The Relative Contribution of Body Fat and Fat Pattern to Blood Pressure Level

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SUMMARY Although the association between body weight and blood pressure is irrefutable, body fat mass and blood pressure level may not necessarily be directly related. To clarify the relative contribution of fat mass to blood pressure level, we analyzed data on 399 adults consecutively entering a weight control program. Although most subjects were notably overweight (mean ideal body weight 177%), the population represented a wide spectrum of body weights and blood pressure levels. Study parameters included body fat mass (by total body water, $^{40}$K, and Steinkamp formula), lean body mass, body build (chest to height ratio), fat cell number and size from bilateral buttock biopsy specimens, upper fat pattern by arm to thigh circumference ratio, and central fat pattern by subscapular to triceps skinfold ratio. Our results concurred with previously noted correlations between obesity and blood pressure (as mean arterial pressure): weight ($r = 0.44$), percentage of body fat ($r = 0.19$), and absolute fat mass ($r = 0.38$; all $p < 0.01$); however, lean body mass, age, and body build correlated highly with both fat mass and mean arterial pressure, thereby confounding this relationship. Multivariate analysis was performed to evaluate the relative contribution of fat mass to mean arterial pressure in the presence of these and other potentially confounding variables. Lean body mass, age, body build, and an upper body fat pattern were found to contribute significantly to the variation in mean arterial pressure ($p < 0.01$). In their presence, percentage of body fat, absolute fat mass, central fat pattern, fat cell characteristics, and age of onset of obesity did not significantly improve the predictability of mean arterial pressure. Thus, blood pressure level may be related to the aforementioned correlates of obesity and to body fat pattern rather than to fat mass per se. (Hypertension 7: 578-585, 1985)

KEY WORDS • obesity, blood pressure • lean body mass • fat pattern • body build • fat cell number and size • weight • hypertension

CONSIDERABLE epidemiological and clinical evidence exists to support an association between obesity and hypertension. The increased prevalence of obesity among hypertensive subjects and hypertension among the obese is well documented. In addition, prospective trials have demonstrated blood pressure lowering with weight reduction. Several observations lead us to question the presumed causal relationship of obesity and hypertension: (1) the majority of the obese are not hypertensive, (2) not all cross-sectional studies show a significant association between indices of obesity and blood pressure level, and (3) the positive coefficients of correlation are consistently small, with indices of obesity explaining only 3 to 14% of the variability in blood pressure level. In addition, several cross-sectional studies using a limited number of prognostic variables suggest the existence of confounding variables in the association of fatness and blood pressure. Thus, the association between indices of obesity and blood pressure may simply be an artifact of the mutual dependence of obesity and blood pressure on other variables such as age and body build.

This study was undertaken to identify variables that might be indirectly responsible for the observed asso-
ciation of body weight and blood pressure and to determine, in their presence, the relative contribution of fatness to blood pressure level. In addition, we examined the data for the existence of other factors independently predictive of blood pressure level.

Methods

Study subjects included 399 adults (87 men, 312 women) consecutively admitted to the Weight Control Unit of the Obesity Research Center, St. Luke's–Roosevelt Hospital Center, New York, between December 1977 and May 1981. Seventy-four subjects who were taking antihypertensive medication, including diuretics for any reason, were omitted from the analyses involving blood pressure level. Patients were either self-referred or physician-referred for weight control. Because there was a fee for services often not reimbursed by health insurance, subjects probably were more affluent than the obese population in general.

Age, race, sex, age of onset of obesity, current blood pressure lowering medications, and presence of hypertension among first-degree relatives were obtained from a questionnaire completed on admission to the Weight Control Unit. Height and 12 other anthropometric measurements (skinfold thickness at thorax, abdomen, upper arm, and subscapular region; length of arm and thigh; and circumference at waist, iliac crest, wrist, upper arm, chest, and thigh) were obtained in the fashion described by Steinkamp et al., all on the right side of each subject. Measurements were carried out by a laboratory technician who had been trained for more than 6 months. Lengths and circumferences were examined with good reproducibility; within individuals, each of the skinfold measurements had a coefficient of variation of 15 to 20%. Blood pressure measurements were taken in the right arm after a brief period of rest in the sitting position. A large adult cuff size was used when the subjects were wearing an examining gown.

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Results, subsequent analyses were performed combining all blood pressure groups (high, intermediate, and normotensive subgroups were comparable (see Results), subsequent analyses were performed combining all blood pressure groups (high, intermediate, and normal).

To test the relative contribution of selected study parameters to the variability of blood pressure level, multivariate analyses were performed with a multiple linear regression model using a stepwise variable selection procedure. The independent variables in the regression procedure were potential correlates of blood pressure level and were selected to minimize multicollinearity, or interdependence, such that two parameters did not describe a common phenomenon. For example, if arm to thigh circumference ratio were selected as a variable in the model to describe upper body fat pattern, then waist to hip ratio would not be used because of their high interdependence and correlation.
The adequacy of the model was confirmed by inspecting separate plots of the residuals against the predicted values and the residuals against the independent variables as described in Draper and Smith.

Results

Characteristics of Patient Population

The majority of subjects were white (94% white, 5% black, 1% other) women (312/399 or 78%). A broad range of age, weight, and blood pressure was present, as shown in Figures 1 and 2. All subjects entered the Weight Control Unit because of a concern about body weight; however, 19 of the 399 subjects (5%) were actually within a normal body weight range (80–120% of ideal body weight) and 1 subject was less than 100% of ideal body weight. A family history of hypertension was present in 43% of the subjects.

The study population is described in Table 1. The results of every parameter were available in 218 of the 399 subjects. Their characteristics were similar to the total population, as shown in Table 1.

Univariate Correlates of Blood Pressure

The correlation coefficients of the various study parameters and systolic, diastolic, and mean arterial pressure (MAP) are given in Table 2. Of note was the finding that measures of body fatness (fat mass and percent body fat) had weaker correlations with blood pressure than did measures reflecting body build (chest to height ratio, weight, and weight to height indices). The magnitude and relative order of the correlations were essentially identical for the subpopulation of 218 individuals for whom all study parameters were available.

Table 1: Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>Mean ± sd</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>399</td>
<td>41 (41) ± 12 (12)</td>
<td>14–74 (17–74)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>399</td>
<td>103 (104) ± 27 (26)</td>
<td>53–213 (60–205)</td>
</tr>
<tr>
<td>% Ideal body weight</td>
<td>399</td>
<td>177 (177) ± 41 (40)</td>
<td>96–332 (101–332)</td>
</tr>
<tr>
<td>% Body fat*</td>
<td>374</td>
<td>39 (40) ± 8 (8)</td>
<td>14–58 (21–58)</td>
</tr>
<tr>
<td>Age of onset of obesity (yr)</td>
<td>399</td>
<td>17 (17) ± 12 (12)</td>
<td>1–67 (1–67)</td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td>320+</td>
<td>126/82 (128/82) ± 18/11 (17/10)</td>
<td>80–200/50–121 (96–200/50–121)</td>
</tr>
</tbody>
</table>

*From total body water.
+Subjects taking antihypertensive medications are excluded.
TABLE 2. Univariate Correlates of Mean Arterial Pressure (MAP) for all Subjects, Normotensive Subjects (MAP < 93.3 mm Hg), and Hypertensive Subjects (MAP ≥ 106.7 mm Hg)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>All subjects (n = 233-320)</th>
<th>Normotensive (n = 79-118)</th>
<th>Hypertensive (n = 45-61)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Build (chest to height ratio)</td>
<td>0.47* (0.46*±0.43*)</td>
<td>0.34*</td>
<td>0.34*</td>
</tr>
<tr>
<td>Weight to height indices (W, W/H, W/H2, H/W, % ideal W)</td>
<td>0.42-0.45* (0.42-0.43*/0.38-0.42*)</td>
<td>0.25-0.31*</td>
<td>0.15-0.20</td>
</tr>
<tr>
<td>Body fat mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>By total body water</td>
<td>0.38* (0.37*±0.35*)</td>
<td>0.30*</td>
<td>0.33*</td>
</tr>
<tr>
<td>By ⁴ᴷK</td>
<td>0.37* (0.32*±0.37*)</td>
<td>0.29*</td>
<td>0.24*</td>
</tr>
<tr>
<td>By Steinkamp formula</td>
<td>0.43* (0.40*±0.40*)</td>
<td>0.33*</td>
<td>0.29*</td>
</tr>
<tr>
<td>Age</td>
<td>0.33* (0.39*±0.24*)</td>
<td>0.23*</td>
<td>0.21</td>
</tr>
<tr>
<td>Lean body mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>By total body water</td>
<td>0.38* (0.31*±0.38*)</td>
<td>0.07</td>
<td>0.10</td>
</tr>
<tr>
<td>By ⁴ᴷK</td>
<td>0.23* (0.19*±0.24*)</td>
<td>0.07</td>
<td>0.24</td>
</tr>
<tr>
<td>Fat pattern†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper</td>
<td>0.33* (0.32*±0.31*)</td>
<td>0.21*</td>
<td>0.43*</td>
</tr>
<tr>
<td>Central</td>
<td>0.20* (0.20*±0.17*)</td>
<td>0.13</td>
<td>-0.02</td>
</tr>
<tr>
<td>Adipocyte characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat cell size</td>
<td>0.20* (0.21*±0.16*)</td>
<td>0.19*</td>
<td>0.11</td>
</tr>
<tr>
<td>Fat cell number</td>
<td>0.18* (0.16*±0.17*)</td>
<td>0.18</td>
<td>0.02</td>
</tr>
<tr>
<td>Body fat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>By total body water</td>
<td>0.19* (0.22*±0.15*)</td>
<td>0.31*</td>
<td>0.20</td>
</tr>
<tr>
<td>By ⁴ᴷK</td>
<td>0.28* (0.26*±0.27*)</td>
<td>0.27*</td>
<td>0.29*</td>
</tr>
<tr>
<td>By Steinkamp formula</td>
<td>0.13* (0.16*±0.08)</td>
<td>0.28*</td>
<td>0.12</td>
</tr>
<tr>
<td>Type of obesity (by age onset)</td>
<td>-0.01 (0.03/ -0.04)</td>
<td>-0.02</td>
<td>-0.09</td>
</tr>
</tbody>
</table>

W = weight; H = height; ⁴ᴷK = natural isotope of potassium measured by whole body counter.

*p < 0.05.
†Upper pattern defined by arm to thigh circumference ratio; central pattern defined by subscapular to triceps skinfold ratio.

Because selection of individuals for a genetic predisposition to hypertension could increase the association between measures of fatness and blood pressure, subpopulations with and without a family history of hypertension were examined, as shown in Table 3. There were no significant differences in the slopes of the regression lines between MAP and any of these variables by analysis of covariance. Thus, the relationship between MAP and these measures of fatness appears to be unrelated to a family predisposition to hypertension.

Because body build was estimated as the chest to height ratio, which may have been affected by chest wall fatness, the effect of the latter was removed by stratification of the population according to subscapular skinfold thickness. The correlation of body build and MAP remained highly significant (p < 0.01) at skinfold thickness levels of less than 20, 20 to 40, and greater than 40 mm. In addition, analysis of covariance controlling for subscapular skinfold thickness showed a highly significant regression of MAP on chest to height ratio (p < 0.001). Hence, the association of chest to height ratio and MAP appeared to be independent of chest wall fatness as measured by subscapular skinfold thickness.

Arm to thigh circumference ratio was chosen as the measure of upper body fat pattern, and subscapular to triceps skinfold ratio as the measure of central body fat pattern. Justification for selecting these two indices of...
body fat patterning was twofold: (1) the indices do not overlap in delineating an upper and a central pattern of obesity (i.e., the arm to thigh ratio describes an upper versus lower pattern, while both measures are peripheral; the subscapular to triceps ratio describes a central versus peripheral pattern, while both measures are upper) and (2) neither index had a significant correlation with percentage of body fat \( (r = 0.08 \text{ and } 0.003 \text{ respectively}) \). By contrast, a previously used index, waist to hip ratio, does not distinguish between upper and central fat patterns, and it had a significant correlation with percentage of body fat \( (r = -0.22, p = 0.0001) \). Although both of the chosen indices correlated with MAP, they were only weakly associated with each other \( (r = 0.14, p = 0.01) \), which suggests that upper and central fat patterns may be distinct.

Of interest was the finding that age of onset of obesity was not significantly associated with the distribution of body fat (as determined by these or 9 other ratios of anthropometric measurements descriptive of body fat pattern). The only exceptions were the weak but significant correlations \( (r = 0.2-0.3) \) among men between age of obesity onset and three trunk to thigh circumference ratios.

Several variables confounded the association of blood pressure and fatness. Lean body mass correlated with MAP \( (r = 0.38, p < 0.0001) \) and absolute fat mass \( (r = 0.43, p < 0.0001) \). Body build correlated with MAP \( (r = 0.47, p < 0.0001) \) and absolute fat mass \( (r = 0.76, p < 0.0001) \). Age correlated with MAP \( (r = 0.33, p < 0.0001) \) and percentage of body fat \( (r = 0.11, p = 0.04) \), although it did not correlate significantly with absolute body fat mass \( (r = -0.01) \).

**Analysis of the Independent Contribution of Fatness to Blood Pressure**

Since univariate correlations revealed the confounding associates of fatness and blood pressure noted above, a multivariate procedure was needed to quantitate the relative contribution of each parameter to blood pressure level when examined in the presence of the others. Twelve study parameters were analyzed using a stepwise regression model among the 218 subjects who had the results of all study parameters available (see Table 4). As the results of this analysis were essentially identical when examined separately for systolic, diastolic, and mean arterial pressure, only the results of the latter are reported. Body build (chest to height ratio) and body mass index (weight/height\(^2\)) were entered into the model by substitution because of their high intercorrelation (among men, \( r = 0.94 \); among women, \( r = 0.85 \)).

Four parameters were found to be highly predictive of blood pressure level: lean body mass, age, lateral body build, and an upper body fat pattern. Presence or absence of a family history of hypertension contributed to a smaller extent. Substitution of lean body mass by \(^{40}\)K technique for that by total body water did not alter the results. Nor did substitution of body mass index as the measure of body build alter the results. These five parameters explained 37% of the variation of MAP. In their presence, the power of the model to predict blood pressure level was not significantly increased by body fat mass, percentage of body fat, central body fat pattern, fat cell number or size, age of onset of obesity, or sex. The regression equation for this model is as follows: MAP = 66.76 + 0.40 lean body mass (kg) + 0.26 age (yr) + 26.42 chest to height ratio + 10.14 arm to thigh ratio + 2.19 (if family history of hypertension).

As the order of entry of each of the parameters into the stepwise regression procedure can potentially alter the results, the maximum R\(^2\) model (to examine all combinations of variables) was also employed and gave identical results. Finally, to be certain that percentage of body fat was given the best opportunity to correlate with MAP, this parameter was forced into the regression model first. Even this procedure failed to increase its relative contribution to the variability of MAP.

**Discussion**

The well-documented association between weight and blood pressure observed in heterogeneous groups may be due to the mutual dependence of weight and blood pressure on confounding variables such as age and body build. Age correlates with both weight and blood pressure among industrialized populations, and has been found to account for as much as 35% of the variation in MAP. 45% of the variation in percentage of body fat. Knowledge of the confound-
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The effect of body build on the association of obesity and hypertension dates back to the early studies of Robinson and Bruce,11, 22, 23 who documented a strong association between chest to height ratio and relative weight, which suggested a tendency toward obesity in lateral build groups. At the same time, persons with a lateral build had 4 to 11 times the frequency of hypertension compared with persons of linear build. When body build was controlled, weight accounted for less than 1% of the variation in blood pressure. Several investigators have documented stronger associations of blood pressure with weight or weight to height indices than with more direct indices of fatness, such as skinfold thickness.1, 24, 25 Stanton et al.9 reported similar results and also showed that percentage of body fat (derived from skinfold measurements) in the presence of age and body mass index had little effect on the accuracy of classifying individuals as hypertensive or normotensive. In the present study we used a similar method of multivariate analysis but included parameters of body fat pattern, fat cell characteristics, family history of hypertension, and more sophisticated measures of body composition. We found that the relative contribution of measures of fatness to blood pressure level was insignificant compared with the effects of lean body mass, age, a lateral body build, an upper body fat pattern, and a family history of hypertension. Collectively, these data suggest that the well-documented association between weight and blood pressure may be due to age or to some aspect of body composition other than fatness per se.

To our knowledge, this is the first study to demonstrate in both univariate and multivariate models of analysis an association between lean body mass and blood pressure level. Data from the 1971 to 1975 National Health and Nutrition Examination Survey26 support our findings that skinfold thickness did not correlate with blood pressure as closely as did the body mass index, which suggests that non-fat components may be more important. Most obese individuals have an increased lean body mass.27 Although a physiological explanation for the relationship between lean body mass and blood pressure is not evident, it is quite possible that the changes in lean mass that accompany changes in body weight27 could directly affect blood pressure levels. In contrast to our findings, Berglund et al.28 and Siervogel et al.21 were unable to document a significant correlation between lean body mass and blood pressure. Other than their use of fewer subjects, it is difficult to explain the difference in results.

Although fatness itself may not be a major factor relating to blood pressure level, distribution of body fat appears to have an independent contribution. Blair et al.29 evaluated the association of triceps and subscapular skinfold thickness with blood pressure and found that only the subscapular skinfold (implying central adiposity) was a better predictor of blood pressure level. Similarly, Stallones et al.30 developed a fat patterning index to describe the relative distribution of trunk versus extremity fat and showed a greater tendency for increasing systolic blood pressure with adiposity of the trunk. With the large number of anthropometric measurements available for analysis in this study, we were able to consider a variety of ratios in an attempt to best describe body fat patterns. Evaluation of the two ratios selected (arm to thigh circumference and subscapular to triceps skinfold) suggested that (1) an upper body fat pattern may be distinct from a central fat pattern, (2) the upper fat pattern is more likely to be predictive of a higher blood pressure level, and (3) there is no unique body fat pattern associated with early or late onset of obesity.

The relationship between blood pressure and fat cell characteristics (number and size) has been examined by several investigators. In mixed populations of normal and hypertensive individuals, neither Berglund et al.24 nor Siervogel et al.21 found a significant association between fat cell size and blood pressure level, regardless of whether the biopsy site was the abdomen28 or buttock.21 Only in a subgroup of normotensive subjects did Berglund et al.28 find a positive association. The importance of the site of biopsy relative to pathological states is suggested by the data of Kissebah et al.31 In a study of adipose tissue morphology, fat patterning, and metabolic profiles, they noted increased fat cell size in the abdominal region in subjects with upper body compared with lower body obesity, and highly significant correlations between fat cell size and glucose (r = 0.41) and insulin (r = 0.87) responses to a glucose challenge. On the other hand, fat cell size determined from a thigh biopsy specimen was not significantly different between groups with upper and lower fat patterns and was not associated with the metabolic parameters. In our study of a mixed population unselected for blood pressure level, we were able to document a weak but significant univariate association of both fat cell size (0.20) and fat cell number (0.18) with blood pressure level. When evaluated in the presence of the other study parameters, however, neither fat cell size nor number contributed significantly to the variability in blood pressure level, which suggests that fat cell morphology as described from buttock biopsy specimen is a relatively unimportant associate of blood pressure level. It could be, however, that the site of biopsy limits the significance of these data.

Recognizing the findings of Pietinen et al.,32 who showed that a family history of hypertension was an important factor in determining the strength of the association between weight and blood pressure level, we specifically tried to evaluate the role of heredity on the relationship between fatness and blood pressure. We found that the presence or absence of a family history of hypertension made little difference in the relationship between weight or body fatness and blood pressure level. Our study population included a wide spectrum of degrees of obesity but clearly differed from that of Pietinen et al.,32 which contained a larger proportion of normal weight individuals.

A potentially important limitation of our study was the unavailability of dietary variables in the analysis. Dietary sodium,33 potassium,34 calcium,35, 36 fat,37 and
alcohol, alcohol, as well as foods of animal origin, have been shown to be potential contributors to blood pressure level. On the other hand, recent data by Stanton et al. suggest that these variables may have contributed little to the variation of blood pressure level. In the presence of age and body mass index they found that the accuracy of classifying individuals as hypertensive or normotensive was not increased significantly by inclusion of dietary intake of fat, sodium, calcium, potassium, and alcohol. All dietary variables combined accounted for less than 1% of the variance in blood pressure.

The present data suggest that the blood pressure level of a population consisting predominantly of obese, affluent, white women is unlikely to reflect fatness per se relative to their lean body mass, age, body build, distribution of body fat, or their family history of hypertension. The evident implication is that fat loss in itself would be expected to have a relatively small effect on blood pressure level. There is ample data indicating that weight reduction results in blood pressure reduction; however, the hypertensive response to weight loss within individual studies is not uniform, and several studies report no significant relationship between change in body fatness and change in blood pressure. If excess body fat were a major determinant of increased blood pressure, a significant, permanent, and proportionate reduction in blood pressure would be expected with loss of excess body fat. This remains unproved, although the data of Reisin et al. suggest that some factor associated with reduced body weight may account for blood pressure reduction. Animal studies and clinical observations suggest that negative energy balance may be more closely related to the fall in blood pressure than are changes in body fat. Factors that have been or should be considered contributors to the hypertensive response to weight reduction include the fall in levels of insulin, triiodothyronine, and sympathetic nervous system activity; changes in intake of sodium, potassium, fat, alcohol, and foods of animal origin; altered exercise patterns; and — as yet untested — changes in lean body mass. Pending the availability of appropriate prospective studies to clarify the hemodynamic response to weight reduction, the results of the present investigation warrant caution in the assumption that body fat itself is a major determinant of blood pressure level among obese hypertensive individuals.

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