

# Left Ventricular Geometric Patterns In Obese Nigerian Adults: An Echocardiographic Study.

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## Abstract

Background Obesity has recorded a global rise in its prevalence in the past decade and now constitutes a world wide epidemic. Left ventricular hypertrophy (LVH), and abnormal left ventricular geometry have also been associated with obesity. Prospective studies have shown that left ventricular geometric patterns have prognostic implications with the worst prognosis associated with concentric hypertrophy followed by eccentric hypertrophy and concentric remodeling. It is therefore necessary to know what pattern of hypertrophy is associated with obesity and the relationship between degrees of obesity and the development of left ventricular hypertrophy and abnormal geometry in Nigerians. Method: A total 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. Anthropometric data were obtained from all subjects and body mass index (BMI) was calculated. Based on BMI, the subjects were classified as obese and non obese. 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. Echocardiography was done on all subjects after physical examination. Left ventricular mass was calculated using the Devereux modified ASE cube formula and this was indexed to height.<sup>2</sup> The relative wall thickness and the presence or absence of echocardiographic LVH was used to identify the geometric patterns. Results- The Left ventricular geometric patterns in the obese group were as follows: - 16.7% had normal geometry, 12.6% had concentric remodeling, 46.0% had eccentric hypertrophy and 24.7% had concentric hypertrophy. In the control group, 71.3% had normal geometry, 22.5% had concentric remodeling, 3.7% had eccentric hypertrophy and 2.5% had concentric hypertrophy. In the BMI subgroups, the prevalence of abnormal geometry worsened progressively with the degree of obesity such that in class III obesity, 100% had abnormal geometry and none had normal geometry. Conclusion Obesity is associated with eccentric left ventricular hypertrophy and the risk of its development worsens with the degree of obesity.

## INTRODUCTION

There is a rising global epidemic of obesity<sup>1,2</sup> in the past decade. This epidemic began in the 1980s and has been tracked through the end of the century.<sup>3,4</sup> In this view, it is expected that adverse health consequences of obesity will escalate in the near future world wide.

Obesity is a disease in which excess body fat has accumulated to such an extent that health may be negatively affected.<sup>5</sup> Although often viewed as equivalent to increased body weight, this should not be the case. Obesity is more effectively defined by assessing its linkage to morbidity and mortality.

Obesity can be quantified through several classifications but the most widely accepted classification of obesity is the World Health Organization (WHO) criteria based on BMI<sup>2</sup>. The WHO accepts a BMI of 18.5 to 24.5kg/m<sup>2</sup> as normal,

25.0 to 29.9kg/m<sup>2</sup> as overweight and obesity is defined by a BMI of 30.0kg/m<sup>2</sup> or more. Further sub classification of the BMI in obesity has been documented and a BMI of > 40kg/m<sup>2</sup> is referred to as class III obesity or morbid obesity<sup>2</sup>.

Cardiovascular diseases constitute one of the major adverse consequences of obesity out of which left ventricular hypertrophy (LVH) is an independent risk factor for stroke, myocardial infarction and sudden death.<sup>6</sup> The presence of obesity further worsens the risk of developing these life threatening conditions.

The left ventricle (LV) hypertrophies in a variety of geometric patterns. In some cases the left ventricle dilates with the hypertrophy while in other cases it does not. In obesity, cardiac output is elevated due to increased blood volume causing a chronically elevated preload condition. This in turn increases ventricle size with dilatation, increase

in wall stress and left ventricular mass (LVM) leading to the development of eccentric ventricular hypertrophy.<sup>7</sup> This pattern of eccentric hypertrophy is said to be the commonest type of geometric pattern in the obese.<sup>8</sup>

The different geometric patterns are classified based on the left ventricular mass index (LVMI) and relative wall thickness (RWT). The left ventricular mass index is the left ventricular mass in grams derived from echocardiographic (echo) measurements which is indexed to height, height<sup>2</sup>, height<sup>2.7</sup> or body surface area. Relative wall thickness is the ratio of wall thickness to cavity diameter. Based on the LVMI and RWT, four geometric patterns are identified as follows:

1. (a) Normal geometric pattern - Normal LVMI and normal RWT
2. (b) Concentric remodeling - Normal LVMI and increased RWT
3. (c) Eccentric hypertrophy - Increased LVMI and normal RWT
4. (d) Concentric hypertrophy - Increased LVMI and increased RWT

Prospective studies have shown that LV geometric forms have prognostic implications with the worst prognosis associated with concentric hypertrophy followed by eccentric hypertrophy and concentric remodeling.

Obesity is known to be associated with abnormal left ventricular geometry<sup>8,10</sup> but there is paucity of studies on the pattern of LV geometry in the obese population in our African environment. It is the need to find out what pattern of left ventricular geometry predominates in the obese population in our environment and why this is so, that informed the need for this study.

### MATERIALS AND METHODS

150 obese subjects and 80 non obese were selected from healthy and eligible staff of the University of Port Harcourt Teaching Hospital (UPTH) and patients' relations in a cross sectional survey carried out from January 2008 to August 2008. The UPTH is one of the tertiary hospitals in Nigeria. It is located in Port Harcourt, Southern Nigeria and with in the Niger delta sub-region. Informed consent in written form was obtained from all eligible subjects before they were used for the study. Ethical approval was obtained from the ethical

committee of the UPTH before commencing the studies.

Anthropometric data including weight and height were obtained from all subjects. The weight and height were obtained using a weighing scale and stadiometer and body mass index was calculated. Blood pressure was measured on at least two occasions and fasting blood sugar estimations were done to identify normotensive, normoglycemic subjects aged between 18-65 years who were included in the study. Exclusion criteria included diabetic and hypertensive subjects, individuals with a history of heart disease including congestive cardiac failure, individuals with other causes of left ventricular hypertrophy and those in the overweight category with BMI > 25.0kg/m<sup>2</sup> to 29.9kg/m<sup>2</sup>. Based on BMI the subjects were classified as obese and non obese as follows: Subjects with BMI > 30kg/m<sup>2</sup> were taken as obese. Those with BMI between 18.5kg/m<sup>2</sup> and 24.9kg/m<sup>2</sup> were taken as non obese controls.

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 internal diameter in diastole, 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)<sup>11</sup>

The left ventricular mass (LVM) was calculated using the American Society of Echocardiography formula modified by Devereux<sup>12</sup> (Devereux – modified ASE cube formula) as follows:

- $LVM (g) = 0.8 (1.04(IVSd + LVIDd + PWTd)^3 - LVIDd^3) + 0.6$
- Where IVSd = interventricular septal thickness in diastole
- PWTd = posterior wall thickness in diastole.
- LVIDd = Left ventricular internal diameter in diastole.

The left ventricular mass was indexed to height<sup>2</sup> as proposed by Rosa and associates<sup>13</sup>. The relative wall thickness (RWT) was calculated as  $2 \times PWTd/LVIDd$ . Increased relative wall thickness was taken as  $RWT \geq 0.45$ . Left ventricular hypertrophy (increased LVMI) was defined as  $LVM/H^2 >$

77.7g/m<sup>2</sup> in males and >69.8g/m<sup>2</sup> in females.<sup>14</sup>

The LV geometric classification was based on the evaluation of the LV mass index (LVMI) and relative wall thickness (RWT) as follows:

1. (1) Normal geometry- Normal LVMI with RWT < 0.45
2. (2) Concentric Remodeling - Normal LVMI with RWT ≥ 0.45
3. (3) Eccentric hypertrophy – Increased LVMI with RWT < 0.45
4. (4) Concentric hypertrophy – Increased LVMI with RWT ≥ 0.45.

Statistical analysis was performed using the statistical packages for social sciences (SPSS) version 11 while statistical calculations were carried out using the computer programmed for Epidemiologists (PEPI) version 4.0.

The comparative analysis of the continuous variables was conducted using student t-test while the categorical variables were compared using the independent chi-square test. Values of p<0.05 were considered statistically significant.

**RESULTS**

A total of two hundred and thirty (230) participants 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 age range of 22-58 years. Eighty 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 were no significant differences in the mean ages or height of the obese and non obese subjects. However the weight, body mass index and body surface area were significantly higher in the obese than the control groups as shown in table 1

**Figure 1**

Table 1 Demographic Characteristics of Obese And Controls

Characteristic	Obese (n = 150) Mean (SD)	Non-obese (n = 80) Mean (SD)	t-test	p-value
Age (Years)	42.54 (8.60)	43.11(8.58)	0.48	0.632
Weight (kg)	96.26(11.37)	66.86(8.11)	20.50	<0.001*
Height (m)	1.64(0.08)	1.70(10.09)	0.07	0.942
BMI (kg/m <sup>2</sup> )	36.03 (6.04)	23.07(1.57)	18.84	<0.001*
BSA (m <sup>2</sup> )	2.01 (0.15)	1.77(0.15)	11.56	<0.001*
WC (cm)	106.65(8.92)	80.91(5.79)	23.31	<0.001*
HC (cm)	112.05(10.62)	86.84(6.77)	19.24	<0.001*
WHR	0.95 (0.06)	0.93(0.07)	2.27	0.024*

*\*Statistically significant*

BMI = Body mass index, BSA = Body Surface Area,  
WC = Waist circumference, HC = Hip circumference  
WHR = Waist hip ratio. \* = Statistically significant

The echocardiographic parameters, namely IVSd, LVIDd, PWTd and RWT, LVM, LVMI were significantly higher in the obese than non obese group (table 2).

**Figure 2**

Table 2 Echocardiographic features in obese and controls

Characteristic	Obese (n = 150) Mean (SD)	Non-obese (n = 80) Mean (SD)	t-test	p-value
LAD (mm)	37.56 (3.91)	33.27(5.69)	6.73	<0.001*
IVSd(mm)	12.46(3.00)	9.56(2.47)	9.38	<0.001*
LVIDd(mm)	51.13(4.72)	45.36 (3.87)	9.38	<0.001*
PWTd (mm)	11.07(1.52)	9.41 (2.02)	7.01	<0.001*
RWT	0.46 (0.07)	0.42(0.09)	3.73	<0.001*
IVS/PWTd	1.15(1.32)	1.05 (0.29)	2.33	<0.001*
LVM (g)	240.82 (69.68)	147.31 (51.35)	10.57	<0.001*
LVM/H <sup>2</sup> (g/m <sup>2</sup> )	90.09(28.61)	51.18(17.72)	11.08	<0.001*
LVM/BSA (g/m <sup>2</sup> )	118.96(31.21)	83.46(28.90)	8.43	<0.001*

*\*Statistically significant*

LAD = Left atrial dimension, 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/H<sup>2</sup> = Left ventricular mass indexed to height<sup>2</sup>,  
LVM/BSA= Left ventricular mass indexed to body surface area.

The left ventricular geometric patterns in the obese and control groups are shown in table 3 as follows:-

**Figure 3**

Table 3 left ventricular geometric patterns in obese and controls using LVM/ H

Geometric Pattern	Obese (n=150) Frequency (%)	Non-obese (n=80) Frequency (%)	$\chi^2$	p-value
Normal	25(16.7)	57(71.3)	67.757	<0.001*
Concentric remodeling	19(12.6)	18(22.5)	3.737	0.053
Eccentric Hypertrophy	69(46.0)	3(3.7)	43.308	<0.001*
Concentric hypertrophy	37(24.7)	2(2.5)	18.206	<0.001*

\*Statistically significant

In the obese group, 16.7% had normal geometry, 12.6% had concentric remodeling, 46.0% had eccentric hypertrophy and 24.7% had concentric hypertrophy. In the control group, 71.3% had normal geometry, 22.5% had concentric remodeling, 3.7% had eccentric hypertrophy and 2.5% had concentric hypertrophy. There was statistical difference in the geometric patterns of the obese and control subjects. For normal geometry, ( $\chi^2= 67.76$ ,  $p< 0.001$ ) eccentric hypertrophy ( $\chi^2=43.31$ ,  $p<0.001$ ) and concentric hypertrophy ( $\chi^2 = 18.21$ ,  $p <0.001$ ). There was no significant difference in the frequency of concentric remodeling in the two groups.

When the obese subjects were classified into BMI subgroups and their various geometric patterns sought, results showed that in class I obesity, 30.1% had normal geometry while 69.9% had abnormal geometry, in class II obesity 5.1% had normal geometry while 94.9% had abnormal geometry, in class III obesity, none had normal geometry while 100% had abnormal geometry. In the control group with normal BMI, 71.3% had normal geometry while 28.7% had abnormal geometry. Concentric remodeling was highest in obese class I (20.5%), eccentric hypertrophy was highest in obese class III (77.8%), and concentric hypertrophy was highest in obese class II (62.7%). These are shown in table 4.

**Figure 4**

Table 4 Frequency distribution of the Geometric Patterns for the various classes of BMI in the obese.

Geometric	Class I Frequency (%)	Class II Frequency (%)	Class III Frequency (%)
Normal Geometry	22 (30.1)	3 (5.1)	0 (0)
Concentric Remodeling	15 (20.5)	3 (5.1)	1 (5.6)
Eccentric Hypertrophy	18 (24.7)	37 (62.7)	14 (77.8)
Concentric Hypertrophy	18 (24.7)	3	3 (16.7)
<b>Total</b>	<b>73</b>	<b>59</b>	<b>18</b>

**Total number of subjects = 150**

Class I obesity = 30 -34.9kg/m<sup>2</sup>

Class II obesity = 35 -36.9kg/m<sup>2</sup>

Class III obesity =  $\geq 40$ kg/m<sup>2</sup>

**DISCUSSION**

The present study has demonstrated that obesity is associated with left ventricular hypertrophy and abnormal left ventricular geometry. This agrees with studies by Lavie et. al<sup>10</sup>. which showed that abnormal LV geometry and LVH progressively increased with more obesity. The pattern of left ventricular remodeling is also of prognostic importance as certain geometric patterns have been associated with worse outcomes than others.<sup>9</sup> Obesity produces an increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by excess body weight. Incremental increases in left ventricular filling pressure and volume overtime produce chamber dilatation leading to increased wall stress. This predisposes to an increase in myocardial mass and to left ventricular hypertrophy characteristically of the eccentric type.<sup>7,15</sup>

The fact that eccentric hypertrophy is associated with obesity has been demonstrated in this study also. Eccentric LVH was highest in the total obese group with a frequency of 46.0% and highest also in obese class III subgroup with a frequency of 77.8%. Within the pathophysiological concept established especially in studies by Alpert et. al<sup>8</sup>. and Alexander<sup>16</sup>, eccentric hypertrophy is associated with obesity. However this study also identified a significant proportion of concentric hypertrophy (24.7%) in the obese subjects who were apparently normotensive by casual blood pressure measurement. Many authors agree that in obese subjects with systemic hypertension, there is a double stimulus to develop hypertrophy which could either be concentric or mixed.<sup>17</sup> A 24 hour ambulatory blood pressure monitoring

should therefore be recommended in the obese to detect cases of masked hypertension and nocturnal hypertension. Masked hypertension occurs in individuals in whom office readings underestimate out of office blood pressure readings and occurs in ten percent of patients with high blood pressure.<sup>18</sup> It clearly increases cardiovascular risk. Nocturnal hypertension occurs in individuals whose blood pressures fail to dip at night during sleep. Nocturnal hypertension increases the aggregate hemodynamic load on the cardiovascular system and is a much stronger predictor of cardiovascular outcome than office measurements.<sup>19</sup>

Left ventricular geometric patterns predict mortality and a multivariate analysis in one study showed that abnormal LV geometry including increased RWT and LVMI were independent predictors of mortality.<sup>10</sup> Hence concentric remodeling which is associated with increased RWT but normal LVMI is also a cardiovascular risk.<sup>20</sup> A higher frequency of concentric remodeling (22.5%) was identified in the controls than the obese (12.6%). This is probably due to the increased blood volume and ventricular dilatation associated with the pathophysiology of obesity.

It was also noted that the possibility of having abnormal LV geometry progressively increased with the degree of obesity such that in class III obesity, none of the subject had normal geometry and 100% had abnormal with 77.8% of this being the eccentric type. Studies have shown that in each BMI subgroup, mortality progressively increases with abnormal LV geometry from normal concentric remodeling, eccentric hypertrophy and concentric hypertrophy<sup>10</sup>. Thus an individual in class III obesity with concentric hypertrophy is at a higher risk than a class II individual with the same geometric pattern. This emphasizes the need for early treatment programs to be commenced on obese subjects to prevent progressive weight gain which carries increased risk for the development of abnormal geometric pattern and increased mortality. Concentric and eccentric hypertrophy are associated with increased left ventricular mass which carries independent risk of stroke, arrhythmias and sudden death<sup>21</sup>

It is apparent from this study that a variety of adaptations and alterations in cardiac structure occur as excessive adipose tissue accumulates even in the absence of systemic hypertension and underlying organic heart disease. It is therefore recommended that health education programs should be organized by medical practitioners, nutritionists and government in public institutions and in the community

warning individuals on the predisposing factors to obesity and their consequences. Medical practitioners should be alert to capture cases of obesity and manage them properly using diet and exercise as preliminary measures in primary, secondary and tertiary health institutions. Weight loss can regress left ventricular hypertrophy<sup>22</sup> there by reversing the abnormal left ventricular geometry in the obese subject with eccentric ventricular hypertrophy. This should be instituted early to prevent the complications of obesity and abnormal left ventricular geometry.

Echocardiography should also form part of routine investigation for obesity to enable early identification of patients with abnormal left ventricular geometry and early institution of weight loss measures there by curbing an entity of high cardiovascular risk. As already mentioned above, a 24 hour ambulatory blood pressure monitoring is also recommended in the obese.

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