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Central aortic pulsatile hemodynamics in obese premenopausal women

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Abstract

Obesity is a known risk factor for cardiovascular disease (CVD) but the mechanism by which obesity contributes to cardiovascular risk is not well understood. Arterial stiffness is a CVD risk factor associated with obesity. We studied 16 obese body mass index (BMI > 30) and 10 lean (BMI < 25) healthy premenopausal women. We measured fasting glucose, insulin, and lipids, blood pressure, and arterial tonometry to assess arterial stiffness. Obese women had higher glucose, insulin, total cholesterol and triglyceride levels, blood pressures, cardiac output, and peak flow. Characteristic impedance was lower (146 ± 31 [(dyne · s) · cm⁻⁵] vs. 187 ± 48 [(dyne · s) · cm⁻⁵]; $P = .01$), aortic diameter was greater (2.54 ± 0.20 cm vs. 2.29 ± 0.21 cm; $P < .01$), and peripheral pulse pressure was similar in obese compared with lean women. Obesity in premenopausal women is associated with increased cardiac output and peak aortic flow. Increased aortic diameter in obese women was associated with reduced characteristic impedance, potentially preventing an increase in peripheral pulse pressure despite elevated flow, which suggests proximal aortic remodeling. When aortic remodeling and compensation for increased hemodynamic demands are limited by environmental or genetic interference, hypertension or CVD may result.

Keywords

Arterial stiffness; aortic compliance; tonometry; body mass index

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Dr. Gary F. Mitchell is the owner of Cardiovascular Engineering, Inc. This company designs and manufactures devices that measure vascular stiffness and these devices are used in clinical trials that evaluate the effects of diseases and interventions on vascular stiffness. He receives a salary as a result of this relationship.

Introduction

Obesity is a well-established risk factor for cardiovascular disease.¹ Previous studies have demonstrated that obese individuals have a 2 to 4-fold increased risk of developing coronary artery disease and stroke compared to non-obese patients.² This increased risk was originally believed to be the result of comorbidities linked to obesity, such as hypertension, dyslipidemia, insulin resistance, and diabetes. However, recent data suggest that at least part of the increased incidence of cardiovascular disease is secondary to obesity per se, independent of these other traditional risk factors.³

The mechanism by which obesity mediates cardiovascular disease is not well understood. Cardiovascular disease risk factors generally exert adverse effects on the structure or function of the arterial vessel wall, either directly or through effects on endothelial function.⁴ Arterial wall integrity can be assessed by evaluating abnormalities in pulsatile and steady-flow hemodynamics.⁵ One of the benefits of these noninvasive measurements is that derangements in vascular function can be detected at a preclinical stage, thereby providing the opportunity for early intervention and subsequent risk factor modification.

Although arterial stiffness has been examined in populations at increased risk for cardiovascular disease, including diabetes mellitus,⁶ hypertension,⁷ and postmenopausal women,⁸ there are limited data exploring the hemodynamic characteristics of, specifically, premenopausal women and their risk for cardiovascular disease. Prior studies have examined the association of obesity and arterial stiffness in populations that included women from adolescence to older age.^{9,10} Wildman and colleagues demonstrated that obesity was associated with increased aortic PWV before age 60, but the difference was attenuated in older individuals.¹⁰ In contrast, Zebekakis et al found the opposite relations wherein higher BMI was associated with increased pulse wave velocity (PWV) only in older women and was unrelated to BMI in men at any age.⁹ Thus, relations between obesity and arterial function remain unclear.

Subsequently, our aim was to characterize central hemodynamics and arterial stiffness in a population of obese premenopausal women. We employed arterial tonometry, a well-validated technique,^{11,12} as our method for assessing vascular stiffness and arterial compliance. We studied obese and lean premenopausal women to test the hypothesis that obese premenopausal women would exhibit abnormalities in vascular function, manifest as increased arterial stiffness that will not be evident among the lean women.

Methods

Women were screened at random from Brigham and Women's Hospital, and the local Boston community. Women were included in the study if they met the inclusion criteria that included age between 18 and 40 years, BMI < 25 (lean group) or BMI ≥ 30 (obese group), regular menstrual cycles, and good health, free of thyroid, cardiac, or renal disease. Women were excluded from the study if they were pregnant, lactating, or using oral contraceptive pills. The Human Research Committee of Brigham and Women's Hospital approved the study protocol and all participants provided written informed consent prior to participation.

All study procedures were performed at the Brigham and Women's Hospital. The participants were studied during the first 10 days of their menstrual cycle in the morning after an overnight fast of 8 to 10 hours. Anthropometric measurements, including height and weight, were performed and BMI was formally calculated. Blood was drawn for glucose, insulin, total cholesterol, triglyceride, high-density lipoprotein (HDL), and low-density lipoprotein (LDL).

Participants were then taken to the echocardiography suite for acquisition of the hemodynamic data. They were first placed in the supine position for 10 minutes of rest prior to study procedures. Supine auscultatory blood pressures were obtained by using a computer-controlled device (Cardiovascular Engineering, Inc, Waltham, MA) that automatically inflated the cuff (Hokanson SC12, D. E. Hokanson, Inc, Bellevue, WA) to a user preset maximum pressure and then precisely controlled deflation at 2 mm Hg/sec. The same large cuff (Hokanson SC12) was used to obtain the hemodynamic pressures in all of the study participants per standard protocol.¹¹ Blood pressures were obtained 3 to 5 times at 2-minute intervals in order to acquire three sequential readings that were within 5 mm Hg of each other for both systolic and diastolic measurements. Arterial tonometry and electrocardiogram (ECG) measurements were obtained from the brachial, radial, femoral, and carotid arteries using a custom transducer.

Each participant was then placed in the left lateral decubitus position in order to best capture echocardiographic parasternal long axis views of the left ventricular outflow tract (LVOT). Next, duplicate acquisitions of simultaneous tonometry of the carotid artery and pulsed Doppler of the LVOT from an apical 4-chamber view were recorded. The body surface distances from the suprasternal notch to the brachial (SSN-B), radial (SSN-R), femoral (SSN-F) and carotid (SSN-C) recording sites were measured with a tape measure. All data were digitized during the primary acquisition (ECG and tonometry pressures at 1000 Hz, audio at 12 kHz, and video at 30 frames/sec) then transferred to CD-ROM and sent to the Core Lab at Cardiovascular Engineering, Inc, for analysis. The aortic root diameter of each participant was measured twice, each by an investigator blinded to the participant group, and the average of the two measurements was used in analysis.

The hemodynamic data analyses were performed as previously described by Mitchell and colleagues.¹¹ Tonometry waveforms were signal-averaged using the ECG as the reference.¹³ The systolic and diastolic blood pressure averages were used to calibrate the peaks and troughs, respectively, of the signal-averaged brachial pressure waveform. Mean arterial pressure was calculated by digitally integrating the calibrated brachial pressure waveform. Diastolic blood pressures and mean brachial pressures were used to calibrate the carotid, radial, and femoral pressure tracings.¹⁴ PWV in the peripheral sites was calculated by determining the delay between the appearance of the pressure waveform foot in the carotid and brachial PWV, carotid and radial PWV, and carotid and femoral PWV sites.¹⁵ Cardiac output and cardiac index, calculated as cardiac output/weight, were determined in each participant from LVOT Doppler and cross-sectional area. Characteristic impedance (Z_c) was estimated in the time domain from the ratio of change in pressure to change in flow in early systole, prior to return of the reflected pressure wave.¹⁶ Amplification was calculated as the

ratio of brachial pulse pressure and central pulse pressure. Augmentation index (AI) was calculated as previously described.¹⁷

In order to maximize the accuracy of the data, several corrections and modifications were made to the measurements. Because of potential inaccuracy in tape measure determination of SSN-F distance in the obese group, we calculated the SSN-F distance in the lean women as a fraction of height and used this average fraction (0.30) to calculate the carotid femoral transit distance. We then used this average lean measure of carotid femoral transit distance to calculate carotid and femoral PWV for all subjects. The distance between recording sites was corrected for parallel transmission in the aorta and carotid by subtracting SSN-C from SSN-B, SSN-R, and SSN-F, then dividing by the respective foot to foot transmission delays. The central forward wave amplitude (P_f) was defined as the difference between pressure at the waveform foot and pressure at the first systolic inflection point or peak of the carotid pressure waveform. The distance between recording sites was corrected for parallel transmission in the aorta and carotid by subtracting SSN-C from SSN-B, SSN-R, and SSN-F, then dividing by the respective foot to foot transmission delays.

All blood samples were placed immediately on ice and centrifuged in a refrigerated (4°C) centrifuge at 2000 RPM for 15 minutes. The plasma was then frozen at -70°C until assays were performed. Serum glucose was measured by the glucose oxidase method (Beckman glucose analyzer, Fullerton, CA). Serum insulin was measured by Chemiluminescence assay (Beckman access Chemiluminescence-new protocol, Chaska, MN). Insulin resistance was evaluated by both the glucose to insulin (G/I) ratio and the homeostasis model assessment of insulin resistance (HOMA) index, calculated as $HOMA = (glucose [mg/dL] \times insulin [\mu U/mL]) / 405$.¹⁸ Total cholesterol and HDL were measured by cholesterol oxidase method, triglyceride by glycerol oxidation (Olympus, Irving, TX), and the level of LDL cholesterol was calculated according to Friedewald's formula ($LDL = [Total\ cholesterol - (triglyceride/5)] + DL$).

The non-hemodynamic data analyses were performed using Stata 8 statistical software (Stata Corp, College Station, TX). The data were tested for normal distribution using the Shapiro-Wilk test. Two-sample t-tests with equal variances were used to compare parameters between the obese and lean women. Correlations were measured using Pearson's correlation coefficient. Data are expressed as the mean $\pm$ standard deviation of the mean. A *P* value less than .05 was considered statistically significant.

Results

Baseline characteristics of the study sample are summarized in Table 1. Sixteen obese and 10 lean Caucasian women were studied. The two groups were age-matched and the BMI was significantly greater in the obese group, consistent with study design. Obese women had higher peripheral systolic and diastolic blood pressures ($P < .001$ for both) compared with the lean women although both groups were normotensive.

Metabolic variables are summarized in Table 2. Fasting glucose ($P < .002$) and insulin ($P < .01$) levels were significantly higher in obese participants compared with the lean

participants. The G/I ratio and HOMA index were both significantly greater in the obese compared with the lean group ($P = .02$ for both). In addition, for all the women, BMI positively correlated with fasting glucose ($r = 0.56$, $P < .003$), insulin ($r = 0.66$, $P = .001$), and HOMA ($r = 0.62$, $P < .001$), and negatively correlated with G/I ratio ($r = -0.55$, $P = .003$). Total cholesterol, triglyceride, and LDL levels were significantly higher in obese compared with lean participants. There was also a trend towards a lower HDL level in the obese compared with the lean participants.

Hemodynamic data are shown in Table 3. Central and peripheral pulse pressures were similar between the two groups despite the higher peripheral systolic and diastolic blood pressures among the obese women shown in Table 1. Obese participants had approximately 47% higher cardiac output and 35% higher peak flow compared with lean women. The carotid and brachial PWV, and carotid and radial PWV were similar between the two groups. The carotid and femoral transit delay did not differ between the obese and lean women (75.1 ± 13.8 msec vs. 75.2 ± 9.2 msec; $P = .98$). Carotid and femoral PWV did not differ between the two groups ($P = .53$). In addition, P_f did not differ between the two groups.

The Z_c was significantly lower in obese women ($P = .01$) when calculated as a function of volume flow. When calculated using flow velocity, there was no difference in the Z_c between the two groups (Table 3). Total arterial compliance was similar between the two groups. Left ventricular outflow tract diameter was similar, whereas aortic diameter was significantly larger in the obese women. Moreover, when examining the relationship between the metabolic parameters, fasting glucose, insulin, and HOMA, and the hemodynamic parameters PWV and Z_c , there were no significant correlations observed in either the lean or obese groups.

Additional analyses of the hemodynamic parameters were performed in order to evaluate for the potential impact of baseline differences between the two groups. We observed that augmentation index and amplification did not differ between the lean and obese, but heart rate was higher in obese women ($P = .01$). Because higher heart rate reduces AI and increases amplification,¹⁹ we adjusted for heart rate but still did not observe significant differences in either AI or amplification between the lean and obese groups. There were no differences between the two groups in Z_c , amplification, peripheral resistance, or AI after adjusting for weight.

Discussion

Our study extends the current literature on obesity and central vascular function by examining central aortic pressure-flow relationships as well as regional PWV in obese but otherwise healthy premenopausal women. We observed a significant decrease in Z_c in obese compared with lean women when examined by volume flow, but not velocity flow relationships. We observed an increase in heart rate, cardiac output, and mean arterial pressure in the obese women. Although systemic vascular resistance was lower in obese women, the reduction in resistance was not sufficient to offset the increase in cardiac output and prevent the observed increase in mean arterial pressure. We observed that both

peripheral and central pulse pressures were comparable between the obese and the lean women, even though peripheral blood pressures were elevated among the obese women. Additionally, the aortic root diameter was significantly larger in obese women and correlated with BMI, findings consistent with previous reports.^{9,10}

We propose that lower Z_c in obese women may be due to aortic remodeling necessary to maintain a normal pulse pressure in the setting of increased hemodynamic demands. That is, as a woman becomes obese, she experiences an increase in cardiac output and mean arterial pressure. Subsequently, in response to the increased cardiac output and increased shear stress in the proximal aorta, or as a consequence of increased mean arterial pressure, aortic diameter may increase. Aortic dilation reduces Z_c because of the strong dependence of Z_c on aortic diameter. Furthermore, when Z_c was normalized for body size by using flow velocity rather than volume flow, there was no difference between the groups. Similarly, the amplitude of P_f , which should be independent of body size because pressure is not dependent on size, was comparable between groups. This finding suggests that remodeling of the aorta to a larger diameter in the obese group, with no change in aortic wall stiffness, reduced volume-based Z_c and compensated for the increase in peak flow and cardiac output. Subsequently, the obese women were able to maintain pressure pulsatility at a level comparable to that found in the lean group.

Increased aortic diameter has been reported by Danias and colleagues in their investigation of obese males.²⁰ Although this study demonstrated that both the ascending and abdominal aortic maximal cross-sectional areas measured by magnetic resonance imaging (MRI) were higher in the obese compared with the lean age-matched controls, they failed to demonstrate any difference in arterial compliance, pressure-strain elastic modulus, or stiffness index in the ascending aorta between the two groups. In our study, we focused only on the ascending aorta and confirmed the findings of increased aortic diameter.

Our study did not demonstrate a difference in CF-PWV in our population of otherwise healthy obese women. Although a prior study showed an association between BMI and CF-PWV in middle-aged and older women,⁹ that study did not find an association between BMI and CF-PWV in the younger age range that would be comparable to our population. In contrast, Wildman et al found a moderate increase in CF-PWV in young obese individuals.¹⁰ One possible source of error involves use of a tape measure to assess the distance from the suprasternal notch to the femoral artery. This distance may be overestimated in obese individuals. We have avoided this problem by estimating the distance as a percentage of height. Further studies using a caliper that provides an unbiased estimate of the true linear distance should help resolve this issue.

We observed that fasting glucose, insulin, and HOMA were all higher in the obese women, consistent with the literature.²¹ Although we did not observe correlations between these metabolic parameters and the hemodynamic parameters, PWV and Z_c , this may have been due to the limited sample size in the groups. These relationships may be explored further in future investigations.

Besides sample size, there are other limitations to consider. One potential limitation could be the reproducibility of our study techniques. However, these techniques have been found to be highly reproducible in other studies of both lean and obese subjects.²² Additionally, although BMI does provide an indication of overall body mass, it is not a direct measure of visceral fat, which is more closely correlated with cardiovascular disease risk.²³ We were therefore unable to relate the vascular changes found in obese women specifically to visceral adiposity, which would require other measures using computed tomography or dual energy x-ray absorptiometry.

Additionally, because our primary aim in this study was to clarify the mechanisms underlying hemodynamic changes occurring in the central vasculature in obese premenopausal women, we did not provide extensive characterization of insulin resistance. Although the performance of clamp studies or even oral glucose tolerance testing would have provided more insight into the degree of glucose intolerance of these women, we did examine the G/I ratio and HOMA which have been shown to correlate well with insulin resistance.¹⁸

Conclusion

In conclusion, we observed that obesity in premenopausal women is associated with an increase in hemodynamic demand with substantial increase in cardiac output and peak flow. The increase in cardiac output was associated with an incomplete compensatory reduction in peripheral vascular resistance, leading to increased mean arterial pressure in obese women. We observed increased aortic diameter and reduced Z_c with higher BMI, suggesting remodeling of the proximal aorta, which did compensate for increased peak flow and maintained pulse pressure at a level comparable with that of lean women. The extent of central vasculature remodeling and the ability to compensate for the increase in hemodynamic demands in the central vasculature might be modulated by age and environmental or genetic factors. Further investigation is necessary to determine whether inadequate aortic diameter remodeling in response to hemodynamic stress imposed by obesity will lead to vascular abnormalities resulting in increased cardiovascular disease risk in older women or in other higher risk groups.

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Table 1

Baseline characteristics

	Obese (n = 16)*	Lean (n = 10)*	P
Age, y	31 ± 6	30 ± 5	.6
Weight, kg	104.3 ± 11.5	60.6 ± 4.7	<.0001
Height, cm	166.0 ± 3.5	166.5 ± 5.5	.8
BMI	37.8 ± 5.2	22.9 ± 1.8	<.0001
SBP, mm Hg	117 ± 8	104 ± 6	<.001
DBP, mm Hg	67 ± 8	57 ± 4	<.001

BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure.

* Data are given as mean ± SD.

Table 2

Metabolic variables

	Obese (n = 16)*	Lean (n = 10)*	P
Glucose, mg/dL	87 ± 7	74 ± 11	<.002
Insulin, μ U/mL	21.5 ± 9.9	11.5 ± 5.3	<.01
G/I ratio	5.1 ± 2.5	7.7 ± 3.3	.02
HOMA index	1.4 ± 1.2	0.4 ± 0.3	0.02
Total cholesterol, mg/dL	195 ± 29	165 ± 28	<.01
Triglycerides, mg/dL	138 ± 81	67 ± 25	<.01
HDL cholesterol, mg/dL	45 ± 10	52 ± 8	.08
LDL cholesterol, mg/dL	122 ± 20	98 ± 31	.02

G/I, glucose to insulin; HDL, high density lipoprotein; HOMA, homeostasis model assessment of insulin resistance; LDL, low density lipoprotein.

* Data are given as mean ± SD.

Table 3

Hemodynamic variables

Variable	Obese (n = 16)*	Lean (n = 10)*	P
MAP, mm Hg	87 ± 8	75 ± 4	<.001
PPP, mm Hg	50 ± 8	47 ± 6	.4
CPP, mm Hg	44 ± 7	39 ± 7	.1
Amplification, fold-change	1.15 ± 0.12	1.21 ± 0.16	.22
Heart rate, beats/min	70 ± 9	60 ± 9	.01
Cardiac output, mL/sec	84.3 ± 12.3	57.5 ± 13.1	<.001
Cardiac index, (mL/sec)/kg	0.82 ± 0.13	0.96 ± 0.25	.06
Peripheral resistance, (dyne · sec) · cm ⁻⁵	1406 ± 253	1812 ± 386	.003
Peak flow, mL/sec	337 ± 52	250 ± 28	<.001
Forward wave amplitude, mm Hg	37.8 ± 5.8	35.7 ± 7.4	.42
Characteristic impedance (dyne · sec) · cm ⁻⁵	146 ± 31	187 ± 48	.01
Characteristic impedance (dyne · sec) · cm ⁻³	729 ± 124	768 ± 237	.59
Augmentation index, %	13.0 ± 13.1	5.5 ± 8.8	.12
LVOT diameter, cm	2.01 ± 0.12	1.92 ± 0.12	.08
Aortic root diameter, cm	2.54 ± 0.20	2.29 ± 0.21	<.01
Carotid-brachial PWV, m/sec	6.3 ± 1.1	6.4 ± 1.3	.82
Carotid-radial PWV, m/sec	7.5 ± 1.0	8.7 ± 1.9	.06
Carotid-femoral PWV, m/sec	5.8 ± 1.0	5.6 ± 0.7	.53

CPP, central pulse pressure; LVOT, left ventricular outflow tract; MAP, mean arterial pressure; PPP, peripheral pulse pressure; PWV, pulse wave velocity.

* Data are given as mean ± SD.