The Relationship Between Measures Of Obesity And Echocardiographic Determinants Of Left Ventricular Hypertrophy In Nigerian Adults
O IC, A OS, O OJ, U DI

Citation
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Abstract
Background
Obesity is becoming a global epidemic and the prevalence is increasing not only in the developed countries but also in developing countries like Nigeria. The main adverse consequences are cardiovascular diseases, type 2 diabetes and several cancers. Left ventricular hypertrophy (LVH) is a cardiovascular consequence of obesity and it is an independent risk factor for stroke, myocardial infarction and sudden death. It is therefore necessary to know to what extent obesity is responsible for the development of LVH and what measures of obesity are implicated. Method: A total number of two hundred and thirty (230) subjects were recruited for the study. One hundred and fifty (150) were obese while eighty (80) were non obese controls. Body mass index (BMI), waist circumference (WC) and waist-hip ratio (WHR) were measured and calculated as the case may be. Based on their BMI, the subjects were classified as obese and non obese. Blood pressure measurements on at least two occasions and fasting plasma glucose estimates were done to identify normotensive, normoglycemic subjects who were included in the study. Echocardiography was done on all subjects after physical examination. The Left Ventricular Mass was calculated using Devereux modified ASE cube formula and this was indexed to height \(^2\) and to body surface area. Results BMI and WC were strongly correlated with all echocardiographic parameters with BMI having the strongest correlation with LVM/H \(^2\) \((r=0.708, p<0.001)\) in males and \((r=0.799, p<0.001)\) in females. This was followed by WC \((r=0.678, p<0.001)\) in males and \((r=0.646, p<0.001)\) in females. WHR was weakly correlated with LVM/H \(^2\) \((r=0.213, P=0.040)\) in males and \((r=0.218, p=0.011)\) in females. Conclusion and Recommendation Body mass index (BMI) and waist circumference (WC) had stronger correlations with echocardiographic determinants of left ventricular hypertrophy compared to Waist hip ratio (WHR) in this study. BMI and WC should therefore be the preferred measures of obesity for the assessment of the impact of obesity on left ventricular mass and hypertrophy.

INTRODUCTION
Obesity is becoming a global epidemic and the prevalence is increasing both in the developing world and in numerous developing nations. This represents a situation that demands urgent attention if its potential morbidity and mortality are to be avoided.

Obesity represents a state of excess adipose tissue mass, and currently, overweight and obesity are classified by body mass index (BMI). BMI (weight in kilograms/height \(^2\) in meters) is frequently used as a surrogate measure of fatness in children and adults. In adults, overweight is defined as BMI of 25.0 to 29.9 kg/m\(^2\) while obesity is defined as BMI of ≥30 kg/m\(^2\). Through the use of BMI, the epidemic of obesity that began in the 1980s has been tracked through the end of the century. Data from the USA National Health and Nutritional Examination Survey (NHANES1999-2000) established that 30.5% of the United States population were obese. Rising trends in obesity was also noted in the Multinational Monitoring of Trends and Determinants in Cardiovascular disease (MONICA) project with 15% of men and 22% of women in Europe being reported as obese. Similar data are now being reported from many developing countries particularly those in Asia and, to a lesser extent, those in Africa.

Though data from Africa on this issue is scanty, a clear trend of profoundly increased BMI is noticed when people from Africa migrate to North Western hemispheric countries. Comparison of these indices among Nigerians and
Ghanaians residing in their native countries show these trends profoundly\(^4\). Studies in Nigeria have also demonstrated a rising trend in the prevalence of obesity\(^5,10\).

Obesity can be quantified through the use of BMI as mentioned above. However BMI does not take into consideration regional distribution of adiposity some of which may correlate with cardiovascular risk. Hence other measures of obesity such as waist circumference (WC) and waist to hip ratio (WHR) were introduced. Waist circumference greater than 102 cm in men and greater than 88 cm in women and waist to hip ratios greater than 0.9 in men and 0.85 in women are both used as measures of central/visceral/abdominal types of obesity\(^11\).

Obesity is associated with a host of co-morbidities that significantly increase the potential morbidity and mortality associated with the condition. The main complications of obesity are cardiovascular diseases, type 2 diabetes mellitus and several cancers. Left ventricular hypertrophy is a cardiac consequence of obesity. This is a condition in which the cardiac muscle of the left ventricle becomes enlarged in response to increased pressure or volume overload. In obesity, cardiac output is elevated due to increased blood volume causing a chronically elevated preload condition which in turn increases ventricle size, wall stress and left ventricular mass (LVM) leading to the development of eccentric ventricular hypertrophy\(^12\). Left ventricular hypertrophy is an independent risk factor for myocardial infarction, stroke, arrhythmias and sudden death.\(^13,14\)

The first studies that found an independent association between obesity and an increase in left ventricular mass in the 1960s\(^15\) were later confirmed by echocardiographic studies.\(^16\) Echocardiography (echo) provides visualization of structural and functional changes which appears long before the detection of overt clinical disease.\(^16\) In Africa, we have few studies on the relationship between obesity and the development of left ventricular hypertrophy as determined by echocardiography. The echocardiographic diagnosis of LVH in obese apparently healthy normotensives and institution of weight loss measures to regress LVH implies early diagnosis and early treatment. This would prevent life threatening complications of obesity and underscores the need for this study.

**MATERIALS AND METHODS**

A cross sectional survey of 150 obese and 80 control subjects drawn from healthy and eligible staff of the University of Port-Harcourt Teaching Hospital (UPTH) and patients’ relations was carried out between January 2008 and August 2008. UPTH is one of the tertiary hospitals in Nigeria. It is located in Port-Harcourt, Southern Nigeria and within the Niger delta sub-region. Informed written consent was obtained from all subjects prior to commencing the studies. Ethical approval was obtained from the UPTH ethical committee before commencing the studies. Anthropometric data including weight, height, waist circumference and hip circumference were obtained from all eligible subjects. The weight and height were obtained using a weighing scale and stadiometer and body mass index was calculated. The waist circumference was measured using a flexible tape at the level of the iliac crests\(^17\), and the hip circumference was measured at the level of the external margins of the anterior superior iliac spines\(^18\). Blood pressure was measured on at least two occasions and fasting blood sugar estimations were done to identify normotensive, normoglycemic subjects who were included in the study. Exclusion criteria included diabetic and hypertensive subjects, individuals with history of heart diseases including congestive cardiac failure, well trained athletes and individuals in the overweight category with BMI of ≥ 25.0 kg/m\(^2\) to 29.9 kg/m\(^2\).

Based on their BMI, the subjects were classified as obese and non-obese as follows: Subjects with BMI ≥ 30 kg/m\(^2\) were taken as obese and those with BMI between 18.5 kg/m\(^2\) and 24.9 kg/m\(^2\) were taken as non-obese controls. Abdominal obesity was identified as WC >102 for men and >88 cm for women; and also WHR > 0.9 for men and > 0.85 for women. Echocardiography was performed at rest using Aloka prosound SSD 4000 echo machine with a 2.5 MHz transducer. Physical examination was performed on all subjects before echocardiography. Two dimensional targeted M-mode measurements of the left ventricular end diastolic dimension, interventricular septal thickness in diastole, and posterior wall thickness in diastole were measured just beyond the tip of the mitral valves as recommended by the American Society of echocardiography (ASE)\(^19\). The left ventricular mass was calculated using the American Society of Echocardiography formula modified by Devereux\(^20\) also called the Devereux- modified ASE cube formula as follows:

\[
LV\ mass\ (g) = 0.8 (1.04 (IVSd + LVIDd + PWTd) - LVIDd^3) + 0.6
\]

Where IVSd = interventricular septal thickness in diastole.
LVIDd = left ventricular internal diameter in diastole
PWTd = posterior wall thickness in diastole.

The left ventricular mass was indexed to height $^2$ and also to body surface area (BSA). Left ventricular hypertrophy (increased LVMI) was defined as $LVM/H^2 > 77.7 \text{g/m}^2$ in males and $>69.8 \text{g/m}^2$ in females and $LVM/BSA > 134 \text{g/m}^2$ in males and $>110 \text{g/m}^2$ in females.

Statistical analysis was performed using the Statistical Packages for Social Sciences (SPSS) version 11 while statistical calculations were carried out using the computer software program for epidemiologists (PEPI) version 4.0.

The comparative analysis of the continuous variables was conducted using student-t test while the relationship between the measures of obesity and echocardiographic determinants of left ventricular hypertrophy was analysed using bivariate correlation analysis (Pearson’s Correlation Coefficient test). Values of $p < 0.05$ were considered statistically significant.

**RESULTS**

A total of two hundred and thirty (230) subjects were recruited for the study. One hundred and fifty (150) were obese consisting of 52 males and 98 females with mean age of 42.54 (± 8.60) years and range of 22-58 years. Eighty (80) non-obese controls were used consisting of 41 males and 39 females with mean age of 43.11 (± 8.58) years and age range of 22-58 years. There was no significant difference in the mean ages or height of the obese and non-obese subjects. However, the weight, BMI, BSA, WC, HC, WHR were all significantly higher in the obese subjects than control group ($p<0.05$) as shown in table 1.

The echocardiographic parameters namely the IVSd, LVIDd, PWTd and LVM were also significantly higher in the obese than the control group. The IVSd was noted to be thicker than the PWTd in the obese group, but in the control group, the IVSd and PWTd were about the same measurement. This is shown in table 2.
When the participants were pooled together and the measures of obesity correlated with echocardiographic parameters, strong correlations between BMI, WC was noted with all echo parameters namely IVSd, LVIDd, PWTd, LVM, LVM/H² and LVM/BSA. The strongest correlation was noted between BMI and LVM/H² (r=0.708, p<0.001) in males and (r=0.799, p<0.001) in females. This was followed by WC, (r=0.678, p<0.001) in males and (r=0.646, p<0.001) in females. WHR was weakly correlated with LVM/H² (r=0.213, p=0.040) in males and (r=0.218, p=0.011) in females. Correlation between BMI, WC and IVSd were stronger than those with the PWTd. These are shown in table 3.

When the participants were separated according to their BMI subgroups from normal to class III obesity, the mean LVMI using LVM/H² increased progressively from 54.00g/m² to 113.34g/m² in males and from 48.17g/m² to 112.25g/m² in females as shown in tables 4 and figure 1.

### Table 2 Echocardiographic features in obese and controls

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Obese (n = 150) Mean (SD)</th>
<th>Non-obese (n = 80) Mean (SD)</th>
<th>t-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD (mm)</td>
<td>37.56 (3.81)</td>
<td>33.27 (5.69)</td>
<td>6.73</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>IVSd (mm)</td>
<td>12.46 (3.00)</td>
<td>9.56 (2.47)</td>
<td>9.38</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>51.13 (7.12)</td>
<td>45.36 (3.87)</td>
<td>9.38</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>PWTd (mm)</td>
<td>11.07 (1.52)</td>
<td>9.41 (2.02)</td>
<td>7.01</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>RWT</td>
<td>0.46 (0.07)</td>
<td>0.42 (0.09)</td>
<td>3.73</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>IVS/PWTd</td>
<td>1.15 (1.32)</td>
<td>1.05 (0.29)</td>
<td>2.33</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>249.82 (69.68)</td>
<td>147.31 (51.35)</td>
<td>10.57</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVM/H² (g/m²)</td>
<td>90.66 (28.61)</td>
<td>51.18 (17.72)</td>
<td>11.08</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVM/BSA (g/m²)</td>
<td>118.96 (31.21)</td>
<td>83.46 (28.90)</td>
<td>8.43</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

IVSd = Interventricular septal thickness in diastole,
LVIDd = Left ventricular internal diameter in diastole,
PWTd = Posterior wall thickness in diastole,
LVM = Left ventricular mass,
LVM/H² = Left ventricular mass indexed to height²,
LVM/BSA = Left ventricular mass indexed to body surface area,
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**Figure 4**
Table 4 Relationship between degrees of BMI and LVM/H in participants

<table>
<thead>
<tr>
<th>BMI (kg/m²)</th>
<th>Male (n = 93)</th>
<th>Female (n = 137)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (18.50-24.99)</td>
<td>Mean (SD) [54.00 (21.28) [41]</td>
<td>Mean (SD) [48.17 (12.68) [39]</td>
</tr>
<tr>
<td>Obese Class I (30.00-34.99)</td>
<td>[84.54 (17.01) [29]</td>
<td>[67.91 (13.64) [44]</td>
</tr>
<tr>
<td>Obese Class II (35.00-39.99)</td>
<td>[95.75 (18.01) [21]</td>
<td>[102.21 (25.87) [38]</td>
</tr>
<tr>
<td>Obese Class III (≥40.00)</td>
<td>[111.34 (17.92) [2]</td>
<td>[112.25 (43.21) [16]</td>
</tr>
</tbody>
</table>

BMI = Body mass index,

\[LVM/H^2 = \text{Left ventricular mass indexed to height}^2\]

**Figure 5**
Figure 1 – Relationship between BMI sub groups and LVMI in participants

CI - Class I obesity = 30 - 34.9 kg/m²
CI - Class II obesity = 35 – 39.9 kg/m²
CIII - Class III obesity = ≥ 40 kg/m²

BMI - Body Mass Index

This is further depicted in the correlation coefficient in table 5 where class III obesity correlated strongly with LVM/H² (r=0.710, p<0.001) followed by class II obesity (r= 0.450, p<0.001). No correlation was noticed between normal BMI and echo parameters.

**Figure 6**
Table 5 Correlation of degrees of obesity in terms of BMI (kg/m) with echocardiographic parameters.

<table>
<thead>
<tr>
<th>Echocardiographic Feature</th>
<th>Normal (18.50-24.99) [n=90]</th>
<th>Obese Class I (30.00-34.99) [n=73]</th>
<th>Obese Class II (35.00-39.99) [n=89]</th>
<th>Obese Class III (≥40.06) [n=18]</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVSd (mm)</td>
<td>-0.081 (0.481)</td>
<td>-0.069 (0.562)</td>
<td>0.359 (0.005) **</td>
<td>-0.010 (0.969)</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>0.123 (0.285)</td>
<td>0.205 (0.082)</td>
<td>0.233 (0.053)</td>
<td>-0.164 (0.516)</td>
</tr>
<tr>
<td>PWTd (mm)</td>
<td>-0.99 (0.023)</td>
<td>-0.10 (0.074)</td>
<td>0.075 (0.570)</td>
<td>0.498 (0.055)</td>
</tr>
<tr>
<td>RWT</td>
<td>-0.142 (0.218)</td>
<td>0.275 (0.018)*</td>
<td>0.265 (0.119)</td>
<td>0.251 (0.296)</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>-0.003 (0.778)</td>
<td>0.011 (0.225)</td>
<td>0.394 (0.002) **</td>
<td>0.012 (0.965)</td>
</tr>
<tr>
<td>LVM/H² (g/m²)</td>
<td>-0.040 (0.729)</td>
<td>0.050 (0.620)</td>
<td>0.450 (0.004) **</td>
<td>0.710 (&lt;0.001) **</td>
</tr>
<tr>
<td>LVM/BSA (g/m²)</td>
<td>-0.112 (0.331)</td>
<td>0.034 (0.772)</td>
<td>0.391 (0.002) **</td>
<td>0.356 (0.148)</td>
</tr>
</tbody>
</table>

IVSd = Inter ventricular septal thickness in diastole,

LVIDd = Left ventricular internal diameter in diastole,

PWTd = Posterior wall thickness in diastole

RWT = Relative wall thickness,

LVM = Left ventricular mass

LVM/H² = Left ventricular mass indexed to height²

LVM/BSA = Left ventricular mass indexed to body surface area,

Waist circumference of >102 cm in men and >88 cm in women and waist to hip ratio >0.9 in men and >0.85 in women are classified as abdominal obesity. Abdominal obesity as measured by WC strongly correlated with echo parameters in females but not in males. There was no correlation between abdominal obesity as measured by
WHR and echo parameters. These are shown in tables 6 and 7.

**Figure 7**
Table 6 Correlation of degrees of WC (cm) with echocardiographic parameters.

**Figure 8**
Table 7 Correlation of degrees of WHR with echocardiographic parameters.

**DISCUSSION**
Considering the rising global epidemic of obesity,\(^1\)\(^,\)\(^2\) it is likely that adverse health consequences of excess adiposity will escalate in the near future. Left ventricular hypertrophy is one of the cardiac complications of obesity and echocardiographically determined LVH is a powerful independent predictor of cardiovascular morbidity and mortality.\(^14\)

The present study has demonstrated that obesity is associated with increased left ventricular wall thickness (IVSd and PWTd), increased left ventricular internal diameter (LVIDd), increased left ventricular mass and hypertrophy (LVM, LVH). From the study, the mean IVSd and PWTd were similar in the non obese group in table 2 (9.56mm and 9.41mm) respectively as demonstrated in other studies\(^21\) but in the obese group the IVSd is thicker than the PWTd (12.46mm and 11.07mm) respectively. This suggests that obesity targets the interventricular septum more than the posterior wall. This is further supported in table 3 where correlations between the measures of obesity and IVSd were stronger than those with posterior wall (PWTd).
The mechanism of cardiac hypertrophy in obesity involves both haemodynamic and metabolic factors. Obesity produces an increase in total blood volume and cardiac output caused by the increased metabolic demand induced by excess body weight. This leads to ventricular chamber dilation, increase in wall stress, myocardial mass and left ventricular hypertrophy.

The relative impacts of the measures of obesity namely BMI, WC and WHR on echocardiographic determinants of left ventricular hypertrophy showed that BMI and WC strongly correlated with all the echo parameters namely IVSd, LVIDd, PWTd, LVM and LVMI when the subjects were pooled together. Weaker correlation was noticed with WHR. When the subjects were separated according to BMI subgroups, strong correlations with LVM/H were noticed with class II and class III obesity (table 5). Progressive increases in BMI were also associated with progressive increases in the LVMI (figure 1). This was more prominent in females probably because females were more in number in the obese group and also because females have a higher percentage of body fat than males at similar BMI.

For abdominal obesity, strong correlation with echo parameters was noticed in females with abdominal obesity as measured by waist circumference but not in males (table 6). There was no correlation between abdominal obesity as measured by WHR and echo parameters (table 7). The waist circumference (WC) should therefore be the preferred measure of obesity for assessing the impact of abdominal obesity on echocardiographic parameters.

The findings of this study agree with that of the Framingham heart study where body mass index was found to be strongly correlated with left ventricular mass, left ventricular wall thickness and left ventricular internal dimension after adjusting for blood pressure and age. The Framingham study showed that body mass index was associated with prevalence of echocardiographic left ventricular hypertrophy particularly in subjects with BMI exceeding 30kg/m².

Similar findings were also noted by Sukmoko et al. who found significant correlation between BMI, WC and LVM but differs slightly from those of Avignon et. al. who found correlations between BMI, WHR and LVM. One feature common in all the above is the BMI which showed strong correlation with LVM and even stronger correlations with LVM/H² in males and females (r=0.708, p<0.001 and r=0.799, p<0.001) respectively.

Pathologic hypertrophy as against physiologic hypertrophy is an independent risk factor for cardiovascular events such as stroke, heart failures, arrhythmias and sudden death. Alterations in cardiac structure may be present even without any clinical signs of heart disease. It is apparent from this study that a variety of adaptations and alterations in cardiac structure occur as excessive adipose tissue accumulate even in the absence of systemic hypertension or underlying organic heart disease. To meet increased metabolic needs in obesity, circulating blood volume, plasma volume and cardiac output all increase. The increase in blood volume in turn increases venous return to the right and left ventricles, eventually producing dilatation of these cavities, increasing wall tension. This leads to LVH, which is accompanied by a decrease in diastolic chamber compliance, eventually resulting in an increase in left ventricular filling pressure and progressive ventricular enlargement. As long as LVH adapts to LV chamber enlargement, systolic function is preserved. When LVH fails to keep pace with progressive left chamber dilation, wall tension increases more and systolic dysfunction ensues. This then leads to heart failure, increased risk of arrhythmias and sudden death.

Weight reduction regresses left ventricular hypertrophy and this occurs regardless of whether the subjects have normal or high blood pressure. There is strong evidence that weight loss in overweight or obese individuals reduces the risk for diabetes and other cardiovascular diseases. Weight loss can be mediated through diet, exercise, drugs or bariatric surgery. It is usually generally advised that people with BMI ≥ 30kg/m² should be counseled on diet, exercise and other relevant behavioral interventions and set a realistic goal for weight loss. If these goals are not achieved, pharmacotherapy can be offered. Drug therapy may consist of drugs like sibutramine and orlistat.

In those with BMI > 40kg/m² who fail to achieve their weight loss goals (with or without medications) and who develop obesity complications, referral for bariatric surgery is indicated.

In conclusion, obesity is an independent predictor of left ventricular hypertrophy. BMI and WC had stronger correlations than WHR with echo parameters in this study. BMI and WC should therefore be the preferred measures of obesity for the assessment of the impact of obesity on left.
ventricular mass and hypertrophy. Weight loss can regress LVH and weight loss measures should be instituted early enough to regress LVH and prevent the complications of obesity.

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**References**

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Author Information

Okpara IC
Department of Internal Medicine, Benue State University

Adediran OS
Department of Internal Medicine, Benue State University

Odia OJ
Department of Internal Medicine, University of Port-Harcourt Teaching Hospital

Uchenna DI
Department of Internal Medicine, University of Port-Harcourt Teaching Hospital