alone or hypertension alone. Weight reduction has been shown to lower blood pressure, and it may bring about a more favorable prognosis in obese hypertensive persons. Possible mechanisms that may be responsible for the frequent association between obesity and hypertension have been discussed. Irrespective of the underlying pathophysiologic mechanisms, the adverse metabolic and hemodynamic effects of obesity upon hypertension impose an extra burden and strain on the circulatory system and compromise its functional adequacy. Although it is not precisely known to what extent weight reduction alone may be effective in controlling or preventing the lesser degrees of hypertension, the control of obesity should be an intrinsic part of any therapeutic or preventive antihypertensive regimen.

Additional Indexing Words:
Obesity Blood pressure measurement
Epidemiology Adrenocortical steroids
Hypertension Hemodynamic effects
Weight reduction Prognosis

ELEVATED blood pressure and obesity predispose to coronary heart disease.\(^1\) \(^2\) The control of these two factors is, therefore, an essential part of any coronary heart disease prevention program. It is likely that the detrimental effect of obesity is mediated, at least in part, by the association between blood pressure and weight levels. In fact, the frequent coexistence of these two common conditions suggests that there is a causal relation between them so that weight gain constitutes one kind of environmental stress that brings a genetic predisposition toward hypertension into the open. A parallel situation exists in the case of diabetes, which may sometimes be controlled by weight reduction, implying that, conversely, overeating can precipitate it.

To prevent coronary heart disease, there must be a constant search for environmental factors that may be modified to delay the onset and progression of atherosclerosis and its consequences. The present review attempts to summarize some of the key evidence that links hypertension and obesity and to provide a scientific basis for the belief that weight control is likely to be an important ingredient

---

From the Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, Michigan.

Dr. Chiang is a recipient of an International Postdoctoral Research Fellowship (5 FOJ-TW 1025) and Dr. Epstein is the recipient of a Research Career Award (HE-K8-6748) from the U. S. Public Health Service, Bethesda, Maryland.
of any program for the control of hypertension in the community and in individual patients.

**Prevalence of Overweight and Hypertension**

In the United States about 30% of men and 40% of women over the age of 30 are 20% or more above "desirable weight" as defined in the tables published by the Metropolitan Life Insurance Company.\(^3\) The 1959 Build and Blood Pressure Study of the Society of Actuaries showed that 6% of men (ages 15 to 69) and 11% of women (ages 15 to 69) were 20% or more in excess of "average weight" (for height and age).\(^4\) The difference in the percentage of persons 20% overweight by one of these standards depends obviously on the definition of overweight and the standard used.

Weight is gained more frequently at certain ages or physiological periods. In women, this is most likely after the completion of growth—about age 20, during pregnancy, and after the menopause.\(^5\) Men tend to gain weight between the ages of 25 and 40 years.\(^6\) It has been shown that persons overweight as children, teenagers, or young adults are likely to remain or become more overweight throughout life.\(^1\)

Overweight, as defined above, is the ratio of actual weight to average or desirable weight (specific for age, sex, height, and body build).\(^4\) An individual may be overweight on account of musculature or bony structure rather than excess fat, so that overweight and obesity are not necessarily synonymous. Average weight tables for men and women aged 15 to 65 years are provided in the 1959 Build and Blood Pressure Study of the Society of Actuaries.\(^4\) Desirable weight tables have been constructed by the Metropolitan Life Insurance Company,\(^3\) derived from the 1959 Build and Blood Pressure Study. The National Center for Health Statistics, Public Health Service, has published data on height, weight, and selected body dimensions based on a nationwide adult population sample instead of an insured population.\(^6,\ 7\)

Since the component of overweight that can be reduced is fat, determination of relative fatness is of practical significance. Obesity, defined as excessive body fatness, can be measured in many ways. Some methods are quite complex, such as densitometer or hydrometry studies and determination of whole body potassium content; others are relatively simple, including measurement of subcutaneous skin folds, and soft-tissue x-rays.\(^8\)–\(^16\) The Committee on Nutritional Anthropometry recommended triceps and subscapular skinfolds as methods for characterizing over-all fatness\(^14\) since subcutaneous fat makes up half of the total body fat content.\(^17\) For practical purposes, triceps skinfold measurements alone are considered adequate as being simple, reasonably precise, and reproducible.\(^16\) Beyond 9 years of age, fat accounts for a higher percentage of total body weight in females than in males. Body fatness for both sexes increases with age and, in adult life, increases at a faster rate for men than women; yet, women at all ages are on the average fatter than men. The extent to which overweight measures obesity depends on the correlation between these two measurements, which varies according to body build. The level at which obesity assumes clinical and prognostic significance is not easy to define. Much of this information comes from insured populations and cannot be easily extrapolated to the population at large. It would probably be conservative to estimate the prevalence of significant obesity in the United States, depending on age and sex, as being at least somewhere between 20 and 30%.

The prevalence of hypertension, like the prevalence of obesity or overweight, depends on the level one chooses as the line of division between "normal" and "abnormal." If a systolic blood pressure of 160 mm Hg or greater and/or diastolic blood pressure of 95 mm Hg or greater are chosen,\(^18\) data from the Tecumseh Study indicate that 27% of men and 37% of women aged 40 to 59 are "hypertensive."\(^19\)

**Relationship between Blood Pressure and Body Weight**

In this section, three questions will be discussed: (1) what is the over-all correlation between blood pressure and body weight,
(2) what proportion of hypertensive persons are overweight, and (3) is the correlation between blood pressure and body weight a methodologic artifact?

**Correlations between Blood Pressure and Body Weight**

Clinical and epidemiological investigations of the relationship between body weight and blood pressure from different parts of the world are summarized in table 1. Most population studies tend to show a rise of blood pressure with increase of body weight or adiposity. The correlation tends to be higher in countries where obesity is more common. Epstein reviewed geographic differences in various parts of the world, classified the age-blood pressure trends into groups according to the slope of pressure rise with age and suggested that populations who show the least rise of blood pressure with age are those with lower average weight. Several studies that do not show a constant relationship between body weight and blood pressure have been reported from primitive societies or in social groups where either the range of body weight is narrow or hypertension is rare. Seasonal variation of blood pressure must be taken into account, at least in subarctic climates, where seasonal changes in skinfolds were correlated with variations in blood pressure.

The magnitude of the association between blood pressure and body weight in countries where obesity is very common as in the United States, or common as in Scandinavia, may be illustrated by data from four studies. In the Tecumseh Study in Michigan, the correlation coefficients between systolic pressure and relative weight were 0.29 for those aged 20 to 29; 0.33 for the group aged 30 to 39; 0.23 for those 40 to 49; and 0.21 for those 50 to 59; in the older age groups 60 to 69 and 70 to 79 correlation coefficients were only 0.13 and 0.05 respectively. For diastolic blood pressure correlation coefficients were highest for those aged 30 to 39 (0.32) and lowest for those aged 60 to 69 (0.05); for the other age groups, the values were 0.28 (20 to 29), 0.25 (40 to 49) and 0.26 (50 to 59). In the Framingham study in Massachusetts, the correlation coefficient between systolic blood pressure and relative weight at ages 30 to 59 was 0.3 for both sexes. In an extensive Scandinavian study, the blood pressure difference between those 20% underweight and those 20% overweight averaged 16.9 mm Hg for systolic and 9 mm Hg for diastolic pressure (11,063 men and 3,721 women, age 20 to 60). In another large study by Boe and his colleagues, in Norway, among 67,976 adult men and women, there was an increase in systolic and diastolic pressure of 3 and 2 mm Hg, respectively, for a 10 kg increase in body weight.

The relationship between blood pressure and body weight has been shown to be greater in women, in those with a family history of obesity and hypertension, in the extremely obese, and in those less than 60 years old. Systolic blood pressure shows a greater association with body weight than diastolic pressure. Higher blood pressure was found in nonsmokers than smokers who are less heavy. In other studies, hypertensive patients were heavier than normotensive controls. In addition, relative weight was correlated significantly with the prevalence of hypertensive retinopathy, as well as cardiovascular complication in hypertensive patients. Blood pressure has been shown to correlate with the ponderal index and skin fold thickness. Both Whyte and Kannel and his associates have noted blood pressure to be more strongly and primarily correlated with body weight rather than body fat and concluded that the correlation with body fat is a secondary phenomenon due to the association between weight and degree of fatness.

It is of interest that in at least one population in which hypertension and obesity are both very common, no correlation between the two variables was found; these findings from a study among American Negroes in South Carolina remain to be explained and confirmed by other investigations. In the Evans County (Georgia) study, blood pressure was found to be higher in Negro women, but not in men with higher body weight. Miall and
Table 1

<table>
<thead>
<tr>
<th>Senior author</th>
<th>Study population</th>
<th>Significant findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larimore 1923*</td>
<td>417 factory workers; B.P. and body build</td>
<td>The asthenic had the lowest blood pressure; the sthenic had the highest blood pressure</td>
</tr>
<tr>
<td>Marks 1924 (44)†</td>
<td>Metropolitan Life Insur.; men age 28–42 years, U.S.A.</td>
<td>B.P. 7/6 mm Hg higher in persons 25% overweight; overweight had higher blood pressure</td>
</tr>
<tr>
<td>Huber 1927 (46)</td>
<td>1,332 healthy military personnel, U.S.A.</td>
<td>2/3 of persons over age 40 with 10 lbs. overweight or more had elevated blood pressure; 49% of those 10% underweight or more with low systolic pressure of 110 mm Hg or less; 18% of those overweight with high systolic blood pressure over 140 mm Hg</td>
</tr>
<tr>
<td>Hartman 1929 (39)</td>
<td>2,042 persons age over 15 years, males, 659; females, 1,083; Mayo Clinic</td>
<td>Systolic B.P. increases with increase of weight in both sexes; diastolic B.P. increases with increase of weight in females only</td>
</tr>
<tr>
<td>Robinson 1939 (33)</td>
<td>10,883 healthy persons; 7,478 males, 3,405 females, U.S.A.</td>
<td>Obese females and males have 6 and 1 1/2 times more hypertension respectively; B.P. increases with overweight, and low in underweight in all age groups of both sexes</td>
</tr>
<tr>
<td>Short 1939 (47)</td>
<td>3,516 healthy persons applying for periodic health examination; U.S.A.</td>
<td>More hypertension among overweight persons; B.P. difference of 11/4 mm Hg between overweight and underweight in the 50–59 age group</td>
</tr>
<tr>
<td>Boynton 1948 (35)</td>
<td>75,258 University students (U. of Minnesota), 43,800 males, 31,258 females</td>
<td>Mean systolic and diastolic B.P. rises as weight increases in each age group; more marked in systolic B.P.; systolic B.P. higher in persons with positive family history</td>
</tr>
<tr>
<td>Green 1948 (48)</td>
<td>1,260 obese patients age 11–70, U.S.A.</td>
<td>Hypertension more frequently noted in mild to moderate overweight; age distribution of obese hypertension did not differ from essential hypertension in general</td>
</tr>
<tr>
<td>Master 1953 (49)</td>
<td>1,000 private patients age 20–64; 169 with hypertension</td>
<td>In males, more hypertension in overweight than the general population, ratio: 32 : 14; in females, no difference in hypertension</td>
</tr>
<tr>
<td>Thomas 1955 (36)</td>
<td>266 Johns Hopkins Medical students; study of family relation of overweight and hypertension</td>
<td>Family study showed high correlation between hypertension and obesity among parents, grandparents, uncles, and aunts</td>
</tr>
<tr>
<td>Bjerkedal 1957 (28)</td>
<td>14,784 adult population, 11,063 males, 3,721 females, Norway</td>
<td>Small increase of mean systolic and diastolic B.P. with increase of relative weight in all age groups; no excessive accumulation of hypertension in overweight persons</td>
</tr>
<tr>
<td>Boe 1957 (29)</td>
<td>67,976 adult population, 27,718 males, 40,258 females; Bergen, Norway</td>
<td>Overweight has negligible effect on hypertension; 3/2 mm Hg increase in systolic-diastolic pressure for each 10 kg increase of body weight</td>
</tr>
</tbody>
</table>
Table 1 (continued)

<table>
<thead>
<tr>
<th>Senior author</th>
<th>Study population</th>
<th>Significant findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Padmavati 1959</td>
<td>1,132 low socioeconomic; Delhi, India</td>
<td>In low S-E class, B.P. rise with body weight, but not with age; in high S-E class, B.P. rises with both weight and age; systolic pressure rises 0.2–0.3 mm Hg per pound weight increase, diastolic 0.2 mm Hg per pound weight increase in both groups</td>
</tr>
<tr>
<td>Build &amp; B.P. Study 1959</td>
<td>Metropolitan Life Insurance Study 4,900,000 policy holders, U.S.A.</td>
<td>Excessive mortality of overweight persons esp. in younger age group, male sex and hypertensive; mortality mainly due to cardiovascular causes</td>
</tr>
<tr>
<td>Nutrition Survey of Armed Forces 1960</td>
<td>1,333 military personnel age 20–49, Philippines</td>
<td>Both systolic and diastolic pressures increase with each increment of relative weight</td>
</tr>
<tr>
<td>Stamler 1961</td>
<td>Peoples Gas Co. employees, 1,329 men age 40–49; Chicago, Ill., U.S.A.</td>
<td>Twofold increase in hypertension in overweight persons than in underweight persons</td>
</tr>
<tr>
<td>Epstein 1965</td>
<td>8,641 adult population, age 20–79; Tecumseh, Michigan, U.S.A.</td>
<td>Significant correlation between B.P. and relative weight and skin folds; hypertensive heart disease significantly correlated with relative weight in females only</td>
</tr>
<tr>
<td>Doyle 1961</td>
<td>Waterside worker, Australia</td>
<td>B.P. rises with skinfolds in younger age, up to 40 years; not in older people over 60 years</td>
</tr>
<tr>
<td>Hunter 1962</td>
<td>Polynesians, Cook Island</td>
<td>Significant correlation between obesity and hypertension and hypercholesteremia but coronary heart disease is rare</td>
</tr>
<tr>
<td>Truedsson 1962</td>
<td>Healthy individuals, Sweden</td>
<td>Skin folds correlate with blood pressure in females, but not in males</td>
</tr>
<tr>
<td>Palmai 1962</td>
<td>Subarctic area study of seasonal variation of B.P.</td>
<td>Seasonal changes of B.P. correlation with changes of subcutaneous skinfolds</td>
</tr>
<tr>
<td>Whyte 1959, 1965</td>
<td>100 healthy men 20–24 years, Australia</td>
<td>B.P. correlates significantly with bulk in young Australians, no correlation in older Australians. In New Guinea, B.P. does not rise with age, not related to relative weight; people are lean.</td>
</tr>
<tr>
<td>Reid 1966</td>
<td>676 van drivers of General Postoffice age 40–59, England</td>
<td>B.P. of nonsmokers higher than smokers; the body weight is also higher in the nonsmokers</td>
</tr>
<tr>
<td>Shaper 1967</td>
<td>Three east African tribes, Northern Kenya</td>
<td>B.P. no rise with increase of age or weight in two tribes, the third tribe showed B.P. increase with weight, also increased prevalence of hypertension; the last tribe showed increased ponderal index and consumed more carbohydrates</td>
</tr>
</tbody>
</table>
Table 1 (continued)

Relation of Body Weight and Blood Pressure

<table>
<thead>
<tr>
<th>Senior author</th>
<th>Study population</th>
<th>Significant findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boyle 1967</td>
<td>2,184 persons,</td>
<td>B.P. rises with increase of weight in white people, but no significant difference in weight/B.P. relation in Negro population who have more hypertension</td>
</tr>
<tr>
<td></td>
<td>Charleston Heart Study, U.S.A.</td>
<td></td>
</tr>
<tr>
<td>McDonough 1967</td>
<td>3,102 subjects age 15–74, Evans County, Ga., U.S.A.</td>
<td>More hypertension in Negro population than white. In the Evans County (Georgia) study blood pressure was found to be higher in Negro females, but not males with higher body weight</td>
</tr>
<tr>
<td>Chiang 1967</td>
<td>Taiwan Cardiovascular Study, 1,822 men age 40–59, Taiwan</td>
<td>Hypertensive subjects average 5 kg heavier than age-matched normotensive controls; the hypertensives have significantly thicker skin-folds</td>
</tr>
<tr>
<td>Tibblin 1967</td>
<td>Hypertension, 855 men, age 60, Goteborg, Sweden</td>
<td>B.P. correlated with obesity; hypertensive retinopathy and LVH more in the obese group (3-fold increase)</td>
</tr>
<tr>
<td>Keys 1967</td>
<td>Cooperative study in 7 countries</td>
<td>B.P. significantly correlates with skin folds except Serbian village of Velike Krnja, where there was no correlation; the men are underweight and thin</td>
</tr>
<tr>
<td>Kannel 1967</td>
<td>The Framingham study, 5,127 men and women age 30–62, U.S.A.</td>
<td>More hypertensives in the overweight men and women; B.P. correlates with relative weight throughout all age groups (30–62)</td>
</tr>
<tr>
<td>Aleksandrow 1967</td>
<td>10% of adult population, Poland</td>
<td>B.P. rises with weight increase; more hypertension in obese group</td>
</tr>
</tbody>
</table>

* Date of publication.
† Reference numbers are in parentheses.

his associates32 have made a careful analysis of the relation between blood pressure and body weight, based on a large population sample in South Wales; a correlation was confined to younger persons, in accordance with a similar tendency already noted above in the Tecumseh population.26 Their statement32 that the cause and effect relationship between the two parameters needs further investigation is well justified. In the meantime, summarizing the data presented in this section, the evidence is overwhelmingly in favor of the view that blood pressure and body weight are positively correlated.

Frequency of Overweight among Hypertensive Persons

According to most reports, overweight occurs more frequently in hypertensive than normotensive subjects.21–26, 29–36, 39, 42, 44–55 In the Framingham study, the over-all prevalence of "obesity," defined as a "Framingham Relative Weight" of 120 or greater (that is, 20% above the height and sex-specific median), was about 9% among men aged 30 to 59. From the data presented, it may be calculated that about 6% of the men were hypertensive, according to the definition used. Among these men, the prevalence of obesity was between 13 and 19%, depending on age, whereas the prevalence of obesity among normotensive controls (disregarding borderline hypertensive subjects in the population) varied between 2 and 4% in this age range. A similar trend has been demonstrated in the Tecumseh Community Health Study.26 In the latter study, 33% of the participants (both sexes, age 40 to 59) in the upper quintile for blood pressure were in the upper quintile for relative weight.26, 43 Overweight women generally have more hypertension than overweight men. Robinson and associates,33 in a study of 10,833 persons, showed that obese women have hypertension 1½ times more often than obese men.
OVERWEIGHT AND HYPERTENSION

The frequency of overweight among hypertensive persons will obviously vary according to the definitions used for these two variables. Using definitions that, from the public health point of view, are neither too liberal nor too conservative, one may estimate that at least between one fifth and one third of all hypertensive subjects in the adult population are overweight. Since such persons have an increased mortality from cardiovascular and cerebrovascular diseases, this fact has preventative implications as far as blood pressure lowering through weight reduction is concerned, as will be discussed later.

Comparison of Direct Intra-Arterial and Indirect Auscultatory Methods for Blood Pressure Measurement

Most observers agree that blood pressure increases with body weight or skin folds. The problem is whether the increase is true or an artifact resulting from the influence of large arm girth, thick subcutaneous fat, or both.

Ragan and Bordley compared 51 persons by simultaneous measurement of intra-arterial blood pressure in one arm and the indirect auscultatory method in the other; 13 of the 51 cases had aortic insufficiency; it was found that indirect blood pressure tended to be lower than the direct readings for subjects with thin arms and higher for subjects with thick arms. Pickering and his colleagues used the same data, excluding the 13 cases with aortic insufficiency, to draw up a regression equation and a table for correction of the indirect sphygmonanometer readings. Miall and Oldham used this equation for correcting cuff readings by arm circumference in an epidemiological study but also showed a high correlation between arm circumference and weight (r = 0.99). In 1952, Bjerkedal studied the blood pressure of 14,784 adults in Norway. He found a small increase of blood pressure with body weight and an excess of hypertension in obese persons, concluding that this effect was due to the influence of arm girth as postulated by Ragan and Bordley, and Pickering and associates. However, careful review of his paper indicates that there are more individuals with substantially increased blood pressure than could be expected from the small artifact attributed to increased arm girth.

Recent studies by Raftery and Ward and Holland and Humerfelt, comparing the direct intra-arterial and indirect auscultatory blood pressures failed to confirm the observations of Ragan and Bordley; the difference between intra-arterial and cuff blood pressure, on the one hand, and arm circumference or skinfold thickness, on the other, were not correlated but a significant correlation between arm circumference and the direct arterial pressure was found. Berliner and co-workers studied simultaneous intra-arterial and indirect cuff blood pressure of 100 subjects, 61 were obese and 39 nonobese. While the indirect method generally, but not consistently, underestimated systolic and overestimated diastolic pressure, among lean and moderately obese persons the discrepancies were usually of a minor degree; even in the extremely obese persons, large arm girth did not regularly cause falsely high readings so that a correction for arm circumferences could not be applied routinely. Similar studies by Karlefors and his associates and Nagle showed good agreement between indirect and direct blood pressures at rest and during exercise. Kannel and associates, in the Framingham study, compared upper arm and forearm pressures in the same persons and found similar readings whether or not their arm girths were large. Alexander studied a group of grossly obese patients with average weight over 300 pounds with simultaneous direct intra-arterial and indirect cuff pressure measurement; the blood pressure agreed to within 20 mm Hg systolic and 10 mm Hg diastolic on half of the occasions, and the cuff method gave false low readings as frequently as high readings; most of the patients were frankly hypertensive on the basis of intra-arterial readings. Reid and co-workers, in a survey of British postal workers, found no significant partial correlation between blood pressure and arm circumferences. Khosla and Low observed from a regression analysis of systolic and diastolic pressure, age, weight, and arm circumference...
in a population sample of employed men aged 45 to 69 years that the relationship between arm circumference and sphygmomanometer reading is indirect and due to the high correlation of arm circumference with body weight, which, in turn, is related to blood pressure. Lowe et al. analyzed indirect blood pressure readings in a population of over 5,000 employed men, aged 15 to 69 years; he found that, for a given age, sphygmomanometer readings increase with body weight and that weight and arm circumference are highly correlated. When the blood pressure is corrected for weight, the effect of arm circumference upon blood pressure is negligible. Karvonen and later King have suggested that discrepancies between direct and indirect readings could be minimized by using a wide cuff in obese individuals.

Tibblin, in a cohort study of hypertension in men, found body weight and skinfolds not only related to the level of blood pressure, but also to the severity of hypertensive retinopathy. The prevalence of subjects with hypertensive eye-ground changes increased about three times, with an increase in subscapular skinfolds and body weight; the fundus changes were independent of arm circumference. Stamler also found body weight to be correlated not only with blood pressure, but also with hypertensive heart disease, as demonstrated by electrocardiographic or x-ray evidence of cardiomegaly.

In summary, sources of variation in measuring blood pressure by the indirect sphygmomanometer method, as pointed out by Rose and others, are multiple, arm circumference being only one source of possible variation in the recording of blood pressure. Recent studies on the effect of arm circumference on pressure readings appear to have shown that the discrepancy is minor in the majority of cases, and the difference between direct intra-arterial and indirect cuff readings varies in either direction in obese as well as nonobese subjects (table 2). The relationship between arm girth and sphygmomanometer pressure readings is likely to be an indirect one due to high correlation between body weight and arm circumference, so that the cuff blood pressure reading should not be corrected for arm circumference. Correcting blood pressure by arm circumference will obscure the important influence of body weight on blood pressure in epidemiological studies. Furthermore, it is concluded that the relationship between body weight and blood pressure is not a methodological artifact, due to falsely high blood pressure readings in heavier persons with fatter arms.

Overweight as a Risk of Developing Hypertension and Hypertensive Heart Disease: Longitudinal Studies

Few longitudinal studies on the relation of overweight and the risk of subsequent hypertension have been published. Levy and his colleagues did a retrospective study of the medical records of 22,741 officers in the United States Army. An officer was considered to be overweight if he was heavier by 20 pounds or more than the standard given in Army regulations. Two and a half as many men developed sustained hypertension in the overweight as compared to normal controls after the age of 45 in a total experience of 33,921 persons-years. When transient tachycardia and transient hypertension were added to overweight, the ratio of subsequent development of sustained hypertension was 12.3 fold. The ratios of retirement and death from cardiovascular-renal diseases for the same group were 4.1 and 2.0 times higher, respectively, than for the normal controls.

Stamler analyzed data on 746 men in the labor force of a Chicago utility company over a period of 20 or more years. A strong relationship between the incidence of hypertensive disease in middle age and the rate of increase of body weight in a 20-year period was shown. Men at desirable weight as young adults and exhibiting little or no weight gain over the ensuing decades had the lowest incidence rate of hypertensive disease, whereas men above desirable weight as young adults and with significant weight gains over the next 20 years had a 4 to 5 fold higher incidence rate of hypertensive disease. In another group of 594 employees of the same company
### Table 2

#### Comparisons Between Direct and Indirect Blood Pressure Measurements

<table>
<thead>
<tr>
<th>Names of authors</th>
<th>Patients studied</th>
<th>Direct Methods</th>
<th>Indirect Methods</th>
<th>Differences between direct and indirect blood pressure</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holland Humerfelt (60)</td>
<td>47 patients and staff (not obese)</td>
<td>Hansen capacitance manometer left arm</td>
<td>London School of Hygiene; <strong>sphygmomanometer three</strong> 12 cm × 24 cm cuff</td>
<td>Direct blood pressure higher than indirect</td>
<td>Direct blood pressure correlated with arm circumference</td>
</tr>
<tr>
<td>Ragan Bordley (56)</td>
<td>51 patients (13 with aortic insufficiency)</td>
<td>Optical manometer</td>
<td>13 cm cuff and mercury manometer</td>
<td>Direct higher in individuals with thin arms, lower in individuals with fat arms. When grossly obese and aortic insufficiency patients excluded 83% agreement</td>
<td>Blood pressure over-estimated in obese and under estimated in non-obese</td>
</tr>
<tr>
<td>Berliner Fujiy Lee Yildiz Garnet (61)</td>
<td>100 patients, 61 obese</td>
<td>Sanborn Electromanometer</td>
<td>Baumanometer cuff measured 13 × 20 cm tapered sleeve</td>
<td>Direct systolic is generally higher; in obese, however, cuff pressure is higher; indirect diastolic is always higher but differences are of small magnitude except in extremely obese</td>
<td>Cuff pressure underestimates systolic and overestimates diastolic</td>
</tr>
<tr>
<td>King (67)</td>
<td>Number not mentioned; young healthy men</td>
<td>Statham P23 Db strain-gauge on Electronics for Medicine DR8 recorder</td>
<td>Specially made and molded rubber bladders compare with direct; cuffs 26 cm or 42 cm long, 13 cm wide</td>
<td>Direct generally higher systolic, lower diastolic; considerable difference with shorter cuff and less significant difference with longer cuff</td>
<td>A cuff at least 42 cm long is recommended for measurement of B.P. in an adult</td>
</tr>
<tr>
<td>Alexander and Dennis (37) (38)</td>
<td>35 extremely obese persons</td>
<td>Cournand needle</td>
<td>Not mentioned</td>
<td>Direct systolic generally higher, diastolic generally lower, relationship variable</td>
<td>A high reading by cuff usually indicates hypertension</td>
</tr>
<tr>
<td>Raftery and Ward (59)</td>
<td>50 young healthy females</td>
<td>Inductance manometer and ultraviolet recorder</td>
<td>Cuff size 14 × 17.5 cm London School of Hygiene Sphygmomanometer</td>
<td>The indirect-direct differences were not related to the level of blood pressure, arm skin thickness or arm circumference. Good agreement between phase I indirect systolic pressure and direct systolic pressure. Phase V indirect pressure rather than phase IV agreed better with direct diastolic pressure</td>
<td>Indirect method under-records more at high level of systolic pressure than at normal or low level</td>
</tr>
</tbody>
</table>
Table 3

*Effect of Weight Reduction on Blood Pressure*

<table>
<thead>
<tr>
<th>Senior author</th>
<th>Study group</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benedict 1918* (86)</td>
<td>25 normal men studied; on low calorie diet until 10-12% weight loss, then maintained reduced weight</td>
<td>B.P. drop observed in 11 men. Average B.P. drop from 120/83 to 102/69 mm Hg. 3 borderline hypertensions: B.P. before weight loss 142/90, 130/90, 120/90; after weight loss B.P. on 22nd day: 100/65, 100/70, 90/70 mm Hg</td>
</tr>
<tr>
<td>Preble 1923 (87)</td>
<td>1) 194 obese patients, wt. loss at least 10 lbs. 2) 1,000 cases, with 62 hypertensions, B.P. over 200 mm Hg</td>
<td>There was uniform B.P. drop with wt. loss; Mean B.P. drop 154/96 to 133/86 mm Hg; after weight loss, mean B.P. drop from 219/129 to 170/108 mm Hg</td>
</tr>
<tr>
<td>Terry 1923 (88)</td>
<td>63 obese patients, mean age 45, mean wt. 199 lbs. 58% with hypertension, mean B.P. 196/103 mm Hg; on 1,200 calorie diet for 1 yr.</td>
<td>One year after weight reduction, mean B.P. drop from 196/103 to 170/95 mm Hg</td>
</tr>
<tr>
<td>Bauman 1928 (89)</td>
<td>183 obese patients, on low-calorie and low-salt diet, observed for 9.8 months</td>
<td>48 out of 101 with significant systolic B.P. reduction; 72 out of 161 with significant diastolic B.P. reduction</td>
</tr>
<tr>
<td>Master 1929 (78)</td>
<td>91 females and 8 males from obesity clinic, 10-58 years of age, wt. 170-225 lbs; 67% with systolic pressure over 150 mm Hg</td>
<td>Wt. reduction associated with B.P. drop, (more marked with systolic pressure), also with slowing of heart rate. In 53 patients, wt. loss 25-30 lbs., B.P. drop from 20-30 systolic and 15-20 diastolic. Regain of wt. followed rise of B.P. Wt. loss also associated with improved exercise tolerance</td>
</tr>
<tr>
<td>Fellows 1931 (90)</td>
<td>294 obese Metropolitan Life Insurance Co. employees, on 1,200 calorie diet; in 18 cases thyroid was added</td>
<td>Weight loss associated with significant B.P. drop; 30 hypertensives showed favorable course 5 years after weight reduction. 75% of 294 with initial weight loss. 5 years later, 193 re-examined, only 21% maintained low weight, 79% regained weight in whole or part</td>
</tr>
<tr>
<td>Wood 1939 (74)</td>
<td>Animal experiment 8 dogs: 4 normal, 4 with experimental hypertension</td>
<td>Marked rise of systolic blood pressure when caused to gain large amount of wt. by feeding a diet composed of beef fat in both normal and hypertensive dogs. B.P. drop with diet restriction and loss of wt. Less change in diastolic pressure</td>
</tr>
<tr>
<td>Evans 1952 (82)</td>
<td>100 consecutive obese patients, 61 are hypertensive (systolic B.P. 140-180 mm Hg)</td>
<td>75% of the hypertensives showed a fall of B.P. to normal of less than 135 mm Hg with weight reduction. Weight reduction has no effect on obese individuals with severe hypertension</td>
</tr>
<tr>
<td>Fletcher 1954 (81)</td>
<td>38 obese hypertensive females (20% overweight or more), B.P. over 150 systolic and/or 100 diastolic mm Hg. Initial diet 600 calories/day, later to 1,000 calories/day. Average</td>
<td>For 38 obese hypertensives with systolic over 150 mm Hg, mean weight loss was 32 pounds over 6.4 months, mean systolic pressure drop was 32 mm Hg. For 30 obese females out of the 38 with diastolic over 100 mm Hg as well, mean wt. loss over 6.5 months was 33 lbs., and</td>
</tr>
</tbody>
</table>
### Table 3 (continued)

#### Effect of Weight Reduction on Blood Pressure

<table>
<thead>
<tr>
<th>Senior author</th>
<th>Study group</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dahl 1958</strong></td>
<td>age 54.9 years, 21 obese hypertensive females who failed to lose weight and 20 obese normotensive females were used for control</td>
<td>diastolic pressure drop was 16.5 mm Hg. No significant B.P. changes observed in the control group of 21 obese hypertensives and 20 obese normotensive females. A group of obese normotensive females who lost weight also showed B.P. drop</td>
</tr>
<tr>
<td><strong>Salzano 1958</strong></td>
<td>12 obese hypertensive patients 20-100% overweight having hypertension for 2-14 years. Low-salt diet with 85-105 mg Na/daily, and low calorie 600-800 calorie/daily. The low-salt diet and low-calorie diet were alternated to make a single change at a time, calorie or salt</td>
<td>Group 1: Low-calorie, no salt restriction only one out of four with B.P. reduction</td>
</tr>
<tr>
<td><strong>Olson 1959</strong></td>
<td>16 normotensive obese persons, 12 females, 4 male with 14-85% overweight. <strong>Weight reduction 2 lbs/wk</strong></td>
<td>Group 2: Low-salt, no calorie restriction all four patients showed a significant B.P. drop more than usual experience. Obese hypertensive seems more sensitive to salt restriction</td>
</tr>
<tr>
<td><strong>Adlersberg 1946</strong></td>
<td>54 obese hypertensive patients on 1,200 calorie diet, no drug or dehydration, seen 1940-1941, 15 patients re-examined 1944</td>
<td>Group 3: Low-calorie diet at first and low-salt diet added later: Only one out of four with B.P. reduction with low calorie diet alone at first; when low salt diet was added, all four showed significant reduction of blood pressure</td>
</tr>
<tr>
<td><strong>Keys 1947</strong></td>
<td>34 healthy men on 1,600 calorie diet for 6 months, (control wt. 69.4 kg)</td>
<td>Average systolic and diastolic pressure reductions were 12/8 mm Hg respectively; 81% showed significant systolic pressure reduction, 65% showed significant diastolic pressure reduction</td>
</tr>
<tr>
<td><strong>Green 1948</strong></td>
<td>1,260 obese patients. 149 (11.8% hypertensive) age 30-60</td>
<td>No reduction of blood pressure in normotensive group with a weight loss of 14 lbs. over 4-8 months period; 75% of hypertensive group showed significant reduction of blood pressure after weight reduction</td>
</tr>
<tr>
<td><strong>Brozek 1948</strong></td>
<td>Observations on European countries: Russia (Leningrad), Holland and Germany during World War II with drastic food shortage</td>
<td>Average weight loss was 23½ lbs.; 72% with significant B.P. drop; 28% with no change of B.P. Those who maintained low weight had favorable prognosis. Wt. reduction had no effect on retinopathy</td>
</tr>
<tr>
<td></td>
<td>Mean B.P. drop 106/69 to 97/64 mm Hg. Mean pulse rate 56 to 37 per minute <strong>Mean VO₂ reduction 228 to 145 ml/min</strong></td>
<td>Heart size also reduced</td>
</tr>
<tr>
<td></td>
<td>Weight reduction brought ½ of the obese hypertensives to normal B.P. Influence of weight reduction on B.P. is inconsistent</td>
<td></td>
</tr>
</tbody>
</table>

*Circulation, Volume XXXIX, March 1969*
<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Diet/Findings</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kempner</td>
<td>1949</td>
<td>High carbohydrate diet, low salt and high potassium</td>
<td></td>
</tr>
<tr>
<td>Wilhelmj</td>
<td>1951</td>
<td>Animal experiment. 4 normal dogs with 13 fasting periods. Mean wt. loss 4 kg</td>
<td></td>
</tr>
<tr>
<td>Martin</td>
<td>1952</td>
<td>37 middle-aged obese patients wt. 212–218 lbs; 18 hypertensive, 19 normotensive; Average wt. loss 42 lbs., for normotensive, and 36 lbs. for hypertensives</td>
<td></td>
</tr>
</tbody>
</table>

Reduction of blood pressure with this diet, probably due to low salt content as well as weight reduction.

1. B.P. change during fasting period:
   - dog 1: 127/62 to 86/35 mm Hg
   - dog 2: 112/66 to 86/51 mm Hg
   - dog 3: 102/60 to 74/45 mm Hg
   - dog 4: 111/43 to 82/37 mm Hg
2. Associated with slowing of heart rate
3. The fall of B.P. to stable state is influenced by preceding nutritional state, longer to achieve stable B.P. if previous diet is luxurious, shorter if previous diet is poor

13 of 18 normotensives showed no change of B.P., 4 with systolic and/or diastolic pressure drop, one with B.P. rise; 12 of 19 hypertensives showed no change of B.P., 2 with both systolic and diastolic pressure drop, 5 with systolic fall alone

* Date of publication

followed up from 1954 to 1957, the incidence rate of hypertension and hypertensive cardiovascular disease was twice as high in the overweight group. Kannel and his associates reported on the development of hypertension in the Framingham study. Of 5,127 men and women aged 30 to 62 years at the initial examination and followed for more than 12 years, subsequent development of hypertension was related not only to gross obesity but to relative weight, as determined on the initial examination. Thus, the risk of developing hypertension in the group 20% overweight was eight times greater than in the group 10% underweight, and the risk of developing hypertensive cardiovascular disease was about 10 times greater in the group 20% overweight as compared to all others. Men at desirable weight as young adults who reported little or no weight gain over a 20-year period had one fifth the incidence of hypertension (\( \geq 150/90 \)) as compared to those who had a significant gain of weight in the same period. In the Framingham study, after a 12 year follow-up period, the incidence of hypertension (\( \geq 160/95 \)) was also shown to be significantly higher in persons who gained weight after their twenty fifth birthday. While 8% of those who had gained from 4 to 15 pounds were hypertensive, almost 20% of the group gaining more than 29 pounds were hypertensive. The 12-year incidence of hypertension could be related to both obesity and to initial relative weight. It was also noted that the initially obese but otherwise normal subjects developed diabetes, cerebrovascular disease, and coronary heart disease more often.

In summary, with few exceptions, increase of relative weight over time is associated with a rise of blood pressure in population studies. Obese hypertensive patients experience a greater risk of coronary heart disease, cerebrovascular disease and mortality than persons with either hypertension or obesity alone. The risk of subsequent development of sustained hypertension in young adults with obesity or those becoming obese in the ensuing decades is greater than among young adults with normal or less than normal weight who remain lean.

**Effect of Weight Reduction on Blood Pressure**

Experimental and clinical observations on the effect of weight reduction in 19 studies are summarized in chronological sequence in table 3. Animal experiments in normal and
hypertensive dogs have shown that weight reduction by fasting is followed by a fall in both systolic and diastolic pressure and slowing of heart rate.\textsuperscript{74} When the animals regained weight by feeding a rich diet, the blood pressure and heart rate returned to previous levels. The preceding nutritional state of the animal is related to the degree of blood pressure fluctuation during weight reduction and duration of achieving stable low pressure level.\textsuperscript{74, 75} The animals’ systolic blood pressure is affected more markedly than diastolic pressure.

During a period of drastic food shortage in World War II, it was observed that the frequency of hypertension and hypertensive cardiovascular complications was markedly reduced in Russia, Holland, and Germany during the war and reversed thereafter.\textsuperscript{76} In one human experiment, 34 healthy men were on a low-calorie diet for 6 months and lost about a quarter of their control body weight; there was marked reduction of blood pressure, heart rate, oxygen consumption, and decrease of cardiac size with reversal when normal weight was regained.\textsuperscript{77}

Clinical studies on obese hypertensive patients have demonstrated a general but variable and inconsistent fall of blood pressure with weight reduction.\textsuperscript{78, 91} It is more consistent for systolic blood pressure,\textsuperscript{78–80} in obese woman,\textsuperscript{81} with mild to moderate hypertension,\textsuperscript{82} substantial and prolonged weight loss,\textsuperscript{82} and in combination with salt restriction.\textsuperscript{84} Prolonged weight reduction in some obese hypertensive patients was said to have a favorable effect on the course of the disease.\textsuperscript{83} In one study, 38 hypertensive women who lost 32.4 pounds on the average showed an average blood pressure fall of 32.8 mm Hg systolic and 16.5 mm Hg diastolic; a control group without any change of weight had no decrease in blood pressure.\textsuperscript{8} The effect of caloric restriction on obese hypertensive patients is enhanced by low-salt intake.\textsuperscript{84, 85} Dahl and co-workers\textsuperscript{84} found obese hypertensive patients more sensitive to salt restriction than non-obese hypertensives; they concluded that most low-calorie diets also contain less sodium chloride, and the pressure-lowering effect is mainly due to salt restriction.

In summary, in spite of the fact that the effect of weight reduction varies and difficulty is encountered in keeping patients on diet, weight reduction lowers blood pressure in a considerable proportion of obese hypertensive patients. It is desirable to prescribe weight reduction in combination with salt restriction as part of a therapeutic regimen for obese hypertensive patients.

**Overweight and Hypertension: Common Mechanisms and Pathophysiological Changes**

The mechanism of the frequent coexistence of overweight and hypertension is unknown. Possible mechanisms and influencing factors are summarized in Table 4. Alexander and associates,\textsuperscript{37, 38} studying a series of 40 extremely obese persons with average body weight of 300 pounds, showed that the hypertensive individuals in this series had increased cardiac output and normal peripheral resistance. They postulated that an increased stroke volume is required to pump blood through an expanded vascular bed in the obese individual. Blood volume, oxygen consumption, and the transverse diameter of the heart

<table>
<thead>
<tr>
<th>Table 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Overweight and Hypertension: Summary of Possible Mechanisms and Influencing Factors</strong></td>
</tr>
<tr>
<td><strong>I. Hemodynamic:</strong></td>
</tr>
<tr>
<td>1) Increased $V_{O_2}$ and A-V $O_2$ difference, more so during exercise\textsuperscript{38, 92, 93}</td>
</tr>
<tr>
<td>2) Increased cardiac output with normal peripheral resistance \textsuperscript{31, 32, 38}</td>
</tr>
<tr>
<td>3) Increased blood volume and plasma volume \textsuperscript{38}</td>
</tr>
<tr>
<td><strong>II. Hormonal:</strong></td>
</tr>
<tr>
<td>1) Increased adrenocortical function \textsuperscript{37, 98}</td>
</tr>
<tr>
<td>2) Imbalance of renin-angiotensin-aldosterone system \textsuperscript{96}</td>
</tr>
<tr>
<td><strong>III. Environmental:</strong></td>
</tr>
<tr>
<td>1) Physical inactivity leading to reduced energy expenditure and overweight \textsuperscript{41, 94}</td>
</tr>
<tr>
<td>2) Overeating and high-salt intake \textsuperscript{84}</td>
</tr>
<tr>
<td><strong>IV. Genetic:</strong></td>
</tr>
<tr>
<td>1) Endomorph body type \textsuperscript{38, 29, 33, 43, 44, 65}</td>
</tr>
<tr>
<td>2) Family history of obesity and hypertension \textsuperscript{35, 36}</td>
</tr>
</tbody>
</table>
also were noted to increase with body weight. Proger and Dennig found increased oxygen consumption in four moderately obese patients, but there was no significant difference in cardiac output and A-V oxygen difference as compared to the controls. Taylor and his colleagues, in a study of healthy adults, found basal cardiac output related to body weight (r = 0.54); and A-V oxygen difference positively correlated to body fat (r = 0.63). Whyte suggested that high blood pressure in the obese is a result of increased cardiac output, which is forced into a reservoir (comprising the aorta and large arteries), which does not increase with body weight. In individuals with diseased inelastic aortae, the effect of increased cardiac output on blood pressure would be disproportionately greater. Fletcher suggested that the higher blood pressure in obese persons may be a physiological response to the greater metabolic burden and mechanical handicap imposed by obesity. These suggestions indicate that the mechanism of the hypertension related to obesity may be different from essential hypertension in which cardiac output is normal and peripheral resistance increased.

Morris and Crawford, in a national necropsy survey of 5,000 cases, noted that hypertensive heart disease was present in light workers five times more frequently than in heavy workers; obese individuals tend to be more inactive physically. Tibblin has suggested that hypertension and obesity may be the result of a third set of antecedent events, such as physical activity. Hartroft, in a comparative autopsy series, found renal weight in obese hypertensive persons to be normal, but significantly reduced in the whole series of hypertensive individuals. Obese persons consume not only food above energy requirement but may also ingest more salt. According to Dahl and associates, blood pressure levels of obese persons are especially sensitive to dietary salt restriction. Conn suggested that the common triad of obesity, hypertension, and impairment of carbohydrate tolerance in middle aged men may be related to a form of primary aldosteronism that activates the renin-angiotensin-aldosterone system.

Mild degrees of hypoxia and increased A-V oxygen difference associated with obesity lead to increased red-cell mass and increased blood volume. Another common finding in obesity is increased corticosteroid secretion. In pharmacological doses, corticosteroids increase cardiac output. It is not clear whether the modest increase in 17-hydroxysteroid and 17-ketosteroid secretion in obesity could explain vascular hypertension or whether the increase in steroid production is a response to increased circulating blood volume.

Regardless of the underlying pathophysiological mechanism, the hemodynamic and metabolic changes in obesity may exert an adverse effect on hypertension. The increased cardiac output and blood volume in obesity, when imposed upon persons with hypertension, may further strain the circulatory system and compromise its functional adequacy. These adverse effects are diagramatically illustrated in figure 1, which illustrates the various pathophysiological mechanisms that obesity may add to hypertension. Weight reduction in the obese hypertensive may remove the extra metabolic and hemodynamic burdens that

![Figure 1](http://circ.ahajournals.org/)

A suggested mechanism for the adverse hemodynamic effect of obesity in hypertensive subjects.

*Circulation, Volume XXXIX, March 1969*
obesity imposes upon hypertension. It seems plausible that weight reduction would diminish circulatory strain, thus preventing accelerated decompensation in hypertensive heart disease among the obese, especially when coupled with appropriate antihypertensive therapy.

Prognosis of Overweight and Hypertension

The Build and Blood Pressure Study in 1959 showed excessive mortality in overweight persons among all age and blood pressure groups. When mortality is assessed by height and weight levels for three blood pressure groups (normal, moderate, and more severely elevated) and three age groups (15 to 39, 40 to 49, 50 to 69 at policy issue), the excess mortality is greatest in the younger age group, due primarily to cardiovascular disease. Even slight and moderate overweight is associated with excess of mortality in the more severely hypertensive group. According to Lew, at levels of systolic blood pressure of 140 to 160 mm Hg and diastolic pressure of 90 to 100 mm Hg, overweight curtails the expectation of life among individuals who are both overweight and hypertensive as compared to hypertensive subjects who are not overweight. However, longevity is much less affected among women than men at corresponding degrees of overweight and hypertension.

Overweight and hypertension are also associated with an excess of coronary heart disease and cerebrovascular disease, especially among men. Levy and his colleagues showed that retirement from active service and death from cardiovascular renal disease were more common among Army officers in the obese-hypertensive group than in the obese or hypertensive groups alone. Severity of hypertensive retinopathy and cardiovascular complications were related to obesity in Tibblin's study of hypertensive disease in men aged 50. However, in other clinical series of patients with more severe hypertensive disease, Bechgaard, Frant, and Mathisen found mortality in obese hypertensive subjects no higher than in nonobese hypertensive persons. In fact, obese hypertensive women may have a relatively better chance of long-term survival. In a 10-year follow-up study of a small number of hypertensive patients without retinal change or only arteriolar narrowing at the Mayo Clinic, Breslin and colleagues showed a slightly greater mortality in individuals 30% overweight but in individuals with sclerosis and exudates, the prognosis was slightly better in the obese group.

Clinical studies are highly selective. The difference between these studies and the life insurance company experience may be due to the fact that the actuarial study dealt with blood pressures in the range from 140 to 160/90 to 100 mm Hg, whereas blood pressures in the clinical investigations were higher. Blood pressures over 160/100 mm Hg apparently overshadow the effect of overweight on mortality and the course of severe hypertensive disease may be altered but little by weight reduction. However, in individuals with a moderate degree of blood pressure elevation associated with overweight, weight reduction may significantly change the course of the disease. Obese persons who are able to reduce their weight comprise a relatively small and selected proportion of the total group. Nevertheless, data from the Build and Blood Pressure Study indicate a lowering of the mortality ratio among such men; there were too few women in the series for analysis. Among those called "moderately obese" (averaging 25% overweight), the mortality ratio was 100% in those who lost weight, as compared with a ratio of 128% in the total group; the corresponding figures in the "markedly obese" (averaging 35 to 40% overweight) were 96% and 151%, respectively.

It is difficult to extrapolate these findings to what might be the effect of weight reduction in the population at large. For this reason, there is a great need for documenting the potential benefits of weight loss among the obese and obese hypertensive subjects by means of further study. However, the converging evidence from a large variety of sources summarized in this review strongly suggests that the impact of weight reduction in the general population would be very considerable.
and lessen appreciably the burden of mortality and also morbidity from cardiovascular diseases.

References

19. Tecumseh Community Health Study: Unpublished data.
OVERWEIGHT AND HYPERTENSION


52. NUTRITION SURVEY OF THE ARMED FORCES: A Report by Interdepartmental Committee on Nutrition for National Defense. Washington 25, D. C., U. S. Gov't Printing Office. Thailand (1960); Republic of China (1960); Ethiopia (1959); Philippines (1957); Uruguay (1963); Alaska (1959); Chile (1960).


89. BAUMAN, L.: Obesity: Recent reports in the literature and results of treatment. JAMA 90: 22, 1928.
Overweight and Hypertension: A Review
BENJAMIN N. CHIANG, LAWRENCE V. PERLMAN and FREDERICK H. EPSTEIN

Circulation. 1969;39:403-421
doi: 10.1161/01.CIR.39.3.403
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/39/3/403

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/