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CONCENTRIC LEFT VENTRICULAR REMODELLING AND AORTIC STIFFNESS: A COMPARISON OF OBESITY AND HYPERTENSION

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Abstract

Background: Increased thoracic ascending aortic stiffness is thought to contribute to concentric left ventricular hypertrophy and increased mortality, a pattern seen in hypertension. As such, aortic stiffness and increased left ventricular mass are candidates by which obesity increases cardiovascular risk. However, obesity is characterized predominantly by increased abdominal aortic stiffness and with eccentric left ventricular hypertrophy.

Methods: We aimed to establish whether or not, in addition to these changes, there is also an element of concentric remodeling in obesity that was predicted by ascending aortic stiffness. 301 subjects underwent cardiovascular magnetic resonance imaging to measure regional aortic distensibility and left ventricular morphology. To compare obesity with hypertension, subjects were separated into groups by hypertensive status and body mass index.

Results: In comparison to normotensive subjects, hypertension was linked with concentric remodeling (a 17% increase in left ventricular mass: volume ratio (LVM:VR), (p<0.001) and reduced ascending aortic distensibility (by 64%,p<0.001). LVM:VR was negatively correlated with ascending aortic distensibility (R-0.36,p<0.01). Obesity, in the absence of hypertension, was associated with elevated left ventricular mass when compared to normal weight normotensive subjects (by 27%, p<0.01), in an eccentric pattern with cavity dilatation (p<0.01). However, LVM:VR was also 14% larger than in normal weight normotensive subjects (p<0.01), indicative of additional concentric remodeling. LVM:VR in obesity was, however, not correlated with ascending aortic distensibility when adjusted for mean arterial pressure (R=-0.14,p<0.14).

Conclusion: In summary, despite the predominantly eccentric pattern of left hypertrophy in obesity there is a concentric element of hypertrophy that, unlike in hypertension, is not linked to increased ascending aortic stiffness.

Introduction

Obesity *per se* is a well recognized risk factor for cardiovascular disease, (1, 2) exerting independent adverse effects on the cardiovascular system. (2, 3) Despite this well documented link, the mechanisms by which obesity modulates cardiovascular risk are not well understood. As obesity is associated with increased arterial stiffness and left ventricular hypertrophy, (4-8) both powerful predictors of mortality in multiple patient groups, (9) these have become candidate mechanisms to explain at least part of the increased mortality seen in obesity. In addition, weight loss in the setting of obesity has been shown to improve outcome and is associated with improvements in aortic stiffness and with reduced left ventricular mass. (10)

Aortic stiffness is generally thought to result in increased mortality via its haemodynamic effects. Mechanical fatigue and fragmentation of the elastin fibers is believed to result in dilatation of the proximal aorta and transfer of load to stiffer elements of the aortic wall, such as collagen. As a consequence, aortic wall stiffness and pulse wave velocity are increased, resulting in the premature arrival of reflected pressure waves in late systole rather than diastole, (11) which increases central pulse pressure and as a consequence systolic blood pressure. (12) Higher systolic blood pressures increase left ventricular afterload, increasing myocardial work and resulting in concentric left ventricular hypertrophy, increased oxygen demand and subendocardial ischaemia.(13,14) This then links aortic stiffness to increased mortality via concentric left ventricular hypertrophy.

Indeed, obesity is linked to a predominantly eccentric pattern of hypertrophy rather than the concentric pattern of hypertrophy which would be expected with increased proximal aortic stiffness. Furthermore, eccentric hypertrophy is less predictive of cardiovascular events than concentric remodeling. (15,16)

Hence, although attractive as potential mediators of cardiovascular risk, it is feasible that obesity related changes in aortic stiffness, being predominantly in the abdominal aorta, are not acting to increase pulse pressure and produce concentric left ventricular hypertrophy, but rather that obesity is linked, via increased blood volume, to an eccentric pattern of hypertrophy and independently produces distal aortic stiffness via obesity dependent processes which have no bearing on left ventricular mass.

We aimed to establish whether or not, in addition to the well-documented changes of eccentric hypertrophy and distal aortic stiffness in obesity, there was an element of concentric left ventricular remodeling that was predicted by increased ascending aortic stiffness.

In order to do this we used cardiovascular magnetic resonance imaging to examine the relationship between regional aortic distensibility and left ventricular morphology in a cohort of normotensive subjects across a wide range of body mass index measures and compared this to a cohort of hypertensive subjects where the interplay between aortic stiffness and concentric left ventricular hypertrophy is more established.

Methods

Ethics and Study Cohort

The study was approved by the local ethics committee, and informed written consent was obtained from each patient. In total, 301 subjects met criteria for inclusion into the study and were split in to two cohorts, normotensive and hypertensive, each with three subgroups according to body mass index.

Normotensive Subjects 172 healthy subjects (60% female, BMI range 18.5-44.8kg/m^2) were included into the normotensive subset of the study. All normotensive subjects were screened for the presence of identifiable cardiac risk factors and excluded if they had

a history of cardiovascular disease, definite hypertension, diabetes, current smoking (or a greater than 2 pack year history of smoking), or use of cardiac medications. All subjects were normotensive at the time of scanning (taken as an average of three supine measures over ten minutes under 140/90mmHg, (Model; MandauTM digital sphygmomanometer, P.M.S Instruments Ltd, United Kingdom). Subjects were excluded if they had either a diabetic range fasting glucose level ($\geq 6\cdot7$ mmol) or a fasting total cholesterol level $\geq 6\cdot5$ mmol, a history of coronary artery disease (CAD), or of cardiac chest pain or valvular heart disease. For analysis subjects were separated into three groups according to WHO body mass index category (normal weight normotensive, n = 79), overweight normotensive, (n = 42) and obese normotensive (n = 51)).

Hypertensive Subjects 129 subjects (female 60%, BMI range 19.9 – 48.5) were recruited to this study. Subjects were deemed hypertensive if a history of hypertension was present or hypertension was confirmed on 24hr blood pressure monitoring (Model, TM2420 PMS Instruments Ltd, Maidenhead, UK) or clinic blood pressure monitoring as above MandauTM digital sphygmomanometer [P.M.S (Instruments) Ltd, United Kingdom]. Subjects were excluded if they had a history of diabetes, hypercholesterolaemia, established cardiovascular disease, coronary disease or had a history of smoking, current smokers were also excluded. As with the normotensive cohorts subjects were separated into three groups according to body mass index (normal weight hypertensive BMI 18.5 (n = 31), overweight hypertensive (n = 51) and obese hypertensive (n=47)).

Aortic Imaging

All MR scans for the assessment of left ventricular morphology and aortic distensibility were performed on a 1.5 Tesla MR system (Siemens Healthcare, Erlangen, Germany) (17). Indices

of aortic function were assessed using an SSFP cine sequence with the following parameters: TR 42 ms, TE 1.4 ms, FOV read 380 mm, in plane resolution 1.97 mm, slice thickness 7 mm. Based on sagittal-oblique pilot images aligned with the aortic arch, aortic cine images were acquired in transverse planes at 3 levels: the crossing of the pulmonary arch through 1) the ascending thoracic aorta (Ao), 2) descending thoracic aorta (PDA) and 3)12 cm below this slice in the abdomen (DDA) as previously described (18). The abdominal cine images were piloted perpendicular to the orientation of the abdominal aorta. During the acquisition of the images, a brachial blood pressure was recorded (DINAMAP 1 846-SX, Critikon Corp) to provide the systolic (P_s) and diastolic (P_d) aortic pressures at the same time as the volume images were being acquired.

Left Ventricular Imaging

All imaging was prospectively cardiac gated with a precordial four lead ECG and acquired during end expiration breathold. Images were acquired using a steady state free precession (SSFP) sequence with an echo time (TE) of 1·5ms, a repetition time (TR) of 3·0ms, temporal resolution 47·84ms and a flip angle of 60° as previously described. (14-16) SSFP cine sequences were used to acquire localisation images followed by a SSFP left and right ventricular short axis stack of contiguous images with a slice thickness of 7mm and an interslice gap of 3mm.

Data Analysis

Left Ventricle

Image analysis for left ventricular volumes and mass was performed using Siemens analytical software (ARGUS©). The short axis stack was analysed manually contouring the endocardial borders from base to apex at end-diastole and end-systole. The epicardial

border was contoured at end-diastole to yield myocardial mass. Left ventricular mass (g) was calculated as the epicardial volume minus the endocardial volume multiplied by 1·05 (specific gravity of myocardium). The inter-observer and intra-observer coefficient of variation for left ventricular mass measures with this method is excellent, and has been previously reported. (19)

Aortic Imaging

Aortic cross sectional area in systole $_{(Amax)}$ and diastole $_{(Amin)}$ was calculated using an automated in house software program within Matlab 6.5©, and vascular distensibility was calculated as previously described. (20) Aortic distensibility (A_D) represents the relative change in area of the aorta per unit pressure, taken here as the pulse pressure and is calculated according to the formula: Aortic Distensibility = $(A_{max} - A_{min})/A_{min}/(P_{max} - P_{min})$, where A_{max} = maximal (systolic) area (mm^2), A_{min} = minimal (diastolic) area (mm^2), P_{max} = systolic blood pressure (mm Hg), and P_{min} = diastolic blood pressure (mmHg). The coefficient of variation of this automated method is 0.32% for intra-study repeat analysis and 2.18% for inter-study repeated acquisition and analysis.(21)

Statistical Analysis

All statistics were analysed using a commercial software package (SPSS 15; SPSS, Chicago, Ill). All results are presented as the mean ± standard deviation. All data were subjected to Kolmogorov-Smirnov tests to establish normal distribution of the data. Data groups were compared using a one way ANOVA technique with post hoc Bonferroni correction. Any differences were considered significant at $p < 0.05$. The associations between left ventricular mass: volume ratio and aortic distensibility were analysed initially

without any adjustments (crude model) and then with adjustments for potential confounders (adjusted models). Because left ventricular structure is known to be affected by gender, body mass index and blood pressure these variables were considered in the adjusted models.

Results

Anthropometric data for the study groups is shown in Table 1.

Blood Pressure

There was no significant difference between systolic or diastolic blood pressures between the normotensive subgroups ($p > 0.99$, Table 1). Pulse pressure was also similar between the normotensive groups ($p>0.99$). As expected, both systolic and diastolic blood pressure measurements were larger in the hypertensive groups than in the normotensive groups ($p<0.01$), but did not differ between the body mass index-separated subgroups. Pulse pressure followed a similar pattern, being greater in the hypertensive groups than in the normotensive groups but similar between hypertensive body mass index-separated subgroups (intergroup analyses; $p < 0.01$ and $p>0.99$ respectively). As expected, all hypertensive subgroups had higher systolic, diastolic and pulse pressure readings than their normal weight normotensive counterparts (Table 1).

Body Mass Index

As expected, both obese groups had larger body mass index values than overweight and normal weight groups ($p<0.001$). All overweight groups had larger body mass indices than normal weight groups ($p<0.001$). Normal weight, overweight and obese normotensives were well matched in body mass index to normal weight, overweight and obese hypertensives (Table 1).

Aortic Size

<u>Diastolic Aortic Measurements</u>

Overweight normotensive and obese normotensive subjects had larger diastolic aortic cross-sectional areas than normal weight normotensive subjects at all three levels of the aorta. (p<0.001 for all analyses). Overweight and obese normotensive subjects did not differ in diastolic aortic measurements at any level (p>0.99). Normal weight, overweight and obese hypertensive subjects all had larger diastolic aortic measurements than normal weight normotensive subjects (p<0.001, for all analyses) but diastolic aortic measurements between these hypertensive subgroups were similar (p>0.99 for all subgroup analysis, Table 1). Overweight and obese hypertensive patients were seen to have larger diastolic aortic areas than normotensive overweight and obese subjects (p<0.001, Table 1).

Regional Distensibility Measures

Although there was clearly a stepwise decrease in aortic distensibility with increasing body mass index at all three levels of the aorta measured, when analysed in body mass index-separated subgroups, the reduction in aortic distensibility between normal weight normotensive subjects and overweight normotensive subjects did not reach statistical significance (p >0.10 for all analyses). Obese normotensive subjects had lower aortic distensibility than normal weight normotensive subjects at all levels recorded, with the absolute reduction being largest in the abdominal aortic area (ascending aorta by 22%, proximal descending aorta by 20%, abdominal aorta by 27%, p<0.01). Compared to normal weight normotensive subjects, normal weight, overweight and obese hypertensive subjects had substantially reduced aortic distensibility at all three levels (Table 1). In contrast to the normotensive groups, the largest reductions in distensibility were in the ascending aorta

(normal weight hypertensive by 65 %, overweight hypertensive by 65 %, obese hypertensive by 61 %, p< 0.001, Table 1 & Figure 1). All hypertensive subgroups had similar but reduced aortic distensibility measurements (p>0.99 for all subgroup analyses). Overweight and obese hypertensives had lower aortic distensibility measures than their overweight and obese normotensive counterparts at all three levels measured (p<0.001). The effects of obesity and hypertension were seen to be additive in reducing aortic distensibility with aortic distensibility being not only significantly lower in obese normotensives than that recorded in normal weight normotensives but also significantly lower in obese hypertensives than obese normotensives. This pattern was seen across all three levels of the aorta (p<0.001 for all analyses, Table 1).

Left Ventricular Characteristics

Absolute Left Ventricular Mass

Left ventricular mass was larger in both overweight and obese normotensive subjects than normal weight normotensive counterparts (by 21 % and 27 % respectively, p<0.001). Left ventricular mass was also larger in overweight and obese hypertensives than normal weight normotensives (by 27 % and by 33 % respectively, p<0.001). Overweight and obese hypertensives had similar left ventricular mass measurements to their normotensive counterparts (p>0.99 and p= 0.83 respectively).

Left Ventricular Mass: Volume Ratio

Amongst normotensive subjects, left ventricular mass: volume ratio was greater in both overweight and obese than in those with normal weight (p=0.02 & p=0.003 respectively). All hypertensive subgroups had larger left ventricular mass: volume ratios than normal weight normotensives (p< 0.001). Importantly, obese hypertensives had larger left ventricular

mass: volume ratios than obese normotensives (by 15%, p=0.02 Table 1 & Figure 1),
suggesting that obesity is acting in an incremental way to increase concentric remodeling.

Aortic Distensibility and Left Ventricular Mass: Volume Ratio: Crude Associations

To assess the effects of hypertension on regional aortic distensibility, and reduce the impact
of body mass index, all normal weight subjects including all hypertensive subjects were
pooled into a 'hypertension subset' for analysis (n=120, female 60%, blood pressure
range SBP 90- 183mmHg, DBP 55-105mmHg). Pearson correlation revealed that ascending
aortic distensibility, proximal aortic distensibility and abdominal aortic distensibility were
negatively correlated with left ventricular mass volume ratio (Table 2).

To assess the effects of body mass index on left ventricular mass: volume ratio, and reduce
the effects of blood pressure, all normotensive subjects were pooled into a 'normotensive
subset' for this analysis including all levels of body mass index (n=172, 60% female, BMI
range 18.5- 44.8kg/m^2). Pearson correlation revealed that ascending aortic distensibility
and proximal descending aortic distensibility were negatively correlated to left ventricular
mass: volume ratio (Table 2). There was no correlation between abdominal aortic
distensibility and left ventricular mass volume ratio (p=0.10).

To assess the combined effects of obesity and hypertension all hypertensive patients across
all body mass index values were pooled into a 'combined subset' for this analysis (n=129,
female 58%, BMI range 19.9-48.5 kg/m^2, BP range SBP 115-211 mmHg, DPB 63 -1 13mmHg).
Pearson correlation revealed that in this subset there were no significant correlations between
ascending aortic distensibility, proximal aortic distensibility or abdominal aortic
distensibility and left ventricular mass volume: ratio (Table 2).

Aortic Distensibility and Left Ventricular Mass: Volume Ratio: Adjusted Associations

In the hypertensive subset, despite only including normal weight subjects, there was a significant positive association between body mass index and left ventricular mass: volume ratio (Table 2). When adjusted for gender, body mass index and mean arterial pressure the negative correlation between ascending aortic distensibility and left ventricular mass: volume ratio remained significant (R= -0.33, p=0.01). As prescription of beta-blockers and/ or ace-inhibitors (ACEi) potentially has effects on vascular elasticity, we performed analysis adjusting for this in addition to above. This again showed that, when adjusting for gender, body mass index, mean arterial pressure and beta-blocker/ACEi usage, the negative correlation between ascending aortic distensibility and left ventricular mass: volume ratio remained significant (R= -0.24, p=0.02). This suggests that in the hypertensive subset increased ascending aortic stiffness is acting to produce increased concentric hypertrophy independent of body mass index and blood pressure.

Despite only including normotensive subjects into the normotensive subset there was a significant positive correlation between all blood pressure measurements (systolic blood pressure, diastolic blood pressure, mean arterial pressure and pulse pressure) and left ventricular mass: volume ratio (Table 2). After adjusting for mean arterial pressure there was no significant association between ascending aortic distensibility and left ventricular mass: volume ratio in the normotensive subset (R= -0.14, p=0.14). The positive correlation between body mass index and left ventricular mass: volume ratio remained after adjusting for gender, mean arterial pressure and ascending aortic distensibility (R= 0.35, p=0.01). This suggests that obesity is indeed acting to produce an additional concentric element of hypertrophy, and that this process is independent of the effect of mean arterial blood pressure. Unlike the hypertensive subgroup, however, this pattern is not related to ascending

aortic stiffness.

Discussion

Given the global increase in obesity and its association with mortality, further understanding of the ways in which obesity acts to increase mortality is of great clinical importance. Candidate mechanisms to explain this are aortic stiffness and increased left ventricular mass, both of which are present in obesity and have been shown to be predictors of cardiovascular events in many patient groups. However, obesity is linked to a predominantly distal pattern of increased aortic stiffness primarily affecting the abdominal aorta, quite different to that seen in hypertension and other patient cohorts, and to predominantly eccentric hypertrophy rather than the concentric pattern which reportedly stronger links to mortality. (22) To date, no study has investigated whether ascending aortic stiffness is linked to a concentric remodeling process in obesity in a pattern similar to that reported in hypertension. We have analysed the relationship between regional aortic stiffness (as assessed by aortic distensibility) and left ventricular remodeling (as assessed by left ventricular mass: volume ratio) in a group of normotensive obese subjects and compared this to a group of normal weight hypertensive subjects.

We have shown, in contrast to hypertension, where increased ascending aortic stiffness is related to concentric remodeling independent of the effects of systolic blood pressure and body mass index, that despite the fact that additional concentric remodeling does occurs in obesity, independent of systolic blood pressure, this is not related to increased ascending aortic stiffness when the effects of systolic blood pressure were removed.

The most widely accepted mechanism by which aortic stiffness increases mortality involves

degradation of elastin fibers in the proximal aorta resulting in dilatation and resultant transfer of load to stiffer collagenous elements. The increased stiffness accelerates reflected pressure waves which consequently arrive in late systole rather than diastole, (11) increasing central pulse pressure and systolic blood pressure (12) resulting in concentric left ventricular hypertrophy, increased oxygen demand and subendocardial ischaemia. (13,14) In keeping with this, as expected, the hypertension subset had the greatest aortic dilatation and reduction in distensibility measures in the ascending aorta and, in addition to this, ascending aortic stiffness was seen to be negatively correlated with left ventricular mass: volume ratio, in agreement with the proposed mechanism outlined above.

In comparison to the hypertension subset where concentric remodeling predominated, the effect of increasing obesity in the normotensive subset was a more eccentric pattern of hypertrophy (as evidenced by significantly lower left ventricular mass: volume ratio). However, the left ventricular mass: volume ratios were still significantly elevated in the overweight and obese normotensive cohorts when compared to their normal weight normotensive counterparts confirming that, despite a predominantly eccentric pattern of hypertrophy, an element of concentric hypertrophy is also present. Elevated blood pressure appears not to be the cause of this concentric hypertrophy as values were well within the normal range, similar for all normotensive groups. In addition, body mass index remained positively correlated with left ventricular mass: volume ratio when adjusted for mean arterial pressure.

As with the hypertensive cohort, but to a lesser extent, the ascending aorta in the obese normotensive group was seen to be significantly dilated when compared to normal weight normotensive subjects, and was significantly stiffer, again to a lesser degree than in hypertension. Furthermore, ascending aortic distensibility was correlated negatively with left

ventricular mass: volume ratio on crude analysis. Although initially this would indicate that ascending aortic stiffness in obesity is related to increased left ventricular mass: volume ratios and thus to an additional element of concentric remodeling, when the effects of blood pressure were removed this relationship became non significant.

This suggests that obesity is acting to produce left ventricular hypertrophy via two distinct haemodynamic effects. Firstly, the excess fat mass associated with obesity is known to increase metabolic demand and, thus, both cardiac output and total blood volume are elevated. These circulatory changes are known to cause left ventricular geometric remodeling in the form of cavity dilatation (confirmed again in this study), which leads to a compensatory eccentric left ventricular hypertrophy in response to increased wall stress. (23,24) Secondly, this study has shown, in obesity, that in addition to the eccentric hypertrophy there is an element of concentric remodeling that is related to body mass index, independent of systolic blood pressure and not related to proximal aortic stiffness. An explanation for this may be that the inter-relationships between obesity and left ventricular mass are more complex than simple haemodynamic effects, and involve adipokine-mediated mechanisms. Increased visceral and subcutaneous adiposity is known to cause higher levels of serum leptin, a hallmark of human obesity, and hyperinsulinaemia, both of which have been linked to ventricular hypertrophy in humans and in animal models, and as such it is likely that there are multiple processes occurring in parallel to cause left ventricular mass increases in obesity. In addition, when obesity and hypertension coexist, we have shown that left ventricular mass volume ratio is greater than that recorded in hypertension alone suggesting an additive effect of the two pathologies in producing left ventricular hypertrophy.

Interestingly, this study has highlighted that the pattern of distensibility changes in obesity

differ from those in hypertension, with a predominantly distal pattern of stiffness seen in obesity and a predominantly proximal pattern seen in hypertension. The reasons for this are not known but aortic distensibility changes in the setting of obesity have been attributed to a number of factors that are not present in hypertension including; hyperleptinaemia (25), external physical compression from adipose tissue, (26) elevated circulatory inflammatory cytokines (27) and increased free fatty acid levels (28). In addition to this, obese individuals have an excess of abdominal visceral fat, which not only is a better predictor of cardiovascular and metabolic risk than total body fat alone but is also linked to altered vascular function. (29) Therefore, it is likely that obesity- related processes known to affect vascular function are affecting all sections of the aorta, but their effect is greatest on the areas which are least elastic, i.e. the abdominal sections.

Limitations

The duration of hypertension and obesity has not been recorded in this study and will have an impact on left ventricular and aortic measures. In addition a proportion of the hypertensive subjects were on blood pressure lowering medication (normal weight 36%, overweight 37%, obese 34%) which may again have had influences on left ventricular measures. However, the percentage of subjects taking blood pressure lowering medication was not different between hypertensive subgroups and therefore is unlikely to have biased one particular group.

Conclusion

This study has shown, in agreement with published data, that proximal aortic dilatation and increased proximal aortic stiffness is linked to concentric left ventricular hypertrophy in

hypertension, in line with the concept of aortic-ventricular coupling. We have also shown that despite the predominantly eccentric pattern of hypertrophy in obesity, there is a concentric element of hypertrophy that occurs independent of systolic blood pressure but is not linked to increased ascending aortic stiffness. As ascending aortic stiffness is thought to be one of the mechanisms by which cardiovascular risk is increased, via increased left ventricular concentric remodeling, it is unlikely that obesity is acting via similar mechanisms given the lack of relationship between ascending aortic stiffness and left ventricular mass: volume ratios reported in this study in obesity.

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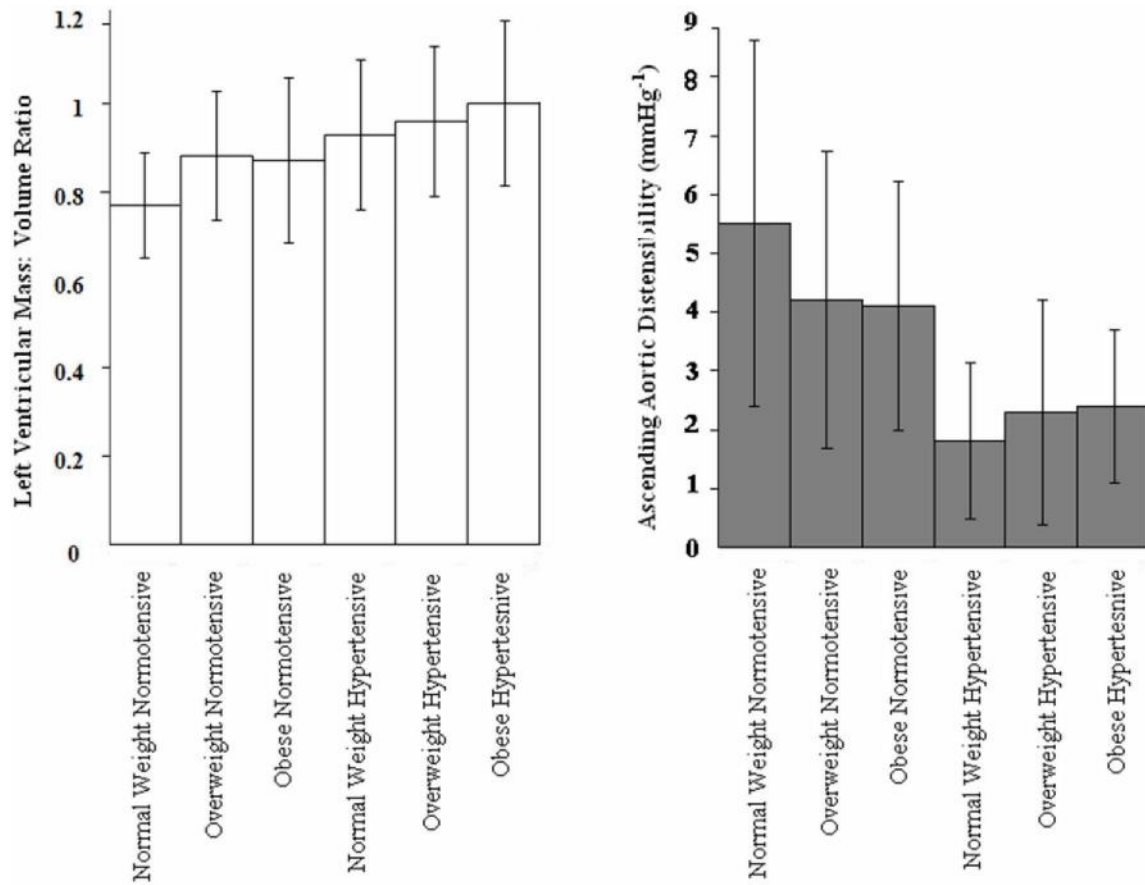
Figure

Figure 1. Left Ventricular: Mass Volume Ratio and Ascending Aortic Distensibility across the

Study Groups

Table 1. Study groups anthropometric, left ventricular and aortic parameters (statistical significances described in text)

	Normal Weight Normotensive N =79	Overweight Normotensive N =42	Obese Normotensive N=51	Normal Weight Hypertensive N=31	Overweight Hypertensive N=51	Obese Hypertensive N =47
Body Mass Index (kg/m²)	22 ± 2	27 ± 1	35 ± 4	23 ± 1	28 ± 1	34 ± 4
Systolic Blood Pressure(mmHg)	117±11	123±12	120±13	142±15	149±15	151±18
Diastolic Blood Pressure(mmHg)	73±8	77±9	76±9	83±9	85±9	87±11
Pulse Pressure (mmHg)	44 ± 8	45 ± 9	45 ± 10	61 ± 16	65 ± 15	64 ± 16
Diastolic Ascending Aortic Area (mm²)	489 ± 146	620 ± 148	598 ± 162	698 ± 148	732 ± 193	746 ± 219
Systolic Ascending Aortic Area (mm²)	595± 152	725± 155	700± 168	753± 138	805± 192	821±226
Diastolic Proximal Descending Aortic Area	295 ± 78	342 ± 90	336 ± 98	379 ± 77	396 ± 94	388 ± 104
Systolic Proximal Descending Aortic Area	356 ± 80	399 ± 90	396 ± 102	420 ± 79	446 ± 100	443 ± 120
Diastolic Abdominal Aortic Area (mm²)	220 ± 60	268 ± 75	259 ± 60	340 ± 99	324 ± 67	319 ± 73
Systolic Abdominal Aortic Area (mm²)	287 ± 65	330 ± 79	324 ± 67	394 ± 98	386 ± 83	372 ± 71
Ascending Aortic Distensibility (mmHg⁻¹)	5.5 ± 3.1	4.2 ± 2.5	4.1 ± 2.1	1.8 ± 1.3	2.0 ± 1.9	2.2 ± 1.3
Proximal Descending Aortic Distensibility	5.1 ± 2.1	4.5 ± 2.1	4.1 ± 1.7	2.3 ± 1.3	2.3 ± 1.2	2.4 ± 1.3
Abdominal Aortic Distensibility (mmHg⁻¹)	7.6 ± 3.1	6.3 ± 3.4	5.7 ± 2.4	3.2 ± 1.9	3.3 ± 1.8	3.3 ± 1.9
Left Ventricular Ejection Fraction (%)	68 ± 6	68 ± 6	70 ± 7	72 ± 7	71 ± 9	72 ± 6
Left Ventricular Mass(g)	101±26	120±28	125±32	114±23	132±36	138±41
Left Ventricular End Diastolic Volume (ml)	134 ± 31	137 ± 26	145 ± 23	124 ± 26	140 ± 45	138 ± 27
Left Ventricular Mass/Volume Ratio	0.77 ± 0.12	0.88 ± 0.15	0.87 ± 0.19	0.93 ± 0.17	0.96 ± 0.17	1.00 ± 0.19

Table 2. Correlations of Left Ventricular Mass: Volume Ratio across Different Groups

	LV Mass: Volume Ratio		
	Normotensives (all BMI) (n= 172)	Normal Weight (normotensive and hypertensive) (n=120)	Hypertensives (all BMI) (n=129)
Ascending Aortic Distensibility (mmHg-1)	-0.19*	-0.39*	-0.01
Proximal Descending Aortic Distensibility (mmHg-1)	-0.14	-0.41*	0.01
Abdominal Aortic Distensibility (mmHg-1)	-0.10	-0.37*	-0.01
Pulse Pressure (mmHg)	0.20*	0.32*	-0.11
Systolic Blood Pressure (mmHg)	0.35*	0.31*	-0.05
Diastolic Blood Pressure (mmHg)	0.27*	0.01	0.11
Mean Arterial Pressure (mmHg)	0.36*	0.23*	0.05
Body Mass Index (kg/m^2)	0.3*	0.27*	0.09

*= $p<0.05$