A STUDY OF THE PATTERN OF OBESITY AND ITS PRIMARY CO-MORBIDITIES AMONG ADULT PATIENTS ATTENDING GENERAL OUTPATIENT CLINIC OF FEDERAL MEDICAL CENTRE OWERRI, IMO STATE.

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BY

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NOVEMBER 2012.
DECLARATION

It is hereby declared that this work is original unless otherwise acknowledged. The work has neither been presented to any College for Fellowship Award nor has it been submitted elsewhere for publication.

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DEDICATION
This book is dedicated to my late parents Ogbuefi and Iyom Mrs. Francis Anakonobi Ikwudinma, their life foundation sustained me and to my senior sister Elizabeth who played a major role in what I am today.
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LIST OF ABBREVIATIONS AND ACRONYMS

AIDS: Acquired Immune Deficiency Syndrome
AgRP: Agouti-Related Peptide
BMI: Body Mass Index
BC: Before Death of Christ
BP: Blood Pressure
°C: Degree celsius
cm: Centimetre
COD: Cholesterol oxidase
CV: Coefficient of variation
CHD: Coronary Heart Disease
CART: Cocaine, Amphetamine Regulated Transcript
DM: Diabetes Mellitus
DHS: Demographic Health Survey
DNA: Deoxyribonucleic acid
dl: Decilitre
EDTA: Ethylene Diamine Tetra-acetic acid
FFA: Free Fatty Acid
FBS: Fasting Blood Sugar
FMC: Federal Medical Centre
GOD: Glucose Oxidase
GOPD: General Outpatient Department
GLU: Glucose
GOPC: General Outpatient Clinic
H: Height
hr: hour
HDL-C: High Density Lipoprotein Cholesterol
HIV: Human Immunodeficiency Virus
IBW: Ideal Body Weight
IFCC: International Federation of Clinical Chemists
JNC: Joint National Committee
Kg: Kilogram
Kcal: Kilocalorie
LH: Lateral Hypothalamus
LDL-C: Low Density Lipoprotein Cholesterol
m: Meter
mg: Milligram
mph: Meter Per Hour
ml: Milliliter
MDG: Millennium Development Goals
NCD: Non-Communicable Diseases
NO: Nitric Oxide
NIDDM: Non-Insulin Dependent Diabetes Mellitus
No: Number
OD: Optical Density
POMC: Pro-Opiomelanocortin
TBF: Total Body Fat
TC: Total Cholesterol
TG: Triglyceride
Rpm: Revolution per minute
USPSTF: United States Preventive Services Task Force
US: United States
USA: United States of America
VMH: Ventromedial Hypothalamus
VLDL: Very Low Density Lipoprotein
WHO: World Health Organization
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<td>W:</td>
<td>Weight</td>
</tr>
<tr>
<td>WC:</td>
<td>Waist Circumference</td>
</tr>
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<td>WHR:</td>
<td>Waist to Hip Ratio</td>
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<td>%:</td>
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SUMMARY

Obesity has become a growing and serious non-communicable medical condition in Nigeria. This study was therefore carried out to determine the pattern of obesity and its primary co-morbidities among adult patients attending General Outpatient Clinic of Federal Medical Centre, Owerri.

This study was a hospital-based cross-sectional study carried out from May 2010 to July 2010. A total of 2,391 patients were screened for obesity using BMI criteria and 618 of them met the selection criteria. Of these 618 obese patients, a subsample of 206 were systematically selected by using sampling interval of 1:3. These 206 obese patients were screened for hypertension, diabetes mellitus and dyslipidaemia. The awareness of their obese condition and knowledge of lifestyle modifications for obese condition were also assessed. Data were collected using clinical data collection proforma and pretested structured interviewer administered questionnaires.

The prevalence of obesity was 25.8%. Most of the obese patients (68%) had class I obesity. Most of the obese patients belonged to the age group 48 – 57 years and were married (67.5%), had tertiary education (38.8%), traders (30.6%) and belonged to low socio-economic class (67.5%). There was significant association between marital status and degree of obesity. Dyslipidaemia (43.2%) was the commonest primary co-morbidity. The systolic (r=0.21) and diastolic (r=0.27) hypertension, triglyceride (r=0.082), low density lipoprotein cholesterol (r=0.001) and fasting blood sugar (r=0.081) were correlated with BMI. Furthermore, most of the respondents were not aware of their obese condition (74%) and had no knowledge of lifestyle modification for obese condition (55.8%).
This study had shown that obesity and its primary co-morbidities are emerging as a serious health problem among the study population with class I obesity as the most common pattern and dyslipidaemia, the commonest primary co-morbidity. Anthropometric determination of obesity as baseline clinic assessment in all patients and routine screening of its primary co-morbidities are advocated. Education on healthy lifestyle practices for obesity, its prevention and control are recommended.
CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND

Obesity is culturally and socially acceptable among blacks and Nigerians and therefore not usually recognized as a medical problem.\textsuperscript{1,2,3} However, it is now widely seen as a rapidly growing health risk globally\textsuperscript{4} and in 1997 was recognized by World Health Organization (WHO) as a health problem that poses a serious threat to public health worldwide. Obesity is defined as an excess of adipose tissue\textsuperscript{5} resulting in Body Mass Index (BMI) $\geq 30\text{kg/m}^2$. Previously, it was considered to be the direct result of a sedentary lifestyle and chronic ingestion of excess calories. Although sedentary lifestyle and chronic ingestion are important causes, in some cases, there is now evidence for strong genetic influences on the development of obesity.\textsuperscript{6}

The incidence and prevalence of obesity have increased significantly during the past two decades in developed countries and this trend is rising\textsuperscript{7} and increasing markedly over the past twenty years.\textsuperscript{8} Affecting an alarming 50-60\% of nations’ population not only in United States of America (32.2) but also Mexico (24.4 for male, 34.5 female), Egypt (33.1 for female) and South Africa (10.1 for male, 27.9 for female),\textsuperscript{9} also a high prevalence was reported in pacific Islands, parts of middle East and some developed countries.\textsuperscript{10} In Nigeria, a study had reported a prevalence rate of 16.3\% in a riverine community Okriaka in Rivers state and 14\% in Portharcourt Rivers State.\textsuperscript{11}
Obesity is an emerging problem in segments of sub-Saharan African society particularly where lifestyles are becoming urbanized and Westernized.\textsuperscript{12} The problem of obesity is widespread and is not only in the western world but also in the poorest countries.\textsuperscript{7} Obesity is reported to be associated with increased risk of premature death, heart disease, high blood pressure and diabetes mellitus.\textsuperscript{5,7,13} Indeed, many disorders occur with greater risk in obese people, primarily the most important being hypertension, type 2 diabetes mellitus (DM) and hyperlipidemia.\textsuperscript{5,14} Data has shown association of obesity with hypertension, DM and hyperlipidemia under the umbrella disorder called metabolic syndrome.\textsuperscript{15}

As developing countries such as Nigeria continue to combat communicable diseases such as HIV/AIDS, tuberculosis leading to longer life expectancies, there is also emerging an unprecedented epidemic of obesity and its primary co-morbidities.\textsuperscript{16,17} Globally, rising trends in morbidity and mortality related to chronic non-communicable disease such as obesity and its metabolic co-morbidities have led the World Health Organization and other international and national organizations to device strategies for chronic non-communicable disease prevention and control.\textsuperscript{18} Efforts should be made to prevent weight gain in order to improve the quality, lifespan and reduce health costs associated with obesity and its co-morbidities.

Obesity is largely associated with lifestyle which can be modified through centralized health policy.\textsuperscript{19} Some professional bodies and organizations like US Preventive Services Task Force (USPSTF) recommend that clinicians screen all adults patients for obesity and offer intensive counseling and behavioural interventions to promote sustained weight loss for obese adults.
1.2 STATEMENT OF THE PROBLEM AND PROBLEM ANALYSIS

Obesity is a growing health problem globally. Even in mild degrees, it has serious medical consequences. It is a medical risk factor for a number of diseases including diabetes mellitus (DM), ischaemic heart diseases, high blood pressure and dyslipidaemia. On average, obesity reduces life expectancy by 6 to 7 years, a BMI of 30-35 reduces life expectancy by 2 to 4 years, while severe obesity (BMI ≥ 40) reduces life expectancy by 10 years. Obesity increases the risk of mortality; a body weight of 10% above average is accompanied by an 11% increase in women, while if the body weight is 20% above average the excess mortality rises to 20% and 10% for men and women respectively. Other metabolic consequences of obesity include impaired glucose tolerance, impaired fasting glucose and hyperuricaemia, all of which are disease conditions with negative impact on cardiovascular morbidity and mortality.

The 1980’s and 1990’s witnessed alarming increase in obesity across the globe especially in developed countries; where among the leading causes of preventable death, obesity ranks second only to smoking and could soon surpass it. People in sub-Saharan Africa including Nigeria appear not to perceive obesity as a health risk. In most African communities, obesity has been considered as a positive health value and never as sign of disease. Even more, it is considered as an external sign of wealth, prosperity and as a cultural pattern of beauty by the media. To challenge such socio-cultural belief is difficult when potential role models such as health workers are obese and comprehensive data on prevalence of obesity is scanty especially in developing countries like Nigeria. Thus obesity, a disease previously
thought to have low prevalence in Nigeria because of its association with wealth and affluence has risen in prevalence over the last decade or so to levels that now constitute an epidemic threat.

In the present, there had been upsurge in the number of obese patients who have obesity related primary co-morbidities presenting at the General Out Patient Clinic of Federal Medical Centre Owerri. These primary co-morbidities worsen the patient’s prognosis with many developing acute and chronic complications of hypertension, diabetes mellitus and dyslipidaemia.

In recent times, there is tremendous increase in the number of fast food outlets (junk food) and heavy consumption of glucose sweetened soft drinks and beverages across the country\textsuperscript{25,26} especially in urban areas of the country such as Owerri. Also, there is high level of commercial marketing of energy dense, nutrient poor foods that are specifically targeted at young adults. This type of marketing strategy contributes to unhealthy diet that promotes obesity and also undermines various healthy lifestyle intervention strategies.

Furthermore, conversion of most recreational facilities in the study area into commercial uses and church activities and abandonment of our local vigorous life for sedentary life pattern, rapid and unplanned urbanization/modernization of lifestyle have further compounded the burden of obesity. Most patients come to clinic because of other medical conditions other than obesity, because they do not feel their overweight/obesity as a problem or wish to lose weight making obesity treatment one of the most difficult problems in clinical medicine.
Also clinicians, though aware of obesity and its associated health risks, have not made it a routine to assess for obesity and screen for its primary co-morbidities in the clinic through simple anthropometric measurements. They do not have time to educate and enlighten their patients on healthy lifestyle. In addition, inadequate reimbursement and lack of proper training in counselling skills have in general made physicians able to provide only limited care for their patients needing health education and weight control guidance. Data from this study will help improve and provide opportunities for such services to the patients. Furthermore, these preventive measures are yet to be embraced on a large scale in developing countries compared to developed countries. The near absence of these in Global Millenium Development Goals (MDGs) does not help the situation. \(^\text{27}\) This type of study has not been done in Owerri hence there is lack of knowledge of pattern of obesity in the environment.

1.3 **RELEVANCE OF THE STUDY TO FAMILY MEDICINE**

The enormous and rising burden of obesity and its medical consequences in developing countries such as Nigeria has informed the decision for this study in our environment. \(^\text{28}\) This study will help to determine the prevalence of obesity, and also assist in understanding the magnitude of the problem posed by obesity and in assessing the present intervention strategies to curb it. The study will inform the need and necessity to review the current morbidity of non-communicable diseases especially obesity in Nigeria.

Obesity should be considered as a disease entity in its own right and would require treatment even in the absence of other co-morbid conditions because of the
morbidity associated with untreated obesity. The study will help to direct more effort in the prevention and control of obesity and its associated co-morbidities as envisioned in the WHO Global Strategy on diet, physical activity and health, ensuring that people have access to healthy diets and get involved in physical activities. In our families, urban, semi-urban and rural communities, developing and developed nations, people die each year from medical consequences of obesity despite its largely modifiable risk factors. Since developing countries have fewer resources to manage obesity and its co-morbidities than developed countries, it is important to identify through epidemiological data interventions that are effective, inexpensive, widely practicable and culturally acceptable. Given the chronic nature of most diseases associated with obesity and by extension the huge cost of treatment, the prospects look grim for the already under funded and ill equipped African health care system to deal with a new epidemic alongside existing ones such as HIV/AIDS, tuberculosis and malaria.

In the past, studies have shown that diabetes mellitus and certain other obesity related conditions occur to a markedly greater than average extent in many minority populations. A high risk body fat distribution (central obesity) occurs to a greater extent in some minority populations than in whites. Because of situational and cultural factors, effective obesity preventive and treatment approaches may need to be defined on ethnicity specific bases. Increased attention to obesity as it occurs in and affects diverse ethnic groups can help to address critical minority health issues. Such efforts can also broaden and enrich aspects of obesity research for which models based on white population are inappropriate or limited, hence the need for
local institutionalized related studies. Clearly continued and more effective population wide efforts to prevent and treat obesity are needed.

Obesity does not only form part of the metabolic syndrome but can worsen the major components of this syndrome by causing poor glycaemic control, increased insulin resistance, dyslipidaemia and hypertension, therefore the necessity for the study for appraisal and re-appraisal especially in the control of obesity and attendant co-morbidities.

In Nigeria, obese patients frequently present to the general medical practitioners and this study will help to sensitize them on the need to routinely assess for obesity. It has been suggested that obesity can be used to identify without clinical diagnosis those who are mostly at risk of several chronic non-communicable diseases.

The early recognition of obesity by family physicians is quintessential to its management whilst identifying its common primary co-morbidities avails great opportunities for prevention and control. This will help obese patients who most often do not present obesity as the reason for encounter or may avoid medical care because of fear of embarrassment for their body image. Also data from the study will conscientize family physicians to help patients identify and address barriers to compliance, such as social and environmental cues to unplanned eating.

The study will enlighten the family physicians on the magnitude of obesity and its primary correlates in the environment. It will also improve the knowledge and awareness of patients to the burden of obesity and its associated co-morbidities.

Moreso, unless there is a significant attitudinal changes in our society concerning the
ubiquitous supply of high caloric foods and our increasingly inactive lifestyle, the situation will worsen which the study is expected to directly or indirectly ameliorate.

The results obtained from the study will assist health policy makers enact appropriate laws and regulations to help control negative lifestyle and proliferation of fast food outlets in the study area. The prevention of obesity will have a major health implication by reducing the burden of hypertension, diabetes mellitus and dyslipidaemia through dietary management, improved physical activities and lifestyle modifications. This study will tremendously achieve positive universal, selected and targeted preventions in affected individuals who are obese to prevent further weight gain. Indirectly, their families and community in general will benefit from the education on healthy lifestyle based practices.

1.4 AIM OF THE STUDY

This study is aimed at determining the pattern of obesity and its primary co-morbidities among adult patients attending General Outpatient Clinic of Federal Medical Center, Owerri.
1.5 OBJECTIVES

1. To determine the prevalence of obesity using BMI anthropometry among the study population.

2. To describe the pattern of obesity using BMI anthropometry among the study population.

3. To describe the demographic characteristics of the obese patients.

4. To determine the prevalence of specific primary co-morbidities of obesity such as hypertension, diabetes mellitus and dyslipidaemia among the study population.
CHAPTER TWO

LITERATURE REVIEW

2.1 DEFINITION

Traditionally, the term “obesity” was defined as an excess proportion of body fat in relation to observed norms in the population by age, sex and height regardless of weight.\textsuperscript{33} That is, a person of normal weight carrying a higher than normal proportion of body fat would be defined as obese. Furthermore, it can be defined as body weight of about 20\% above that of the standard height and weight table for the community.\textsuperscript{34} Currently, these definitions have changed. Rather than referring independently to excess body weight and body fat, present definitions refer to an elevation in an individual’s body mass index, which determination comprehends both weight and height. Obesity is defined as body mass index (BMI) of 30 or more.\textsuperscript{35} It can also be defined in terms of waist circumference of 102 centimeter (cm) or more in men or 88cm or more in women.\textsuperscript{11}

2.2 Historical Perspective

Obesity is from a Latin word obesitas, which means “stout, fat or plump”. Esus is the past participle of edere (to eat), with ob (over) added to it. The Oxford English Dictionary documents its first usage in 1611 by Randle Cot Grave. The Greeks were the first to recognize obesity as a medical disorder.\textsuperscript{36} Hippocrates wrote that corpulence is not only a disease itself, but the harbinger of others.\textsuperscript{2}Also, the Indian surgeon Sughruta (6\textsuperscript{th} century BC) related obesity to diabetes mellitus and heart disorders.\textsuperscript{37}

Obesity was historically viewed as a sign of wealth and prosperity. Thus, it was common among high officials in Europe in the middle ages and Renaissance\textsuperscript{38} as well
as in Ancient East Asian civilizations. With the onset of industrial revolution it was realized that the military and economic might of nations were dependent on both the body size and strength of their soldiers and workers.\textsuperscript{38,39} Increasing the average body mass index from what is now considered underweight to what is now the normal range played a significant role in the development of industrialized societies. Height and weight thus both increased throughout the 19th century in the developed world. During the 20th century, as populations realized their genetic potential for height, weight began increasing much more than height, resulting in obesity.\textsuperscript{40} In modern western culture, excess weight is often regarded as unattractive and obesity is commonly associated with various negative stereotypes. People of all ages can face social stigmatization and may be targeted by bullies or shunned by their peers, hence a reason for discrimination.\textsuperscript{41} Public perceptions in the western societies regarding what is healthy body weight and ideal body weight have significantly changed since the beginning of 20th century, while slimness began to be seen as the standard for ideal body weight.\textsuperscript{39} In contrast, what is viewed as healthy body weight changed in the opposite direction with increasing body weight regarded as normal. This was attributed to increasing rates of adiposity leading to increased acceptance of extra body fat as being normal.\textsuperscript{42} However in many African countries, weight gain is culturally perceived as a symbol of wealth, beauty and fertility.\textsuperscript{2,39} Moreso, obesity is viewed as a sign of good health and well-being in many parts of Africa like Nigeria, especially since the emergence of HIV epidemic.\textsuperscript{2}
2.3 Prevalence of Obesity

The increasing prevalence of obesity is gaining worldwide attention. Countries with food security and under nutrition problems are paradoxically not spared due to the fact that maldistribution of resources and affluence co-exist with abject poverty.\(^{43}\)

Before the 20\(^{th}\) century, obesity was rare\(^{36}\) and in 1997, the World Health Organization (WHO) formally recognized obesity as a global epidemic\(^{40}\) and in 2005 the WHO estimated that at least 400 million adults (9.8%) are obese.

While many industrialized countries have experienced increasing obesity rates, the United States are among the highest in the world. Estimates of the number of obese American adults have been steadily expanding from 19.4% in 1997, 24.5% in 2004\(^{44}\) to 26.6% in 2007\(^{45}\) and if the current trend continues 41% will be obese by 2015.\(^{46}\)

Also in America between 1986 and 2000, the prevalence of severe obesity (BMI≥ 40kg/m\(^2\)) quadrupled from one in two hundred Americans to one in fifty; extreme obesity (BMI≥ 50kg/m\(^2\)) in adults increased by a factor of five from one in two thousand to one in four hundred.\(^{47}\)

Moreso, data has reported prevalence by states in US of highest rate in West Virginia (30.6%) and least rate of 18.4% in Colorado.\(^{48}\)

Lifestyle changes related to increasing trends in consumer consumption of energy dense high calorie food, soda and junk foods are implicated as associated links to the observed increased prevalence rates in America. Also severe obesity in the United States, Australia and Canada are increasing faster than the overall rate of obesity.\(^{49}\)

Furthermore, a cohort study\(^{50}\) of rapid increase in obesity in population samples from three countries- America, Jamaica and Nigeria between 1994 to 1999 reported
prevalence rates of 51%, 24% and 5% respectively. It further reported that weight gain differed significantly by country, with Jamaica having a larger weight gain approximately four times larger than the corresponding weight gain in US and Nigeria; also that even though low prevalence was reported in Nigeria, the studied rural population sample gained as much weight as those followed in the US during the period. Rapid cultural changes, decreasing rural population and character of social development not necessarily the level of economic activity were attributed as driving force in increasing obesity rate in the poor and middle income countries like Nigeria and Jamaica. However, the study did not provide comparable measures of physical activities, nutritional patterns and obesity prevention activities; also the individual studied may differ from their weight trajectory from the other segment of the population.

Similarly, the prevalence of obesity in Ghana for the population 18 years and above was 5.5% in 2003 and varied across regions and socio-demographic characteristics; which when compared to conditions like tuberculosis and HIV/AIDS with prevalence around 3%, is high and alarming. Further studies reported obesity prevalence rate of 16.1% in Greater Accra and 14.1% among adults aged 25 years and above in Accra. While diet (kenkey) and sedentary lifestyle were observed as the likely explanation for the high obesity rate in Greater Accra, the high prevalence observed in the entire Ghanaian population was attributed to urbanization, modernization, affluence and changing lifestyles.

In developing countries such as Nigeria, obesity is regarded as a disease of affluent societies. In Nigeria, early data in the middle and later part of the last century
suggested a low prevalence, however reports from other studies indicate increasing prevalence. A study had documented alarming prevalence rates of 71.6% females and 50.5% males in a population of hypertensive patients in Abuja Nigeria, figure similar to that in developed countries. In another population based study, among type 2 diabetics, 83% were obese all suggested marked increase in prevalence comparable to what is obtained in developed countries. These changes were attributed to rapid and unplanned urbanization, change from local dietary pattern to western style diet which is driven by the proliferation of fast food outlets in major cities across Nigeria.

Furthermore in Nigeria, a study conducted in urban areas of Rivers State South Nigeria in 2006 showed prevalence rates of 16.3% and 14% with more prevalence among females and young adults (<40 years). These finding were attributed to older age, female sex and socio-cultural practices among the people.

Further studies in two communities of suburban Northern Nigeria reported a prevalence of 13.1% (11.2% males and 22.0% females), which is worrisome for a population made up of about 90% being physically active peasant farmers. The influence of agricultural machinery, use of herbicides (hence less physical activity in tilling the soil), availability of modern transportation and change in eating habits such as increase in the intake of calorie-laden beverages were blamed for the high prevalence of obesity among the studied population.
2.4 Factors Affecting Obesity

2.4.1 Age and Sex

The World Health Organization (WHO) 2005 reported higher rates of obesity among women than men. The rate of obesity also increases with age up to 60 years. \(^{51,52}\)

In England and Wales, there have been increases in the prevalence rates of obesity from 6% and 8% respectively for men and women in 1980 to 8% and 12% in 1990 and to over 21% for both in 2000. \(^{60}\)

In America, although heterogeneity in obesity prevalence currently exists among U.S sub-populations, the rate of change among adults is virtually identical across social class, gender and race. \(^{61}\) The mechanism for this trend is rooted in macrosocial changes in consumption and associated behaviours, however there is relatively little empirical research relating change in socio-cultural patterns to age-related weight gain.

Marked obesity rate among minority US population than in the whites had been reported and in some cases exceeding prevalence rate in the white by three times. \(^{62}\) The markedly high prevalence of obesity is more pronounced in women than in men. These findings were due to differences in socioeconomic status, regional and urban or rural resident patterns.

Also, while some studies in Cuba in 1980s showed age variations in obesity rate ranging from 8% to 39% in men and 20% to 47% in women, \(^{63}\) others reported less obesity rates of 7.1% in men and 10.2% in women. These observations were reported to be due to high intake of energy dense macronutrient-poor foods,
sedentary lifestyle, cultural and traditional practice of viewing obesity as a positive self image and beauty.

Other studies done within Africa such as one done in four urban areas of Cameroon also reported high prevalence of obesity in women (19.5%) than men (6.5%) and the difference particularly large in the younger age groups of (15-34 years), where the ratio of obese women to obese men was over five. Also the study reported that prevalence varied greatly with age and obesity BMI increases greatly with age until 45-54 years. Other studies in urban Cameroon also reported obesity rate of 5.4% in men and 17.1% in women which were lower than the findings in the previous study.

Moreso, study in Ghana reported high obesity rate in females (7.4%) compared to males (2.8%). Similarly, Ghana Demographic and Health surveys (DHS) showed that prevalence of Obesity or overweight among adults (non pregnant) women across the country increased 2.5 fold in ten years from 10% in 1993 to 25.3% in 2003. Crucially the 2003 DHS data showed that there were more obese women (25.3%) than malnourished women (9%).

In addition, WHO 2003 sponsored National Obesity Survey in Ghana showed higher obesity rates in southern compared to northern regions, among women compared to men, among married than unmarried individuals and among older compared to younger individuals and obesity increased with age up to 64 years. Also a cohort study in Ghana reported that 6.1% women were morbidly obese.

In the past, there were few studies on obesity in Nigeria and the West African sub region. Earliest studies on obesity conducted in Nigeria among population sample in
Lagos reported obesity rates of 8.3% and 37.7% for males and females respectively.\textsuperscript{69} Also another study done in Enugu among undergraduate students revealed obesity prevalence of 21% (8.1% male and 13.1% female), with more females having class I obesity 52.7% and class II obesity 4.6% compared to males with 33.6% and 3.8% for class I and II respectively.\textsuperscript{70} These findings were attributed to the type of food available in most eating houses on campus which are energy dense nutrient poor foods especially targeted on young adults, decreased level of physical activity and limited duration of time spent on exercise, however the lower level adult age of 17 years used in the study may account for more obese subjects recruited in the study.

In another study\textsuperscript{11} done in two riverine urban communities Rivers state Nigeria, reported higher obesity rates in both females (89.4% and 62.1%) and males (10.26% and 37.9% ) with most in middle age group. The reasons for these findings include the “iria” native practice of keeping women in fattening rooms for aesthetic reason, general, physical inactivity of women and age of subject as increasing age is associated with decreased physical activity.

Further study conducted in suburban northern Nigeria reported high obesity rates of 11.2% male to 22.0% female;\textsuperscript{58} and further observed that in diabetic and non diabetic populations, obesity tends to occur more commonly among females than males. Cultural and genetic factors were believed to play a role, although the study did not find out the factors responsible for the observed findings. Also, a study done in Lagos western Nigeria reported a prevalence of obesity in type 2 diabetes of 18.6% with class I, 14.3% in class II and 4.3% in class III obesity.\textsuperscript{20} Moreso,
frequency of obesity in type 2 DM was higher in females 25.9% compared to 10.6% males; the obese type 2 DM were predominantly females 72.9% with female to male ratio of 2.7 to 1 in contrast to female to male ratio of non obese type 2 DM of 0.9 to 1. The study\textsuperscript{20} re-emphasized the increasingly apparent predominance of obesity in women compared to men of African origin with DM as demonstrated by other studies which reported approximately 4.4 fold increase in abdominal adiposity in women (75%) compared to men (17%) with type 2 DM in Trinidad.\textsuperscript{71} Also another study done in Lagos Nigeria, showed obesity was present in 24.1% of hypertensive-diabetics as compared to 14.7% normotensive diabetics.\textsuperscript{72} In addition, the study also found that all patients with class III obesity were females and compared similarly with a study in USA (2000) which reported a prevalence of class III obesity 2.2% and highest (6.0%) amongst the black women.\textsuperscript{73}

2.4.2 Rural/Urban

The prevalence of obesity and obesity related morbidities in developing countries are relatively low but are changing rapidly with urban and rural variations.\textsuperscript{18} For example, a study\textsuperscript{11} done in Rivers state among urban and rural residents, showed a prevalence of 14.0% in urban Port-Harcourt while in rural riverine area of Okirika was 16.3%.

However, the prevalence of obesity is more noticeable in urban areas.\textsuperscript{74,75} It is currently estimated that as much as 20-50% of urban populations in Africa are classified as either overweight or obese,\textsuperscript{76,77} and that by 2025 three quarters of the obese population worldwide will be in non-industrialized countries.\textsuperscript{17} This was attributed to the fact that modern societies seem to be converging on a pattern of
diet high in saturated fats, sugar and refined foods and low in fibre, usually termed
the “western diet”. Urbanization and socio-economic transformation comes with
increased access to energy dense foods and less strenuous jobs resulting into many
people having a positive energy balance and hence becoming overweight or obese.

17,29,51,77-80

In addition, a study done to examine changes over time on the prevalence of
obesity among urban areas in seven African countries showed a steep rise in the
prevalence of obesity an increase by an average of nearly 5% per year. This
reinforces the observation that obesity is on the increase in urban areas of Africa
and lends support to the WHO warning on an impending dual epidemic of
communicable and non-communicable diseases in Africa in the near future.17 This
trend being observed in spite of rampant poverty in urban areas due to access to
cheap foods with a high content of fat and sugar among the urban poor is easier
than among the rural population.82-84

In the context of rapid urbanization in Africa, primarily driven by rural-urban
migration, the new migrants have been shown to adapt to new urban lifestyle which
ultimately predisposes them to becoming obese even though their socio-economic
status might be lower than that of long term residents.81,85,86 Indeed some studies
have shown that recent migrants to cities tend to have a Higher Body Mass index
(BMI) than rural residents and those with longer urban environment exposure.87

With increasing urbanization, there might be a shift of the obesity burden to sections
of the poor urban population who may not have the knowledge or financial
resources to adopt healthier lifestyles coupled with erroneous perception of
obesity as symbol of high social status.90,91
2.4.3 Marital Status

A study\textsuperscript{81} had found that young and unmarried women were less likely to be obese compared to their married counterparts. This finding is also supported by similar studies.\textsuperscript{11,51} These findings were attributed to the perception of obesity as a positive symbol of high social status, ignorance to recognize body size as health problem and the fact that single women are less likely to be multiparous which is associated with higher risk of obesity.

2.4.4 Socio-Economic Status

A study\textsuperscript{81} has shown that women of higher socio-economic status (household wealth and women education) were more likely to be obese than their poorer counterparts in line with other studies;\textsuperscript{50,74,77} also, women who engage in income generating activities (working) were more likely to be obese. Similar findings were reported in studies\textsuperscript{64,79,92,93} which showed a strong positive relationship between socio-economic status and obesity among men, women and children. This contrasts with another finding which reported increase in obesity among women of lower socio-economic status. These observations maybe due to the criteria for household wealth measures (household amenities and possessions) used in the study which may not be appropriate for detecting changes in household wealth over time as household asset and characteristics may not change in the short term secondary to changes in household income.

Very few studies have examined the changing dynamics of obesity and socio-economic status over time, making it difficult to assess the socio-economic differentials in the rate of progression to obesity in urban Africa. Further research
using other measures like income or expenditure might improve the understanding of changes in obesity over time by economic differentials.

Also, a study in Ghana\textsuperscript{52} reported high obesity rate among urban high class residents compared with the low class residents. Obesity rate was higher among the employed compared to self employed or the volunteer workers and among people whose jobs are sedentary in nature.

\subsection*{2.4.5 Educational Status}

A study\textsuperscript{81} found that based on educational attainment, non educated women have higher obesity rate compared to the more educated women at level of secondary degree or higher education. In contrast, a positive association was reported between obesity and duration of education (used as proxy for socio-economic status) in a Cameroonian study.\textsuperscript{64} Likewise, studies in Ghana\textsuperscript{52,67}reported higher prevalence in populations with tertiary education and high educational status compared with less literate or illiterate populations.

However, developed countries show an inverse (negative) relationship between education and obesity particularly among women; the lower the education or the social class, the higher the prevalence of obesity. These findings were attributed to westernization of lifestyle, reduced physical activities, more sedentary life and adaptations of high energy, high fat diets. As these becomes more general in developing countries, the familiar inverse association of obesity with socio-economic and educational class will emerge.
2.4.6 Income level

Obesity prevalence rates have also been increasing in many low and middle income countries.\textsuperscript{94-96} In an international comparative study of 85 countries mean levels of BMI and other cardiovascular (CV) risk factors were found to have an asymmetric upside down U shaped association with a country’s stage of development, whereby mean BMI levels were highest in middle income countries.\textsuperscript{95} A longitudinal analysis of 3 cohorts studies done in countries (Jamaica, Nigeria and United states) demonstrate significantly more rapid weight gain among individuals living in a middle income country example Jamaica and confirm prior evidence based on cross-sectional prevalences.\textsuperscript{50} Cross sectional analysis of obesity prevalence reflect the accumulated history of caloric excess but are relatively insensitive to the dynamic processes that may reflect recent changes in obesity promoting exposures.

In some countries like Nigeria, where average citizens have unlikely seen any substantial increase in their material standard of living over the last two decades,\textsuperscript{97} thus the lifestyle changes required to fuel weight gain do not require general economic development, and instead may reflect the penetration of market-based consumption patterns into stagnating or declining economies. A review of large scale surveys on diet, activity and obesity showed that the speed of dietary and activity pattern shifts is great resulting in major shifts in obesity on a worldwide basis and that the burden of obesity is shifting towards the poor.\textsuperscript{88}
2.5 Hazards of Obesity

2.5.1 Health Hazards of Obesity

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems. Excess body weight is the sixth most important risk factor contributing to the overall burden of disease worldwide. In 1997, the WHO published a landmark document recognizing obesity as a worldwide disease that poses a serious threat to public health. In USA obesity was set to overtake smoking in 2005 as the main preventable cause of illness and premature death. The effect of obesity on health including cardiovascular disease and diabetes have been documented. Moderate to severe obesity is a risk factor not only for coronary heart disease (CHD), but also for other chronic diseases. Studies have shown strong association of obesity with hypertension in Nigerian diabetics. Obesity arising early in adulthood or occurring over a long period of follow-up convey particularly strong risks of coronary heart disease (CHD). It is a leading preventable cause of death worldwide, with increasing prevalence in adults and children. Authorities view it as one of the most serious public health problems of the 21st century.

Evidence to support the view that some obese people eat little yet gain weight due to a slow metabolism is limited; on average, obese people have a greater energy expenditure than their counterparts due to the energy required to maintain an increased body mass. It was once thought the health problem of affluent western world has now increased in prevalence in many developing countries, and has
been described as a time bomb for the future explosion in the frequency of cardiovascular disease, type 2 diabetes mellitus and dyslipidaemia.\textsuperscript{7}

As the developed world grapples with a proportionately high burden of non-communicable (chronic) diseases, such as obesity, developing countries and countries undergoing socio-economic transition are experiencing a mixed epidemic of non-communicable and communicable diseases.\textsuperscript{104} In many developing countries, research and investment in health has been mainly devoted to infectious diseases, despite the growing need to address chronic diseases such as obesity with more effort and resources.\textsuperscript{17} Deaths from infectious disease, maternal and perinatal conditions and nutritional deficiencies combined are projected to decline by 3\% over the next 10 years, while at the same time deaths due to chronic diseases are projected to increase by 17\%.\textsuperscript{17} As a result, it is estimated that of the projected 64 million deaths worldwide in 2015, 41 million (64\%) will result from chronic diseases unless urgent action is taken.\textsuperscript{17} Similarly, obesity has considerable impact on the psychological health of its sufferers as it generates a feeling of inferiority and inadequacy both of which may lead to further weight increase.

Industrialization and modernization among others have contributed to an increased life expectancy, and at the same time, contributed to an increased morbidity and mortality from the diet related non-communicable diseases mostly associated with obesity.\textsuperscript{88,105} This is not only in affluent societies, but also in many sectors of populations in low income societies. Obesity, a well known disease, has emerged with new epidemic features as a negative outcome of the different patterns of
transition and as an unhealthy well identifiable common factor for Non-Communicable Diseases (NCD) and its increasing rate of morbidity and mortality. Obesity has reached epidemic proportion worldwide especially in the developed nations both in children and adults. It has dramatically increased in prevalence in most industrialized countries and at the same time, many developing countries have undergone an economic transition from societies characterized by subsistence agriculture to increased urbanization and industrialization. In addition, modern societies seem to be converging on a pattern of diet high in saturated fat, sugar and refined foods and low in fibre, usually termed the "western diet".

2.5.2 Metabolic Hazards of Obesity

The potential metabolic hazards of obesity have been documented extensively particularly in relation to its common primary co-morbidities such as cardiovascular diseases, type 2 diabetes mellitus and dyslipidaemia. The presence of metabolic risk of obesity is related to the duration and degree of obesity and to the location of excess fat (visceral fat is more metabolically active than subcutaneous fat and hence more deleterious). However, medical risk of obesity is highly associated with the distribution of the body fat and abdominal fat is considered at least as important a medical risk as the total amount of body fat.

While obese subjects are prone to hypertension, hypertensive subjects also appear to be prone to weight gain and future weight gain is significantly greater in hypertensive subjects than in normotensive subjects. It is likely that obesity, hypertension and metabolic abnormalities interact and potentiate their individual impact on cardiovascular risk. These obesity related metabolic abnormalities and
impairment of cardiovascular function may be present even at a younger age and
progress asymptptomatically for decades before clinical manifestations set in.

In addition, body fat distribution is an additional dimension of obesity related risk,
independent of overall weight or total body fat. Upper body fat, particularly intra-
abdominal fat, has been associated with an increased risk for diabetes and
cardiovascular diseases in several cohorts,\textsuperscript{111,112} including several minority groups.
Where relevant data exist, obesity is associated with increased levels of
cardiovascular risk factors in ethnic minorities in America. These minority
populations have a relatively higher prevalence of obesity related diseases than do
white populations, particularly regarding diabetes mellitus.

Further findings from a national cohort of US adults showed that for every kilogram
of increase in weight, the risk for diabetes increases by 4.5\%.\textsuperscript{113} Also, another
national cohort study of US adults reported similar findings and a weight gain over a
10 years period was strongly associated with increased risk for diabetes.\textsuperscript{113} Although
obesity contributes to several major cardiovascular risk factors, the greatest impact
of current increases in body weight has been on diabetes.\textsuperscript{101,114} Excess body weight
is the primary underlying epidemiologic measure associated with diabetes risk,
however the distribution of the diabetes epidemic is not wholly consistent with
variations in BMI across populations.\textsuperscript{115} In general, populations with recent rapid
increase in obesity appear to have higher than predicted diabetes rates.

\textbf{2.5.3 Economic Impact of Obesity}

Obesity and their health consequences may have significant direct and indirect
economic impact. The direct medical costs include preventive, diagnostic and
treatment services related to obesity. Indirect costs include morbidity costs (value of income lost from decreased productivity, restrictive activity and absenteeism) or mortality costs (value of future income lost due to premature deaths). In USA alone, consumers spent $40 billion to $100 billion annually on weight loss products and services in search of effective therapies.\textsuperscript{116} Because many weight loss interventions are unproven and untested, health professionals often lack information with which to recommend a certain therapy or to monitor a patient once therapy is chosen. In addition, obesity leads to many problems including disadvantages in employment, reduced work performance and increased business costs and decreased productivity\textsuperscript{117} affecting all levels of society from individuals, corporations to governments. Some research has shown that obese people are less likely to be hired for a job and are less likely to be promoted.\textsuperscript{41} In 2000, the extra weight of obese passengers cost airlines US $275 million.\textsuperscript{118} Also other specific industries such as food and allied products are witnessing rising negative economic concerns.

### 2.6 Types of Obesity

Different types\textsuperscript{119} of obesity have been variously described. These include generalized obesity which is uniform deposition of excess fat throughout the body; android obesity- excess deposition of fat over the waist; gynoid obesity- excess deposition of fat over the hip and thighs and superior (central) obesity which is excess deposition of fat over the face, neck and upper part of the trunk and the limbs are thin.

In addition, obesity can be further divided into hyperplastic characterized by increased number of fat cells\textsuperscript{34} or combination of both hypertropic-hyperplastic
obesity. Hyperplastic obesity is common in children (juvenile onset obesity) whereas hypertrophic is characterized by enlarged fat cells and is more common in adult onset obesity.\textsuperscript{120}

Moreso, it can be classified based on percentage increase in overweight:\textsuperscript{34} mild cases (20\% to 40\% overweight), moderate cases (41\% to 100\% overweight) and severe cases (over 100\% overweight).

Other types of obesity based on criteria used include: central or peripheral obesity based on distribution of body fat; metabolic or regulatory obesity based on its pathogenic mechanism; childhood, early childhood, gestational and middle-aged obesity types according to age of onset of obesity; and classification according to related cause: genetic, hypothalamic, dietary, endocrine and physical inactivity types of obesity.\textsuperscript{34}

\textbf{2.7 Grading of Obesity}

The WHO in 2010 classified obesity\textsuperscript{121} based on associated risk of co-morbidities using the BMI criteria into obesity (BMI \( \geq 30 \)), class I (moderate obesity) BMI 30-34.9, class II (severe obesity) BMI 35.0-39.9 and class III (very severe obesity) BMI \( \geq 40.0 \). In contrast, as Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations redefined obesity; Japanese have defined obesity as any BMI greater than 25\textsuperscript{122} while China used a BMI of greater than 28.\textsuperscript{123}
2.8 **Aetiology of Obesity**

At individual level, a combination of excessive caloric intake and a lack of physical activity is thought to explain most cases of obesity. Some of these obesitogenic individual behaviours include high fat diets, frequent snacks in between standard meals, consumption of energy dense foods (sweets, ice-cream, soft-drinks), alcohol, cultural stay in fattening rooms prior to marriage. In contrast, increasing rates of obesity at a societal level are felt to be due to an easily accessible and palatable diet, increased reliance on cars and mechanized manufacturing. Total calorie consumption has been found to be related to obesity and increasing extra calories came from an increase in carbohydrate consumption (mostly sweetened beverages in young adults) rather than fat consumption. In some countries like America, the primary source of these extra carbohydrates are sweetened beverages which now accounts for almost 25% of daily calories in young adults. As societies become increasingly reliant on energy dense, big portion, fast food meals, the association between fast food consumption and obesity becomes more concerning; and in United States consumption of these fat food meals and calorie intake from these meals quadrupled between 1977 and 1995. Mechanized agriculture and subsidization of agricultural source of these processed high calorie food has made it cheaper and more available compared to fruits and vegetables in Europe and America.

A limited number of cases are due primarily to genetic, medical reasons or psychiatric illness. Similar to many other medical conditions, obesity is the result of an interplay between genetic and environmental factors. Polymorphism in various genes controlling appetite and metabolism predisposes to obesity when sufficient calories are present. More than 41 of these sites have been linked to the
development of obesity when a favourable environment is present. The percentage of obesity that can be attributed to genetics varies, depending on the population examined, from 6% to 85%. Obesity is a major feature in several syndromes such as Prader-Willi Syndrome, Bandel-Biedl Syndrome, Cohen Syndrome and Momo Syndrome. In people with early-onset severe obesity (onset before 10 years of age and a body mass index over three standard deviations above normal) 7% harbor a single point DNA mutation. Studies that have focused on inheritance patterns rather than upon specific genes have found that 80% of the offspring of two obese parents were obese, in contrast to less than 10% of the offspring of two parents who were of normal weight. Also adopted children demonstrate a close relationship between their body mass index and that of their biologic parents and genetic determinants of some types of obesity have now been established.

While genetic influences are important to understanding obesity, they cannot explain the current dramatic increase seen within specific countries or globally. The shifts on causal factors of obesity on general and global basis may be attributed to combination of various social determinants such as wealth, cultural and behavioural attitudes, urbanization, family size (woman’s risk increase by 7%/child while man is 4%/child) and smoking (quitters of smoking gain an average of 4.4kg for men and 5.0kg for women over a ten year period).

Also sedentary lifestyle plays a significant role in obesity. Worldwide there has been a large shift towards less physically demanding work, this is primarily due to increasing use of mechanized transportation and a greater prevalence of labour saving technology in the home. The World Health Organization reported that people
worldwide are taking up less active recreational pursuits; while a study in Finland\textsuperscript{134} found an increase, but a study from the US found leisure time physical activity has not changed significantly.\textsuperscript{135}

Furthermore, certain physical and mental illness and the pharmaceutical substances used to treat them can increase the risk of obesity.\textsuperscript{2,136} Although obesity is not regarded as a psychiatric disorder, obesity risk is higher in patients with psychiatric disorders than in persons without psychiatric disorders.\textsuperscript{137} Medical illness that increase obesity risk include several rare genetic syndromes, some congenital and acquired syndromes- Hypothyroidism, Cushing’s Syndrome, Growth Hormone Deficiency, and eating disorders- binge eating disorder and night eating syndrome.

The drugs that may cause obesity or changes in body composition include insulin, oral contraceptives, steroids, phenothiazines, antidepressants, antipsychotics, thiazolidinediones, sulfonylureas, anticonvulsants (phenytoin and valproate).\textsuperscript{2,34}

Moreso, association between viruses and obesity has been found in human and several different animal species and difference in gut flora in obese and lean individuals can affect the metabolic potential contributing to obesity.\textsuperscript{138}

Other possible contributors to recent increase in obesity include: insufficient sleep, endocrine disruptors (environmental pollutants that interfere with lipid metabolism), decrease variability in ambient temperature, increased rates of smoking (suppresses appetite), drugs (eg antipsychotics), pregnancy at a later age, epigenetic risk factors passed on generationally, assortative mating leading to increased concentration of obesity risk factors.\textsuperscript{139}
2.9 Pathophysiology of Obesity

Pathophysiologically, many possible mechanisms are involved in the development and maintenance of obesity; and there is a neurophysiological basis for the regulation of body weight. A protein hormone leptin, (a 167 amino acid peptide), has been observed to have important effects in regulating body weight, metabolism and reproductive function. It is produced in men exclusively by adipose tissue but in women also by the placenta. Other mediators include ghrelin, insulin, orexin, PYY3-36, cholecystokinin, neurotensin, bombesin, adiponectin and adipokines. Leptin and ghrelin influence on appetite are complementary with ghrelin (produced in stomach) modulating short-term appetitive control and leptin (produced by adipocytes) mediating long-term appetitive control. The hormonal effects are mediated through their actions on central nervous system particularly the hypothalamus, the brain area central to the regulation of food intake and energy expenditure. These hormones act through several circuits (like the melanocortin pathway) in the area of the hypothalamus, the arcuate nucleus, that has outputs to the lateral hypothalamus (LH) and ventromedial hypothalamus (VMH), the brain’s feeding and satiety centers respectively. In the arcuate nucleus, leptin acts on and regulates two neuronal sites: the neuropeptide Y (NPY) and agouti-related peptide (AgRP) which stimulates feeding and inhibits satiety and the pro-opiomelanocortin (POMC) and Cocaine, Amphetamine-Regulated transcript (CART) which stimulates satiety and inhibits feeding. Thus in leptin signaling either via leptin deficiency or resistance leads to over feeding and may account for some genetic and acquired forms of obesity. Human obesity is associated with high serum leptin concentration suggesting leptin resistance rather than leptin deficiency.
The enzyme lipoprotein lipase synthesized in adipocytes induces obesity by causing deposition of fat in adipose tissues.\textsuperscript{119} In obesity, the adipocytes number and size are increased in middle age resulting in obesity, but with excess calorie intake, the number and size can increase at any age.\textsuperscript{119} With weight gain in obese individuals, adipocytes increase in size due to accumulation of triglycerides and synthesis of more leptin.

## 2.10 Pathophysiology of Obesity and its Primary Metabolic Co-Morbidities

### 2.10.1 Pathophysiology of Obesity and Diabetes Mellitus

Study had shown serum leptin levels to correlate with some markers of the metabolic syndrome in obese individuals and could serve as a risk factor for cardiovascular disease.\textsuperscript{142} High circulating leptin concentrations in obesity may cause insulin resistance by inhibiting insulin signaling.\textsuperscript{34} Also studies have recognized leptin, adipokines, resistin and adiponectin as link between obesity and type 2 diabetes.\textsuperscript{143,144} During stress, the increased release of cathecholamines leads to increased FFA levels. The increased free fatty acids results in the generation of oxidant stress molecules, depression of the nitric oxide (NO) production, insulin resistance and hyperinsulinaemia. Also, it can result in the inhibition of hepatic clearance of insulin leading to peripheral insulin sensitivity, a feature of non insulin dependent diabetes mellitus (NIDDM) common among obese patients.

### 2.10.2 Pathophysiology of Obesity and Hypertension

Although the precise mechanism linking obesity to hypertension and increased cardiovascular risk are not fully understood, obesity may lead to hypertension and cardiovascular disease by activating the renin-angiotensin-aldosterone system, by
increasing sympathetic activity and leptin resistance by increased procoagulatory activity, by endothelial dysfunction and through subclinical inflammation. Also, peripheral hyperinsulinaemia results in sodium retention and stimulation of sympathetic nervous system leading to hypertension common among obese patients. Further mechanisms include increased renal sodium re-absorption, causing a shift to the right of the pressure natriuresis relationship and resulting in volume expansion.\textsuperscript{145}

2.10.3 Pathophysiology of Obesity and Dyslipidaemia

It has been observed that the site of distribution of excess fat could bear important relationship to the development of some medical consequences of obesity such as cardiovascular diseases. Recent research has shown functional differences between adipose tissue in the abdominal region and gluteal region. Truncal obesity is considered to be more dangerous because its adipose tissues are more lipophylic and generate more free fatty acids when metabolized. During stress, there is release of catecholamines which preferentially cause increased lipolytic activity together with depressed lipoprotein lipase activity in abdominal adipocytes. The free fatty acids (FFA) released as a consequence of this enter the portal vein resulting in a high concentration in the liver. Thus by above mechanism, hypertrophy of the abdominal adipose mass would result in the exposure of the liver to excessive FFA concentrations.

The high level of FFA in the liver favour the development of hypertriglyceridaemia by their esterification in the liver and resulting in secretion of very low density lipoprotein (VLDL). Both of these factors predispose to development of coronary heart disease common among obese patients.
2.11 Diagnostic Criteria

The evaluation of fatty mass and definitions of obesity use a range of approaches, some of which are complex or invasive and are inapplicable outside of specialized clinical practice to identify candidates for weight management. Consequently in these situations, indirect estimates obtained from weight and height formulae are routinely used for the assessment of obesity. Indirect measurements are not only easily obtainable; they have been shown to be reasonably accurate.

In routine clinical practice and epidemiological studies the most commonly and widely used measure to define obesity is the Body Mass Index (BMI). The BMI is defined as weight (in kg) divided by height (in meters) squared. It provides an estimate of generalized adiposity that can be compared across studies and populations. Many studies have found a correlation between BMI and densitometry estimates of body fat composition in adult populations. The validity of BMI as a measure of adiposity is further supported by its association with obesity related risk factors such as blood triglycerides, total cholesterol, blood pressure and fasting blood glucose levels. In Brazil, the National Health and Nutritional Examination Surveys have shown that increase in BMI is usually associated with increase in prevalence of diabetes mellitus, hypertension and dyslipidaemia. A study has shown BMI to be most suitable index derived from weight and height for the assessment of obesity in Nigerian populations. Also it is a special and sensitive predictor of total body fat. The study reported that BMI was the only index (among others: weight-height ratio (W/H), Rohrer’s index (W/H³) and Ponderal index (H/W1/3) not significantly correlated with height in both sexes while being...
strongly positively correlated with weight, in contrast the other indices have strong correlation with height,\textsuperscript{148} thus the most suitable index of obesity. This is similar to earlier studies which found BMI to be least height biased obesity index in both sexes.\textsuperscript{154,155} A most appropriate index derived from weight and height is one that is most strongly positively correlated with weight and minimally correlated with height and ought to be independent of height.\textsuperscript{153} Also a study\textsuperscript{156} has found BMI to be a sensitive and specific predictor of percentage total body fat (TBF \%) obtained from underwater weighing, giving credence to its use to assess total body fat. In Nigeria, studies have shown that BMI measurement increases progressively with age.\textsuperscript{157}

The use of BMI to determine obesity has often been criticized because of the inability of this measure to discriminate between weight from body fat and weight from other body structures such as muscle and bone mass. Moreso, BMI could be a less valid indicator of adiposity among the elderly who tend to have a shift of fat from peripheral to central sites. For such population and with evidence of health risk associated with abdominal fat, waist indices, measure of central obesity is preferred. Nevertheless a study that assessed obesity related risk as determined by BMI and waist circumference among adult Nigerians reported greater proportions of obesity related risk factors with BMI than abdominal obesity as determined by waist circumferences.\textsuperscript{11} In addition, BMI is a commonly used measure that has been strongly related to adiposity and strongly predictive of risk of future disease.\textsuperscript{158}

Other physical measurements used to assess obesity include Ideal Body Weight (IBW), Waist Circumference (WC) and Waist-Hip Ratio (WHR). The WHR is useful in assessing obesity in people who are very muscular and with WC is a better measure of central obesity and both have been shown to be strongly associated with
cardiovascular disease risk factors such as diabetes and hypertension in many populations.\textsuperscript{152,159} However, various large scale prospective studies have shown that anthropometric measurements except WHR were strongly correlated with each other\textsuperscript{94} and will provide comparable information. The correlation of indices of overall and central obesity is highly suggestive of an association between increased overall obesity (as measured by BMI) with increased visceral fat (as measured by WC).

Obesity can be assessed more accurately by direct measures such as Skin Fold Thickness, Densitometry, Computerized Axial Tomography Scan, Magnetic Resonance Imaging and Dual Energy X-ray Absorptiometry,\textsuperscript{160} but these measures are too laborious, time consuming and impracticable for field epidemiology or clinical practice.

\subsection*{2.12 Treatment of Obesity}

\subsubsection*{2.12.1 Non Pharmacological Treatment of Obesity}

There is a clear need to develop a global strategy for managing the increasing number of obese subjects in the community. Although diet programs may produce weight loss over a short term,\textsuperscript{161} effective long term weight loss necessitates persistent changes in dietary quality, energy intake and physical activity\textsuperscript{2} and making these changes part of a person’s lifestyle.\textsuperscript{162} The most economical and main treatment of obesity is non-pharmacological and consists of dieting and physical exercise.\textsuperscript{119,124,163} This requires use of multi-disciplinary approach to weight control, setting realistic goals, maintaining record of goals, instructions, weight progress charts, assessing efficacy of weight loss measures and integrating obesity control measures into the overall management of its concomitant co-morbidities.\textsuperscript{163}
Diet restriction remains the most common method of obesity reduction; increased physical activity alone is not thought to be enough strategy for obesity reduction. Some studies show that weight loss induced by increased daily physical activity without caloric restriction substantially reduces abdominal obesity and insulin resistance in men although exercise without weight loss reduces abdominal fat and prevents further weight gain.

Dietary treatment involves reduction in energy intake of 500 to 600 calories less than the energy expenditures of the individual, reduction in fat intake, avoiding frequent snacks and diet with concentrated sugars. Also eating healthy diets consisting of complex carbohydrates with enough protein, adequate amount of fibre, vitamins and minerals. This aim to achieve weight reduction of 1kg per week, with initial loss of 10% of the patients body weight over six months.

Study had found weight loss to be associated with a significant reduction of blood pressure and had beneficial effects on the associated risk factors. It was found that a modest reduction in body weight can cause a meaningful reduction in the activity of the renin-angiotensin-aldosterone systems in the circulation and in adipose tissue which makes a major contribution to the blood pressure decrease. Weight loss of 5% is associated with the reduction of angiotensinogen levels by 27%, renin by 43%, aldosterone by 31%, angiotensin-converting enzyme activity by 12% and angiotensinogen expression by 20% in adipose tissue. Study had shown that weight loss of only 5%-10% improved blood pressure, lipid levels and glycaemic control as well as symptoms of depression, anxiety and eating related psychopathology. In another study of obese patients, an average weight loss of
6.5% yielded significant reductions in blood pressure (11.1/5.8mmHg), triglycerides (94mg/dl), glucose (17mg/dl) and total cholesterol (37mg/dl).\textsuperscript{167} Weight loss has been shown to improve endothelial function,\textsuperscript{168} decrease sympathetic activity and improve baroreflex function.

Some studies had found significant reduction in mortality in certain populations with weight loss. It was shown that over the short term (weeks or months), intentional weight loss in obese individuals reduced risk factors for and improved symptoms of obesity-related conditions. \textsuperscript{169,170} In obese women without obesity related illness a weight loss of greater than 9kg was associated with a 25\% reduction in mortality.\textsuperscript{171} A recent review concluded that certain subgroups such as those with type II diabetes and women show long term benefits in all cause mortality; while outcomes for men do not seem to be improved with weight loss.\textsuperscript{172} Subsequent study had shown benefits in mortality from intentional weight loss in those who had severe obesity.\textsuperscript{173}

Diets to promote weight loss are generally divided into four categories: low fat, low carbohydrate, low calorie and very low calorie. A meta-analysis of six randomized controlled trials found no difference between three of the main diet types (low calorie, low carbohydrate, low fat) with a 2-4kg weight loss in all studies.\textsuperscript{161} However, at two years these three methods resulted in similar weight loss irrespective of the macronutrients emphasized.\textsuperscript{174} In other randomized control trials over 24 weeks and by one year on low carbohydrate diet program compared to conventional diet and low fat diet had found modest to greater weight loss and reduction in serum triglycerides, increase High Density Lipoprotein (HDL) cholesterol level and glycaemic control compared to the other groups.\textsuperscript{175,176} Compared with low-
fat diet, a low-carbohydrate diet program had better participant retention and greater weight loss; and during active weight loss, serum triglyceride levels decreased more and high density lipoprotein cholesterol level increased more with the low-carbohydrate diet than with the low-fat diet. The magnitude of weight loss compared favorably with that achieved with use of weight loss medications such as orlistat (decrease of about 9% at six months) and sibutramine (decrease of about 8% at six month). However, the inability of the study to distinguish the effects of the low-carbohydrate diet and those of the nutritional supplements provided and lack of long-term follow up of the groups limit the generalization of the above findings.

With use, muscles consume energy derived from both fat and glycogen. Due to the large size of leg muscles, walking, running and cycling are the most effective means of exercise to reduce body fat. During moderate exercise, equivalent to a brisk walk, there is a shift to greater use of fat as a fuel. A randomized controlled trial found that exercising alone led to limited weight loss, but in combination with diet resulted to a 1kg weight loss over diet alone and 1.5kilogram loss was observed with a greater degree of exercise.

The more intense the exercise program, the more the weight loss and energy expenditure. Mild exercise like sitting and standing leads to 80kcal/hr to 120kcal/hr energy expenditure; moderate exercise like fast walking, swimming, tennis playing leads to 250kcal/hr to 350kcal/hr energy expenditure; while vigorous exercise like cycling and running both at 10 metres per hour(mph) leads to 600kcal/hr and 800kcal/hr respectively.
In addition, an observational study on the impact of 20-year changes in leisure-time physical activity on metabolic syndrome parameters found that an increase in sporting activity or physical activity had a beneficial effect on metabolic syndrome parameters.\textsuperscript{178} Also further intervention studies on reduction in obesity and related co-morbid conditions after diet induced weight loss or exercise induced weight loss in men have observed similar reductions in abdominal obesity, visceral fat and insulin resistance when weight loss induced by diet restriction are compared with weight loss induced by exercise alone,\textsuperscript{164} and further demonstrate that exercise without weight loss is a useful method for reducing abdominal fat and preventing further increase in obesity. However the study was not conducted in obese women. Moreover, a meta-analysis of controlled clinical trials on effect of exercise on glycaemic control and body mass in type 2 diabetes mellitus found that exercise training reduces glycosylated haemoglobin (HbAic) by an amount that should decrease the risk of diabetic complications with no significant change in weight compared to the controlled group\textsuperscript{179} and underscores the importance of exercise in the treatment of metabolic complications of obesity. Exercise training decreases hepatic and muscle insulin resistance and increases glucose disposal through a number of mechanisms that may not necessarily be associated with body weight changes.\textsuperscript{179} Weight loss program often promote behavioural, lifestyle changes and diet modification. This may involve eating smaller meals, cutting down on certain types of food and making a conscious effort to exercise more.
2.12.2 Pharmacological Treatment of Obesity

Apart from lifestyle (non pharmacological) measures for obesity, anti-obesity medications produce modest weight loss, maintenance and are not very popular nor easily available in most developing countries of the world. Most of these medications are currently not in use due to their side effects and adverse reaction but are still available illegally off-label or on short-term basis because their long term safety and health benefits remain unclear. The principle behind their use is to enhance thermogenesis or act centrally to suppress appetite as well as enhance thermogenesis.\(^\text{180}\)

The thermogenic/metabolic stimulants used include thyroxine, caffeine and ephedrine. Because thyroxine use requires close supervision and expert management, it is not advisable for use in treatment of obesity except in hypothyroidism, while caffeine and ephedrine are of limited usefulness in management of obesity due to their side effects which include insomnia, agitation and tachycardia.\(^\text{180}\)

Another group of anti obesity drugs used are the anorexants like phantamine, amphetamine, fenfluramine, denfenfluramine.\(^\text{181}\) They induce and maintain the feeling of satiety and anorexia. Their use in obesity treatment is limited due to their side effects which include cerebral stimulation, insomnia, agitation, arrhythmia, addiction, pulmonary hypertension and severe cardiac valvular damage.

Also, the usefulness of certain anti-obesity drugs depends upon the co-morbidities present. Metformin is used in obese diabetics because it may lead to mild weight loss while thiazolidinediones decrease central obesity.\(^\text{182}\)
Currently, the newer anti-obesity medications include Orlistat (xenical) which acts by reducing intestinal fat absorption by inhibiting pancreatic lipase; Sibutramine (meridian) which acts in the brain to inhibit deactivation of the neurotransmitters norepinephrine, serotonin and dopamine thereby decreasing appetite; and Rimonabant (acomplia) which acts via specific blockage of the endocannabinoid system. Over a long term, average weight loss on Orlistat is 2.9kg, Sibutramine is 4.2kg and Rimonabant is 4.7 kg. Also Orlistat and Rimonabant use lead to reduced incidence of diabetes and all the three new drugs improve fasting lipid levels. However there is little information on how these drugs affect the long-term complications or outcomes of obesity. The adverse effects of these drugs which limit their use in the treatment of obesity include: annoying and socially unacceptable gastrointestinal effects (loose stools, fecal incontinence, flatulence, dry mouth, oily spotting), insomnia, tachycardia, raised blood pressure for orlistat and increased risk of heart attacks and strokes in patients with cardiovascular disease for sibutramine.

Also due to continued concerns about adverse effects and lack of evidence about long-term safety, pharmacological treatments are used as adjuncts to strategies for changing lifestyle only on selected patients (those with BMI above 30kg/m2 and obesity related co-morbidities) after consideration of trade-off between potential benefits and harms. Currently, no prescription weight-loss drugs are approved for long-term use and non can provide sustained weight loss without recommended lifestyle changes.
2.12.3 Surgical Treatment of Obesity

Though surgery is not often recommended for the management of obesity because of frequent complications associated with it such as pulmonary embolism, gastrointestinal leak, deep venous thrombosis and wound infection,\textsuperscript{186} cost of surgery and required necessary skills are not readily available. However according to the clinical practice guidelines of the American College of Physicians, surgery is the most effective treatment for severely obese persons (BMI above 40kg/m\textsuperscript{2}) who fail to lose weight through diet and exercise or who have serious obesity-related health problems.\textsuperscript{187}

The criteria for surgical treatment of obesity include: patient age 18 years or older with morbid obesity (BMI≥40 or between 35 and 40 with major weight related morbidities); patients who have already had intensive management in specialized obesity clinics; patients who have failed to maintain weight loss after trying appropriate non-surgical measures; patients with no clinical or psychological contraindication to anaesthesia in surgery; and patients who understand and are committed to long-term follow-up.\textsuperscript{187}

Weight loss surgeries are termed either malabsorptive or restrictive. Malabsorptive operations which are more common, decrease the amount of food that can be ingested, as well as the calories and nutrients the body absorbs. The most common malabsorptive operations are the Roux-en-Y gastric bypass (a rerouting past the duodenum and jejunum and can be performed in an open procedure or laparoscopically) and the biliopancreatic diversion. Restrictive operations which
reduce stomach size include adjustable gastric banding and vertical banded gastroplasty.\textsuperscript{188}

In comparing the effects of the two types of surgeries on weight reduction outcome, studies have found gastric bypass procedures led to significantly greater weight loss (89\% vs 64\% of excess weight at three years, and 30\% weight loss at one year after surgery) and less weight regain than restrictive surgeries.\textsuperscript{189} In addition, surgery for severe obesity is associated with long term weight loss and decreased overall mortality. A study reported a weight loss of between 14\% and 25\% at 10 years and a 29\% reduction in all cause mortality when compared to standard weight loss measures.\textsuperscript{190} Also, weight loss after surgery is associated with reduction in metabolic syndrome-related abnormalities\textsuperscript{54} and a marked decrease in the risk of diabetes mellitus and cardiovascular disease.\textsuperscript{190}

Other forms of surgery for obesity include wiring the jaws to allow only fluid diet to be taken by straw and liposuction.\textsuperscript{191} The effects of these forms on weight loss are less well determined. However regaining lost weight after removal are common with these types of surgical treatment.

\textbf{2.12.4 Control of Obesity}

Obesity is a preventable chronic health condition and can be controlled through a multidisiplinary approach from the individual to the various cadres of the society. Prevention is the best treatment of obesity by careful dietary monitoring, lifestyle choices along with regular physical activities.\textsuperscript{124,163}

The various strategies necessary to prevent obesity and promote positive lifestyle changes at the national level include: setting up of national priorities to curb non
communicable diseases such as obesity, tax on foods with low nutritional value (for example soft drinks, confectionaries and snack food), subsidies for “healthy” foods, funding research to prevent or treat obesity, health care financing like reimbursement for interventions for health promotion and change in lifestyle. 192,193

At the state, local government and community level, the prevention strategies include: support for safe and convenient venues for physical activities such as walking paths and bicycle lanes, sponsorship of local public education campaigns and events to promote physical activities (for example walking groups) and organization of grassroots lobbying efforts. 194

Also at school and work place levels, the prevention strategies include: provision of requirements for daily physical education, banning of soft drink and snack machine, inclusion of topics on promoting health in the curriculum, family involvement and provision of wellness programs at the work place. 192-194

Furthermore, at the individual and health professional levels, the prevention strategies include: education and counseling to promote self awareness, motivation and monitoring (diet and physical activity diary), family and social support groups, guideline development for best practice and identification of high risk individuals and groups. 26,163

In addition, prevention strategies at the mass media and food industry includes: promotion of healthy lifestyles and realistic body images, dissemination of health information, labeling on packaged foods to identify ingredients and nutrient contents, inclusion of healthy food choices on restaurant menu and restriction on advertisement for low nutritional value foods that target children. 191
Once obesity develops, the main control intervention strategies include: sustained diet education, behavior oriented counseling, reinforcement and follow up, monitoring, stress management, stimulus control, problem barriers identification and solving contingency management and social support.\textsuperscript{163}

Further control interventions for chronic care and management of obese adults include: improved clinical information system, developing weight management team, team training and feedback; developing a self management support such as maintenance support, sourcing of self help materials and individual or group education skills, training and development; and development of community resources and partnership such as referrals for additional resources, liaison with community based programs and organizational leadership in the community for action or policy development.

Much of the western world has created clinical practice guidelines (clinical protocols) in an attempt to address rising rates of obesity. Generally, the guidelines are made to address the prevention and management of obesity at both the individual and population levels in both children and adults. A clinical practice guideline by the US Preventive Services Task Force (USPSTF) recommended intensive behavioural and dietary counseling in those with obesity and other known risk factors for cardiovascular and diet related chronic disease. These intensive counselling can be delivered by primary care clinicians or by referrals to other specialists such as nutritionists or dieticians\textsuperscript{195}. \textsection
CHAPTER THREE
MATERIALS AND METHODS

3.1 STUDY AREA

Federal Medical Centre Owerri, is located in Owerri, capital of Imo state, South-East of Nigeria. Imo state is bounded on the North by Anambra and Abia states, on the South by Rivers state, on the East by Abia state and on the West by Anambra and Rivers state. Imo state has a population of 3,927,563 people (1,976,471 males and 1,951,092 females) and a population density of 711.6 persons per square kilometers according to 2006 Population Census. The annual population growth rate of Imo state was 3.0% in 2003. Also Owerri Municipal where FMC Owerri is situated has a population of 125,337 (male 60,882 and female 64,455). It is about one hour drive to the two major commercial towns in the South East, namely Aba, Onitsha and Port-Harcourt in South South.

Two seasons are prominent in the state, namely rainy and dry seasons. The dry season starts in November and lasts till March while rainy season starts in April and ends in October. The mean monthly temperature of Imo state during the dry season is 34°C while it is 30°C in rainy season. It has relative humidity of about 60 to 80% throughout the year. The mean annual rainfall is between 1500 and 2200 millimeters.

Economic and social activities are low compared to industrial and commercial cities like Aba and Onitsha. It has one of the lowest cost of living indices in Nigeria as well as a peaceful and hospitable setting. It is endowed with abundant mineral and agricultural resources, with farming as the predominant occupation of the indigenous
population. It has three Federal higher educational institutions and two state higher educational institutions within the capital city, in addition to rapid commercialization and industrialization within the Owerri Municipal. These have boosted the social life in the city with attendant increase in the number of hotels, banks, rest houses, fast food outlets and beer parlours. The main native diet is ofe Owerri soup with pounded yam and Ugba. The main tourist attraction is Oguta lake. Imo state is culturally homogenous to a large extent and predominantly inhabited by Ibos. The major religion of the people is Christianity and most public parks and sports arenas are gradually being used mainly for church activities in Owerri.

### 3.2 HEALTH FACILITY

Federal Medical Centre Owerri, is located in Owerri Municipal. The hospital was established in 1903 as a military hospital but has since then passed from military to Shell BP, then to the government of old Eastern Nigeria. It then passed on to East-Central State government, to Imo state Government and finally to the Federal Government of Nigeria. The Federal Government of Nigeria took over the hospital in 1995 and changed its status from a general hospital to a tertiary hospital with the mandate to provide clinical services, training and research.

FMC, Owerri serves as a referral centre for primary and secondary public health Institutions as well as private and mission hospitals in Imo, Abia, Anambra and Rivers states. The centre provides primary, secondary and tertiary health care. It has facilities for emergency medical, general out-patient, family planning, physiotherapy services and community psychiatric unit. It also offers medical and surgical specialist services in surgery, internal medicine, family medicine, pediatrics,
laboratory medicine, obstetrics and gynaecology, ophthalmology, otolaryngology and dental surgery. The centre runs residency training in family medicine, internal medicine, surgery, pediatrics, ophthalmology, otolaryngology, pathology and radio-diagnosis. The hospital has 340 bed capacity with bed occupancy rate of 90%. The out patient clinic attendance in 2009 was 230,948. A total of 46,713 patients attended GOPC and 37,434 of them were new patients.

3.3 CLINIC SETTING
The General Out Patient Department (GOPD) of Federal Medical Centre Owerri is fully manned by consultant Family Physicians and resident doctors who attend to patients. The General Out Patient Clinic (GOPC) from hospital records attends to between 150 to 180 new patients per clinic day which translates to about 3,000 to 3,600 new patients per month.

3.4 STUDY POPULATION
The study population was made up of all new adult patients aged 18 years and above, with BMI ≥30kg/m² who gave consent to participate in the study.

3.5 STUDY DURATION
The study was carried out from May 2010 to July 2010

3.6 STUDY DESIGN
This was a hospital based descriptive study.

3.7 SAMPLING METHOD
A systematic sampling method was used to select the subjects. Obese patients who met the selection criteria were registered each consulting day in the register
prepared for the study by the author. This list of obese patients was taken as the sample frame. A one in three systematic sampling of the obese patients who were registered daily over the expected study period was used. This gave the expected number of obese patients during the study period of 618. The estimated sample size was 206, thus giving sampling fraction of 1/3 and sampling interval of 1 in 3. The first registered obese patient was selected by balloting from the first 3 registered patients as the starting number for the systematic sampling, and subsequently every 3rd registered patient on the register was selected for the study until the sample size was met.

3.8 SELECTION CRITERIA

Patients were selected based on the following inclusion and exclusion criteria.

3.8.1 INCLUSION CRITERIA

1. Patients aged 18 years and above using age group intervals of 18-27, 28-37, 38-47 etc.
2. Patients with BMI $\geq$ 30kg/m$^2$.
3. Patients who gave consent for the study.

3.8.2 EXCLUSION CRITERIA

1. Patients who were critically ill.
2. Pregnant women and patients with ascites or other forms of obvious edema.

3.9 SAMPLE SIZE

A sample size of 206 patients was used in the study. The sample size was determined using the formula below.\cite{198}
\[ N = \frac{Z^2 \cdot pq}{d^2} \]

where

N: Minimum sample size

Z: the normal standard deviation usually set at 1.96 which corresponds to 95% confidence interval.

P: The proportion or the target population estimated to have a particular characteristic. In a study done in Port Harcourt Nigeria, the prevalence of obesity was 14%. Thus 14% (0.14) was used in the study to give minimum sample size estimate.

q: 1.0 – P (1.0 -0.14)

d: Degree of accuracy desired usually set at 0.05.

Hence \( N = (1.96)^2 \times (0.14) \times (0.86) \)

\( (0.05)^2 \)

\[ N = 185 \]

However, a sample size (\( N_s \)) of 206 was used for the study to take care of non-response in the study. The selected sample size \( N_s \) was calculated considering an anticipated response rate of 90% (0.9). This was calculated by dividing the original calculated sample size (\( N \)) by the anticipated response rate as follows

\[ N_s = \frac{N}{0.9} \]

Where \( N \) = Minimum sample size

\( N_s \) = Selected sample size

0.9 = anticipated response rate.

Substituting in the above formula

\[ N_s = \frac{185}{0.9} = 206. \]
3.10 PRETESTING OF THE QUESTIONNAIRE INSTRUMENT

The questionnaire was pretested by the researcher on patients attending the GOPC Federal Medical Centre Umuahia, which has similar status to the GOPC Federal Medical Centre Owerri. A total number of 30 obese patients were haphazardly recruited for the pretest which lasted for about three days. Aspects of the questionnaire examined include:

i. The sectional and general patients understanding of the questions in the questionnaire and their willingness and response to questions asked.

ii. The appropriate time to administer the questionnaire during the doctor-patient interaction; and the time duration needed to administer the questionnaire, take measurements and samples and record the outcomes.

iii. Also the general and specific logistics support needed for efficient instrument administration and sample collection was evaluated.

iv. In addition, sample collection procedures were examined to assess validity and reliability of results that will be generated/recorded in the questionnaire. This was done by repeating the same procedures on same patients by the researcher and comparing the results recorded.

At the end of pretest activity, necessary adjustments were made on the questionnaire before carrying out the research on the proposed patients attending GOPC FMC Owerri by the researcher.

3.11 SAMPLE COLLECTION

Sample collection was done by the researcher during the study after informed verbal explanation and consent was obtained from the patient. The collection of samples for the study was standardized to ensure reliability by using the same
procedure/method of sample collection, same type of specimen containers and relevant laboratory analysis done within one hour of specimen collection. Both good laboratory practice and standard operating procedures were observed throughout the procedure. The samples were analyzed by only one senior chemical scientist in the chemical pathology laboratory of F.M.C. Owerri.

3.12 DIAGNOSTIC (SAMPLE) PROCEDURE

3.12.1 Obesity: Weight and Height Anthropometry

Weight was taken (in kilogram) with the patient standing erect bare-foot on a validated stadiometer weighing scale by Techmel and Techmel USA TT 120, in minimal clothing, with pockets free of objects that might add to weight eg keys, rings, mobile phones, wallets etc. The weight was measured to the nearest 0.1kg. The validity of the scale was checked every day before sample collection with a known weighted object (10kg).

The height was taken with a height meter combined with the weighing scale (Techmel and Techmel USA TT 120) with patient standing upright looking straight forward, with back straight, heels against the scale, without shoes, caps or scarves. The pointer of the height meter was pressed firmly against the scalp and read off on the meter scale to the nearest 0.5cm. The manufacturers standard operating procedures were followed (appendix one). The Body Mass Index (BMI) was calculated by dividing the measured weight in kilograms (kg) by the height in meters (m) squared, that is weight (kg)/height (m²).
3.12.2 HYPERTENSION: Blood Pressure Measurement

The blood pressure was taken by the investigator using auscultatory method with standard mercury in glass Accuson sphygmanometer. Prior to the measurement, the patient was seated and rested for 5 minutes in sitting position on a chair that supported the back comfortably. The left arm muscles were relaxed and the forearm supported with the cubital fossa at the heart level. A cuff of suitable size was applied evenly to the exposed arm to cover its major blood vessels. The cuff rapidly inflated until the manometer reading was about 30mmHg above the level at which the pulse disappeared and then slowly deflated. During this time, the Korotkoff sounds were monitored using a littman’s stethoscope placed over the brachial artery. The systolic blood pressure was taken when the first heart sound was heard (korotkoff phase 1) and diastolic blood pressure level taken when heart sounds disappeared (korotkoff phase V). The blood pressure was also measured in the right arm as described for the left arm in order to rule out significant inter-arm blood pressure difference and the arm that gave higher reading was subsequently used. The systolic and diastolic blood pressure was taken on the arm that gave the higher reading twice separated by an interval of 2 minutes. The three readings were recorded and the mean value calculated. The same instruments (Accusson sphygmanometer and Littman stethoscope) were used throughout the study according to manufacturer’s guidelines (appendix two) and checked daily before use (on a student nurse of known BP daily) to ensure validity.

3.12.3 DIABETES MELLITUS

Two milliliter (2ml) of blood was drawn from each subject after 8-12 hours fast with adequate disinfection of the skin over the venepuncture site by the investigator and
the blood sample put into a specimen bottle containing sodium fluoride oxalate. This was then labelled, registered and taken to the laboratory within one hour after collection for analysis. Only fasting plasma glucose was used in the study for ease of analysis. The fasting plasma glucose was determined by the glucose oxidase method. The reagents used were manufactured by Giesse Diagnostic snc, via Cervinara, 45 Colle Prenestino Rome Italy. The reagent has a measure range/linearity of 6.97-800mg/dl and sensitivity of 1mg/dl=0.00251 A at 510nm.

Good laboratory practice and manufacturer’s standard operating procedures were followed (appendix three). Subjects who had eaten before the test were given another time to come for the test. The results of the test were made known to them and then subsequently they were educated and counselled appropriately.

3.12.4 DYSLIPIDAEMIA

A 5ml venous blood sample after overnight fast was drawn from the antecubital fossa of each of the subjects with adequate disinfection of the skin over the venepuncture site. The samples were collected by the investigator. The serum was collected for analysis after the blood was allowed to clot and separated in the 5ml plain vacutainer bottle. The sample was taken to the laboratory within one hour after collection for analysis. Total cholesterol and triglyceride levels were determined using reagents manufactured by Bio Systems S.A. Costa Brava 30, Bercelona (Spain) with quality system certified according to EN ISO 13485 and EN ISO 9001 standards; with sensitivities for TC and TG of 1.75mA. dl/mg=67.6mA.L/mmol and 1.2mA. dl/mg= 112mA. L/mmol respectively. HDL-C level was determined using reagents manufactured by Randox laboratories Ltd, Ardmore, Diamond Road, Crumlin, Co. Antrim, United Kingdom. In all the above
tests, good laboratory procedures and manufacturer’s standard operating procedures were followed (Appendix four, five, six). The value of LDL-C was calculated from the Friedwald’s formula: \( \text{LDL-C (mg/dl)} = \text{TC} - \text{HDL} - (\text{TG}/5) \). Subjects who had eaten before the test were given convenient time to come for the test. Also, the results of the test were made known to them and subsequently they were educated and counseled appropriately.

### 3.13 DIAGNOSTIC CRITERIA

#### 3.13.1 Diagnosis of Obesity

Obesity is defined as \( \text{BMI} \geq 30 \text{kg/m}^2 \) and categorized into: class 1 obesity (mild obesity) = BMI 30-34.9; class II obesity (moderate obesity) = BMI 35-39.9 and class III obesity (severe obesity) = BMI ≥40.

#### 3.13.2 Diagnosis of Hypertension

Hypertension is defined as systolic and/or diastolic blood pressure \( \text{BP} \geq 140/90 \text{mmHg} \) in tandem with JNC VII report on prevention, evaluation and treatment of high blood pressure in adults aged 18 years and older.

#### 3.13.3 Diagnosis of Diabetes Mellitus

A fasting venous plasma glucose of \( \geq 126 \text{mg/dl} \) after over night fast on two occasions was taken as diabetes mellitus.

#### 3.13.4 Diagnosis of Dyslipidaemia

Dyslipidaemia (abnormal lipid profile) is defined as serum Total Cholesterol (TC) \( \geq 200 \text{mg/dl} \) (5.17mmol/l), Triglyceride (TG) \( \geq 150 \text{mg/dl} \) (1.7mmol/l), Low Density Lipoprotein Cholesterol (LDL-C) \( \geq 100 \text{mg/dl} \) (2.58mmol/l) and High Density Lipoprotein Cholesterol (HDL-C) \( < 40 \text{mg/dl} \) (1.03mmol/l).
3.14 DATA COLLECTION

Data was collected on every clinic day using the structured and pre-tested questionnaire (appendix seven) that was completed through oral interview, by the author, on new patients that agreed to participate in the study after explanation and informed consent had been signed (appendix eight). The stadiometer, sphygmomanometer and Littman stethoscope were checked everyday to ensure validity of the instruments. The blood pressure measurements, samples for fasting plasma glucose and lipid profile were done as enumerated in the diagnostic (sample) procedure. The socio-demographic variables, anthropometric variables, blood pressure measurements, fasting plasma glucose, fasting lipid profiles and socio-economic class were recorded in the questionnaire. The socio-economic status were classified into upper, middle and lower socio-economic strata (appendix nine) to suit Nigerian environment. Patients awareness of his/her obese condition, source of awareness of the information on the obese condition and knowledge of various methods of lifestyle modification for obese condition were assessed and recorded in the questionnaire. The knowledge level on methods of lifestyle modification for obese condition was graded into: No knowledge (none of the 3 ways), low knowledge (1 out of 3 ways), moderate level (2 out of the 3 ways) and high level (knows all the 3 ways) were recorded.

3.15 DATA ANALYSIS

Data was analyzed using statistical package for the social sciences (SPSS) version 17. Relevant means and standard deviations were calculated alongside with
appropriate frequencies. Findings were presented in frequency tables and charts including pie and bar charts.

Further analyses were done to assess their awareness, knowledge of obese condition and knowledge of lifestyle measures for obese condition. Also test of association between awareness of obese condition and socio-demographic characteristics and obesity and co-morbid conditions and socio-economic factors were done using chi-square test. Also bi-variate analysis was further done to ascertain the relationship between blood pressure, fasting blood sugar, lipid profile and BMI. P-value < 0.05 was assumed to be statistically significant.

3.16 ETHICAL CONSIDERATION

Ethical clearance certificate (appendix ten) was obtained from the Ethical Committee of the Federal Medical Centre, Owerri. The consent form for the study were also signed by the subjects. The subjects’ identities were not published or disclosed. The patients included in the study were informed of their BMI, blood pressure, fasting plasma glucose level and lipid profile level. They were educated and counselled on lifestyle modifications such as weight loss, healthy diet and exercise. In addition, those found to have obesity related co-morbid conditions were referred appropriately.

3.17 FUNDING

The research was funded by the research grant from the hospital and supplemented by the researcher. Necessary instruments and materials were provided by the department.
CHAPTER FOUR

SUMMARY OF RESULT

A total of 2,391 patients were seen within the study period out of which 618 of them were obese, thus giving a prevalence rate of 25.8%. However, a subsample of 206 obese patients were systematically selected by using every third obese patient (1 in 3) for the study. Among the 206 obese patients, 139 (68%) had class I obesity (i.e BMI: 30-34.9), 48 (23%) had class II obesity (i.e BMI: 35-39.9) and 19 (9%) had class III obesity (i.e BMI: ≥40).

The age of the obese patients ranged from 19 to 76 years with mean age of 44.71 years ±13.41 years. There were 33 (16.0%) males and 173 (84.0%) females, with male to female ratio of 1:5.2. Majority of the obese patients were married (67.5%), Christians (98.5%), had tertiary education (38.8%), engaged in trading (30.6%), with majority earning between N10,000 to N45,000 per month (42.7%) and belonged to lower socio-economic class (67.5%).

Eighty-eight (42.7%) of the obese patients were hypertensive; while thirty-three (16.1%) of the obese patients were diabetic and eight-nine (43.2%) of the obese patients had dyslipidemia with most having low HDL cholesterol (49.4%), and 34.5% among them had multiple abnormal lipid levels.
Majority 152 (74%) were not aware of their obese condition while only fifty-four (26%) were aware of their obese condition. Majority (75.9%) of those who were aware of their obese condition knew by self. Most 115 (55.8%) had no knowledge of lifestyle modifications for their obese condition and none (0.0%) had high knowledge level. The findings and further details of the results are enumerated below.
There were 33 (16.0%) males and 173 (84%) females with male to female ratio of 1:5.2. Most of the patients were females (Table I).

**TABLE 1: SEX DISTRIBUTION OF THE PATIENTS:**

<table>
<thead>
<tr>
<th>Sex</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>33</td>
<td>16.0</td>
</tr>
<tr>
<td>Female</td>
<td>173</td>
<td>84.0</td>
</tr>
<tr>
<td>Total</td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
The age of the patients ranged from 19 years to 76 years. The age group 48-57 years constituted the highest number of obese patients 49 (23.8%) followed by the age group 28-37 years with 48 (23.3%), while the age group 68-77 years constituted the least number of obese patient 11 (5.3%). The mean age of all the patients was 44.71 years±13.41 years. While the mean age of the males and females were 50.5±2.6 years and 43.6±1.0 years respectively (Table 2).

**TABLE 2: AGE DISTRIBUTION OF THE PATIENTS**

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-27</td>
<td>23</td>
<td>11.2</td>
</tr>
<tr>
<td>28-37</td>
<td>48</td>
<td>23.3</td>
</tr>
<tr>
<td>38-47</td>
<td>47</td>
<td>22.8</td>
</tr>
<tr>
<td>48-57</td>
<td>49</td>
<td>23.8</td>
</tr>
<tr>
<td>58-67</td>
<td>28</td>
<td>13.6</td>
</tr>
<tr>
<td>68-77</td>
<td>11</td>
<td>5.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
Table 3 below showed thirteen (6.3%) of the patients had no formal education while 38 (18.4%) of them had primary education; however a greater proportion of them had tertiary education 80 (38.8%).

**TABLE 3: DISTRIBUTION OF THE PATIENTS BY EDUCATIONAL STATUS**

<table>
<thead>
<tr>
<th>Educational Status</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No formal</td>
<td>13</td>
<td>6.4</td>
</tr>
<tr>
<td>Primary</td>
<td>38</td>
<td>18.4</td>
</tr>
<tr>
<td>Secondary</td>
<td>57</td>
<td>27.7</td>
</tr>
<tr>
<td>Tertiary</td>
<td>80</td>
<td>38.8</td>
</tr>
<tr>
<td>Post-tertiary</td>
<td>18</td>
<td>8.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Table 4 below showed most of the respondents were married 139 (67.5%) while 28 (13.6%) were single. The widowed were 36 (17.4%) while the separated/divorced constitute 3 (1.5%)

**TABLE 4: DISTRIBUTION OF THE PATIENTS BY MARITAL STATUS:**

<table>
<thead>
<tr>
<th>Marital status</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single</td>
<td>28</td>
<td>13.6</td>
</tr>
<tr>
<td>Married</td>
<td>139</td>
<td>67.5</td>
</tr>
<tr>
<td>Widowed</td>
<td>36</td>
<td>17.4</td>
</tr>
<tr>
<td>Separated/Divorced</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
The patients were predominantly Christians 203 (98.5%) while 3 (1.5%) were moslems (table 5).

**TABLE 5: DISTRIBUTION OF PATIENTS BY RELIGION**

<table>
<thead>
<tr>
<th>Religion</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Christian</td>
<td>203</td>
<td>98.5</td>
</tr>
<tr>
<td>Moslem</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Others: Traditionalist, Atheist</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
The predominant occupations among the subjects were trading 63 (30.6%) and civil servants 60 (29.1%). While 5 (2.4%) were involved in farming and 28 (13.6%) were unemployed. The students/apprentice were 14 (6.8%), artisans 12 (5.8%) and health workers 4 (1.9%) as depicted in the table 6 below.

**TABLE 6: DISTRIBUTION OF PATIENTS BY OCCUPATION**

<table>
<thead>
<tr>
<th>Occupation</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trader</td>
<td>63</td>
<td>30.6</td>
</tr>
<tr>
<td>Civil Servant</td>
<td>60</td>
<td>29.1</td>
</tr>
<tr>
<td>Unemployed</td>
<td>28</td>
<td>13.6</td>
</tr>
<tr>
<td>Student/Apprentice</td>
<td>14</td>
<td>6.8</td>
</tr>
<tr>
<td>Artisans</td>
<td>12</td>
<td>5.8</td>
</tr>
<tr>
<td>Professionals</td>
<td>7</td>
<td>3.4</td>
</tr>
<tr>
<td>Retired civil servants</td>
<td>7</td>
<td>3.4</td>
</tr>
<tr>
<td>Farmer</td>
<td>5</td>
<td>2.4</td>
</tr>
<tr>
<td>Health workers</td>
<td>4</td>
<td>1.9</td>
</tr>
<tr>
<td>Clergy</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Others-Publisher/Contra/Sports</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Primary sources of income were through self effort 82 (39.8%) and government 65 (31.6%) as depicted in the table 7 below.

**TABLE 7: DISTRIBUTION OF SOURCES OF INCOME FOR THE PATIENTS**

<table>
<thead>
<tr>
<th>Source of Income</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self employed</td>
<td>82</td>
<td>39.8</td>
</tr>
<tr>
<td>Government</td>
<td>65</td>
<td>31.6</td>
</tr>
<tr>
<td>Private Sector</td>
<td>13</td>
<td>6.3</td>
</tr>
<tr>
<td>Husband</td>
<td>14</td>
<td>6.8</td>
</tr>
<tr>
<td>Parents</td>
<td>13</td>
<td>6.3</td>
</tr>
<tr>
<td>Children</td>
<td>11</td>
<td>5.3</td>
</tr>
<tr>
<td>Others — church, friends and relatives</td>
<td>8</td>
<td>3.9</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Majority of the patients (modal income level) earn between N10,000.00 and N45,000.00 per month 88 (42.7%) followed by 78 (37.9%) patients that earn less than N10,000.00 while respondents earning N150,000.00 and above constitute 2 (1.0%) as shown in the table 8 below.

**TABLE 8: DISTRIBUTION OF PATIENTS BY MONTHLY INCOME**

<table>
<thead>
<tr>
<th>Income per Month (in Naira)</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10,000</td>
<td>78</td>
<td>37.9</td>
</tr>
<tr>
<td>10,000-45,000</td>
<td>88</td>
<td>42.7</td>
</tr>
<tr>
<td>45,000-150,000</td>
<td>38</td>
<td>18.4</td>
</tr>
<tr>
<td>&gt;150,000</td>
<td>2</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
Table 9 below showed that most of the patients belong to the lower class 139 (67.5%) while 47 (22.8%) constitute middle class and only 20 (9.7%) belong to the upper class.

**TABLE 9: DISTRIBUTION OF PATIENTS BY SOCIO-ECONOMIC CLASS**

<table>
<thead>
<tr>
<th>Socio-economic Class</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower class</td>
<td>139</td>
<td>67.5</td>
</tr>
<tr>
<td>Middle class</td>
<td>47</td>
<td>22.8</td>
</tr>
<tr>
<td>Upper class</td>
<td>20</td>
<td>9.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>206</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Most of the patients 68% were found to have mild obesity while 23% had moderate obesity and only 9% had severe obesity as shown in figure 1 below.

**FIGURE 1: CLASSIFICATION OF OBESITY BASED ON BMI:**
The pattern of co-morbid conditions observed among the patients were diabetes (16.1%), hypertension (42.7%) and dyslipidaemia (43.2%) (figure 2).

**FIGURE 2: PATTERN OF CO-MORBID CONDITIONS AMONG THE OBESE PATIENTS**

![Bar chart showing the percentages of diabetes mellitus, hypertension, and dyslipidaemia among obese patients.](image)
Among the hypertensives, most 67 (76.2%) had both systolic and diastolic hypertension while 13 (14.7%) and 8 (9.1%) had only systolic and diastolic hypertension respectively. Most of the patients with dyslipidemia had low HDL 44 (49.4%), elevated LDL 39 (43.8%) and least number of them 18 (20.2%) had high TC level. However 30 (34.5%) of them had multiple lipid abnormality. Also 33(16.1%) of the obese subjects were confirmed diabetics as shown in the table 10 below.

**TABLE 10: PATTERN OF CO-MORBIDITY AMONG THE OBESE PATIENTS**

<table>
<thead>
<tr>
<th>Types of Hypertension</th>
<th>N=88</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic Hypertension only</td>
<td>13</td>
<td>14.7</td>
</tr>
<tr>
<td>Diastolic Hypertension only</td>
<td>8</td>
<td>9.1</td>
</tr>
<tr>
<td>Both</td>
<td>67</td>
<td>76.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Abnormal Lipid Profile</th>
<th>N=89</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated Cholesterol</td>
<td>18</td>
<td>20.2</td>
</tr>
<tr>
<td>Elevated Triglycerides</td>
<td>25</td>
<td>28.1</td>
</tr>
<tr>
<td>Elevated LDL</td>
<td>39</td>
<td>43.8</td>
</tr>
<tr>
<td>Low HDL</td>
<td>44</td>
<td>49.4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diabetes Mellitus</th>
<th>N=206</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirmed diabetes</td>
<td>33</td>
<td>16.1</td>
</tr>
<tr>
<td>Non diabetes</td>
<td>173</td>
<td>83.9</td>
</tr>
</tbody>
</table>
Most of the obese patients (74%) were not aware of their obese condition while only 26% of the patients were aware of their obese condition (figure 3).

FIGURE 3: DISTRIBUTION OF PATIENTS BASED ON AWARENESS OF OBESE CONDITION
Among those who were aware of their obese condition, most knew through self awareness 43(79.6%), followed by friends/peers 6(11.1%); family members 2(3.7%) while only 3(5.6%) were informed of their obese condition by a health professional (table 11).

**TABLE 11: DISTRIBUTION OF PATIENTS BASED ON SOURCE OF AWARENESS OF OBESE CONDITION**

<table>
<thead>
<tr>
<th>Source of awareness for obese condition</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self</td>
<td>43</td>
<td>79.6</td>
</tr>
<tr>
<td>Friends/peers</td>
<td>6</td>
<td>11.1</td>
</tr>
<tr>
<td>Health professional</td>
<td>3</td>
<td>5.6</td>
</tr>
<tr>
<td>Family member</td>
<td>2</td>
<td>3.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>54</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Most of the obese patients 115(55.8%) had no knowledge of lifestyle modification measures for obesity while 32(15.5%) and 59(28.7%) had low and moderate levels respectively. None of them (0.0%) had high knowledge level of lifestyle modification for their obese condition (table 12).

**TABLE 12: DISTRIBUTION OF LEVEL OF KNOWLEDGE OF LIFESTYLE MODIFICATION FOR OBESE CONDITION**

<table>
<thead>
<tr>
<th>Level of Knowledge of lifestyle modification for obese condition</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No knowledge</td>
<td>115</td>
<td>55.8</td>
</tr>
<tr>
<td>Low knowledge</td>
<td>32</td>
<td>15.5</td>
</tr>
<tr>
<td>Moderate knowledge</td>
<td>59</td>
<td>28.7</td>
</tr>
<tr>
<td>High knowledge</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>206</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
No association was demonstrated between knowledge of lifestyle modification for obesity and awareness of obese condition, however patients with mild and moderate obesity were less likely to be aware of their obese condition ($X^2=8.29$, $p=0.016$) as shown in table 13 below.

**TABLE 13: ASSOCIATION BETWEEN AWARENESS OF OBESE CONDITION, KNOWLEDGE OF LIFESTYLE MODIFICATION AND TYPE OF OBESITY**

<table>
<thead>
<tr>
<th>Level of knowledge of lifestyle modification for obesity</th>
<th>Awareness of obese condition</th>
<th>$X^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes n=54 (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Knowledge</td>
<td>27 (23.4)</td>
<td>88 (76.6)</td>
<td>1.07</td>
</tr>
<tr>
<td>Low knowledge</td>
<td>9 (28.1)</td>
<td>23 (71.9)</td>
<td></td>
</tr>
<tr>
<td>Moderate knowledge</td>
<td>18 (31.0)</td>
<td>41 (69.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Type of obesity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild Obesity</td>
<td>28 (20.1)</td>
<td>111 (79.9)</td>
<td>8.29</td>
</tr>
<tr>
<td>Moderate Obesity</td>
<td>18 (37.5)</td>
<td>30 (62.5)</td>
<td></td>
</tr>
<tr>
<td>Severe Obesity</td>
<td>8 (42.1)</td>
<td>11 (57.9)</td>
<td></td>
</tr>
</tbody>
</table>
Association was demonstrated between awareness of obese condition and marital status ($\chi^2=7.51$, $p=0.023$) and educational status ($\chi^2=8.28$, $p=0.041$). Widowed/separated/divorced were more likely to be aware of their obese condition (table 14).

**TABLE 14: ASSOCIATION BETWEEN AWARENESS OF OBESE CONDITION AND SOCIAL FACTORS**

<table>
<thead>
<tr>
<th>Social factors</th>
<th>Awareness of obese condition</th>
<th>$\chi^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes n=54 (%)</td>
<td>No n=152 (%)</td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>7 (21.2)</td>
<td>26 (78.8)</td>
<td>0.51</td>
</tr>
<tr>
<td>Female</td>
<td>47 (27.2)</td>
<td>126 (72.8)</td>
<td></td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>13 (46.4)</td>
<td>15 (53.6)</td>
<td>7.51</td>
</tr>
<tr>
<td>Married</td>
<td>34 (24.5)</td>
<td>105 (75.5)</td>
<td></td>
</tr>
<tr>
<td>Others (Widowed/separated/divorced)</td>
<td>7 (17.9)</td>
<td>32 (82.1)</td>
<td></td>
</tr>
<tr>
<td><strong>Educational status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal</td>
<td>3 (23.1)</td>
<td>10 (76.9)</td>
<td>8.28</td>
</tr>
<tr>
<td>Primary</td>
<td>8 (21.1)</td>
<td>30 (78.9)</td>
<td>df=3</td>
</tr>
<tr>
<td>Secondary</td>
<td>18 (31.6)</td>
<td>39 (68.4)</td>
<td></td>
</tr>
<tr>
<td>Tertiary</td>
<td>16 (20.0)</td>
<td>64 (80.0)</td>
<td></td>
</tr>
<tr>
<td>Post-tertiary</td>
<td>9 (50.0)</td>
<td>9 (50.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Socio-economic class</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower</td>
<td>33 (23.7)</td>
<td>106 (76.3)</td>
<td>2.46</td>
</tr>
<tr>
<td>Middle</td>
<td>13 (27.7)</td>
<td>34 (72.3)</td>
<td></td>
</tr>
<tr>
<td>Upper</td>
<td>8 (40.0)</td>
<td>12 (60.0)</td>
<td></td>
</tr>
</tbody>
</table>
There is association between marital status and degree of obesity. Those that were Widowed/separated/divorced were more likely to have moderate or severe obesity (table 15).

**TABLE 15: ASSOCIATION BETWEEN SELECTED SOCIAL FACTORS AND OBESITY**

<table>
<thead>
<tr>
<th>Social factors</th>
<th>Classification of Obesity</th>
<th>$\chi^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild (n=139)</td>
<td>Moderate (n=48)</td>
<td>Severe (n=19)</td>
</tr>
<tr>
<td></td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>27 (81.8)</td>
<td>4 (12.1)</td>
<td>2 (6.1)</td>
</tr>
<tr>
<td>Female</td>
<td>112 (64.7)</td>
<td>44 (25.4)</td>
<td>17 (9.8)</td>
</tr>
<tr>
<td><strong>Educational Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>9 (69.2)</td>
<td>2 (15.4)</td>
<td>2 (15.4)</td>
</tr>
<tr>
<td>Primary</td>
<td>28 (73.7)</td>
<td>7 (18.4)</td>
<td>3 (7.9)</td>
</tr>
<tr>
<td>Secondary</td>
<td>36 (63.2)</td>
<td>16 (28.1)</td>
<td>5 (8.8)</td>
</tr>
<tr>
<td>Tertiary</td>
<td>55 (68.8)</td>
<td>19 (23.8)</td>
<td>6 (7.5)</td>
</tr>
<tr>
<td>Post-tertiary</td>
<td>11 (61.1)</td>
<td>4 (22.2)</td>
<td>3 (16.7)</td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>19 (67.9)</td>
<td>7 (25.0)</td>
<td>2 (7.1)</td>
</tr>
<tr>
<td>Married</td>
<td>97 (69.8)</td>
<td>31 (22.3)</td>
<td>11 (7.9)</td>
</tr>
<tr>
<td>Others</td>
<td>23 (59.0)</td>
<td>10 (25.6)</td>
<td>6 (15.4)</td>
</tr>
<tr>
<td>(Widowed/separated/divorced)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Socio-economic Class</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>96 (69.1)</td>
<td>31 (22.3)</td>
<td>12 (8.6)</td>
</tr>
<tr>
<td>Middle</td>
<td>28 (59.6)</td>
<td>13 (27.7)</td>
<td>6 (12.8)</td>
</tr>
<tr>
<td>Upper</td>
<td>15 (75.0)</td>
<td>4 (20.0)</td>
<td>1 (5.0)</td>
</tr>
</tbody>
</table>
No significant association was demonstrated between the co-morbidity and class of obesity using Chi-square test (table 16).

**TABLE 16: ASSOCIATION BETWEEN CLASS OF OBESITY AND CO-MORBIDITY:**

<table>
<thead>
<tr>
<th>Co-morbid conditions</th>
<th>Classification of Obesity</th>
<th>X²</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild n=139 (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Moderate n=48 (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe n=19 (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>56 (63.6)</td>
<td>2.14</td>
<td>0.342</td>
</tr>
<tr>
<td>No</td>
<td>83 (70.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>23 (69.7)</td>
<td>0.10</td>
<td>0.95</td>
</tr>
<tr>
<td>No</td>
<td>116 (67.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>61 (69.0)</td>
<td>2.67</td>
<td>0.263</td>
</tr>
<tr>
<td>No</td>
<td>78 (66.7)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

However, further analysis done using correlations between BMI and average blood pressure (both systolic and diastolic), lipid profile and FBS. Both average systolic (r=0.21, p=0.003) and diastolic (r=0.27, p=0.000) blood pressure were found to be significantly correlated with the patients’ BMI. Also, there was a positive correlation between the patient’s total triglycerides and BMI but was not statistically significant (r=0.082, p=0.240). Among patients’ with mild and moderate obesity there was negative correlation between their HDL level and BMI (r=-0.033, p=0.656), while patients with mild obesity demonstrated weakly positive correlation between LDL and BMI (r=0.001, p=0.99). Patients with moderate and severe obesity were also found to have weak positive correlation between FBS and BMI (r=0.081, p=0.516).
CHAPTER FIVE

DISCUSSION

This study has revealed high prevalence of obesity among the study population with majority (68%) having class I obesity. Most of the obese patients belonged to the age group 48 – 57 years (28.3%), were married (67.5%), had tertiary education (38.8%), were traders (30.6%) and belonged to lower socio-economic class (67.5%). There was significant association between marital status and degree of obesity ($x^2 = 27.99$, df = 2, p value = 0.000). Among the co-morbid conditions studied, dyslipidaemia (43.2%) had the highest prevalence rate. The systolic and diastolic blood pressures, triglycerides, low density lipoprotein cholesterol and fasting blood sugar correlated with BMI. Majority of the subjects were not aware (74%) of their obese condition and had no knowledge (55.8%) of lifestyle modifications for obese condition.

The prevalence rate of 25.8% in this study is higher than that reported in Portharcourt (14.0%),\(^{11}\) Ilorin (13.2%),\(^{204}\) and other parts of Africa like Greater Accra (16.1%)\(^{51}\) and Cameroon (17.1%);\(^{65}\) and also Jamaica (24.0%).\(^{50}\) But lower to reported prevalence of 39% in Ibadan\(^{151}\) and 26.6% reported in USA.\(^{45}\) The relative disparities between the prevalence rates observed in this study and other studies were probably due to epidemiological characteristics of the study population and a reflection of increasing trends in consumption of high calorie and junk foods driven by numerous fast food outlets. The similar prevalence rates reported in developed countries such as USA further underscore the observation that prevalence
rates of obesity in developing countries are attaining epidemic proportions with variations across regions and countries.

The pattern of obesity seen in this study showed highest prevalence of class I obesity (68%) among the study population. This agreed with pattern reported from studies\textsuperscript{151,205} in Ibadan and in other parts of Nigeria.\textsuperscript{11,70,206} Those with class I and II obesity were less likely to be aware of their obese condition. This pattern is probably due to the observation that body build at these levels are culturally and socially desirable and acceptable in the environment and not regarded as a pathological condition.

The finding in this study of higher prevalence rates of obesity among the young and middle age groups is similar to reports from Ibadan,\textsuperscript{205} PortHarcourt,\textsuperscript{11} Calabar,\textsuperscript{157} Cameroon\textsuperscript{64} and Ghana.\textsuperscript{51} The low prevalence rate seen in older age group (5.3%) is in tandem with other studies.\textsuperscript{51,52,64} This trend of obesity probably is from the adopted diet behavioral patterns with more people eating fast food in preference to home food and more sedentary lifestyle perceived as a sign of wealth among the middle aged group in the environment. Also studies had reported that BMI increases with age.\textsuperscript{157,207} However, it had been shown that anthropometric determinants of obesity using BMI criterion could be a less valid indicator of obesity among the elderly who tend to have a shift of fat from peripheral to central sites with progressive distribution of the fat stores more in the visceral region. Moreso, aging is associated with relative reduction in fat free mass especially muscle mass with relative increase in fat mass.\textsuperscript{208} Obesity therefore increases with advancing age as physical activities diminish and central obesity rises as peripheral fat is diverted to
central sites. In such population, waist indices (Waist Circumference, Waist-Hip ratio) a measure of central obesity is preferred.

This study also observed higher prevalence of obesity among females (84.0%) compared to males (16.0%). High female rates were reported in Ibadan (female 50.4%, male 49.6%), PortHarcourt (females 62.1%, males 37.9%), Enugu (females 13.1%, males 8.1%), Ilorin (females 7.8%, males 5.3%) and Northern Nigeria (female 22.0%, male 11.2%); and in countries like Cameroon (females 19.5%, male 6.5%) and Ghana (female 7.4%, male 2.8%). This higher female prevalence rate was also reported in other studies. This may be attributed to socio-cultural factors, changes in the energy density of diets, physical activity and genetic differences between the sexes.

The study also found that married women have higher prevalence of obesity. Furthermore, there was association between marital status and development of obesity. This is in keeping with finding in Ibadan which reported higher prevalence rate of obesity amongst the married patient. Other studies also supported this finding. This is probably due to socio-cultural belief of obesity as evidence of “good husband care”, beauty, general dietary habit of married people (increase sized portion during family meals) in the study area. Also married women are likely to be multiparous which is associated with high risk of obesity.

This study found higher prevalence of obesity among respondents with tertiary education (38.8%), this is similar to the findings in Ibadan (39.5%) and other developing countries. This contrasts with reports of more prevalence of obesity among non-educated in developed countries. This may be due to westernization
of lifestyle, reduced physical activities, more sedentary lifestyle and consumption of high energy, high fat diets commonly seen among the more educated class in Nigeria. However, as these obesity-related factors become more generalized in developing countries, the familiar inverse association of obesity with educational level will probably dominate.

The predominant occupation of the respondents was trading (30.6%); with self employment (39.8%) being the major source of income. Also, it was found that most (42.7%) respondents earned between ₦10,000 to ₦45,000 and most (67.5%) belonged to low socio-economic class. This is in contrast to other studies which showed higher obesity rates among the high income and socio-economic class. It had been found that obesity rates had been on increase in many low and middle income countries, with burden of obesity shifting towards the poor and lower socio-economic class. It portrays the fact that though the material wealth of citizens over the years is still abysmal, the lifestyle habits required to develop and sustain obesity do not require general economic development. Instead, it may reflect the penetration of market based consumption patterns into stagnating or declining economies.

Dyslipidaemia was the commonest (43.2%) primary comorbidity among the study population. The prevalence and pattern of dyslipidaemia in this study are similar to the pattern reported in previous studies with low HDL –C being the most frequent lipid abnormality. It had been shown that dyslipidaemia is becoming an important medical problem in both developed and developing countries and
associated with obesity in a clustering of medical conditions known as dysmetabolic syndrome.\textsuperscript{202,211}

The prevalence rate of hypertension among the obese patients in this study was 42.7\% with majority of the hypertensives (76.2\%) having both systolic and diastolic hypertension. This is comparable to findings from developed countries like UK (42.0\%);\textsuperscript{213} but higher than in other Nigerian studies like Ibadan (35.0\%),\textsuperscript{151} Edo (20.2\%),\textsuperscript{214} Ilorin (27.1\%).\textsuperscript{204} Moreso, this is more than the extrapolated prevalence rate of 20\% from the 1997 nationwide survey of non-communicable diseases in Nigeria\textsuperscript{18} These findings highlight the burden of hypertension among the study population and corroborate the report that prevalence of hypertension is on the increase in Nigeria.\textsuperscript{18,214} According to these reports from Nigeria, hypertension is the commonest non-communicable disease in Nigeria\textsuperscript{18,215} and has become a public health problem worldwide especially in sub-Saharan African where it is increasing in importance as a component of non-communicable disease burden and a major cause of cardiovascular morbidity and mortality.

Diabetes mellitus was seen in 16.1\% of the study population. This is lower than report in Ibadan (19.7\%)\textsuperscript{151} and higher than Lagos (14.7\%).\textsuperscript{72} Dyslipidaemia is a documented risk factor for numerous chronic conditions including diabetes mellitus\textsuperscript{216} most commonly shown in metabolic syndrome. The increase in number of fat cells alters body’s response to insulin, potentially leading to insulin resistance that result in the development of type 2 diabetes mellitus.

Further analysis of data from this study showed association between marital status and degree of obesity as seen in another study.\textsuperscript{205} Also, there was correlation
between BMI and TG, LDL-C levels as reported in other studies.\textsuperscript{11,151} Body mass index also correlated with systolic and diastolic hypertension in agreement with other reports, \textsuperscript{72,151,217,218} and with FBS which is consistent with other reports.\textsuperscript{11,151}

The author found low awareness (26\%) and perception of obese condition among the study population; with only 5.6\% informed by health care professionals. This gave credence to the observation that obesity is socially and culturally acceptable among Nigerians as an indication of good health and affluence and not usually regarded as a medical condition. The study also found association between awareness of obese condition and marital status and educational status. The widowed/separated/divorced were more likely to be aware of their obese condition. This may be due to their quest to be healthy, look slimmer and younger. The more educated will likely acquire more personal information from books, internets and media about obesity. This was seen in the study, as most respondents were aware of obese condition through self effort (79.6\%).

Interestingly, majority of obese population had no knowledge of lifestyle modification for obese condition (55.8\%), and among those with knowledge, none had high knowledge of the lifestyle practice. These are due to patient and health personnel related factors in health care service delivery. With inadequate level of health manpower, poor knowledge of obesity by health workers and time constraints, health personnel sometimes fail to assess for obesity and explain to the obese patients in sufficient detail the health consequences of obesity. This lack of information is not limited to the health consequences of obesity but also the benefits of lifestyle modifications. Information based on knowledge is power, while there is
needs to improve the knowledge of healthcare workers on obesity and its health hazards, this knowledge needs to be passed on to the real beneficiaries—the patients. If the general patients are aware of their obese condition, health hazards of obesity as well as modifiable predisposing factors of obesity, then it may be easier to educate them on lifestyle modifications especially healthy diet, physical activity and behavioural modifications.

5.1 LIMITATIONS OF THE STUDY

Few local studies, literatures, randomized and meta-analyses data were available for referencing in our environment.

The study was hospital based as only patients who presented to the clinic were recruited. This may not be a true representation of what may be obtained in the larger community.

5.2 CONCLUSION

This study had demonstrated high prevalence of obesity among the study population with class I obesity being the most common pattern and dyslipidaemia the commonest primary co-morbidity. The dyslipidaemia, hypertension and diabetes mellitus invariably predispose these obese patients to increase cardiovascular morbidity and mortality. Identification and implementation of patient, family and community-based intervention programmes that are effective, efficient, widely practicable and culturally acceptable are advocated. These intervention measures should be considered by health planners and policy makers in the formulation of health policies and programmes geared towards reducing the burden of obesity and
its primary co-morbidities. Family physicians should therefore make a diagnosis of obesity, screen for its primary co-morbidities and more importantly counsel patients on the need for lifestyle modifications such as weight reduction.

5.3 RECOMMENDATIONS

The study has shown the existence of obesity and its primary co-morbidities among the study population. It is recommended based on the results of this study that:

1. Anthropometric determination of obesity and screening for its common primary co-morbidities should be integrated as part of clinic baseline assessment of adult Nigerian attending General Outpatient clinics.

2. A planned proactive follow up is necessary. Those with primary co-morbidities should be secondary target for risk reduction therapy and appropriate management.

3. Long term targeted primary prevention intervention programmes on obesity should be instituted in the study area.

4. Family physicians working in health care settings should educate and counsel their patients on healthy eating, social habits as well as physical activity.

5. Food industries should include healthy food choices to their menus. Also support for safe and convenient venues for physical activity such as walking paths, bicycle lane and wellness programmes in work place are recommended for implementation by relevant institutions.

6. There should be a comprehensive strategy to address poor awareness of obesity and poor knowledge of lifestyle modifications for obesity among the study population.

7. There should be advertisement restrictions and tax on foods with high energy dense, low nutrient values and subsidies on healthy foods.
In the study area, the following further studies are recommended:

1. To provide more reliable epidemiological and clinical data, further studies on the epidemiology and management of obesity is recommended.

2. To provide idea on burden of obesity and its co-morbidities in the study area, community-based and hospital-based case-control studies are recommended in order to effect population based prevention and control.
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APPENDIX ONE

Stadiometer: Health scale with height manufactured by Techmel and Techmel USA TT120

STANDARD OPERATION PROCEDURE

Specifications-

1. Body weighing

   Maximum Weight 120kgs. (265 lbs).

   Sub-divisions of the weighing dial 0.5kg

2. Height Measuring

   Height Measuring Range 70-190cm. (27 ½ -74 3/4in.)

   Sub-divisions of graduation 0.5cm

How to take reading of weight measurement

This weighing machine is well constructed with great precision and high sensitivity. A self-indicating dial is equipped for easy reading, showing accurate weight both in Metric and in British systems by an indicator which is connected with the lever mechanism by means of gears and coiled spring. When not in use, the indicator should point at the “o” position. If there is any deviation, adjust the indicator by turning the screw under the dial.
How to take reading of height measurement

The height measuring standard is composed of three round tubes of different calibres: the outer tube is rigidly fixed onto the column of the dial, while the middle and the inner tubes are closely inserted therein one after the other with both Metric and British units graduated thereon. On the top of the inner tube, there is a movable headpiece which should be pulled out at right angle with the measuring standard before use. When measuring height, first stretch the inner tube to its full length sufficient for a range of 70-126cm (27 ½-49 ½in). The reading can be obtained from the point where the graduation of the inner tube coincides with upper flange edge of the medium tube. In case the height is over 126cm, then pull out the middle tube for a measuring range of 126-190cm. (49 ½-74 3/4in.), and the reading is to be taken from the point where the graduation of the middle tube and the upper flange edge of the outer tube join together.
APPENDIX TWO

ACCUSON MERCURIAL SPHYGMOMANOMETER

GUIDELINES AND PRECAUTIONS

A mercurial sphygmomanometer should be handled with care. Do not drop the instrument or treat in any way that could result in damage to the manometer. Regular checks should be made to ensure there are no leaks from the inflation system, and that the manometer has not been damaged, resulting in a loss of mercury.

CLEANING THE MANOMETER TUBE

To obtain the best results from a mercurial sphygmomanometer the manometer tube should be cleaned at regular intervals. This will ensure the mercury can move up and down the tube freely, and respond quickly to changes in pressure in the arm cuff.
APPENDIX THREE

FBS Determination: Standard Operation Precautions/guidelines by Giesse diagnostics

GLUCOSE SL

Enzymatic colorimetric method (GOD-POD)

Quantitative determination of glucose in serum, plasma.

PRINCIPLE

The enzymatic methods uses Glucose oxidase (GOD) to catalyze the oxidation of glucose to hydrogen peroxide and gluconic acid. Hydrogen peroxide, when combined with 4- aminoantipyrine and 4 - hydroxybenzoic acid, forms a red dye compound. The intensity of the red colour produced is directly proportional to the glucose quantity in the sample.

SPECIMEN

Unhemolyzed fresh serum, plasma with heparin.

Serum must be separated from clot promptly.

Glucose in serum is stable for 24 hours at 2-8°C, and 8 hours at room temperature.

Shake and bring samples at room temperature before using.

REAGENTS

<table>
<thead>
<tr>
<th>Reagent (A) GLU Liquid Vol = 100/250/1000 ml</th>
<th>Buffer Glucose Oxidase 100 mmol/L</th>
<th>10000 U/L</th>
<th>Peroxidase 2000 U/L</th>
<th>4-AAP 1 mmol/L</th>
<th>4-hydroxibenzonic acid 10 mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard (B) GLU Liquid Vol = 10 ml</td>
<td>Glucose (100 mg/dl)</td>
<td>(5.56 mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**PACKAGE: Collection and Storage**

Store in refrigerator (2-8°C)

Stable until the expiry date indicated on the bottle.

After the unsealing and the taking of the reagent, it is advised to close up the bottle immediately in order to avoid evaporation, direct light exposure and bacteric contamination.

**PRECAUTIONS/DANGER SYMBOLS**

The preparation, according to current regulation, is classified as not dangerous. The total concentration of non active components (preservatives, detergents, stabilizers) is below the minimum required for citation.

Always handle with care, avoid ingestion, avoid contact with eyes, skin and mucous membranes. The samples must be handled as potentially infected from HIV or Hepatitis.

**REAGENT PREPARATION STABILITY**

Ready to use Liquid Reagent. The Reagent must be at room temperature before using.

The Reagent is liquid and rose-coloured. Pale colouring of the reagent (<0.050 O.D.) due to air light exposure doesn’t compromise the working.
REQUIRED MATERIALS NOT PROVIDED

General Laboratory Equipment and Instrumentations.

PROCEDURE

Wavelength: 510mm (500 – 520)

Cuvette: 1 cm light path

Temperature: +37°C

Reading: against blank reagent

Assay Type: End point

Sample/reagent ratio: 1/100

Pipetting in tubes:

<table>
<thead>
<tr>
<th></th>
<th>BLANK</th>
<th>SAMPLE</th>
<th>STD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent (A)</td>
<td>1000µl</td>
<td>1000µl</td>
<td>1000µl</td>
</tr>
<tr>
<td>Distill water</td>
<td>10µl</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample</td>
<td></td>
<td>10µl</td>
<td></td>
</tr>
<tr>
<td>Standard</td>
<td></td>
<td></td>
<td>10µl</td>
</tr>
</tbody>
</table>

Mix, incubate for 10 minutes at 37°C and read sample and standard extinction.

Volumes can be proportionally modified

This methodology describes the manual procedure to use the kit.

For automated procedure, ask for specific application.

Calibration with watery standard may cause a systematic error when using automatic instrumentations.

Human protected calibrator REF. 6002 is advised
**CALCULATION**

*Serum plasma*

\[
\text{OD Sample} \\
\text{Glucose mg/dl} = \text{__________} \times 100 \text{ (Standard Value)} \\
\text{OD Standard}
\]

Standard 100 mg/dl = 5.56 mmol/L.

To convert mg/dl in mmol/L multiply by 0.0556.

**EXPECTED VALUES**

Serum, plasma: 60 - 110 mg/dL \hspace{1cm} 3.33 – 6.1 mmol/L

The abovementioned values are to be considered as a reference. It is strongly recommended that each laboratory establish its own normal range according to its geographic area, according to IFCC protocol.

**WASTE DISPOSAL**

The disposal of the product must be in accordance with local regulation concerning waste disposal.

**QUALITY CONTROL**

It is recommended to execute the quality control at every kit utilization to verify that values are within the reference range indicated by the methodology. For this purpose the use of test serum REF. 6000 (Normal ambit) and REF. 6001 (Pathologic ambit) is advised.
## PERFORMANCE

<table>
<thead>
<tr>
<th>Measure of Range/Linearity</th>
<th>6.97 – 800 mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detection Limit</td>
<td>6.97 mg/dL</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>1 mg/dL = 0.00251 A at 510 nm</td>
</tr>
</tbody>
</table>

## INTRA-ASSAY PRECISION: n=20

<table>
<thead>
<tr>
<th>Level</th>
<th>Mean (M)</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Level</td>
<td>M = 52.2 mg/dL</td>
<td>2.15%</td>
</tr>
<tr>
<td>Medium Level</td>
<td>M = 119 mg/dL</td>
<td>1.59%</td>
</tr>
<tr>
<td>High Level</td>
<td>M = 389 mg/dL</td>
<td>2.30%</td>
</tr>
</tbody>
</table>

## INTER-ASSAY PRECISION: N = 20

<table>
<thead>
<tr>
<th>Level</th>
<th>Mean (M)</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Level</td>
<td>M = 51.0 mg/dL</td>
<td>2.22%</td>
</tr>
<tr>
<td>Medium Level</td>
<td>M = 114 mg/dL</td>
<td>4.54%</td>
</tr>
<tr>
<td>High Level</td>
<td>M = 408 mg/dL</td>
<td>4.69%</td>
</tr>
</tbody>
</table>
APPENDIX FOUR

Total cholesterol (TC) Level: Standard Operation/guideline by BioSystems Co.

Principle of the Method

Free and esterified cholesterol in the sample originates, by means of the coupled reactions described below, a coloured complex that can be measured by spectrophotometry.

\[
\text{chol. Esterase} \\
\text{Cholesterol ester} + \text{H}_2\text{O} \rightarrow \text{Cholesterol} + \text{Fatty acid}
\]

\[
\text{chol. oxidase} \\
\text{Cholesterol} + \frac{1}{2} \text{O}_2 + \text{H}_2\text{O} \rightarrow \text{Cholesterol} + \text{cholestenone} + \text{H}_2\text{O}_2
\]

\[
\text{Peroxidase} \\
2\text{H}_2\text{O}_2 + 4 - \text{Aminoantipyrine} + \text{Phenol} \rightarrow \text{Quinoneimine} + 4 \text{H}_2\text{O}
\]

CONTENTS

<table>
<thead>
<tr>
<th>CONTENTS</th>
<th>COD 11805</th>
<th>COD 11505</th>
<th>COD 11506</th>
<th>COD 11539</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent Standard</td>
<td>1 x 50 mL</td>
<td>1 x 200 mL</td>
<td>1 x 500 mL</td>
<td>1 x 1L</td>
</tr>
<tr>
<td></td>
<td>1 x 5 mL</td>
<td>1 x 5 mL</td>
<td>1 x 5 mL</td>
<td>1 x 5 mL</td>
</tr>
</tbody>
</table>

COMPOSITION

A. Reagent. Pipes 35 mmol/L, sodium cholate 0.5 mmol/L, phenol 28mmol/L, cholesterol esterase > 0.2 U/mL, cholesterol oxidase > 0.1 U/mL, peroxidase > 0.8 U/mL, 4-aminoantipyrine 0.5 mmol/L, pH 7.0.

STORAGE

Store at 2-8°C.

Reagent and Standard are stable until the expiry date shown on the label when stored tightly closed and if contaminations are prevented during their use.

Indications of deterioration:

- Reagent: Presence of particulate material, turbidity, absorbance of the blank over 0.200 at 500 nm (1 cm cuvette).
- Standard: Presence of particulate material, turbidity.

REAGENT PREPARATION

Reagent and standard are provided ready to use.

ADDITIONAL EQUIPMENT

- Thermostatic water bath at 37°C
- Analyzer, spectrophotometer or photometer able to read at 500 ± 20nm

SAMPLES

Serum or plasma collected by standard procedures.

Cholesterol is stable for 7 days at 2-8°C. Heparin, EDTA, oxalate and fluoride may be used as anticoagulants.

PROCEDURE

1. Bring the Reagent to room temperature.

2. Pipette into labeled test tubes: (Note 1)

<table>
<thead>
<tr>
<th></th>
<th>Blank</th>
<th>Standard</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol Standard (S) Sample</td>
<td>-</td>
<td>10 µL</td>
<td>-</td>
</tr>
<tr>
<td>Reagent (A)</td>
<td>1.0 mL</td>
<td>1.0 mL</td>
<td>10 µL</td>
</tr>
</tbody>
</table>
3. Mix thoroughly and incubate the tubes for 10 minutes at room temperature (16-25°C) or for 5 minutes at 35°C.

4. Measure the absorbance (A) of the Standard and Sample at 500nm against the Blank. The colour is stable for at least 2 hours.

**CALCULATIONS**

The cholesterol concentration in the sample is calculated using the following general formula:

\[
\frac{A_{\text{Sample}}}{A_{\text{Sample}}} \times C_{\text{standard}} = C_{\text{Sample}}
\]

If the Cholesterol Standard provided has been used to calibrate (Note 2):

\[
\frac{A_{\text{Sample}}}{A_{\text{Sample}}} \times 200 = \text{mg/dL cholesterol}
\]

\[
\frac{A_{\text{sample}}}{A_{\text{sample}}} \times 5.18 = \text{mmol/L cholesterol}
\]

**REFERENCE VALUES**

The following uniform cut-off points have been established by the US National Cholesterol Education Program and have also been adopted in many other countries for the evaluation of coronary artery disease risk.

<table>
<thead>
<tr>
<th>Cholesterol Level</th>
<th>mg/dL</th>
<th>mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Up to 200 mg/dL</td>
<td>= 5.2mmol/L</td>
<td>Desirable</td>
</tr>
<tr>
<td>200 – 239 mg/dL</td>
<td>= 5.2 – 6.21 mol/L</td>
<td>Borderline High</td>
</tr>
<tr>
<td>&gt; 240 mg/dL</td>
<td>=&gt; 6.24mmol/L</td>
<td>High</td>
</tr>
</tbody>
</table>
QUALITY CONTROL

It is recommended to use the Biochemistry Control Serum level I (cod. 18005, 18009 and 18042) and II (cod. 18007, and 18043) to verify the performance of the measurement procedure.

Each laboratory should establish its own internal quality control scheme and procedures for corrective action if controls do not recover within the acceptable tolerance.

METROLOGICAL CHARACTERISTICS

- Detection limit: 0.3 mg/dL = 0.008 mmol/L
- Linearity limit: 1000 mg/dL = 26mmol/L. For higher values dilute sample ½ with distilled water and repeat measurement.
- Repeatability (within run).

<table>
<thead>
<tr>
<th>Mean concentration</th>
<th>CV</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>121 mg/dL = 3.13mmol/L</td>
<td>1.1%</td>
<td>20</td>
</tr>
<tr>
<td>257 mg/dL = 6.66 mmol/L</td>
<td>0.9%</td>
<td>20</td>
</tr>
</tbody>
</table>

- Reproducibility (run to run):

<table>
<thead>
<tr>
<th>Mean concentration</th>
<th>CV</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>121 mg/dL = 3.13mmol/L</td>
<td>1.9%</td>
<td>25</td>
</tr>
<tr>
<td>257 mg/dL = 6.66 mmol/L</td>
<td>1.0%</td>
<td>25</td>
</tr>
</tbody>
</table>

- Sensitivity: 1.75 mA.dL/mg = 67.6mA.L/mmol
- Trueness: Results obtained with reagent did not show systematic differences when compared with reference reagents (Note 2). Details of the comparison experiments are available on request.

- Interferences: Lipemia (triglycerides 10 g/l) does not interfere. Bilirubin (>10 mg/dL) and hemoglobin (>5 g/L) may affect the results. Other drugs and substances may interfere.

These metrological characteristics have been obtained using an analyzer. Results may vary if a different instrument or a manual procedure are used.
APPENDIX FIVE

Triglycerides (TG) level: Standard operation/guideline by BioSystems Co.

PRINCIPLE OF METHOD

Triglycerides in the sample originates, by means of the coupled reactions described below, a coloured complex that can be measured by spectrophotometry.

\[
\begin{align*}
\text{Lipase} \\
\text{Triglycerides} + \text{H}_2\text{O} & \rightarrow \text{Glycerol} + \text{Fatty acids} \\
\text{Glycerol kinase} \\
\text{Glycerol} + \text{ATP} & \rightarrow \text{glycerol} - 3 - \text{P} + \text{ADP} \\
\text{G}-3-\text{P-oxidase} \\
\text{Glycerol} - 3 - \text{P} + \text{O}_2 & \rightarrow \text{Dihidroxyacetone} - \text{P} + \text{H}_2\text{O}_2 \\
\text{peroxidase} \\
2\text{H}_2\text{O}_2 + 4 - \text{Aminoantipyrine} + 4 - \text{Chlorophenol} & \rightarrow \text{Quinoneimine} + 4\text{H}_2\text{O}
\end{align*}
\]

CONTENTS

<table>
<thead>
<tr>
<th>COD 11828</th>
<th>COD 11528</th>
<th>COD 11529</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Reagent</td>
<td>1 x 50 mL</td>
<td>4 x 50 mL</td>
</tr>
<tr>
<td>B. Standard</td>
<td>1 x 5 mL</td>
<td>1 x 5 mL</td>
</tr>
<tr>
<td></td>
<td>2 x 250 mL</td>
<td>1 x 5 mL</td>
</tr>
</tbody>
</table>

COMPOSITION

A. **Reagent:** Pipes 45mmol/L, magnesium chloride 5 mmol/L, 4-chlorophenol 6 mmol/L, lipase > 100 U/mL, glycerol kinase >1.5 U/mL, glycerol -3-phosphate oxidase > 4 U/mL, peroxidase > 0.8 U/mL, 4-aminoantipyrine 0.75 mmol/L, ATP 0.9 mmol/L, pH 7.0.
S. **Triglycerides Standard:** Glycerol equivalent to 200 mg/dL (2.26 mmol/L) triolein. Aqueous primary standard.

**STORAGE**

Store at 2 – 8°C.

Reagent and Standard are stable until the expiry date shown on the label when stored tightly closed and if contaminations are prevented during their use.

**Indications of deterioration:**

- Reagent: Presence of particulate material, turbidity, absorbance of the blank over 0.150 at 500 nm (1cm cuvette).
- Standard: Presence of particulate material, turbidity.

**REAGENT PREPARATION**

Reagent and Standard are provided ready to use.

**ADDITIONAL EQUIPMENT**

- Thermostatic water bath at 37°C
- Analyzer, spectrophotometer or photometer able to read at 500 ± 20 nm

**SAMPLES**

Serum of plasma collected by standard procedures.

Triglycerides in serum or plasma are stable for 5 day at 2-8°C. Heparin, EDTA, oxalate and fluoride may be used as anticoagulants.

**PROCEDURE**

1. Bring the Reagent to room temperature.
2. Pipette into labeled test tubes: (Note 1)
3. Mix thoroughly and incubate the tubes for 15 minutes at room temperature (16-25°C) or for 5 minutes at 37°C.

4. Measure the absorbance (A) of the Standard and Sample at 500 nm against the Blank. The colour is stable for at least 2 hours.

**CALCULATIONS**

The triglyceride concentration in the sample is calculated using the following general formula:

\[
\frac{A_{\text{Sample}}}{10 \, \mu\text{L}} \times C_{\text{standard}} = C_{\text{Sample}}
\]

\[
A_{\text{Sample}} \times 200 = \text{mg/dL triglycerides}
\]

\[
A_{\text{sample}} \times 2.26 = \text{mmol/L triglycerides}
\]

**REFERENCE VALUES**

The following uniform cut-off points have established by the US National Institute of Health have also been adopted in many other countries for the evaluation of risk.
Up to 150 mg/dL = 1.7mmol/L  
150 – 199 mg/dL = 1.70-2.25 mol/L  
200 – 499 mg/dL = 2.26-5.64 mmol/L  
> 500 mg/dL => 5.65 mmol/L

<table>
<thead>
<tr>
<th>Normal</th>
<th>Borderline High</th>
<th>High</th>
<th>Very high</th>
</tr>
</thead>
</table>

**QUALITY CONTROL**

It is recommended to use the Biochemistry Control Serum level I (cod. 18005, 18009 and 18042) and II (cod. 18007, 18010 and 18043) to verify the performance of the measurement procedure.

Each laboratory should establish its own internal Quality Control scheme and procedures for corrective action if controls do not recover within the acceptable tolerance.

**METROLOGICAL CHARACTERISTICS**

- Detection limit: 1.6 mg/dL = 0.018 mmol/L
- Linearity limit: 600 mg/dL = 6.78 mmol/L. For higher values dilute sample ¼ with distilled water and repeat measurement.
- Repeatability (within run).

<table>
<thead>
<tr>
<th>Mean Concentration</th>
<th>CV</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 mg/dL = 1.13mmol/L</td>
<td>1.7%</td>
<td>20</td>
</tr>
<tr>
<td>245 mg/dL = 2.77 mmol/L</td>
<td>0.7%</td>
<td>20</td>
</tr>
</tbody>
</table>

- Reproducibility (run to run):
<table>
<thead>
<tr>
<th>Mean Concentration</th>
<th>CV</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 mg/dL = 1.13mmol/L</td>
<td>2.6%</td>
<td>25</td>
</tr>
<tr>
<td>245 mg/dL = 2.77 mmol/L</td>
<td>1.7%</td>
<td>25</td>
</tr>
</tbody>
</table>

- Sensitivity: 1.2 mA.dL/mg = 112mA.L/mmol

- Trueness: Results obtained with reagent did not show systematic differences when compared with reference reagents (Note 2). Details of the comparison experiments are available on request.

- Interferences: Hemoglobin (10g/dl) does not interfere. Bilirubin (2.5mg/dL) may affect the results. Other drugs and substances may interfere.

These metrological characteristics have been obtained using an analyzer. Results may vary if a different instrument or a manual procedure are used.
APPENDIX SIX

HDL level: Standard operation/guidelines by Randox laboratories Ltd.

INTENDED USE

For the quantitative in vitro determination of HDL- Cholesterol in serum and plasma.
This product is suitable for manual use.

Supplementary pack for Cholesterol, CHOD-PAP method.

Cat No.

CH 203 R1. Precipitant 4 x 80 ml.

PRINCIPLE

Low density lipoproteins (LDL and VLDL) and chylomicron fractions are precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL (high density lipoprotein) fraction, which remains in the supernatant, is determined.

SAMPLE

Serum, heparinized plasma or EDTA plasma.

REAGENT COMPOSITION

<table>
<thead>
<tr>
<th>Contents</th>
<th>initial concentrations of solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1. Phosphotungstic Acid</td>
<td>0.55 mmol/l</td>
</tr>
<tr>
<td>Magnesium Chloride</td>
<td>25 mmol/l</td>
</tr>
</tbody>
</table>
SAFETY PRECAUTIONS AND WARNINGS

For in vitro diagnostic use only. Do not pipette by mouth.

Exercise the normal precautions required for handling laboratory reagents.

Health and Safety Data Sheets are available on request

The reagents must be used only for the purpose intended by suitably qualified laboratory personnel, under appropriate laboratory conditions.

STABILITY AND PREPARATION OF REAGENTS

R1. Macro assays: Contents ready for use undiluted. Stable up to the expiry date specified when stored at +15 to +25°C.

R1 Semi-micro assays: Predilute the precipitating reagent in the ratio 4 +1 with redistilled water (dilute the contents of 80 ml bottle with 20 ml redistilled water). Stable up to the expiry date specified when stored at +15 to +25°C.

MATERIALS PROVIDED

HDL – Cholesterol Precipitant.

MATERIALS REQUIRED BUT NOT PROVIDED

Randox Lipid Controls:

Level 1 LE 2661 or LE 2668
Level 2 LE 2662 or LE 2669
Level 3 LE 2663 or LE 2670

Randox aqueous Cholesterol Standard Cat. No. ST 1590

Reagent solution for Cholesterol CHOD-PAP Assay

Cat. Nos. CH 200, 201 and 202.
PROCEDURE NOTES

Only clear supernatants on centrifugation must be used. In the case of incomplete sedimentation (turbid supernatant) caused by elevated triglyceride concentrations, the sample should be diluted 1 + 1 with 0.9% NaCl solution and the precipitating step repeated. The result should then be multiplied by 2.

PROCEDURE

1. Precipitation

<table>
<thead>
<tr>
<th>Pipette into centrifuge tubes:</th>
<th>Macro</th>
<th>Semi Micro</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample/Standard</td>
<td>500 µl</td>
<td>200 µl</td>
</tr>
<tr>
<td>Precipitant (R1)</td>
<td>1000 µl</td>
<td>-</td>
</tr>
<tr>
<td>Diluted Precipitant (R1)</td>
<td>-</td>
<td>500 µl</td>
</tr>
</tbody>
</table>

Mix and allow to sit for 10 minutes at room temperature. Then centrifuge for 10 minutes at 4,000 rpm, or 2 minutes at 12,000 rpm.

Separate off the clear supernatant within two hours and determine the cholesterol content by the CHOD-PAP method. The supernatant may be stored up to five days at +2 to +25°C.

2. Cholesterol CHOD-PAP Assay

Wavelength: 500 nm, Hg546 nm

Cuvette: 1 cm light path

Temperature: 20-25°C of 37°C
Measurement: against reagent blank

Only one reagent blank per series is required.

Pipette into test tubes:

<table>
<thead>
<tr>
<th></th>
<th>Reagent Blank</th>
<th>Standard</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distilled Water</td>
<td>100 µl</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Supernatant</td>
<td>-</td>
<td>-</td>
<td>100 µl</td>
</tr>
<tr>
<td>Standard Supernatant</td>
<td>-</td>
<td>100 µl</td>
<td>-</td>
</tr>
<tr>
<td>Reagent</td>
<td>1000 µl</td>
<td>1000 µl</td>
<td>1000 µl</td>
</tr>
</tbody>
</table>

Mix, incubate for 10 minutes at 20-25°C or 5 minutes at 37°C. Measure the absorbance of the sample ($A_{sample}$) and Standard ($A_{standard}$) against the reagent within 60 minutes.

**CALCULATION**

1. **HDL Cholesterol**

When using a factor

<table>
<thead>
<tr>
<th></th>
<th>MACRO</th>
<th>SEMI-MICRO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wavelength</td>
<td>mmol/l</td>
<td>mg/dl</td>
</tr>
<tr>
<td>mmol/l</td>
<td>mg/dl</td>
<td></td>
</tr>
<tr>
<td>500 nm</td>
<td>4.65</td>
<td>180</td>
</tr>
<tr>
<td></td>
<td>5.43</td>
<td>210</td>
</tr>
<tr>
<td>Hg 546 nm</td>
<td>7.09</td>
<td>274</td>
</tr>
<tr>
<td></td>
<td>8.27</td>
<td>320</td>
</tr>
</tbody>
</table>

When using a standard:

Concentration of HDL Cholesterol in supernatant.

\[
\text{Concentration} = \frac{\Delta A_{sample}}{\Delta A_{standard}} \times \text{Conc. Of Standard}
\]
QUALITY CONTROL

Randox Lipid Controls, level 1, level 2 and level 3 are recommended for daily quality control. Two levels of controls should be assayed at least once a day. Values obtained should fall within a specified range. If these values fall outside the range and repetition excludes error, the following steps should be taken.

1. Check instrument settings and light source.
2. Check cleanliness of all equipment in use.
3. Check water, contaminants i.e. bacterial growth may contribute to inaccurate results.
4. Check reaction temperature.
5. Check expiry date of kit and contents.
6. Contact Randox Laboratories Customer Technical support, Northern Ireland (028) 94422413.

INTERFERENCES

The assay is unaffected by icteric samples (bilirubin <30 mg/dl), rheumatoid factors <1000 IU/ml, haemolytic samples (Hb < 500 mg/dl) and lipaemic samples (triglyceride <1200 mg/dl). Lipaemic samples with a triglyceride concentration >1200 mg/dl should be diluted 1+9 with 0.9% (w/v) NaCl before assay. The corresponding result should be multiplied by 10.

EXPECTED VALUES (NCEP GUIDELINES)

<table>
<thead>
<tr>
<th>Mg/dl</th>
<th>mmol/l</th>
</tr>
</thead>
</table>
| <40  | <1.04  | Low  
| ≥60  | ≥1.55  | High |
As HDL cholesterol is affected by a number of factors such as smoking, exercise, hormones, age and sex, each laboratory should establish its own reference ranges.

**LINEARITY**

The test is linear up to a cholesterol concentration of 19.3mmol/l (750 mg/dl). Dilute samples with a cholesterol concentration greater than this 1 + 2 with 0.9% NaCl. Multiply the result by 3.

**SENSITIVITY**

It is recommended that each laboratory established its own range of sensitivity as this is limited by the sensitivity of the spectrometer used.

Under the conditions of the assay a change of 0.004 absorbance units is equivalent to 0.06 mmol/l.
APPENDIX SEVEN

QUESTIONNAIRE

This is a dissertation questionnaire in the Faculty of Family Medicine of the National Post Graduate Medical College of Nigeria on the topic “A study of the pattern of obesity and its primary co-morbidities among adult patients attending GOPC of FMC, Owerri by DR IKWUDINMA AUGUSTINE .O. (S/Reg Dept. of Family Medicine FMC Owerri). This is a requirement for the award of fellow (FMCFM) in the above faculty.

All information given shall be treated confidentially.

Please answer the questions correctly.

Thanks for your co-operation.

1. Serial number..............................................................................................................................................
2. Hospital number..............................................................................................................................................
3. Address............................................................................................................................................................

A. SOCIO - DEMOGRAPHIC INFORMATION

1. Sex: male □ female □
2. Age (years):
   (a) 18-27 □ (b) 28-37 □ (c) 38-47 □ (d) 48-57 □ (e) 58-67 □
   (f) 68-77 □ (g) 78-87 □ (h) 88-97 □ (i) above 97 □
3. Race
   (a) Ibo □ (b) Yoruba □ (c) Hausa □ (d) others (specify) □
4. Educational level
   (a) Non formal education □ (b) Primary level □
   (c) Secondary level □ (d) Post Secondary level □
   (e) Post Graduate level □
5. **Occupation:**
   (a) Unemployed    (b) student/Apprentice    (c) Artisans (specify)    
   (d) Drivers (specify)    (e) Trading (specify)    (f) farming (specify)    
   (g) Civil servant (specify)    (h) HW (specify)    (i) Business executive    
   (j) Professional (specify)    

6. **Martial Status:**
   (a) Single    (b) Married    (c) Widowed    (d) Divorced    (e) separated    

7. **Religious inclination:**
   (a) Christians    (b) Moslem    (c) traditionalist    (d) others specify    

8. **Source of Income**
   (a) Self employed (specify)    
   (b) Government employee (specify)    
   (c) Private sector employee (specify)    
   (d) Others (specify)    

9. **Amount of income per month (income level)**
   (a) < N10,000    (b) N10,000 - N45,000    (c) N45,000 - N 150,000    (d) > N150,000    

10. **Socio-economic class**
   (a) Lower class    (b) Middle class    (c) Upper class    

B. **PHYSICAL MEASUREMENT**

11. (a) Height (metres)  
    (b) Weight (kilograms)  
    (c) BMI (wt.kg/Ht.M^2) kg/m^2  

D **BLOOD PRESSURE MEASUREMENTS (mmHg)**  
    ______________________  1  
    ______________________  2  
    ______________________  3  

    Average = \frac{1+2+3(\text{mmHg})}{3} = \text{---------(mmHg)}
C. BIOCHEMICAL MEASUREMENTS

12. (a) fasting blood glucose ___________ 1(initial) mg/dl

___________  2(confirmatory) mg/dl

(b) Blood lipids

Cholesterol________________ mg/dl

Triglyceride__________________________ mg/dl

HDL cholesterol _____________________ mg/dl

Calculated LDL cholesterol____________ mg/dl

D. ASSESSMENT OF PATIENTS AWARENESS (PERCEPTION)/KNOWLEDGE OF LIFESTYLE MODIFICATION

(a) Are you aware you are obese?

(i) Aware (ii) Not aware

(b) Source of awareness of information on his/her obese condition.

(i) Self (iii) relatives/family members

(ii) friends/peers (iv) Health professionals (v) others (specify)

(c) What are the ways of lifestyle modification for obesity?

(i) Adequate diet

(ii) Adequate physical activities

(iii) Behavioural changes such as drinking of alcohol
APPENDIX EIGHT

CONSENT FORM

I........................................................................of ..........................................................hereby consent to this study. I acknowledge that I have been fully counseled on the implication of the study. I understand that the study is to be carried out solely for the purpose of medical research and I am willing to participate for that purpose on the understanding that I shall be entitled to withdraw this consent any time.

Date..........................................................signed..........................................................

(Patient)

I confirm that I have explained to the patient the purpose and nature of the study and the fact that refusal to participate will not in any way affect his/her normal care by me or any member of the institution. I know the consequences of any false declaration on this or any other form.

Date..........................................................signed..........................................................

(Doctor/Researcher)
APPENDIX NINE

SOCIO-ECONOMICS CLASSIFICATION

1. **UPPER CLASS**: Higher professional occupations, higher managerial and administrative occupations, large employers. Examples: medicine, lawyers, engineers, authors, scientists, architects, captains of industries, directors of businesses.

2. **MIDDLE CLASS**: Lower professional occupations, lower managerial and administrative occupations, small employers. Examples: Teachers, senior civil servants, owners of small business and industries.

3. **LOWER CLASS**: Lower supervisory and technical occupations, routine and semi-routine occupations, manual occupations. Examples: Artisans, drivers, farmers, clerks, factory workers and operators, Market men and women, labourers, casual workers, domestic servants,