Does Obesity Influence Early Target Organ Damage in Hypertensive Patients?

Roland E. Schmieder, MD, and Franz H. Messerli, MD

Background. Various prospective studies have found that lean hypertensive patients have greater cardiovascular morbidity and mortality than obese hypertensive subjects. It was therefore hypothesized that hypertension is more benign when associated with obesity. In the present study, we evaluated effects of obesity on early target organ damage in patients with essential hypertension.

Methods and Results. In a total of 207 subjects, systemic and renal hemodynamics as well as left ventricular structure and function were assessed by measuring cardiac output (indocyanine green dye dilution), renal blood flow (clearance of 131I paraiminohippuric acid), and mean arterial pressure (invasively) and by two-dimensionally guided M-mode echocardiographic findings. Systemic and renal vascular resistance, compliance of the large arteries evaluated by the stroke volume/pulse pressure index, and left ventricular mass served as parameters for early target organ damage. All individuals were categorized into four groups: lean and obese normotensive as well as lean and obese hypertensive subjects. In obese hypertensive patients, total peripheral resistance was significantly lower and stroke volume/pulse pressure index was higher than in the lean hypertensive group, almost reaching values of normotensive control subjects. No effect of obesity on the renal circulation was noted, whereas in hypertension, renal vascular resistance was elevated. The degree of left ventricular hypertrophy was more pronounced in the hypertensive groups than in their normotensive counterparts and progressively increased with obesity. Nevertheless, in obese hypertensive patients, left ventricular function, as measured by fractional fiber shortening and velocity of circumferential fiber shortening, was maintained despite the fact that the heart had been exposed to the double burden of an increased preload (obesity) and afterload (hypertension).

Conclusions. Obesity had a disparate effect on target organs in hypertension. At rest, obesity seemed to mitigate cardiovascular changes in the systemic vascular bed caused by hypertension. However, no such mitigation was observed in the renal vasculature, and left ventricular hypertrophy was even exacerbated by the presence of obesity. Our findings in part negate the concept that obesity is able to exert a protective effect on early target organ damage in hypertensive patients and, in particular, on the heart. (Circulation 1993;87:1482–1488)

Key Words • obesity • hypertension

Hypertension and obesity are disorders that are closely linked.1-3 For many years, it has been well documented that obese patients are more likely to be hypertensive than lean individuals and that interim weight gain is predictive of later onset of hypertension.4,5 Both obesity and arterial hypertension have been identified as independent risk factors for cardiovascular disease in the Framingham Heart Study.6-7 In the Honolulu Heart Program, rates of coronary heart disease and cardiovascular disorders were higher in obese than in nonobese men for both normotensive and hypertensive patients.8 In a prospective follow-up of 8 years, the relative risk for fatal and nonfatal coronary heart disease increased from the lowest to the highest quintile of obesity in hypertensive women.9 The absence of a protective effect in obesity hypertension is supported by a recent analysis of the Framingham Heart Study, in which the risk ratio for cardiovascular events was basically similar in lean and obese subjects.5,10 These studies are in contrast to several large-scale prospective studies that showed obese hypertensives to be at a lower risk of cardiovascular disease than lean hypertensives11-20 and therefore showed that obesity might exert “protective” effects on the cardiovascular system. In particular, in several studies in which the question of whether obesity attenuates the impact of high blood pressure on coronary arteries was addressed, prospective analysis revealed a lower risk for coronary heart disease in obese than in lean hypertensive subjects.16,17,19,20 It is difficult to explain the discordant results of the various studies. A recent review of 11 prospective studies suggested that the risk of major coronary artery disease–related events in lean hypertensive men was not as high as that in obese hypertensive men.21

Ever since Perera and Damon22 reported that accelerated hypertension (defined by papilledema and biopsy-verified arteriolar necrosis of kidney) is more prevalent in lean than in obese women but not men, the concept of the vascular protective effects of obesity in a subgroup of hypertensive patients has been controversial. No study has

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Received October 3, 1992; accepted January 11, 1993.
looked at these protective effects of obesity at an early stage of hypertensive disease, i.e., before cardiovascular death or irreversible target organ damage (World Health Organization stage III) has developed.

Our current investigation extended our previous analyses of the interaction between obesity and hypertension.23–27 Using hemodynamic as well as echocardiographic techniques, we report our experience in more than 200 patients in an attempt to elucidate the question of whether obesity exerts any protective effects on the systemic circulation, renal vasculature, and cardiac structure in early hypertensive disease.

Methods

Study Groups

The study population was composed of 207 subjects (mean age, 42±10 years) consecutively enrolled in the study protocol. All patients with high blood pressure were referred to the outpatient clinic of the Ochsner Clinic (single-center study) to specify the cause and stage of arterial hypertension (tertiary referral center). The normotensive control group that voluntarily participated in the echocardiographic and hemodynamic study had been referred to Ochsner Clinic because of obesity or unspecified gastrointestinal disorders. Arterial hypertension was said to be present if casual blood pressure exceeded 140 mm Hg systolic and 90 mm Hg diastolic on at least three successive measurements taken with a regular cuff for the lean subjects and with a large cuff for the obese subjects.

All normotensive patients had casual pressure values consistently below 140/90 mm Hg when measured in the outpatient clinic. Therefore, individuals whose casual blood pressure was more than or less than 140/90 mm Hg were excluded from the study. Each participant had a complete clinical workup to exclude secondary causes of arterial hypertension, diabetes mellitus, any form of renal disease, or cardiovascular impairment (in particular, coronary heart disease).28 Most patients had never been treated for high blood pressure. In those who had received treatment, anti-hypertensive therapy was discontinued at least 4 weeks before the invasive and echocardiographic study. The protocol of the study was approved by the institutional clinical investigation committee, and informed consent was obtained from each patient.

Participants were classified as "lean" when their body mass index ranged between 19 and 27 kg/m². Body mass index >27 kg/m² was chosen as a cutoff point between "obese" and "lean." Patients whose body mass index was between 27 and 31 kg/m² were graded as "mildly obese," and those whose body mass index was >31 kg/m² were considered "severely obese." These benchmarks of obesity, 27 and 31 kg/m², respectively, were chosen according to the survey of NHANES II.29 The cutoff points of 27 and 31 kg/m² further correspond very closely to being 20–40%, respectively, over the desirable weight for persons of medium build as determined by the Metropolitan Life Insurance Company in 1983.30

### Table 1. Clinical Characteristics in Normotensive Subjects and Patients With Essential Hypertension Divided According to Body Mass Index >27 kg/m² or ≤27 kg/m²

<table>
<thead>
<tr>
<th></th>
<th>Lean Normotension (n=29)</th>
<th>Essential Hypertension (n=61)</th>
<th>Obese Normotension (n=26)</th>
<th>Essential Hypertension (n=85)</th>
<th>ANOVA*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>39±11</td>
<td>44±12</td>
<td>33±11</td>
<td>44±9</td>
<td>0.001</td>
</tr>
<tr>
<td>Sex (male:female)</td>
<td>20:9</td>
<td>31:30</td>
<td>18:8</td>
<td>61:24</td>
<td>0.01</td>
</tr>
<tr>
<td>Race (white:black)</td>
<td>27:2</td>
<td>45:16</td>
<td>22:4</td>
<td>53:32</td>
<td>0.01</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170±20</td>
<td>170±8</td>
<td>171±9</td>
<td>173±9</td>
<td>0.001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69±10</td>
<td>70±9</td>
<td>100±18</td>
<td>94±14</td>
<td>0.001</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23.8±2.4</td>
<td>24.0±2.2</td>
<td>34.0±6.2</td>
<td>31.5±4.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>86±7</td>
<td>112±14</td>
<td>91±5</td>
<td>109±12</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*See text for further details.

### Table 2. Systemic Hemodynamics in Normotensive Subjects and Patients With Essential Hypertension Divided According to Body Mass Index >27 kg/m² or ≤27 kg/m²

<table>
<thead>
<tr>
<th></th>
<th>Lean Normotension (n=29)</th>
<th>Essential Hypertension (n=61)</th>
<th>Obese Normotension (n=26)</th>
<th>Essential Hypertension (n=85)</th>
<th>ANCOVA*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>119±10</td>
<td>158±24</td>
<td>124±7</td>
<td>153±21</td>
<td>0.001</td>
</tr>
<tr>
<td>Diastolic pressure (mm Hg)</td>
<td>69±6</td>
<td>89±11</td>
<td>75±5</td>
<td>88±10</td>
<td>0.001</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>67±9</td>
<td>68±9</td>
<td>71±11</td>
<td>67±10</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.41±1.1</td>
<td>5.28±1.0</td>
<td>6.7±1.6</td>
<td>6.27±2.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>81±17</td>
<td>78±15</td>
<td>96±23</td>
<td>95±21</td>
<td>0.001</td>
</tr>
<tr>
<td>Total peripheral resistance (units)</td>
<td>16.6±4.2</td>
<td>21.8±4.7</td>
<td>14.3±3.7</td>
<td>18.2±4.0</td>
<td>0.001</td>
</tr>
<tr>
<td>Stroke volume/pulse pressure (mL/mm Hg)</td>
<td>1.64±0.48</td>
<td>1.27±0.41</td>
<td>1.94±0.40</td>
<td>1.50±0.47</td>
<td>0.02</td>
</tr>
</tbody>
</table>

bpm, Beats per minute.

*See text for further details.
TABLE 3. Renal Hemodynamics in Normotensive Subjects and Patients With Essential Hypertension Divided According to Body Mass Index >27 kg/m² or ≤27 kg/m²

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>ANCOVA*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normotension (n=29)</td>
<td>Essential hypertension (n=61)</td>
<td>Normotension (n=26)</td>
</tr>
<tr>
<td>Renal plasma flow (mL/min)</td>
<td>670±127</td>
<td>599±125</td>
<td>754±181</td>
</tr>
<tr>
<td>Renal blood flow (mL/min)</td>
<td>1,092±272</td>
<td>970±292</td>
<td>1,244±329</td>
</tr>
<tr>
<td>Renal vascular resistance (units)</td>
<td>84±22</td>
<td>127±48</td>
<td>79±24</td>
</tr>
<tr>
<td>Renal blood supply (RBF/CO; %)</td>
<td>20.5±4.4</td>
<td>18.5±5.6</td>
<td>19.0±5.1</td>
</tr>
</tbody>
</table>

*See text for further details.

Hemodynamics

Systemic and renal hemodynamics were assessed as previously described. In brief, polyethylene tubing was introduced into an antecubital vein and into the brachial artery and advanced to the level of the superior vena cava and the ascending aorta, respectively. Continuous intra-arterial pressure was recorded, and mean arterial pressure was obtained by electrical integration. Cardiac output in the supine position was measured in triplicate using indocyanine green dye. Heart rate was measured by continuous ECG. Renal plasma flow was determined from the disappearance of a single injection of 131I para-aminobiphenyl acid, using the two-compartment model for calculations. Stroke volume, total peripheral resistance, renal blood flow, renal blood supply (renal blood flow divided by cardiac output), and renal vascular resistance were calculated by standard formulas. The vascular compliance of large arteries was estimated by the stroke volume/pulse pressure index.

Echocardiography

Left ventricular structure and function were evaluated by two-dimensionally guided M-mode echocardiography during or immediately after the invasive study. Echocardiographic tracings were recorded in the half-left-sided position in the third or fourth interspace and analyzed by two independent observers according to the recommendations of the American Society of Echocardiography (ASE). Because left ventricular mass by ASE conventions systematically overestimates the true anatomic left ventricular weight, the values for left ventricular mass were corrected by using an equation derived from a comparison of echocardiographic mass with necropsy findings. Fractional fiber shortening and velocity of circumferential fiber shortening were taken as indexes of global left ventricular function.

Statistical Analysis

Comparisons of lean and obese patients as well as of normotensive and hypertensive subjects were made by using two-way ANOVA with subsequent Scheffe’s tests. Because the clinical characteristics such as age, sex, and race differed among the four groups (Table 1), covariance analyses were applied to exclude any concomitant impact of age, sex, and race on our results. Because we did not find significant interactions between obesity and hypertension and sex, race, or age, covariance adjustments were made in Tables 2–7. To allow ease of comparison of the groups, age-adjusted values were calculated for determinants of target organ damage (total peripheral resistance, arterial compliance, renal vascular resistance, and left ventricular mass). χ² analyses were done when indicated. Our results in the text and tables are expressed as mean±1 SD and in the figures as mean±1 SEM.

Results

According to the study design, mean arterial pressure in the two hypertensive groups was different from mean arterial pressure in the two normotensive groups. Mean arterial pressure, however, did not differ within the normotensive and hypertensive groups despite a significant difference in body mass index (Table 1). Height was not different among the four groups, whereas age, sex, and race differed significantly (Table 1). Therefore, only results of the two-way ANCOVA were reported in the following, thereby eliminating any impact of age, sex, and race.

TABLE 4. Cardiac Structure and Function in Normotensive Subjects and Patients With Essential Hypertension Divided According to Body Mass Index >27 kg/m² or ≤27 kg/m²

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>ANCOVA*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normotension (n=29)</td>
<td>Essential hypertension (n=61)</td>
<td>Normotension (n=26)</td>
</tr>
<tr>
<td>Left ventricular wall thickness (mm)</td>
<td>16.8±2.7</td>
<td>20.7±3.0</td>
<td>18.3±2.7</td>
</tr>
<tr>
<td>Diastolic diameter (mm)</td>
<td>47.9±8.6</td>
<td>47.6±6.6</td>
<td>49.6±6.8</td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>173±66</td>
<td>223±64</td>
<td>204±67</td>
</tr>
<tr>
<td>Fractional fiber shortening (%)</td>
<td>36±7</td>
<td>35±9</td>
<td>34±6</td>
</tr>
<tr>
<td>Velocity of circumferential fiber shortening (cm/sec)</td>
<td>1.20±0.30</td>
<td>1.12±0.31</td>
<td>1.11±0.19</td>
</tr>
<tr>
<td>Left ventricular stroke work (mm Hg×mL)</td>
<td>127±30</td>
<td>161±32</td>
<td>157±40</td>
</tr>
</tbody>
</table>

*See text for further details.
When compared with lean subjects, cardiac output was increased in the obese patients ($p<0.001$); this increase was observed in the normotensive and hypertensive groups (Table 2). Elevated cardiac output was predominantly produced by increased stroke volume, noted in the obese patient groups ($p<0.001$), whereas heart rate was similar among lean and obese groups; again, these changes were independent of the level of arterial pressure, whether normotensive or hypertensive.

Conversely, total peripheral resistance was decreased in the obese groups ($p<0.001$) in comparison with their lean counterparts, regardless of whether they were normotensive or hypertensive (Table 2). Compliance of the large arteries, as judged by the stroke volume/pulse pressure index, was higher in the obese groups ($p<0.001$) than in the lean groups regardless of their level of arterial pressure. At a similar body mass index, hypertensive patients had a higher total peripheral resistance ($p<0.001$) and lower arterial compliance ($p<0.02$) than normotensive patients.

Renal plasma flow ($p<0.06$) and renal blood flow ($p<0.05$) were lower in hypertensive patients than in normotensive patients; conversely, renal vascular resistance was clearly elevated in established hypertension ($p<0.01$) (Table 3). Obesity did not affect renal plasma flow, renal blood flow, or renal vascular resistance. Renal blood supply (renal blood flow divided by cardiac output) was found to be low in the hypertensive groups compared with the normotensive groups ($p<0.01$) predominantly because of the reduction in renal blood flow ($p<0.05$). Renal blood supply decreased even more in the presence of obesity ($p<0.001$) because cardiac output was elevated in both obese groups (Table 3).

Left ventricular mass increased progressively with obesity and evidence of sustained hypertension (Table 4). The ANCOVA (excluding any bias of age, sex, or race) revealed significant influences of obesity ($p<0.05$) and hypertension ($p<0.01$) on left ventricular mass. Left ventricular wall thickness was increased with hypertension ($p<0.001$) and with obesity ($p<0.05$). Diastolic diameter tended to be increased in the obese groups ($p=0.08$) regardless of whether they were normotensive or hypertensive. In this stage of hypertensive disease (WHO stage I or II), left ventricular function was preserved in hypertensive patients compared with their normotensive counterparts, and obesity appeared not to impair fractional fiber shortening or velocity of circumferential fiber shortening (Table 4). Left ventricular stroke work, the product of stroke volume and systolic pressure, was most elevated in obese hypertensive patients ($p<0.01$) because of increased stroke volume related to obesity and because of increased systolic pressure in hypertension (Table 4).

To illustrate these results, total peripheral resistance and compliance of the large arteries, both of which are considered to reflect the severity of target organ damage in the systemic circulation, and renal vascular resistance and left ventricular mass were expressed as age-adjusted values in Figures 1–4. Furthermore, obese subjects were subdivided into mildly obese (defined by body mass index $>27\text{ kg/m}^2$ but $<31\text{ kg/m}^2$) and severely obese patients (defined by body mass index exceeding $31\text{ kg/m}^2$). Total peripheral resistance ($p<0.01$) was higher and compliance of large arteries ($p<0.05$) was lower in the lean than in the mildly obese as well as the severely obese groups. This pattern was similar in the normotensive and hypertensive groups, although, of course, total peripheral resistance was elevated in sustained hypertension ($p<0.01$). Age-adjusted values for renal vascular resistance indicated that obesity did not affect renal hemodynamics in either the mildly obese or

**Figure 1.** Bar graph of age-adjusted values for total peripheral resistance (TPR adj) in lean, mildly obese, and severely obese patients. A significant impact of obesity ($p<0.01$) and hypertension was observed ($p<0.01$).

**Figure 2.** Bar graph of age-adjusted values for compliance (COMP adj) in lean, mildly obese, and severely obese patients. A significant impact of obesity ($p<0.01$) and hypertension ($p<0.02$) was evaluated.

**Figure 3.** Bar graph of age-adjusted values for renal vascular resistance (RVR adj) in lean, mildly obese, and severely obese patients. No significant impact of obesity was observed, in contrast to the impact of hypertension ($p<0.01$).
FIGURE 4. Bar graph of age-adjusted values for left ventricular mass (LVM adj) in lean, mildly obese, and severely obese patients. A significant impact of both obesity (p<0.05) and hypertension (p<0.01) was noted.

in the severely obese patients, whereas sustained hypertension did (p<0.01) (Figure 3). The degree of left ventricular hypertrophy was most pronounced if obesity and hypertension coexisted (Figure 4). Age-adjusted values for left ventricular mass indicated that the degree of left ventricular hypertrophy increased progressively with obesity (p<0.05) and sustained hypertension (p<0.01).

Finally, to analyze the impact of obesity in various age, sex, and race groups, we calculated the ANCOVA in the specific subgroups (Tables 5–7). The presence of obesity resulted in a decrease in total peripheral resistance that was more pronounced in women than in men (for men, p<0.11; for women, p<0.001; Table 5), more in black than in white individuals, and more in old than in young subjects (all p<0.05). Renal vascular resistance was not influenced by obesity in various sex, race, and age groups (all not significant) in contrast to hypertension. As outlined above, the degree of left ventricular mass was increased in hypertension if obesity coexisted; however, this exaggeration held true only in the white (p<0.01) and male (p<0.01) subjects and in the young and old subjects, although only slightly (p<0.10), but not in the female or black subjects.

Discussion

Systemic vascular resistance is the hallmark of systemic vascular disease and was documented in a prospective study to be predictive for the development of cardio-

vascular morbidity and mortality along with age and left ventricular mass. In our current study in more than 200 participants, systemic vascular resistance was found to be lower in the obese groups than in their lean counterparts at similar levels of mean arterial pressure. The lower level of total peripheral resistance suggested that obese hypertensive patients were hemodynamically younger than their lean counterparts with regard to the systemic circulation. Conversely, we found that obesity hypertension (as opposed to hypertension in the nonobese) is hemodynamically characterized by an increased cardiac output, mainly due to a rise in stroke volume (which is produced via the Frank-Starling mechanism by an increased preload). Confirming our previous reports, these results indicate that the hemodynamic changes in obesity were already evident in subjects whose body mass index exceeded 27 kg/m².

In hypertension, the vascular compliance of the large arteries was reduced, as indicated by a decreased stroke volume/pulse pressure index. The presence of obesity appeared to counteract this process because this index was higher in obese than in lean normotensive subjects. This pattern was found in all subanalyses categorizing our patients—young versus old, black versus white, and male versus female patients. However, all hemodynamic profile measurements were determined at rest only and therefore do not represent the hemodynamic situation during daily life activities.

In two sets of experiments, we recently subjected lean and obese patients to stress and found that obese patients responded with an exaggerated blood pressure response mediated by an increase in total peripheral resistance. Thus, the seemingly protective hemodynamic effects of obesity observed at rest appeared to be blunted during exposure to stress. This new observation in two different study groups clearly limits the interpretation of data determined at rest and questions their applicability for daily life situations.

In the current study, we did not observe any significant difference in renal blood flow and renal vascular resistance among the lean and obese groups regardless of whether the normotensive or hypertensive groups were compared, which is in contrast to a preliminary report that used analysis by matched pairs. Our current analysis, however, was based on a larger sample of patients and excluded any bias of age, sex, race, height, or mean arterial pressure by ANCOVA and specific subgroup analysis. In addition, a stepwise multiple regression analysis yielded no significant association

| Table 5. Determinants of Target Organ Damage in Lean Versus Obese Patients, Men and Women |
|-----------------------------------------------|---------------|-----------------|-----------------|-----------------|
| Parameters for target organ damage            | Lean (n=20)   | Normotensive    | Hypertensive    | Lean (n=8)      | Normotensive    | Hypertensive    | Lean (n=24)      | Normotensive    | Hypertensive    | Impact of hypertension/ obesity* |
| TPR (units)                                   | 20.2±4.6      | 17.7±3.9        | 16.3±4.0        | 13.8±4.6        | 17.6±4.4        | 24.0±4.0        | 15.1±2.2        | 19.4±4.5        | 0.03/0.001      |
| SV/PP (mL/mm Hg)                              | 1.64±0.49     | 1.32±0.42       | 2.05±0.42       | 1.76±0.53       | 1.33±0.42       | 1.11±0.27       | 1.76±0.31       | 1.27±0.41       | 0.09/0.01       |
| RVR (units)                                   | 79±22         | 123±51          | 69±15           | 116±44          | 90±24           | 130±44          | 98±27           | 147±48          | 0.02/NS         |
| LVM (g)                                       | 170±61        | 219±74          | 226±62          | 253±64          | 163±86          | 217±70          | 178±49          | 231±80          | 0.05/NS         |

TPR, total peripheral resistance; SV/PP, stroke volume over pulse pressure; RVR, renal vascular resistance; LVM, left ventricular mass. *ANCOVA.
between body mass index and renal hemodynamics, thereby confirming the current results. The presence of hypertension, in contrast, clearly reduced renal blood flow and increased renal vascular resistance. Castleman and Smithwick reported that the more renal blood flow was reduced and vascular resistance elevated, the more severe was nephrosclerosis in renal biopsies of patients with essential hypertension. Thus, hypertension but not obesity impaired renal perfusion.

In our study, left ventricular mass was greatest in the obese hypertensive groups. This was particularly significant in white subjects and in men, but it was not found in black subjects and women. The latter appeared to be related to the relatively high left ventricular mass in the lean groups. Concomitantly, end-diastolic diameter was slightly enlarged, reflecting increased preload. Although we observed no impairment of cardiac performance, previous analysis noted a depressed myocardial contractility in patients with eccentric left ventricular hypertrophy of obesity. Recently, we reported peak filling rate, duration of peak filling rate, and left atrial emptying index (indicators of ventricular filling) to be reduced in obese normotensive and obese hypertensive patients in comparison with their lean counterparts. Depressed myocardial contractility and diastolic dysfunction, when sustained, will eventually lead to premature congestive heart failure, particularly in patients with obesity hypertension.

Unloading from the heart the double burden of obesity and hypertension by losing weight and controlling blood pressure should become a major goal of preventive cardiology. Although results from the four prospective studies examining the risks of obesity hypertension were controversial, a closer analysis favored the view that obesity in hypertensive patients is deleterious to the cardiovascular system. It appears that prospective studies might have been biased by the fact that lean subjects tended to have more rapid progression of hypertensive disease and earlier manifestations of cardiovascular complications than obese subjects. Consequently, when all patients with signs of cardiovascular disease at baseline were excluded (thereby also excluding lean hypertensive patients, who were more likely to die of cardiovascular complications), the so-called protective effects of obesity hypertension were no longer evident. Lean and obese hypertensive subjects had similar prognoses of the development of subsequent fatal cardiovascular events. However, the four prospective studies did not report statistical analysis of cardiovascular complications from cerebrovascular disease and end-stage renal disease. When looking at individual data, it appears that the risk for renal failure and cerebrovascular events was increased if obesity coexisted with hypertension.

In conclusion, obesity and arterial hypertension disparately affected structural and functional cardiovascular findings. Perfusion of the kidneys in hypertension was impaired to a similar extent in lean and obese hypertensive patients. Patients with obesity were hemodynamically younger than comparable lean subjects, at least during resting conditions. Left ventricular mass was found to be more markedly increased in obesity hypertension, a fact that indicates an additional risk for cardiovascular events. Thus, our analysis of early target organ damage in obesity hypertension does not support the concept that obesity exerts renoprotective or cardioprotective effects on the cardiovascular system.
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Does obesity influence early target organ damage in hypertensive patients?
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*Circulation*. 1993;87:1482-1488
doi: 10.1161/01.CIR.87.5.1482

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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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