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Beneficial Cardiovascular Effects of Bariatric Surgical and Dietary Weight Loss in Obesity

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Objectives We hypothesized that, in obese persons without comorbidities, cardiovascular responses to excess weight are reversible during weight loss by either bariatric surgery or diet.

Background Obesity is associated with cardiac hypertrophy, diastolic dysfunction, and increased aortic stiffness, which are independent predictors of cardiovascular risk.

Methods Thirty-seven obese (body mass index 40 ± 8 kg/m^2) and 20 normal-weight subjects (body mass index 21 ± 2 kg/m^2) without identifiable cardiac risk factors underwent cardiac magnetic resonance imaging for the assessment of the left and right ventricles and of indexes of aortic function. Thirty of the obese subjects underwent repeat imaging after 1 year of significant weight loss, achieved in 17 subjects by diet and in 13 subjects by bariatric surgery. Seven obese subjects underwent repeat imaging after 1 year of continued obesity.

Results Left and right ventricular masses were significantly increased, left ventricular diastolic function impaired, and aortic distensibility reduced in the obese. Both diet and bariatric surgery led to comparable, significant decreases in left and right ventricular masses, end-diastolic volume, and diastolic dysfunction, and an increase in aortic distensibility at all levels of the aorta, most pronounced distally (e.g., distal descending aorta 5.1 ± 1.8 mm Hg^{-1} × 10^{-3} before weight loss and 6.8 ± 2.5 mm Hg^{-1} × 10^{-3} after weight loss; $p < 0.001$). No improvements were observed in continued obesity.

Conclusions Irrespective of method, 1 year of weight loss leads to partial regression of cardiac hypertrophy and to reversal of both diastolic dysfunction and aortic distensibility impairment. These findings provide a potential mechanism for the reduction in mortality seen with weight loss. (J Am Coll Cardiol 2009;54:718–26) © 2009 by the American College of Cardiology Foundation

Obesity, defined by a body mass index (BMI) >30 kg/m^2, is associated with an increased mortality rate, and even greater risk is associated with a BMI of ≥35 kg/m^2 (1). Ventricular hypertrophy, diastolic dysfunction, and aortic stiffness are present in obesity (2,3); there is a strong relationship between left ventricular (LV) hypertrophy (4), diastolic dysfunction (5), and all-cause mortality on the one hand, and between impairment of aortic elastic function and cardiovascular events in healthy and diseased populations on the other hand (6). Therefore, increased mortality in obesity is likely to be related, at least in part, to the long-term cardiovascular sequelae of increased body weight.

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Not only is obesity related to higher death rates, but also a growing body of evidence suggests that weight loss reduces long-term mortality (7). There is, however, little information on the cardiovascular effects of weight loss in obese persons who have no other identifiable cardiovascular risk factors. To investigate this important question, we undertook a study to assess the effects of substantial weight loss, over 1 year, on the cardiovascular changes present in obesity per se, namely, in the absence of traditional cardiovascular risk factors such as hypertension, diabetes mellitus, and hypercholesterolemia. If LV hypertrophy, diastolic impair-

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ment, and aortic stiffness are indeed reversible in such persons, that would suggest that important predictors of cardiovascular risk can be positively influenced by weight loss over 1 year. Such an observation would provide a potential mechanism for the reduction in mortality associated with weight loss.

There are 2 main methods of weight loss: 1) dietary intervention; and 2) bariatric surgery. Surgical weight loss is now well established to confer better long-term weight management than dietary weight loss and has been shown to reduce long-term mortality (7). Therefore, the utilization of bariatric surgery is rapidly increasing.

Despite this, no study to date has addressed the relative beneficial effects of these different weight loss approaches on cardiac and aortic structure and function. In view of this, the secondary aim of this study was to compare the cardiovascular effects of bariatric surgery and dietary weight loss.

Methods

Ethics and study cohort. The study was approved by the local ethics committee, and informed written consent was obtained from each patient.

Fifty-seven subjects (37 obese, BMI >30 kg/m^2, and 20 normal weight controls, BMI 18.5 to 24.9 kg/m^2) were included in the study. All subjects were screened for identifiable cardiac risk factors and excluded if they had a history of cardiovascular disease, current smoking, hypertension, diabetes (fasting glucose level ≥6.7 mmol), a fasting total cholesterol level ≥6.5 mmol, or use of any medications. All subjects had a normal 12-lead electrocardiogram, normal global and regional resting cardiac function on magnetic resonance (MR) imaging, and did not perform >3 sessions (defined as 30 min) of sweat-producing exercise per week.

Blood tests. Fasting blood tests for glucose, cholesterol, leptin, insulin, free fatty acids, and C-reactive protein were taken on the day of the scan. An estimate of insulin resistance was calculated using the homeostasis model assessment of insulin resistance (HOMA-IR) equation (fasting insulin [μU/ml] × fasting glucose [mmol/l] / 22.5) (8).

MR imaging. All MR imaging scans for the assessment of LV and right ventricular (RV) mass, volumes, ejection fraction, diastolic function, and aortic distensibility were performed on a 1.5-T MR system (Siemens, Erlangen, Germany). Images for ventricular volumes and diastolic function were acquired using a steady-state free precession sequence with an echo time of 1.5 ms, a repetition time of 3.0 ms, in plane resolution 1.5 × 1.5 mm^2, temporal resolution 47.84 ms, and a flip angle of 60°, as previously described (9). All imaging was cardiac gated and acquired during end-expiratory breath hold.

Indexes of aortic function were assessed using a retrogated steady-state free precession cine sequence with the following parameters: temporal resolution 42 ms, echo time 1.4 ms, field of view read 380 mm, in-plane resolution 1.97 mm, and slice thickness 7 mm. Aortic cine images were acquired in transverse planes at 3 levels as previously described (10); the crossing of the pulmonary arch through: 1) the ascending thoracic aorta; 2) the descending thoracic aorta; and 3) 12 cm below the right hemidiaphragm perpendicular to the orientation of the abdominal aorta. Brachial artery blood pressure was recorded during image acquisition to provide pulse pressure.

Abbreviations and Acronyms

BMI = body mass index
HOMA-IR = homeostasis model assessment of insulin resistance
LV = left ventricle/ventricular
MR = magnetic resonance
RV = right ventricle/ventricular

Visceral fat mass, body composition, and percentage excess weight. A single breath-hold, 5-slice, water-suppressed T1-weighted turbo spin echo sequence centered around L5 was acquired (11). Images were manually contoured for visceral fat volume. Percentage excess weight loss was calculated according to the formula: percent excess weight loss = (weight before − weight after [kg]/excess body weight before) where excess body weight = (total body weight − ideal body weight). Ideal body weight refers to the weight if BMI = 25 kg/m^2. Bioelectrical impedance was used to determine total body fat mass and body composition using the Bodystat 1500 analyzer (Bodystat, Douglas, Isle of Man).

Weight loss and continued obesity. Seventeen obese subjects underwent a supervised low glycemic index diet, and 13 underwent bariatric surgery (10 roux-en-Y gastric bypass, 3 laparoscopic adjustable gastric band). Follow-up was 390 ± 33 days. Neither group was enrolled in graded exercise programs. Seven additional obese subjects, initially enrolled in the low glycemic index diet, who were unsuccessful in losing weight, underwent repeat scanning after 1 year (378 ± 44 days) to determine the effects of continued obesity.

Data analysis. Image analysis for LV and RV volumes, mass, and diastolic function was performed using Siemens analytical software (ARGUS). The short-axis stack was analyzed manually, contouring endocardial and epicardial borders from base to apex at end diastole and end systole to determine LV and RV mass (g) and volumes, as described (9). Left ventricular mass was indexed to height, body surface area, and height$^{2.7}$ to enable more stringent allowance for obesity (12).

Manually contouring short-axis slices across the cardiac cycle, volume time curves, diastolic peak filling rate, and time to peak filling rate, both normalized to end-diastolic volume, were derived as described (13).

Aortic cross-sectional area in systole and diastole was calculated using semiautomated in-house software within Matlab version 6.5 (Mathworks, Natick, Massachusetts), which shows a highly reproducible assessment of distensibility (coefficient of variance 0.58%). Aortic distensibility (AD) was calculated according to the formula: AD = ([A_{max} − A_{min}]/A_{min})/(pulse pressure), where A_{max} = maximal systolic area (mm^2), and A_{min} = minimal diastolic area (mm^2). All images were analyzed blinded.