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Association of Weight and Body Composition on Cardiac Structure and Function in the Atherosclerosis Risk in Communities (ARIC) Study

Natalie A. Bello, MD MPH[1,2], **Susan Cheng, MD MPH**[2], **Brian Claggett, PhD**[2], **Amil Shah, MD MPH**[2], **Chiadi E. Ndumele, MD, MHS**[3], **Gabriela Querejeta Roca, MD**[2], **Angela B.S. Santos, MD**[2], **Deepak Gupta, MD**[4], **Orly Vardeny, PhD**[5], **David Aguilar, MD**[6], **Aaron R. Folsom, MD, MPH**[7], **Kenneth R. Butler, PhD**[8], **Dalane W. Kitzman, MD**[9], **Josef Coresh, MD, PhD**[3], and **Scott D. Solomon, MD**[2]

[1] Division of Cardiology, Department of Medicine, Columbia University Medical Center, New York, NY

[2] Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA

[3] Department of Medicine, Johns Hopkins University, Baltimore, MD

[4] Vanderbilt Heart and Vascular Institute, Department of Medicine, Vanderbilt University Medical Center, Nashville, TN

[5] Pharmacy Practice Division, University of Wisconsin School of Pharmacy, Madison, WI

[6] Section of Cardiology, Department of Medicine, Baylor College of Medicine, Houston, TX

[7] Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN

[8] Department of Medicine, University of Mississippi Medical Center, Jackson, MS

[9] Cardiovascular Medicine Section, Department of Internal Medicine, Wake Forest School of Medicine, Winston-Salem, NC

Abstract

Background—Obesity increases cardiovascular risk. However, the extent to which various measures of body composition are associated with abnormalities in cardiac structure and function, independent of comorbidities commonly affecting obese individuals, is not clear. This study sought to examine the relationship of body mass index (BMI), waist circumference (WC), and percent body fat (BF) with conventional and advanced measures of cardiac structure and function.

Methods and Results—We studied 4343 participants of the Atherosclerosis Risk in Communities Study who were aged 69-82 years, free of coronary heart disease and heart failure,

Correspondence to: Scott D. Solomon, MD; Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115; Phone (857) 307-1960; Fax (857) 307-1944; ssolomon@rics.bwh.harvard.edu.

Disclosures

None.

and underwent comprehensive echocardiography. Increasing BMI, WC, and BF were associated with greater left ventricular (LV) mass and left atrial volume indexed to height$^{2.7}$ in both men and women (P<0.001). In women, all three measures were associated with abnormal LV geometry, and increasing WC and BF were associated with worse global longitudinal strain, a measure of left ventricular systolic function. In both sexes, increasing BMI was associated with greater right ventricular (RV) end-diastolic area and worse RV fractional area change (P≤0.001). We observed similar associations for both waist circumference and percent body fat.

Conclusions—In a large, biracial cohort of older adults free of clinically overt coronary heart disease or heart failure, obesity was associated with subclinical abnormalities in cardiac structure in both men and women and with adverse left ventricular remodeling and impaired left ventricular systolic function in women. These data highlight the association of obesity and subclinical abnormalities of cardiac structure and function, particularly in women.

Keywords

obesity; body mass index; echocardiography; remodeling; gender differences

The prevalence of obesity has grown over the past several decades with 68% of U.S. adults currently considered overweight or obese (1). While obesity has been associated with increased all-cause mortality, after adjustment for traditional cardiovascular (CV) risk factors, body mass index (BMI) and waist circumference (WC) remain only minimally associated with coronary heart disease and stroke (1,2,3) There are conflicting data as to whether greater BMI is associated with an increased risk of heart failure (4,5).

Although many of the mechanisms remain unexplained, a growing body of evidence demonstrates there are detrimental effects of obesity on cardiac structure and function (6,7,8,9). The majority of studies examining the effects of obesity on the heart used anthropomorphic measures such as BMI, which does not distinguish between fat and lean tissue and may misclassify individuals with excess fat mass as non-obese, especially in older adults in whom sarcopenia is common. Bioimpedence offers a means of directly measuring body composition and enables body mass to be classified as fat or lean, and may help to elucidate the true relationship between excess body fat and abnormalities of cardiac structure and function. Additionally, waist circumference more directly reflects abdominal obesity, which is associated with increased metabolic abnormalities and cardiovascular risk.

We analyzed variations in body composition (percent body fat) and traditional anthropomorphic measures (BMI and waist circumference) and their association with cardiac structure and function in an elderly, biracial cohort that was free from clinically overt coronary heart disease or heart failure.

Methods

Study Population

The Atherosclerosis Risk in Communities (ARIC) Study is a prospective cohort designed to investigate the causes of atherosclerosis and its related clinical outcomes in 15,792 men and women recruited from four U.S. communities (Forsyth County, NC; Jackson, MS; suburbs

of Minneapolis, MN; and, Washington County, MD) (10). Participants were aged 45 to 64 years at the time of the baseline examination (1987-1989) and were subsequently evaluated during four follow-up clinical visits (Visits 2, 3, 4 and 5) at approximately 3-year intervals, except for visit 5 (15 y interval). Annual telephone follow-up of participants has been conducted since the baseline visit. Institutional review boards from each site approved the study, and informed consent was obtained from all participants.

The present investigation is a cross-sectional analysis of 4343 ARIC participants during visit 5 (2011-2013). Of the 6538 participants at visit 5, we first restricted the sample to 5741 who had complete data for echocardiography, BMI, waist circumference, and percent body fat. We then excluded participants with prevalent coronary heart disease (n=853) or heart failure (n=480), race other than black or white (n=12), as well as those who were underweight at visit 5 (n=53), yielding a final n of 4343.

Ascertainment and Categorization of Body Composition

Standardized anthropomorphic measurements of weight, height, and waist circumference were obtained and bioelectric impedance (measured using the Tanita Body Composition Analyzer, TBF-300A) was utilized to calculate percent body fat, fat mass and lean body mass. Obesity was defined using three different criteria: 1. Body-mass index [defined as body weight (in kilograms) divided by height (in meters) squared] was categorized as normal (18.5 ≥ BMI <25 kg/m2), overweight (25 ≥ BMI <30 kg/m2), or obese (BMI ≥30 kg/m2). 2. Waist circumference (cm) with abdominal obesity categorized defined as waist circumference >102 in men or >88 in women. 3. Percent body fat was defined as obese, if >25% in men or 35% in women (11,12,13).

Echocardiographic Methods and Measurements

Echocardiograms were obtained from participants at all four sites at visit 5 on identically configured systems using a standardized protocol (14). Images were digitally transferred to the Cardiovascular Imaging Core Lab at Brigham and Women's Hospital, Boston, MA for offline analysis. The intra-observer variability (coefficient of variation and intra-class correlation) for key echocardiographic measures has been previously published (14).

Images were obtained in the parasternal long- and short-axis and apical 2- and 4-chamber views. Primary measures of left ventricular (LV) dimensions, volumes, and wall thickness; right ventricular (RV) area; left atrial (LA) dimension, volume, and area; and Doppler measures of mitral inflow, tricuspid regurgitation, and mitral annular relaxation velocities were made in triplicate from the 2D views in accordance with the recommendations of the American Society of Echocardiography (15).

Calculation of Derived Echocardiographic Variables

LV ejection fraction was calculated as 100*(LV end-diastolic volume- LV end-systolic volume)/ LV end-diastolic volume. LV mass was determined according to the ASE recommendations and indexed to height to the power of 2.7. Total LV wall thickness (TWT) was the sum of anteroseptal and posterior wall thickness (PWT), and relative wall thickness (RWT) was calculated as (2*PWT) / LV end-diastolic dimension. LV hypertrophy (LVH)

was defined as LV mass indexed to BSA >95 g/m^2 in women and >115 g/m^2 in men. Normal geometry was classified as RWT ≤ 0.42 and no LVH; Abnormal geometry was defined as the presence of either concentric remodeling (RWT >0.42 and no LVH), concentric hypertrophy (RWT <0.42 and LVH),or eccentric hypertrophy (RWT ≥ 0.42 and LVH). RV fractional area change (%) was calculated as 100*(RV end-diastolic area-RV end-systolic area)/RV end-diastolic area. LA volume was indexed to height to the power of 2.7. Abnormalities of diastolic function were assessed using a previously published approach in a community based cohort (16). Our population was classified using the following schema: normal diastolic function (deceleration time of E wave (DT) >140ms, 0.75<E/A<2 and E/E'<10); mild diastolic dysfunction (DT>140ms, E/A<0.75 and E/E'<10); moderate to severe diastolic dysfunction (0.75<E/A<2 and E/E'≥10; or DT <140ms, E/A>2 and E/E'≥10); and unclassifiable if participants did not fall into one of these three categories. Diastolic function was collapsed into normal diastolic function and abnormal diastolic function (mild or moderate to severe diastolic dysfunction) for all analyses.

Statistical Analysis

Data are presented as mean +/− SD, median [IQR], or N (%). Due to gender specific norms for many of the outcome variables as well as significant interactions between sex and the outcomes all analyses were performed stratified by sex. Interactions with race were tested for in all outcomes. Intergroup comparisons were compared using non-parametric trend, t-test, and χ^2 tests. Skewed variables were analyzed following log-transformation. Unadjusted and adjusted associations were assessed using linear and logistic regression. Restricted cubic spline models were used to assess all relationships for linearity. Chi-square tests were used to compare categorical variables. Potential confounders were identified *a priori* and used to correct all models. Covariates included in the regression models were age, race, heart rate, systolic blood pressure, anti-hypertensive use, diabetes, HbA1c, and current smoking. Bonferroni corrected P-values were used to determine statistical significant for all analyses. A corrected p-value of <0.005 was considered statistically significant for the primary echocardiographic measures reported. All reported p values are 2-sided. STATA v12.1 (College Station, TX) was used.

Results

Patient Characteristics

The characteristics of our study sample (n=4343) are shown in Table 1 and Supplemental Table 1. Compared to participants with a normal BMI, those who were overweight and obese were younger and more likely to be black. The prevalences of diabetes and hypertension were high and increased along with BMI and other measures of obesity. Participants with higher BMIs also had significantly larger waist circumferences as well as fat mass (p for trend <0.001). There was an inverse relationship observed between BMI and current smoking.

Left Ventricular Dimensions

In both women and men, greater anthropomorphic measures of obesity, BMI and WC, were associated with a significantly increased thickness of the left ventricular (LV) walls, larger

LV cavity size, and higher LV mass (**Table 2**). A similar association was seen when percent body fat (BF) was used as a measure of adiposity in women. There was a significant interaction with race in the relationship between left ventricular dimensions and BF in men that was not observed with BMI or WC; a positive correlation between BF and LV dimensions was not seen in black men, though in white men the findings were consistent with those in women (**Figure 1**).

Obese women but not men were significantly more likely to exhibit abnormal LV geometry when compared to participants with a BMI < 30 kg/m2 ($p<0.001$ women, $p=0.49$ men) (**Figure 2**).

Left Ventricular Systolic Function

The presence of obesity was not associated with LV systolic function as assessed by ejection fraction in either women or men. In women but not men, circumferential strain, a more sensitive measure of impaired systolic function, was highly associated with all three measures of obesity. Global longitudinal strain was also significantly associated with WC and BF in women but not men.

Diastolic Function and the Left Atrium

In women but not men, obesity was associated with significantly worse overall diastolic function (**Figure 3**). In both sexes, left atrial volumes increased significantly in the presence of increasing measures of obesity, even when scaled by height (**Table 2**).

Right Ventricular Structure and Function

The size of the right ventricle (RV) as assessed by the RV end diastolic area indexed to $height^{2.7}$ was associated positively with body mass index, and RV systolic function was negatively associated with BMI in both men and women ($p\leq0.001$). The relationship between RV size and function and measures of obesity remained significant across all measures of body composition in women ($p<0.001$). In men, the association between BF and RV size and function was significant in white but not black men.

Discussion

In a community-based, bi-racial population of older adults free of cardiovascular disease, greater metrics of body size and adiposity were associated with subclinical abnormalities in LV structure and function which differed somewhat by sex and race. In women, obesity was associated with a higher LV mass coupled with a disproportionately smaller increase in LV diameter, which resulted in concentric and eccentric hypertrophy with a concurrent decrement in strain and abnormalities of diastolic function. Men also exhibited greater total wall thickness and a positive association between LV diameter and mass in relation to greater BMI and WC, though these findings were not significantly accompanied by body size related impairments of LV function or a particular pattern of remodeling; the relationship with BF was modified by race and among men was found to be significant only in whites.

Similar to the changes that occur in hypertension or as a result of aortic stenosis, obesity is associated with cardiac remodeling. Previous studies using echocardiography have also demonstrated healthy obese men and women are more likely to have increased LV wall thickness and LV mass than their non-obese counterparts which may represent the earliest moment on the path to developing heart failure (8,9,17). There is emerging evidence from animal models that this change in wall thickness may be mediated by leptin, an adipocyte hormone that promotes collagen synthesis (18). Despite these changes in LV structure, greater BMI has not previously been associated with a significant decrement in systolic function as assessed by ejection fraction (8,19), or abnormalities of diastolic function after adjustment for clinical covariates (20).

Our results demonstrate that whether assessed via traditional anthropomorphic measures such as BMI and WC, or newer measures of body composition such as BF, obesity is associated with cardiac geometry and function differently in elderly men compared with elderly women. In particular, women are more likely to display primarily LV hypertrophy coupled with diastolic dysfunction and subclinical decrements in systolic function. Less negative circumferential strain has been shown to add incremental prognostic value for the development of incident heart failure (21) and may partially account for the female predominance of heart failure with preserved ejection fraction (HFpEF). By contrast, more obese men were not observed to exhibit similar patterns of remodeling or subclinical decrements in left ventricular systolic function. This preservation of biventricular systolic function could represent relative protection against the development of clinical heart failure with obesity and a type of ‘obesity paradox’ in men.

Both sexes and races demonstrated similar decrements in RV function and a positive association between RV size and greater body size. There are a paucity of data regarding the echocardiographic relationships between right ventricular size and obesity, though subclinical dysfunction has been described in association with increasing BMI. (7) To our knowledge these data are the first from a large, biracial contemporary cohort to examine RV morphology and function and its relation to obesity. We found that there is a significant decrement in RV systolic function coupled with RV enlargement in overweight and obese individuals that was not explained by comorbid hypertension or diabetes. These changes are probably multifactorial and may result from increased intravascular volume or sleep disordered breathing and warrant further study to elucidate the etiological mechanisms. In addition, as the prevalence of obesity continues to rise, the potential implications of RV dysfunction on long term survival will be increasingly important.

Several limitations of our study warrant consideration. First, this is an ambulatory elderly cohort and the findings may not be applicable to all obese individuals. Our study is cross-sectional and therefore hypothesis generating as to the causal relationship between obesity and HFpEF in women. There may also be a healthy survivor bias observed among the small black male cohort that resulted in their findings differing from the other participants. Additionally, there was a small number of participants who were excluded from the analysis due to missing data. These individuals were slightly older, more likely to be black, and slightly more hypertensive than those included. Multiple comparisons were made between three indices of obesity and echocardiographic measures. To minimize the possibility of in

increased risk of a type I error, we used the highly conservative Bonferroni adjustment to account for multiple testing which may have consequentially resulted in limited power. However, this allows us to focus on the most clinically significant differences. Although echocardiography has been well validated for the assessment of cardiac structure and function, MRI is considered the gold standard for the assessment of the RV. However, our results are consistent with those from a prior study that examined the effect of obesity on the RV using cardiac MRI (22).

In conclusion, we observed in a large community-based cohort of elderly individuals that subclinical abnormalities of cardiac structure function are associated with measures of obesity. Both sexes demonstrated similar increases in LV mass and biventricular size as well as decrements in RV function with obesity, although women manifested subclinical left ventricular systolic dysfunction while men did not. These sex-specific abnormalities may contribute to different patterns of obesity-related cardiovascular disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Clinical Perspective

The prevalence of obesity continues to rise in the US and around the world, and threatens to erase the gains made in the reduction of cardiovascular morbidity and mortality over the past several decades. Detrimental effects on cardiac structure and function are a potential mechanism by which obesity increases future risk of cardiovascular disease. The current study examined an elderly, biracial community-based cohort free from overt cardiovascular disease and found larger body size was associated with alterations in cardiac structure and function which varied by race and sex. Despite similar changes in left ventricular cavity size and mass in both sexes, only obese elderly women exhibited adverse remodeling, left ventricular diastolic dysfunction, and a subclinical decline in systolic function. However, abnormalities of right ventricular size and function associated with obesity were found in both sexes. Future studies are needed to determine whether this sex-specific variation in the cardiac effects of obesity contributes to different patterns of obesity-related cardiovascular disease in men and women, and whether weight loss can reverse these abnormalities.

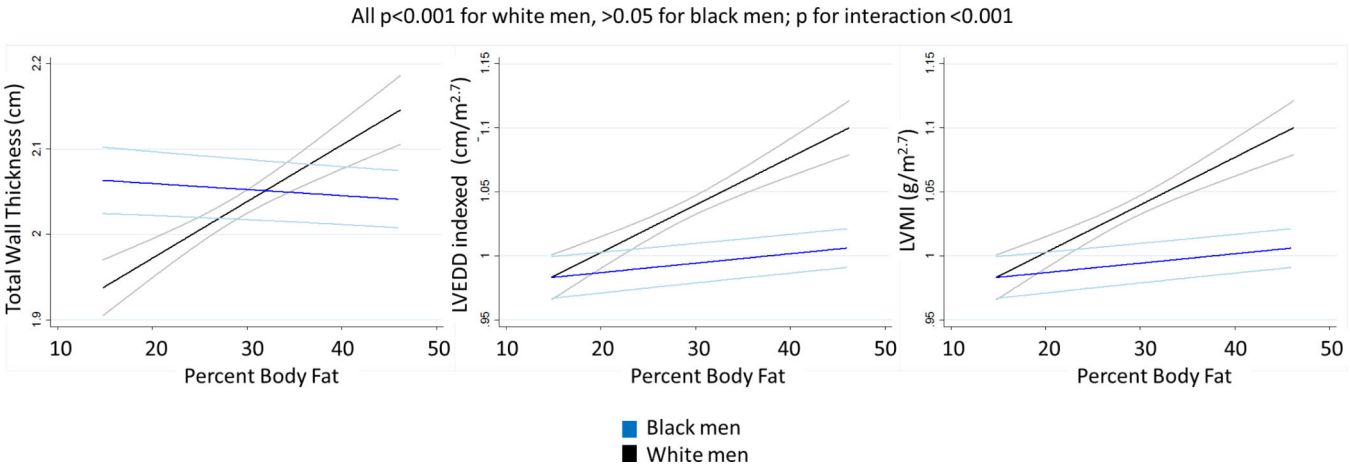

Figure 1.

Higher percent body fat is positively associated with increased LV wall thickness, higher LV mass, and a larger LVEDD in white but not black men.

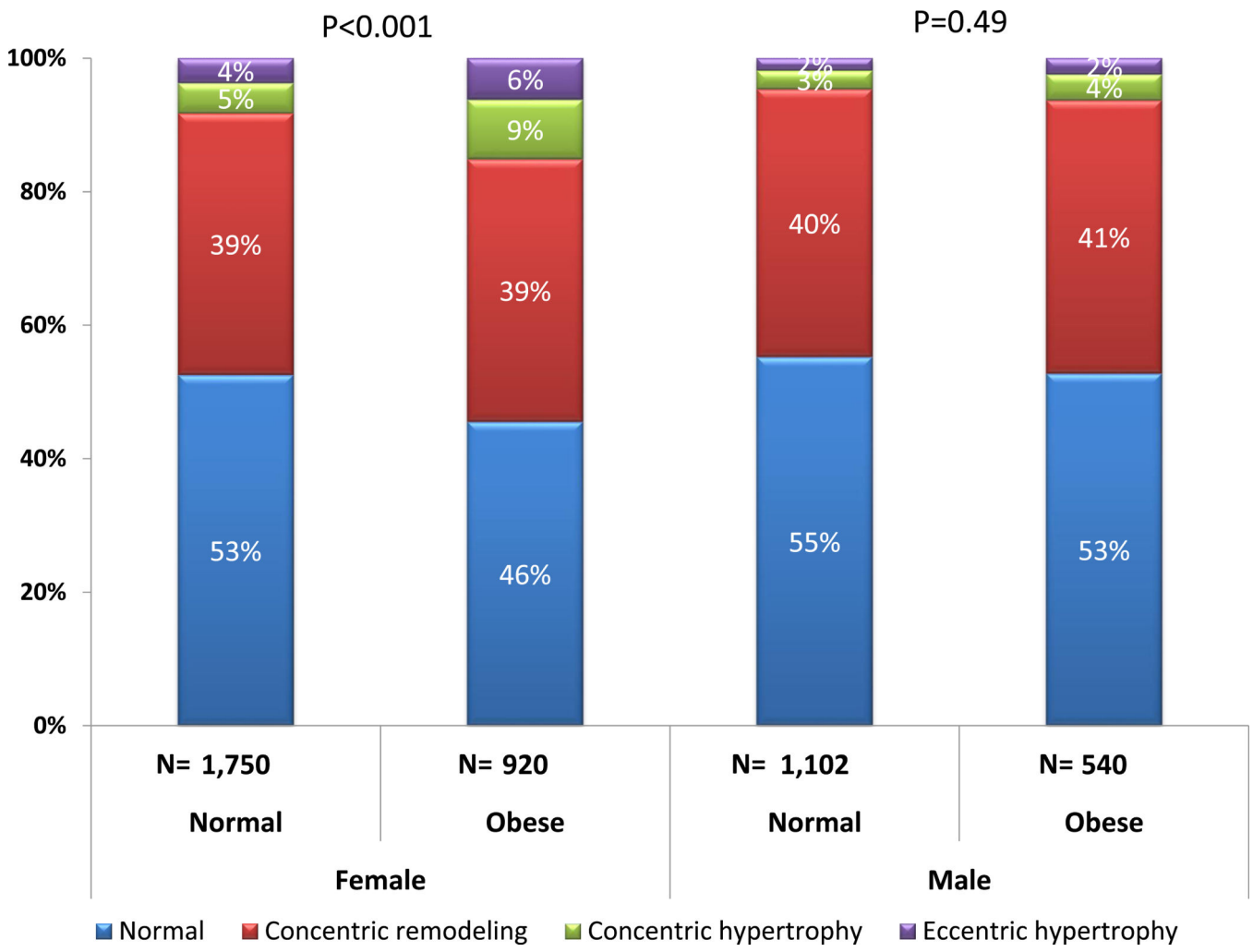

Figure 2.
Obesity is associated with abnormal LV geometry in women but not men.

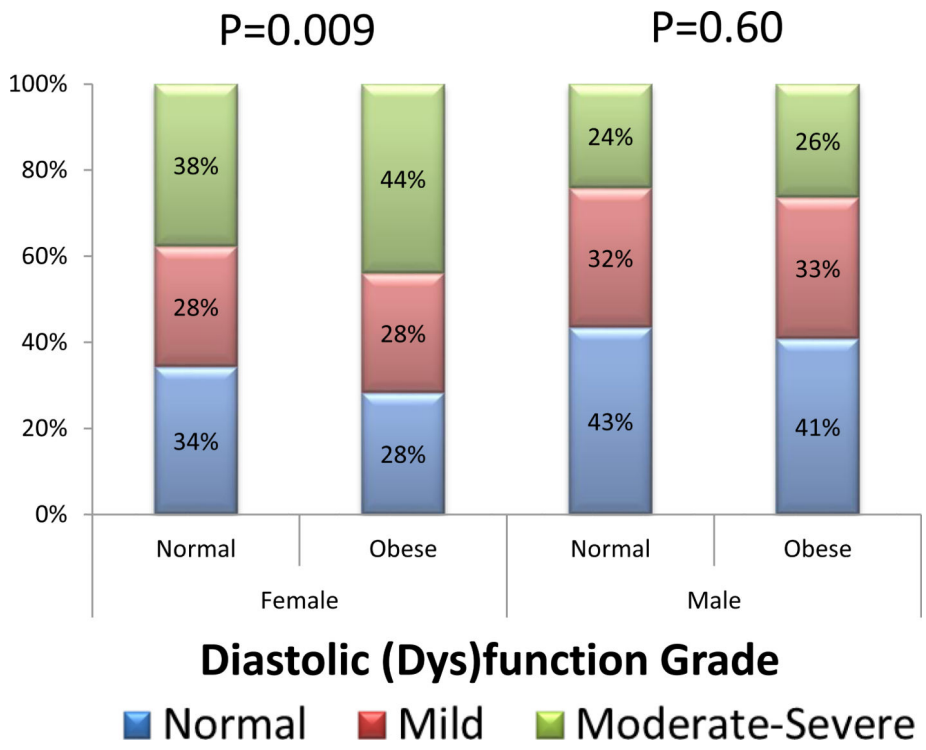

Figure 3.
Obesity is associated with worse diastolic grade in women but not men.

Table 1

Characteristics at Visit 5 Stratified by Body Mass Index Category

	Normal n=1137	Overweight n=1732	Obese n=1474
Clinical Characteristics			
Age (years)	76±5	76 ±5	75±5
Male	377 (33.2%)	732 (42.3%)	547 (37.1%)
Female	760 (66.8%)	1000 (57.7%)	927 (62.9%)
Black	160 (14.1%)	351 (20.3%)	413 (28.0%)
White	977 (85.9%)	1381 (79.7%)	1061 (72.0%)
Center			
Forsyth Co.	327 (28.8%)	392 (22.6%)	259 (17.6%)
Jackson	146 (12.8%)	314 (18.1%)	379 (25.7%)
Minneapolis	390 (34.3%)	563 (32.5%)	408 (27.7%)
Washington Co.	274 (24.1%)	463 (26.7%)	428 (29.0%)
Diabetes mellitus	214 (18.8%)	559 (32.3%)	692 (46.9%)
Hypertension	796 (70.0%)	1378 (79.6%)	1300 (88.2%)
Current Smoker	97 (8.7%)	85 (5.0%)	62 (4.3%)
Lipid lowering therapy	474 (41.7%)	1027 (53.9%)	889 (60.3%)
Heart rate (bpm)	62±10	62±10	64±11
SBP (mmHg)	130±18	131±17	130±17
DBP (mmHg)	65± 10	68±10	69±10
Body Composition			
Height (cm)	165±9	166±10	165±9
Weight (kg)	62±9	76±10	94±14
BMI (kg/m^2)	22.8 ±1.7	27.4±1.4	34.5±4.4
Body fat (%)	28.8±13.9	34.4±7.1	41.3±7.2
Waist circumference (cm)	88±8	99±8	113±11
Waist hip ratio	0.90±0.07	0.94±0.07	0.95±0.08
Laboratory Values			
HbA1c (%)	5.7±0.5	5.9±0.7	6.1±1.0
Total Chol (mg/dL)	194±38	187±42	180±40
LDL (mg/dL)	112±31	110±35	104±34
HDL (mg/dL)	60±15	52±13	49±11
Triglycerides (mg/dL)	108±48	127±62	137±71
hs-CRP (mg/L)	1.27 [0.65,2.56]	1.86 [0.92, 3.80]	2.95 [1.47, 5.62]
NT-proBNP (pg/mL)	141 [75, 243]	105 [56, 201]	101 [54, 196]
eGFR (ml/min/1.73m^2)	71.6±15.4	70.8±16.0	70.9±17.1

Values are shown as mean±SD, median [IQR], or N (%).

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Table 2

Associations between measures of obesity and cardiac structure and function by sex. Model 1 is adjusted for age and race; model 2 is adjusted for age, race, systolic blood pressure, anti-hypertensive use, diabetes, HbA1c, and current smoking. Strain measures were also adjusted for heart rate.

	Women											
				P values								
				BMI			Waist Circumference			Body Fat		
	Normal BMI	Overweight	Obese									
	n= 760	n= 1000	n= 927	unadj	model 1	model 2	unadj	model 1	model 2	unadj	model 1	model 2
Left Ventricular Dimensions												
IVS (cm)	0.94±0.12	1.00±0.14	1.06±0.15	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PWT (cm)	0.84±0.10	0.89±0.11	0.94±0.13	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
LVEDD index (cm/m$^{2.7}$)	1.15±0.14	1.20±0.15	1.24±0.16	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.19	<0.001	0.001
LV RWT (cm)	0.41±0.06	0.43±0.07	0.43±0.08	<0.001	<0.001	0.10	<0.001	<0.001	0.002	<0.001	<0.001	0.002
LV mass index (g/m$^{2.7}$)	31.78±7.18	36.50±7.84	41.78±10.12	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Left Ventricular Systolic Function												
LVEF (%)	66.87±5.17	66.92±5.10	66.67±5.62	0.04	0.17	0.19	0.38	0.36	0.35	0.05	0.23	0.14
Longitudinal strain (%)	−18.63±2.20	−18.37±2.28	−18.28±2.49	<0.001	0.001	0.36	<0.001	<0.001	0.01	<0.001	<0.001	0.04
Circumferential strain (%)	−28.74±3.50	−28.51±3.55	−27.93±3.80	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.02
Left Ventricular Diastolic Function												
LA volume (mL)	38.24±12.61	41.97±12.30	48.34±14.69	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
LA vol index (mL/m$^{2.7}$)	10.76±3.53	11.87±3.34	13.64±4.21	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Peak E (cm/s)	68.35±17.37	66.96±16.46	69.88±17.88	<0.001	<0.001	<0.001	0.01	0.004	<0.001	0.004	0.02	0.02
E/A	0.90±0.29	0.82±0.24	0.83±0.25	<0.001	<0.001	0.04	<0.001	<0.001	0.01	<0.001	<0.001	<0.001
e′ lateral (cm/s)	7.11±2.01	6.79±1.92	6.88±2.01	0.45	0.01	0.43	0.01	<0.001	0.08	0.007	<0.001	<0.001
E/e′ lateral	10.23±3.56	10.52±3.75	10.85±3.80	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.77	0.01	0.10
Right Ventricular Size and Function												
RV FAC (%)	0.55±0.08	0.54±0.07	0.53±0.08	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
RVEDA (cm^2)	16.38±3.76	17.18±3.62	19.00±4.24	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
RVEDA/Ht^2.7 (cm^2/m$^{2.7}$)	4.61±1.06	4.87±1.02	5.38±1.24	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Peak TR velocity (cm/s)	233.99±26.15	238.53±27.08	243.20±31.74	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.04	0.001	0.02

				Men								
				P values								
	Normal BMI	Overweight	Obese	BMI			Waist Circumference			Percent Body Fat		
	n=377	n=732	n=547	unadj	model 1	model 2	unadj	model 1	model 2	unadj	model 1	model 2
Left Ventricular Dimensions												
IVS (cm)	1.03±0.14	1.07±0.16	1.11±0.15	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.02	0.02	0.09
PWT (cm)	0.92±0.14	0.96±0.13	1.00±0.14	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.04	0.02	0.22
LVEDD index (cm/m$^{2.7}$)	0.99±0.13	1.02±0.13	1.06±0.13	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001
LV RWT (cm)	0.42±0.08	0.42±0.07	0.43±0.08	0.05	0.01	0.17	0.10	0.01	0.20	0.55	0.76	0.33
LV mass index (g/m$^{2.7}$)	32.33±7.89	36.22±9.12	40.62±9.61	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Left Ventricular Systolic Function												
LVEF (%)	64.71±6.02	64.66±5.92	64.38±5.56	0.34	0.32	0.54	0.15	0.06	0.13	0.54	0.49	0.71
Longitudinal strain (%)	−17.93±2.35	−17.79±2.40	−17.44±2.45	0.20	<0.001	0.07	<0.001	<0.001	0.04	0.35	0.48	0.85
Circumferential strain (%)	−27.20±3.97	−27.33±3.60	−27.07±3.77	<0.001	0.28	0.90	0.30	0.19	0.89	0.70	0.93	0.59
Left Ventricular Diastolic Function												
LA volume (mL)	47.16±14.93	52.47±21.80	57.10±17.81	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.54	0.32	0.41
LA vol index (mL/m$^{2.7}$)	10.46±3.22	11.70±4.96	12.79±3.98	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.25	0.07	0.11
Peak E (cm/s)	62.43±17.84	62.29±16.53	64.54±17.50	<0.001	<0.001	0.002	<0.001	0.001	<0.001	0.79	0.74	0.96
E/A	0.89±0.28	0.85±0.28	0.83±0.27	0.01	<0.001	0.004	0.02	<0.001	0.01	0.01	0.001	0.004
e′ lateral (cm/s)	7.51±2.10	7.33±2.17	7.09±2.07	0.05	0.001	0.01	0.02	0.001	0.01	0.36	0.09	0.14
E/e′ lateral	8.81±3.26	9.07±3.23	9.68±3.34	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.41	0.12	0.25
Right Ventricular Size and Function												
RV FAC (%)	0.52±0.08	0.51±0.07	0.50±0.08	<0.001	<0.001	0.001	0.001	0.001	0.003	0.15	0.17	0.17
RVEDA (cm^2)	21.86±5.46	22.08±4.90	23.42±5.21	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.99	0.86	0.75
RVEDA/Ht^2.7 (cm^2/m$^{2.7}$)	4.86±1.23	4.92±1.06	5.23±1.16	<0.001	<0.001	<0.001	<0.001	0.001	0.004	0.43	0.34	0.43
Peak TR velocity (cm/s)	231.82±24.51	234.70±25.32	239.30±28.12	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.96	0.84	0.78

Values shown are mean±SD.