UNIVERSITY OF GHANA

COLLEGE OF HEALTH SCIENCES

RELATIONSHIP BETWEEN CAROTID INTIMA MEDIA THICKNESS AND SELECTED ANTHROPOMETRIC VARIABLES

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DEPARTMENT OF ANATOMY

JULY 2018
DECLARATION BY CANDIDATE

I hereby declare that this thesis is a product of my own research undertaken under supervision and has neither been presented in whole nor in part for the award of another degree elsewhere. I take full responsibility for any residual flaws in this work.

Signature: ………………………….. Date: ………../………../………….
(ERIC FATO)

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We hereby declare that the practical work and presentation of this thesis were supervised in accordance with the guidelines on supervision of thesis laid down by the University of Ghana.

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(Professor Clifford Nii Boi Tagoe)
DEDICATION

This thesis is dedicated to my life-coaches, my dad and mum, Mr. Nicholas Kwame Fato and Mrs. Patricia Fato. I owe it all to you. Many Thanks.
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<td>BP</td>
<td>Blood Pressure</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
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<tr>
<td>CV</td>
<td>Cardiovascular</td>
</tr>
<tr>
<td>CVD</td>
<td>Cardiovascular Disease</td>
</tr>
<tr>
<td>CIMT</td>
<td>Carotid Intima Media Thickness</td>
</tr>
<tr>
<td>CCIMT</td>
<td>Common Carotid Intima Media Thickness</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
</tr>
<tr>
<td>CCA</td>
<td>Common Carotid Artery</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>DALY</td>
<td>Disability Adjusted Life Years</td>
</tr>
<tr>
<td>ECA</td>
<td>External Carotid Artery</td>
</tr>
<tr>
<td>GPO</td>
<td>Glycerol Phosphate Oxidase</td>
</tr>
<tr>
<td>HDSS</td>
<td>Health and Demographic Surveillance System</td>
</tr>
<tr>
<td>HDL</td>
<td>High-density Lipoprotein</td>
</tr>
<tr>
<td>HIV</td>
<td>Human Immunodeficiency Virus</td>
</tr>
<tr>
<td>ICA</td>
<td>Internal Carotid Artery</td>
</tr>
<tr>
<td>IDF</td>
<td>International Diabetes Federation</td>
</tr>
<tr>
<td>IMT</td>
<td>Intima Media Thickness</td>
</tr>
<tr>
<td>Acronym</td>
<td>Description</td>
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<tr>
<td>---------</td>
<td>------------------------------------</td>
</tr>
<tr>
<td>LDL</td>
<td>Low-density Lipoprotein</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>NHRC</td>
<td>Navrongo Health Research Centre</td>
</tr>
<tr>
<td>NO</td>
<td>Nitric Oxide</td>
</tr>
<tr>
<td>NCD</td>
<td>Non-Communicable Diseases</td>
</tr>
<tr>
<td>NEFA</td>
<td>Non-Esterified Fatty Acids</td>
</tr>
<tr>
<td>SCAT</td>
<td>Sub-cutaneous Adipose Tissue</td>
</tr>
<tr>
<td>SSA</td>
<td>Sub-Saharan Africa</td>
</tr>
<tr>
<td>TEM</td>
<td>Technical Error of Measurement</td>
</tr>
<tr>
<td>US</td>
<td>Ultrasound</td>
</tr>
<tr>
<td>USS</td>
<td>Ultrasound System</td>
</tr>
<tr>
<td>UK</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>USA</td>
<td>United States of America</td>
</tr>
<tr>
<td>VAT</td>
<td>Visceral Adipose Tissue</td>
</tr>
<tr>
<td>VFA</td>
<td>Visceral Fat Area</td>
</tr>
<tr>
<td>WC</td>
<td>Waist Circumference</td>
</tr>
<tr>
<td>WHtR</td>
<td>Waist to Height Ratio</td>
</tr>
<tr>
<td>WHR</td>
<td>Waist-To-Hip Ratio</td>
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<td>WHO</td>
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ABSTRACT

Background: A large number of studies have emphasized the significance of the association between increased carotid intima-media thickness (CIMT) and increased cardiovascular risk. Incorporation of a measurement of CIMT into the conventional work-up of cardiovascular (CV) risk factors has been suggested to improve prediction of CV risk. In spite of all these studies, there is limited research on CIMT, CVD risk factors and its associations with anthropometric indices of obesity in indigenous African populations.

In order to apply these ultrasound measurements with confidence, there was the need for quantitative data to show the relationship between CIMT, and anthropometric variables in determining cardiovascular disease risk in sub-Saharan African (SSA) population.

Aim: The study determined the association between CIMT and some selected anthropometric variables in assessing cardiovascular disease risk in hypertensive Ghanaians.

Methodology: A study population of 500 adults aged from 40 to 60 years was considered. Common carotid intima-media thickness (CCIMT), visceral adipose tissue (VAT) and subcutaneous adipose tissue (SCAT) ultrasound measurements of the participants were taken at the Clinical Trial Unit of the Navrongo Health Research Centre, Navrongo. Participant’s data on demographics, obesity related anthropometry variables as well as CVD risk variables were collected for statistical analysis.
**Results:** The Spearman correlation conducted showed that CIMT was positively associated with waist to hip ratio (WHR). CCIMT also showed significant association with BP, HDL, LDL, smoking and alcohol intake status.

**Conclusion:** The study established the existence of significant association between CIMT and WHR in assessing CVD risk in hypertensive Ghanaians.
CHAPTER ONE
INTRODUCTION

1.0 BACKGROUND

The common carotid arteries (CCA) are major blood vessels in the neck that supply blood to the brain, head, and neck. There are two carotid arteries, one on either side of the neck. These arteries originate from different arteries, but follow symmetrical courses. The right common carotid originates in the neck from the brachiocephalic trunk; the left from the aortic arch in the thorax. Each divides into an external and internal carotid arteries at the upper border of the thyroid cartilage, at around the level of the fourth cervical vertebra (Netter, 2014; Susan et al., 2016). The internal carotid artery supplies blood to the brain, whilst the external carotid artery supplies blood to the scalp, face and neck (Susan et al., 2016).

Histologically, the walls of the common carotid arteries are made of three layers of tissue (Young, O’Dowd, & Woodford, 2014), namely:

- **Intima**, the smooth innermost layer formed by single layer of flattened endothelial cells.

- **Media**, the muscular middle layer formed by concentric fenestrated sheets of elastin separated by collagenous tissue, relatively few smooth muscle fibres, connective tissue and secretory myocytes.

- **Adventitia**, the collagenous outer layer contains small vasa vasorum which also penetrate the outer half of the tunica media. The arteries are covered by a connective tissue called the carotid sheath.
The intima-media thickness (IMT) corresponds to the intima-media complex, comprising endothelial cells, connective tissue and smooth muscle. The IMT is the site of lipid deposition and plaque formation (Susan et al., 2016). Sonographically determined IMT of the carotid artery is an established surrogate marker for subclinical atherosclerosis; whereas IMT thickening reflects generalized atherosclerosis which is a strong predictor of future cardiovascular events (Lorenz, Schaefer, Steinmetz, & Sitzer, 2010).

Atherosclerosis is the underlying cause of the majority of clinical cardiovascular disease. This is characterized by an accumulation of lipids, inflammatory cells, and development of scar tissue covered by a fibrous cap build within the walls of large and medium-sized arteries (Amasyali & Kilic, 2014). Carotid arterial atherosclerosis disrupts the architecture and function of the common carotid vessels and variably reduces the blood flow to the myocardium (Sharma et al., 2015; Verhagen & Visseren, 2011). It is a progressive process that begins in adolescence, becoming clinically manifest at a later age or, it may occur in a subclinical manner (Sharma et al., 2015).

Hypertension is the leading global preventable risk factor for cardiovascular disease (CVD) and premature death (Bundy & He, 2016). Studies have shown an association between increased common carotid intima-media thickness (CCIMT) and hypertension in elderly and middle-aged subjects (Bonithon-Kopp et al., 2006). A positive association exists between carotid intima-media thickness (CIMT) and the risk of subsequent cardiovascular events in general populations, independent of all major risk factors (Bonithon-Kopp et al., 2006; Matsumoto, Sera, Nakamura, Ueki, & Miyake, 2002). This relationship has promoted the use of CIMT in pathophysiological studies and clinical trials, in which the consideration of CIMT has shifted from a secondary
end point to a surrogate risk factor of cardiovascular events (Bonithon-Kopp et al., 2006).

Effective determination by non-invasive imaging techniques have combined with their predictive power for cardiovascular events to make visceral adipose tissue (VAT) and subcutaneous adipose tissue (SCAT) preferred variables in studies on hypertensive disease risks (Vlachos, Hatziioannou, Perelas, & Perrea, 2007). With respect to obesity-associated disorders, VAT is reckoned to have greater physiological relevance than SCAT in its contribution to consequent disease processes. Visceral adipocytes have distinct secretory and metabolic profiles that distinguish them from subcutaneous adipocytes and contribute to their disease-causing prowess (Loukianos et al., 2014).

VAT is linked to cardiovascular diseases much more than BMI, which has long been considered as an indirect measure of obesity. The gold standard for assessing the levels of VAT and SCAT in the abdomen, which is thought to be the prime adipose region for influencing metabolic disorders (Wajchenberg, 2014a), is the use of Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) scans. However, these techniques are expensive and not available to all researchers. Therefore, waist circumference (WC) has been used as a proxy indicator of VAT due to its ease of measurement and correlation with visceral adiposity (Canoy et al., 2007; Klein, Allison, Heymsfield, Kelley, & Leibel, 2007).

This study will therefore determine quantitative relationship between ultrasound measurements of CCIMT and abdominal adiposity with standard obesity-related anthropometric variables in hypertensive subjects. It is notable that visceral adiposity is an ideal technique used in large epidemiological surveys and has been shown to give accurate measures of CIMT, VAT and SCAT (Vlachos et al., 2007).
BMI is a measure of the body weight relative to height that is associated with body fat and health risk (Ni Mhurchu et al., 2004). Obesity is being recognized as potential threat to health and must be treated early (Ziraba, Fotso, & Ochako, 2009). With obesity increasing, the risk of developing several diseases like hypertension, diabetes, heart ailments, stroke, osteoarthritis, among many others also increases (Ni Mhurchu et al., 2004). Increased body mass index is tightly related to an increased risk of coronary heart disease (Ni Mhurchu et al., 2004).

Several large-scale prospective studies have shown that obese people, defined on the basis of high BMI, have a much greater risk of developing cardiovascular diseases than subjects with normal levels of total body fatness (Kurth et al., 2005; Wu, Chu, & Huang, 2015; Ziraba et al., 2009). It has become increasingly clear that the relationship between obesity and cardiovascular disease depends not only on the amount of body fat but also on its distribution (Ziraba et al., 2009). Individuals with increased fat accumulation in the abdominal region, reflected by high waist-to-hip ratio (WHR), often have higher atherogenic lipid profiles and are at increased cardiovascular risk (Lapidus, Bengtsson, Larsson, Pennert, & Rybo, 1984; Rexrode, 1998).

The rising prevalence of obesity across sub-Saharan Africa (SSA) is associated with increasing levels of obesity-related cardiometabolic diseases including hypertension (Ziraba et al., 2009) and cardiovascular diseases (Akinboboye, Idris, Akinboboye, & Akinkugbe, 2003). Despite such evidence, angiographic and postmortem studies evaluating the association between obesity and cardiovascular disease have provided conflicting results (Ge et al., 2014; Kortelainen & Särkioja, 1997; Solberg & Strong, 1983). There is therefore need for further investigations involving living persons, afforded by CIMT measurements, and indices of obesity.
1.2 PROBLEM STATEMENT

In 2008, CVDs were the most prevalent contributor to mortality in Ghana among all non-communicable diseases (NCDs) as well as the leading cause of institutional deaths accounting for 14.5% of reported total deaths in the country compared to 13.4% deaths from malaria (Ofori-Asenso & Garcia, 2016; Sanuade, Anarfi, & Koram, 2014). The underlying pathology is atherosclerosis, which develops over many years and is usually advanced by the time symptoms occur, generally in middle age (Di Costanzo et al., 2017; Lorenz et al., 2010).

The rate of progression of atherosclerosis is influenced by cardiovascular risk factors: tobacco use, alcohol intake, unhealthy diet and physical inactivity (which together result in obesity), elevated blood pressure (hypertension), abnormal blood lipids (dyslipidaemia), age, sex and elevated blood glucose (diabetes) (Bots, Evans, Riley, & Grobbee, 2003). Continuing exposure to these risk factors leads to further progression of atherosclerosis, resulting in unstable atherosclerotic plaques, narrowing of blood vessels and obstruction of blood flow to vital organs, such as the heart and the brain. The clinical manifestations of these diseases include angina, myocardial infarction, transient cerebral ischaemic attacks and strokes (Bots & Ruijter, 2012; Cooney, Dudina, D’Agostino, & Graham, 2010; D’Agostino et al., 2008).

Carotid ultrasound is a widely used imaging modality for the detection of subclinical atherosclerosis (Shah, 2010). B-mode ultrasound measurement of CIMT is frequently used for non-invasive evaluation of subjects at risk of atherosclerosis (Heiss et al., 1991; Stein et al., 2008).
Some studies have examined the association between various CVD risk factors, anthropometric indices and CIMT (De Michele et al., 2002; Ge et al., 2014; Kamadjeu et al., 2006; Kommuri et al., 2016; Ren et al., 2014), suggesting that atherosclerosis contributes to the pathologic pathways linking obesity and CVD. Moreover, majority of the studies were conducted among mostly overweight or obese adults or adolescents (Folsom et al., 1994; Visscher et al., 2001; Yan et al., 2009), limiting the interpretation of the findings.

It is established that VAT has a strong association with cardiovascular risk (Fain et al., 2014). However, the primary importance of VAT as proxy for subcutaneous abdominal obesity with regard to CVD has been challenged (Abate, Garg, Peshock, Stray-Gundersen, 1995; Goodpaster, Thaete, Simoneau, & Kelley, 1997). Nonetheless, obesity as a risk factor for clinical CVDs in not in dispute, though the underlying mechanism remains unclear.

Emerging clinical and epidemiological evidence indicate that the distribution of adiposity plays an important role in CVD risk, independent of the extent of general adiposity (Canoy et al., 2007). Several studies suggest that anthropometric measures of abdominal adiposity, such as WHR and WC, serve as better predictors of CVD risk than BMI, the primary marker of general adiposity (Yan et al., 2009). Epidemiological evidence has suggested that abdominal obesity accelerates atherosclerotic progression; however, current evidence on the topic is limited and inconsistent (Garcia et al., 2016; Lakka, Lakka, Salonen, Kaplan, & Salonen, 2001; Yan et al., 2009).

Almost all available data on CIMT, VAT and SCAT in relation to CVD were derived from European and Western Caucasian populations, with limited evidence from Africa and populations with a lower BMI (Ni Mhurchu et al., 2004). Need for local studies is
accentuated by the fact that both the risk factors and their relation to CVD endpoints differ among ethnic groups (Cooney et al., 2010; Daar et al., 2007).

While communicable diseases continue to drain health resources in SSA, the rising CVD epidemic poses a new public health challenge, hence the need for cost-effective strategies to detect, predict and prevent CVDs associated with obesity, which this study proposes to address.

1.3 JUSTIFICATION

There is growing evidence that CIMT can be regarded as an indicator of generalized atherosclerosis (Lorenz et al., 2010), and that it may be used as an intermediate end point or proxy end point in trials as a suitable alternative for detecting cardiovascular morbidity and mortality (Lorenz et al., 2010). Ultrasound measurements of VAT, SCAT and CIMT are non-invasive, accurate, inexpensive and ideal for large surveys (Vlachos et al., 2007).

There is limited research on CIMT, VAT, CVD risk factors and its associations with anthropometric indices of obesity in indigenous African populations (Jennings, Lambert, Collins, Levitt, & Goedecke, 2009). In order to apply these measurements with confidence, there is the need for quantitative data to show the relationship between CIMT, VAT and anthropometric variables in determining cardiovascular disease risk in SSA population. There is also the need for cost-effective strategies to detect, predict and prevent CVDs associated with obesity in SSA.
1.4 AIM

The aim of the study was to determine the association between CIMT, VAT and some selected anthropometric variables in assessing cardiovascular disease risk in hypertensive Ghanaians.

1.5 SPECIFIC OBJECTIVES

- To determine association between common CIMT and blood pressure in hypertensive patients.
- To determine association between common CIMT and abdominal ultrasound measurements; VAT, SCAT in hypertension.
- To determine association between common CIMT and some selected anthropometric indices: BMI, WC, WHR and waist-height ratio (WHtR).
- To determine association between blood pressure and the selected anthropometric indices.
- To determine association between common CIMT and CVD risk factors which include; alcohol intake, lipid profile, smoking status, albumin-creatinine ratio (ACR), insulin level and glucose level.
- To determine association between VAT, SCAT and anthropometric indices
1.6 HYPOTHESIS

Increased common carotid intima-media thickness measurements are associated with increasing anthropometric variables in hypertensive Ghanaian adults.
CHAPTER TWO
LITERATURE REVIEW

2.0 CARDIOVASCULAR DISEASES

Cardiovascular diseases (CVDs) are a group of disorders of the heart and blood vessels that include; peripheral arterial disease, coronary heart disease, deep vein thrombosis, cerebrovascular disease, pulmonary embolism, congenital heart disease and rheumatic heart disease (Nangia, Harpreet, Col, & Kaur, 2014).

The increase risk of developing CVD is associated with CV risk factors. CV risk factors in general include demographic characteristics, family history of CVD, smoking, physical inactivity, abnormal lipids and lipoproteins, obesity, hypertension and diabetes (Burger, Pretorius, Fourie, & Schutte, 2016).

The incidence of CVDs is rapidly increasing worldwide and is currently considered to be the leading cause of death in both developing and developed countries (Gaziano, 2017; Mittal & Singh, 2010).

Atherosclerosis is the underlying cause of the majority of clinical cardiovascular events (Verhagen & Visseren, 2011). This generalized inflammatory disease is characterized by an accumulation of lipids, inflammatory cells, and development of scar tissue covered by a fibrous cap build within the walls of large and medium-sized arteries (Alsheikh-Ali, Kitsios, Balk, Lau, & Stanley, 2010).

Individuals with asymptomatic atherosclerosis should preferably be identified at an early stage, so that primary preventive measures can be initiated (Alsheikh-Ali et al., 2010). The development of atherosclerosis usually takes decades, characterised by thickening of the arterial wall as one of the first detectable stages (Alsheikh-Ali et al.,
2010). Carotid ultrasound is a widely used imaging modality for the detection of subclinical atherosclerosis (Shah, 2010). B-mode ultrasound assessment of CIMT is usually used for non-invasive examination of subjects at risk of atherosclerosis. Still, the exact risk of CV events related with an increased CIMT in general populations is not entirely clear (Shah, 2010).

2.1 ANATOMY OF THE COMMON CAROTID ARTERY

In anatomy, the left and right common carotid arteries are arteries that supply the head and neck with oxygenated blood; they divide in the neck to form the external and internal carotid arteries (Scanlon & Sanders, 2007).

The left common carotid artery can be thought of as having two parts: a thoracic (chest) part and a cervical (neck) part. The right common carotid originates in or close to the neck, so contains only a small thoracic portion. Only the left common carotid artery has a substantial presence in the thorax. It originates directly from the aortic arch, and travels upward through the superior mediastinum to the level of the left sternoclavicular joint (Netter, 2014; Susan et al., 2016).

During the thoracic part of its course, the left common carotid artery is related to the following structures: anteriorly, it is separated from the manubrium of the sternum by the sternohyoid and sternothyroid muscles, the anterior portions of the left pleura and lung, the left brachiocephalic vein, and the remains of the thymus; posteriorly, it lies anterior to the trachea, oesophagus, left recurrent laryngeal nerve, and thoracic duct (Moore, Dalley, & Agur, 2014; Netter, 2014).
To its right side below is the brachiocephalic trunk and superiorly the trachea, the inferior thyroid veins, and the remains of the thymus; to its left side are the left vagus and phrenic nerves, left pleura, and lung. The left subclavian artery is posterior and slightly lateral to it (Moore et al., 2014).

The cervical portions of the common carotids are similar and therefore will be described together. Each vessel passes obliquely upward, from behind the sternoclavicular joint to the level of the upper border of the thyroid cartilage, where it divides (Nakamasa et al., 2005; Netter, 2014).

At the lower neck the two common carotid arteries are separated from each other by a very narrow interval which contains the trachea, but at the upper part, the thyroid gland, the larynx and pharynx separate the two arteries (Moore et al., 2014; Netter, 2014).

The common carotid artery is contained in a sheath known as the carotid sheath, which is derived from the deep cervical fascia and encloses also the internal jugular vein and vagus nerve, the vein lying lateral to the artery, and the nerve between the artery and vein, on a plane posterior to both. On opening the sheath, each of these three structures is seen to have a separate fibrous cover (Moore et al., 2014; Netter, 2014).

At approximately the level of the fourth cervical vertebra, the common carotid artery divides into an internal carotid artery (ICA) and an external carotid artery (ECA) (Moore et al., 2014). While both branches travel upward, the internal carotid takes a deeper (more internal) path, eventually travelling up into the skull to supply the brain. The external carotid artery travels more closely to the surface, and sends off numerous branches that supply the head, meninges and neck (Moore et al., 2014).

Behind, the artery is separated from the transverse processes of the cervical vertebrae by the longus colli and longus capitis muscles, the sympathetic trunk being interposed.
between it and the muscles. The inferior thyroid artery crosses behind the lower part of the vessel (Moore et al., 2014).

Medially, it is in relation with the oesophagus, trachea, and thyroid gland (which overlaps it), the inferior thyroid artery and recurrent laryngeal nerve being interposed; higher up, with the larynx and pharynx. Lateral to the artery, inside the carotid sheath with the common carotid, are the internal jugular vein and vagus nerve (Nakamasa et al., 2005; Netter, 2014).

2.2 FEATURES OF THE CAROTID ARTERY

Like all arteries, the carotid arteries are made of three layers of tissue: the Intima (the smooth innermost layer) and the Media (the muscular middle layer), which forms the intima-media complex, and an outer layer called Adventitia (Young et al., 2014), as shown in Figure 1.

The innermost layer, the tunica intima, is the only part of a vessel that is in contact with blood. It is made of simple squamous epithelium called endothelium (Young et al., 2014). This lining is the same type of tissue that forms the endocardium, the lining of the chambers of the heart. Its extreme smoothness prevents abnormal blood clotting. The endothelium of vessels, however, also produces nitric oxide (NO), which is a vasodilator. The tunica media, or middle layer, is made of smooth muscle and elastic connective tissue. Both of these tissues are involved in the maintenance of normal blood pressure, especially diastolic blood pressure when the heart is relaxed (Young et al., 2014). The smooth muscle is the tissue affected by the vasodilator NO; relaxation of this muscle tissue brings about dilation of the vessel. Smooth muscle also has a nerve
supply; sympathetic nerve impulses bring about vasoconstriction (Young et al., 2014). Fibrous connective tissue forms the outer layer, the **tunica adventitia**. This tissue is very strong, which is important to prevent the rupture or bursting of the larger arteries that carry blood under high pressure (Young et al., 2014).

The outer and middle layers of large arteries are quite thick. In the smallest arterioles, only individual smooth muscle cells encircle the tunica intima. As mentioned, the smooth muscle layer enables arteries to constrict or dilate. Such changes in diameter are regulated by the medulla and autonomic nervous system (Young et al., 2014).

The carotid sinus, or carotid bulb extends into the beginning of ICA. The carotid sinus contains sensors that help regulate blood pressure. The carotid artery pulse can normally be felt in the neck by pressing the fingertips against the side of the wind pipe, or trachea (Moore et al., 2014; Young et al., 2014).

![Figure 1. Features of the common carotid artery](https://www.researchgate.net/Intima-media-complex-in-the-CCA-)

*Figure 1. Features of the common carotid artery*
2.3  CIMT

CIMT is measured between the intimal-luminal and the medial-adventitial interfaces of the carotid artery wall represented as a double-line density on an ultrasound image (Tasneem & Ming-Sum, 2014) as shown in Figure 1. The intima-media complex comprises of endothelial cells, connective tissue, and smooth muscle which serves as the site for lipid deposition that eventually leads to a condition known as atherosclerosis (María, Rivera, Ríos, & Díaz, 2016).

Atherosclerosis is considered a pathological alteration of the arteries characterized by the abnormal deposit of lipids and fibrous tissue in the arterial walls, which disrupts the architecture and function of the vessels and reduces, variably, the blood flow to the myocardium (Alsheikh-Ali et al., 2010; María et al., 2016). It is a progressive process that begins in adolescence, becoming clinically manifest at a later age or it may occur in a subclinical manner (Pletcher & Moran, 2017).

2.4  HISTORICAL PERSPECTIVE ON CIMT

Pignoli et al., (1986), reported the first in vitro results of a study investigating the arterial wall thickness with real-time B-mode ultrasound. In that initial study, the distance between two parallel echogenic lines correlated well with the intima-media thickness (IMT) measured on pathologic examination. The authors concluded that B-mode ultrasound represented a useful tool for the measurement of IMT of human arteries in vivo. Subsequently, Persson et al., (1992), demonstrated in an in vivo study that IMT measurement with B-mode ultrasound was highly reproducible. It is partly
because of the easily accessible anatomical position of the carotid arteries, the carotid CIMT has become a frequently used measurement in clinical practice and scientific studies (Persson et al., 1992).

CIMT is a well-studied phenotype of atherosclerosis. Using B-mode ultrasound the CIMT can be assessed quickly, non-invasively and at relatively low cost (Lorenz et al., 2010). Studies have shown that the measurement of CIMT is widely used as an indicator of asymptomatic atherosclerosis and is both used in prospective follow-up studies to investigate the relationship of CIMT with future cardiovascular events (Muntendam, McCall, Sanz, Falk, & Fuster, 2010). In clinical intervention trials it is used to monitor changes of any given treatment (Muntendam et al., 2010). Based on the assumption that change in CIMT is directly correlated with change in cardiovascular risk, CIMT is a frequently used as a surrogate endpoint in clinical trials (Bots & Ruijter, 2012; Dogan et al., 2010).

![Ultrasound image of Intima-media complex in the CCA.](https://www.researchgate.net/Intima-media-complex-in-the-CCA-)

*Figure 2. Ultrasound image of Intima-media complex in the CCA.*
2.5 HYPERTENSION

Hypertension is an increasingly important medical and public health issue worldwide. High blood pressure is estimated to have caused 7.6 million premature deaths (13.5% of the total) and contributed 92 million Disability Adjusted Life Years (DALYs) worldwide in 2001 (Lopez, Alan, Colin, Ezzati, Majid & Murray, 2006). In the year 2000, non-optimal blood pressure was estimated to have caused approximately 7.1 million deaths (12.8% of the total) and contributed 64.3 million DALYs (Lawes et al., 2006). It has been suggested that the prevalence of cardiovascular disease and hypertension is increasing rapidly in SSA (Seedat, 2004). The current prevalence in many developing countries, particularly in urban societies, is said to be already as high as those seen in developed countries (Khor, 2001; Vorster, 2002). SSA is currently battling with communicable diseases such as malaria and HIV, and most governments in the region have limited resources and health budgets (Forouzanfar et al., 2015).

**Blood pressure** is the force the blood exerts against the walls of the blood vessels. Filtration in capillaries depends upon blood pressure; filtration brings nutrients to tissues. Blood pressure is one of the “vital signs” often measured and, indeed, a normal blood pressure is essential to life (Juhola et al., 2013).

The pumping of the ventricles creates blood pressure, which is measured in mmHg (millimetres of mercury) (Pickering et al., 2005). When a systemic blood pressure reading is taken, two numbers are obtained: systolic and diastolic, as in 110/70 mmHg. Systolic pressure is always the higher of the two and represents the blood pressure when the left ventricle is contracting (Pickering et al., 2005). The lower number is the diastolic pressure, when the left ventricle is relaxed and does not exert force. Diastolic
pressure is maintained by the arteries and arterioles and is discussed in a later section (Pickering et al., 2005).

Systemic blood pressure is highest in the aorta, which receives all of the blood pumped by the left ventricle. As blood travels farther away from the heart, blood pressure decreases (Scanlon & Sanders, 2007). The brachial artery is most often used to take a blood pressure reading; here a normal systolic range is 90 to 120 mmHg, and a normal diastolic range is 60 to 80 mmHg (Staessen et al., 2017). In the arterioles, blood pressure decreases further, and systolic and diastolic pressures merge into one pressure. At the arterial end of capillary networks, blood pressure is about 30 to 35 mmHg, decreasing to 12 to 15 mmHg at the venous end of capillaries (Scanlon & Sanders, 2007). This is high enough to permit filtration but low enough to prevent rupture of the capillaries. As blood flows through veins, the pressure decreases further, and in the caval veins, blood pressure approaches zero as blood enters the right atrium (Scanlon & Sanders, 2007).

The upper limit of the normal blood pressure range is now 120/80 mmHg (Graham, Atar, & Johnsen, 2007). The levels of 125 to 139/85 to 89 mmHg, once considered high-normal, are now called “prehypertension,” that is, with the potential to become even higher. A systemic blood pressure consistently higher than the normal range is called hypertension (Staessen et al., 2017). A lower than normal blood pressure is called hypotension. The regulation of systemic blood pressure is discussed in a later section.

Pulmonary blood pressure is created by the right ventricle, which has relatively thin walls and thus exerts about one-sixth the force of the left ventricle (Scanlon & Sanders, 2007). The result is that pulmonary arterial pressure is always low: 20 to 25/8 to 10
mmHg, and in pulmonary capillaries is lower still (Sanders, 2007). This is important to prevent filtration in pulmonary capillaries, which in turn prevents tissue fluid from accumulating in the alveoli of the lungs (Sironi, Maria et al., 2008).

2.6 OBESITY

The report of World Health Organization, (2008), describes obesity as one of the most blatantly visible, yet most neglected, public health issues that threatens to overwhelm both developed and developing countries. WHO now accepts BMI of 25·0 kg/m$^2$ or higher as abnormal. The overweight category is classified as obese when the BMI is 30·0 kg/m$^2$ or more (Guilbert, 2003), which is a risk factor for multiple CVD risk factors, including hypertension, dyslipidaemia, diabetes and the metabolic syndrome (MetS). BMI is a useful indicator of overall adiposity (Odili et al., 2016).

Obesity is a heterogeneous disorder. Obese individuals vary in their body fat distribution, their metabolic profile and degree of associated cardiovascular and metabolic risk (Grundy, 2004; Yan et al., 2009). However, different fat compartments may be associated with differential metabolic risk (Poirier et al., 2005). In particular, the visceral adipose tissue (VAT) compartment may be a unique pathogenic fat depot (Goodpaster et al., 2005; Klein, 2004). VAT has been termed an endocrine organ, in part because it secretes adipocytokines and other vasoactive substances that can influence the risk of developing metabolic traits (Bacha, Saad, Gungor, & Arslanian, 2004; Matsuzawa, 2006; Nielsen, Guo, Johnson, Hensrud, & Jensen, 2004; Wajchenberg, 2014a; Yatagai et al., 2003). While BMI appears to be the most commonly employed metric used for stratifying obesity in clinical practice, mounting
evidence suggests that measures of visceral obesity such as waist–hip ratio (WHR), waist circumference (WC) and waist to height ratio (WHtR) are superior at predicting incident cardiovascular events, a deranged metabolic profile and death (De Michele et al., 2002; Gast et al., 2015; Kommuri et al., 2016; Kortelainen & Särkioja, 1997; Recio-Rodriguez et al., 2012).

The risk of hypertension is up to five times higher among obese people than among those of normal weight, the variability in response reflecting differential genetic susceptibility as well as dietary factors (Hall, 2003). Up to two-thirds of cases of hypertension are linked to excess weight and cross-sectional population surveys suggest that more than 85% of hypertension arises in individuals with BMI values above 25 kg/m2 (Cía, Armario, Badimón, & Redón, 2002). Studies suggest that anthropometric measures of central obesity, such as waist-hip ratio WHR and WC are better predictors of CVD risk than BMI, the primary marker of general adiposity (Yan et al., 2009).

Obesity and hypertension have been confirmed to have a strong association by both clinical and animal studies (Hall, 2003). Evidence points to intra-abdominal obesity as the most important risk factor for hypertension and CVDs (Sironi et al., 2004).

Waist circumference is an easily obtainable but imprecise measure of abdominal adiposity (Brown, Kuk, & Lee, 2015) because it is a function of both the SCAT and VAT compartments. Therefore, assessment of VAT requires imaging with radiographic techniques such as computed tomography (CT) or magnetic resonance imaging (MRI).

Available studies report relations of greater SCAT and VAT with a higher prevalence of impaired fasting glucose (Goodpaster et al., 2005; Oka et al., 2012), diabetes
insulin resistance (Goodpaster et al., 2005; Tulloch-Reid et al., 2004; Wagenknecht et al., 2003), hypertension (Monda et al., 2013; Sironi et al., 2004), lipids (Kobayashi et al., 2001; Lemieux et al., 1995), metabolic syndrome (MetS) and risk factor clustering (Nagaretani et al., 2001).

Several observational studies have reported that waist circumference, a marker of abdominal obesity, was more strongly associated with cardiovascular mortality (Koster et al., 2008; Longo-Mbenza et al., 2007; Paniagua, Lohsoonthorn, Lertmaharit, Jiamjarasrangsi, & Williams, 2008; Staiano et al., 2012) and subclinical atherosclerosis (Recio-Rodriguez et al., 2012; Yan et al., 2009) than body mass index (BMI). In an earlier study, it was shown that the association between insulin resistance and carotid intima-media thickness, a marker of subclinical atherosclerosis, was largely explained by waist circumference, suggesting that abdominal adiposity is responsible for both insulin resistance and atherosclerosis (Longo-Mbenza et al., 2007).

### 2.7 ABDOMINAL ADIPOSITY

Abdominal obesity is considered a fundamental pathology for MetS development, which is associated with increased risk of cardiovascular morbidity and mortality (Després et al., 2008; Fujioka, Matsuzawa, Tokunaga, & Tarui, 1987).

Studies suggest that central obesity poses greater risk of developing future CV events than peripheral or gluteofemoral obesity (Chen, Xu, & Zhang, 2014; Ibrahim, 2010). There are differences between adipose tissue present in subcutaneous adipose tissue and visceral adipose tissue (Fain et al., 2014; Ibrahim, 2010; Wajchenberg, 2014b). Anatomically, VAT accounts for up to 10–20% of total fat in men and 5–8% in women.
(Ibrahim, 2009). VAT is present mainly in the mesentery and omentum, and drains directly through the portal circulation to the liver (Ibrahim, 2010).

The main areas for SCAT are the gluteofemoral regions, back and anterior abdominal wall. About 80% of all body fat is in the subcutaneous area (Linder, Arner, Flores-morales, Tollet-egnell, & Norstedt, 2004; Wajchenberg, 2014a).

VAT compared with SCAT is more cellular, vascular, innervated and contains a larger number of inflammatory and immune cells, lesser preadipocyte differentiating capacity and a greater percentage of large adipocytes (Bruun, Lihn, Pedersen, & Richelsen, 2005).

Studies show that, the high risk of abdominal adiposity may be explained by larger amounts of VAT (Fox et al., 2007; Lear et al., 2007a; Sironi et al., 2004). Waist circumference however, does not distinguish between VAT and SCAT, which have different biological characteristics and functions. VAT secretes more pro-inflammatory adipokines (Gast et al., 2013), and non-esterified fatty acids (NEFA's) per gram adipose tissue than SCAT (Nielsen et al., 2004), and is therefore considered more atherogenic (Després et al., 2008).

Most studies use WC or WHR to define abdominal obesity (Brook, Bard, Rubenfire, Ridker, & Rajagopalan, 2001; Harman-Boehm et al., 2007; Koskinen et al., 2009; Lemieux, Couillard, Alme, Bergeron, & Gaudet, 2000). However, measurement of these circumferences cannot distinguish between visceral and subcutaneous adipose tissue. The standard methods for quantifying visceral fat amount recommended by the International Diabetes Federation (IDF) are MRI and CT (Alberti, Zimmet, & Shaw, 2006). It was reported that for a Chinese population, visceral fat area (VFA) X80 cm² is optimal for detecting two or more metabolic abnormalities. These include
hyperglycaemia, hypertension and dyslipidaemia, using the IDF definition or the 2004 Chinese Diabetes Society definition (Jia & Pan, 2012).

Many longitudinal cohort researches have reported that excess fat in the upper part of the body (abdominal) considered by Wajchenberg (Wajchenberg, 2014a) as “android or male-type obesity,” correlates with increased mortality and risk for disorders such as diabetes, hyperlipidaemia, hypertension, and atherosclerosis of coronary, cerebral, and peripheral vessels than the “gynoid” (lower body or gluteofemoral or peripheral depot) female type of fat distribution (De Michele et al., 2002; Ibrahim, 2010; Larsson, 1991; Zhu et al., 2013). The WHR is simple and convenient for epidemiological studies and provides a useful estimation of the proportion of abdominal or upper-body fat (Brook et al., 2001; Paniagua et al., 2008; Eyben et al., 2003; WHO, 2008). Studies have reported that, the adverse effect of central obesity on metabolic processes is mediated by the intra-abdominal fat depot (Fujioka et al., 1987; Harman-Boehm et al., 2007; Misra & Vikram, 2003; Sironi et al., 2004). For example, the VAT positively correlated with glucose intolerance in the presence of hyperinsulinemia during an oral glucose tolerance examination, suggesting an insulin-resistant state (Poirier et al., 2005). In addition, correlation analyses have shown the independence of the effect of accumulation of deep abdominal fat on glucose tolerance from total adiposity and subcutaneous SCAT and that no relationship was shown between total adiposity and glucose tolerance after control for VAT (Després et al., 2008; Pouliot et al., 1992). In another study of a wide range of total body fat in both healthy young (20) and middle-aged (21) men, it found that the intra-abdominal fat area evaluated by CT was associated with a decrease in insulin sensitivity measured by an euglycemic hyperinsulinemic glucose clamp. In addition to being associated with disturbances in insulin-glucose homeostasis, abdominal obesity has been related to alterations in
plasma lipoprotein-lipid levels (Kobayashi et al., 2001; Lemieux et al., 1995), especially increased plasma triglyceride and low high-density lipoprotein (HDL) cholesterol concentrations, as expected from the relationship of insulin resistance with disturbances in plasma lipid transport and lipoprotein levels (Fox et al., 2007; Sodjinou, Agueh, Fayomi, & Delisle, 2008).

Although the cause-and-effect relationship has not been definitively proven, the evidence available shows that VAT is an essential connection between the many aspects of the metabolic syndrome: glucose intolerance, hypertension, dyslipidaemia and insulin resistance (Wajchenberg, 2014a). That notwithstanding, due to the substantial metabolic heterogeneity still present among obese individuals with similar levels of VAT, it was suggested that genetic predisposition plays a very important part in adjusting the risk related with a given excess of VAT (Alberi et al., 2006; Bouchard, Desprais, & Maurieige, 1993). In this regard, visceral obesity should be regarded a factor that aggravates an individual’s genetic predisposition to the elements of the metabolic syndrome (Purnell, Kahn, Schwartz, & Brunzell, 2001). Even though there is a concurrence that VAT has a strong association with CV risk factors, especially dyslipidaemia and hyperinsulinemia (Larsson, 1991; Molica, Morel, Kwak, Rohner-Jeanrenaud, & Steffens, 2015), the main importance of VAT in relation to SCAT obesity with regard to insulin sensitivity of glucose metabolism has been questioned by Abate et al., (1995) and Goodpaster et al., (1997). Goodpaster et al., (1997) realized that abdominal SCAT was at least as strong a correlate of insulin sensitivity as determined by MRI and CT scan, (evaluated by the euglycemic clamp) as VAT and maintained independent significance after adjusting for VAT. In another study of intervention strategy to promote loss of VAT, measured by CT, Doucet et al., (2002) came to the conclusion that individuals with higher VAT, either through an increase in
body weight or the ability to store fat in the visceral depot, lose more VAT when adjusted to the loss of body fat, regardless of the intervention used (caloric restriction, pharmacological therapy, or exercise) due to the fact that visceral adipocyte has a greater lipolytic rate also in the steady state. In addition, it has been accentuated that the endocrine abnormalities described in obesity, which involve steroid hormones, growth hormone and insulin, may as a matter of fact result in abdominal depot fat accretion. This might cause the MetS in the predisposed individual (Ali & Crowther, 2005; Jensen, 2008).

Previous studies in the general population that directly assessed VAT by CT scan or MRI have shown that more VAT was associated with a larger CIMT (Cooper et al., 2012; Indulekha, Anjana, Surendar, & Mohan, 2011; Lear et al., 2007a; Lo et al., 2006; Wang et al., 2012; Wildman et al., 2011). It was noted that VAT is strongly correlated with total body fat (Martin, Janssens, Caboor, Clarys, & Marfell-Jones, 2003). Therefore, when studying specific effects of VAT, results should be adjusted for total body fat (Wajchenberg, 2014a). To my knowledge, only two of previous studies adjusted for total body fat and showed inconsistent results (Lear et al., 2007a; Oka et al., 2012). In one study VAT contributed to CIMT above total body fat (Lear et al., 2007a), whereas in the other study VAT did not contribute and total body fat was more important in the association with CIMT (Oka et al., 2012). Therefore, the individual contributions of VAT and total body fat to the development of atherosclerosis remain unclear.

Within the abdomen, not only absolute amounts of VAT and SCAT may be important in the development of atherosclerosis, but also their proportion relative to each other. It is hypothesized that excess triglycerides are primarily stored in SCAT and when this depot cannot sufficiently expand, triglycerides will be stored in the visceral area and
eventually in non-adipose tissues such as the liver and skeletal muscle (Martin et al., 2003; Wells, 2007). According to this “lipid overflow” hypothesis (Martin et al., 2003; Wells, 2007), a high relative amount of VAT may thereby indicate dysfunctional SCAT. The ratio of abdominal VAT and SCAT (VAT/SCAT) was recently proposed as a marker reflecting the propensity to store triglycerides viscerally (Kaess et al., 2012). However, when considering this ratio, which can be the same in persons with different amounts of total body fat, it is important to adjust associations for total body fat as well. Furthermore, it is unclear whether the same ratio in persons with different amounts of body fat is associated with similar cardiovascular risk.

2.8 ABDOMINAL ULTRASOUND MEASUREMENTS

Ultrasound measurements of SCAT and VAT, the latter has to do with the distance between the peritoneum and aorta, were measured 5 cm from the umbilicus on the xipho-umbilical line with a 7.5-MHz probe for SCAT and a 3.5-MHz probe for VAT (Armellini et al., 1993). The intra-individual reproducibility of ultrasound measurements was very high both for intra-abdominal and subcutaneous thickness as well as for inter-operators (Pineau et al, 2010; Seven, Thuesen, Linneberg, & Jeppesen, 2016; Stolk et al., 2001).

Several studies demonstrated a highly significant correlation between the intra-abdominal adipose tissue determined by CT and by US. A decade ago, Armellini et al., (1993) found some correlation ($r \approx 0.67$) of intra-abdominal US measurements with CT at the L4-L5 level. In a more recent study, Seven et al., (2016) found a highly significant
correlation between intra-abdominal thickness and CT visceral adipose tissue area ($r \approx 0.89–0.91$) and Radominski, (1998), at The Hospital das Clinicas of São Paulo, Brazil, also observed in 24 subjects an excellent correlation between ultrasonography and CT ($r \approx 0.79$ for subcutaneous abdominal thickness and $r \approx 0.84$ for visceral adipose tissue), again indicating that ultrasound intra-abdominal thickness is a good predictor of visceral abdominal adipose tissue (Goodpaster et al., 1997; Holzhauer et al., 2009; Lee, Hsiao, Yang, Huang, & Chan, 2017; Rasslan et al., 2015). In a cross-validation study, VAT measured by CT was significantly correlated with VAT predicted from an equation using primarily ultrasound VAT ($r \approx 0.84$) (Grima, Guido, Chiavaroli, & Zizza, 2010).
CHAPTER THREE

MATERIALS AND METHODS

3.0 STUDY DESIGN

The study was a cross-sectional ultrasound study. The study employed simple random sampling method to recruit the participants.

3.1 ETHICAL CLEARANCE AND APPROACH TO INFORMED CONSENT

Approval for the study was obtained from the Ethical and Protocol Review Committee of the College of Health Sciences, University of Ghana, Korle-Bu. Approval and permission were also sought from the Navrongo Health Research Centre where the study was conducted. In addition, the participants were made to consent to their participation in the study via the completion of informed consent form (see Appendix I), before the start of the study.

3.2 STUDY SITE

The study was carried out at the Clinical Trial Centre of the Navrongo Health Research Centre (NHRC) in the Kassena-Nankana East District. The study participants were recruited from the Kassena-Nankana East and West districts formally the Kassena-
Nankana District of Upper East Region of Ghana. The area covering the two districts lies between latitude 10.30’ and 11.10’ north and longitude 1.1’ west and cover a total land area of 1675km² along the Ghana-Burkina Faso border. The district is under the Health and Demographic Surveillance system (HDSS) being carried out by NHRC where the study was conducted. As at June 2011 the total population size of the district stood at 153,293 with a population density of 91.5 Km² and 52% being female. The majority ethnic groups in the area are the Kassena and the Nankani. The minority groups in the study area include Bulsas, Dagabas among others (Oduro et al., 2012).

3.3.0 STUDY PARTICIPANTS

3.3.1 Sample size

Considering the nature of the study and population under study, the sample size was determined by the formula below (Chandashekara & Suresh, 2012);

\[ N = \frac{(Z\alpha/2 + Z1-\beta)^2}{\frac{1}{4}[\log\frac{1+r}{1-r}]} + 3 \]

\[ N = (1.96+0.9)^2/ \frac{1}{4}[\log\frac{1+0.3}{1-0.3}] + 3 \]

\[ N = 499.7 \sim 500 \]

Where \( Z\alpha/2 \) and \( Z1-\beta \) are normal deviates for type I error (significance level) and Power of study.

Hence a total sample size of 500 participants with equal proportion of both sexes were recruited for the study.
3.3.2 Inclusion criteria

- Both sexes within the age category of 40-60 years were eligible for participation.
- All individuals permanently resident within the study area were eligible for participation.
- All individuals who were clinically diagnosed and were on anti-hypertensive treatment were eligible for the study.
- All individuals who were clinically diagnosed hypertensive but were not on treatment were eligible for the study.
- There must be willingness of eligible participants to give consent and sign or thumbprint an informed consent document for participation in all the processes of the study.

3.3.3 Exclusion criteria:

The following exclusive criteria were considered;

- Failure to give consent to participate in the study.
- Pregnant women
3.4 QUESTIONNAIRE

Demographic data was collected via interviewer-administered questionnaires that covered areas such as: date of birth, sex, self-reported ethnicity, CVD related risk factors, medical and health histories as captured in Appendix II.

3.5 MEASUREMENT OF BODY WEIGHT, HEIGHT, WAIST CIRCUMFERENCE AND HIP CIRCUMFERENCE

Body weight and height, in light clothing and without shoes were measured using Seca 803 electronic flat scale and a fixed Harpenden Stadiometer (Holtain, UK).

Waist and hip circumferences were measured at the end of a normal expiration with arms relaxed at the sides, directly over the skin or light clothing in standing position. The WC of the participants was measured to the nearest 0.5cm between the lower margin of the 12th rib and the iliac crest using a Seca 201 stretch-resistant metric tape. Hip circumference was measured with the metric tape wrapped around the maximum circumference of the buttocks, with the participants standing with their feet together with weight evenly distributed over both feet according to the guidelines of the WHO, (2008) as indicated in Appendix III.

From these measurements the study was able to determine BMI, WHR, WHtR, and group the study population into underweight, normal weight, overweight and obese for the analysis.
3.6 MEASUREMENT OF VISCERAL AND SUBCUTANEOUS ADIPOSY

Ultrasound was used to determine abdominal adipose depths as proxies for VAT and SCAT. A LOGIQ e ultrasound system (USS) (GE Healthcare, CT, USA) with a 2-5 MHz 3C-RS curved array transducer was used to determine VAT and SCAT. USS VAT thickness was defined as the distance (cm) from the peritoneum to the vertebral bodies, and USS SCAT thickness was defined as the depth (cm) from the skin to the linea alba (De Lucia et al., 2011).

The scan depth was set at 15 cm for the visceral fat measure and 9 cm for the subcutaneous fat measure in order to visualise the relevant anatomical structures. Both measurements were obtained where the vertical line between the xyphoid process and the symphysis intercepts with the waist circumference. All USS measurements were taken by the investigator who is a trained operator. Relative inter-observer technical error of measurement (TEM) was assessed regularly to ensure that the TEM remains below 2% in comparison with another trained operator. TEM was calculated on repeated measurements in 15 individuals (De Lucia et al., 2011). (See Appendix IV).

3.7 MEASUREMENT OF COMMON CAROTID INTIMA-MEDIA THICKNESS

The ultrasound machine was used to evaluate the common carotid artery structure in all participants using high-resolution B-mode ultrasound equipped with a 7.5 MHz linear array transducer. The subjects were made to lie down in a supine position with slight hyperextension and rotation of the neck in the direction opposite the orientation
of the probe. A linear array transducer with a multiple frequency (7 to 12MHz) attached to a high-resolution B mode ultrasound system was used to acquire CIMT images. The ultrasound parameters taken in this study were the maximal CIMT, mean CIMT and minimum CIMT. CIMT was determined as the distance from the media-adventitia interface to the intima-lumen interface on the far wall at both sides (left/right) in a region free of plaques. The two measurements per side were taken according to the standard protocol (see Appendix V).

3.8 MEASUREMENT OF BLOOD PRESSURE

The hypertensive status of the participants was determined and grouped into controlled and uncontrolled hypertension by measuring the individual Blood Pressure (BP) or report of a prior diagnosis of hypertension and current antihypertensive treatment. The BPs were measured using a digital device (Omron M6; Omron, Kyoto, Japan). Appropriate cuff sizes were used, and BPs were measured with participants seated and resting with two minutes break between measurements according to WHO guidelines (Staessen et al., 2017). (See Appendix IV). Three measurements were taken and averaged.

In this study, the BP measurements were grouped into Normal BP, Pre BP, Stage 1 and Stage 2 according to WHO standard (Staessen et al., 2017).
Table 1. Blood Pressure Categories

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>80-90</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>≥160</td>
<td>≥100</td>
</tr>
</tbody>
</table>

3.9 BLOOD AND URINE SAMPLES COLLECTION AND PROCESSING

Overnight fasting venous blood samples were taken by a qualified phlebotomist as outlined in the Table 2. Overnight fasting blood samples were collected from the antecubital vein with minimum stasis. Plasma and serum samples were obtained by centrifugation at 200xg for 20 minutes at 4°C and aliquots were stored at -80°C until assayed (See Table 2 and Appendix VI).

Spot-urine was collected, centrifuged at 1600xg for 5 minutes and aliquots stored at -80°C until assayed (See Table 2).
Vitalab Selectra E Chemistry Analyzer was used to run all the bioassays according the standard protocol (see Appendix 4).

Cholesterol, triglyceride and HDL-cholesterol analyses were performed using a Vitalab Selectra E Chemistry Analyzer. Cholesterol High Performance reagent (cat.no. 704036), Roche Diagnostics) was used to measure cholesterol enzymatically. Triglycerides were analysed enzymatically simultaneously with cholesterol using reagents from the same manufacturer (Triglycerides/GPO, cat. no. 1488872). Triglyceride blanks were measured in CDC surveillance materials using the same reagent, but without lipase. Direct HDL-cholesterol reagent was obtained from Roche Diagnostics (Direct HDL, cat. no. 1661442), and analysed at the same time with the others. If for some reason analyses were delayed, the specimens were kept frozen at -80° C until they are analysed as indicated in Appendix 5.

Results are given in millimol per litre (mmol/L) and were converted to milligrams per decilitre (mg/dL) during analysis. The following ranges were used according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines (National Cholesterol Education Program, 2001) in the study to categorise the data on lipid profile;

- Less than 200 mg/dL: Normal
- 200 to 239 mg/dL: Borderline high
- At or above 240 mg/dL: High

The following are the ranges for **LDL cholesterol** that were used in the study:

- Less than 100 mg/dL: Optimal
- 100 to 129 mg/dL: Near optimal
- 130 to 159 mg/dL: Borderline high
- 160 to 189 mg/dL: High
- 190 mg/dL and higher: Very high

The acceptable range of **HDL cholesterol levels** were values above **40 mg/dL**.

Here are the ranges for triglyceride used in the study:

- Less than 150 mg/dL: Normal
- 150 to 199 mg/dL: Borderline high
- 200 to 499 mg/dL: High
- Above 500 mg/dL: Very high

---

**Table 1. Biochemical assays**

<table>
<thead>
<tr>
<th>Tube</th>
<th>Additive</th>
<th>Quantity</th>
<th>Test</th>
<th>Substance</th>
<th>Aliquot</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red</td>
<td>None</td>
<td>6ml</td>
<td>Total cholesterol</td>
<td>Serum</td>
<td>2 x 1ml</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>LDL</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HDL</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Triglycerides</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Insulin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grey</td>
<td>Sodium Fluoride (Glycolytic inhibitor)</td>
<td>2ml</td>
<td>Blood glucose</td>
<td>Plasma</td>
<td>2 x 0.5ml</td>
</tr>
<tr>
<td>Urine</td>
<td>None</td>
<td>8ml</td>
<td>Creatinine: albumin ratio, total protein, bilirubin</td>
<td>urine</td>
<td></td>
</tr>
</tbody>
</table>
3.10 STATISTICAL ANALYSIS

In order to achieve the aim of the study, the following statistical approach were conducted:

Descriptive statistics was used to analyse the participants’ socio-demographic characteristics, anthropometric and CVD risk factors.

Quantitative variables were reported as mean ± SD. Variables with skewed distributions were presented as median and interquartile range, and log transformed before analysis.

Boxplot method was used in assessing the association between the left and right CIMT and BP, Smoking, alcohol, LDL, total cholesterol, triglyceride and HDL. To further determine if the observed difference in CIMT was different for the various variables, a non-parametric analysis employing Kruskal-Wallis test was conducted. And in the case of CMT and HDL, Maan Whitney test was used.

Boxplot and Kruskal Wallis test were also used to determine the association between BP and the anthropometric variables.

Scatter plot test was used to determine the association between CIMT and quantitative variables such as ACR, VAT, SCAT, BMI, WHR WHtR and WC. Spearman correlation was further used to examine the strength of the linear relationship and to determine if the observed relationship is statistically significant.

The same scatter plot and spearman correlation methods were used to determine the association between VAT, SCAT and BMI, WHR, WC, WHtR.
CHAPTER FOUR
RESULTS

4.0 DEMOGRAPHIC, ANTHROPOMETRIC AND CLINICAL CHARACTERISTICS

The study included 500 hypertension patients aged 40 to 60 years. 250 were males and 250 were females. The entire data was analysed.

Participants were asked to report their gender, age, education, occupation, marital status, smoking status, alcohol status, medical history for socio-demographic data (see table: 3 and 4) by a self-report structure questionnaire. See Appendix II.

Demographic characteristics, anthropometric and biochemical measures of the participants were presented in Table 5 by gender. In general the study had 52 years as mean age (ranged 40-60 years), the mean age of the population by sex were 51.65 (SD ± 5.77) for males and 53.37 (SD ± 5.47) for females. 64.80% of the population never had any form of formal education.

The mean BMI measurements were 21.10 (SD ± 3.49) kg/m2 for males and 22.67 (SD ± 4.14) kg/m2 for females. The data also indicated that 76 (15.20%) participants were underweight, 61 (12.0%) individuals were overweight, 20 (4%) were obese. As high as 343 individuals representing 68.60% of the entire sample size were within the normal body weight (See tables 3 and 5).

Males had a higher standing height, weight, and HDL-cholesterol than females. Females had higher waist circumference, hip circumference, systolic and diastolic blood pressure, total cholesterol level, LDL-cholesterol levels and triglycerides level than males as in presented in Table 5.
Participants who fell under prehypertension condition category were 109 in number representing 21.80% of the sample size. Stage 1 hypertension accounted for the highest observations; 233 (46.60%) followed by Stage 2 hypertension; 152 (30.40%) with 4 (0.80%) being least number of individuals with normal blood pressure. It was observed that participants within the normal blood pressure brackets were among individuals on anti-hypertension drugs.

Table 2. Demographics of the study population

<table>
<thead>
<tr>
<th>Background Characteristics</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
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<td></td>
</tr>
<tr>
<td>Male</td>
<td>250</td>
<td>50</td>
</tr>
<tr>
<td>Female</td>
<td>250</td>
<td>50</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No formal</td>
<td>349</td>
<td>69.80</td>
</tr>
<tr>
<td>Primary</td>
<td>93</td>
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</tr>
<tr>
<td>Secondary</td>
<td>43</td>
<td>8.60</td>
</tr>
<tr>
<td>Tertiary</td>
<td>15</td>
<td>3.00</td>
</tr>
<tr>
<td><strong>Occupation</strong></td>
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<td></td>
</tr>
<tr>
<td>Self-employed</td>
<td>281</td>
<td>56.20</td>
</tr>
<tr>
<td>Formal full-time</td>
<td>21</td>
<td>4.20</td>
</tr>
<tr>
<td>Part-time</td>
<td>6</td>
<td>1.20</td>
</tr>
<tr>
<td>Informal</td>
<td>1</td>
<td>0.20</td>
</tr>
<tr>
<td>Unemployed</td>
<td>190</td>
<td>38.00</td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married or co-habited</td>
<td>6</td>
<td>1.20</td>
</tr>
<tr>
<td>Married/living together</td>
<td>345</td>
<td>69.00</td>
</tr>
<tr>
<td>Divorced/separated</td>
<td>29</td>
<td>5.80</td>
</tr>
<tr>
<td>Widow/widower</td>
<td>120</td>
<td>24.00</td>
</tr>
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</table>
Continuation of Table 2. Demographics of the study population

<table>
<thead>
<tr>
<th>Background Characteristics</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
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<tr>
<td><strong>BMI category</strong></td>
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<tr>
<td>Underweight</td>
<td>76</td>
<td>15.20</td>
</tr>
<tr>
<td>Normal weight</td>
<td>343</td>
<td>68.60</td>
</tr>
<tr>
<td>Overweight</td>
<td>61</td>
<td>12.0</td>
</tr>
<tr>
<td>Obesity</td>
<td>20</td>
<td>4.00</td>
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<tr>
<td><strong>Smoking status</strong></td>
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<tr>
<td>Never smoked</td>
<td>33.4</td>
<td>66.80</td>
</tr>
<tr>
<td>Current smoker</td>
<td>104</td>
<td>20.80</td>
</tr>
<tr>
<td>Former smoker</td>
<td>61</td>
<td>12.20</td>
</tr>
<tr>
<td><strong>Alcohol status</strong></td>
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<td></td>
</tr>
<tr>
<td>Never consumed</td>
<td>72</td>
<td>14.40</td>
</tr>
<tr>
<td>current non-problematic</td>
<td>163</td>
<td>32.60</td>
</tr>
<tr>
<td>current problematic</td>
<td>169</td>
<td>33.80</td>
</tr>
<tr>
<td>former consumer</td>
<td>95</td>
<td>19.00</td>
</tr>
<tr>
<td><strong>Hypertension status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>4</td>
<td>0.80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>109</td>
<td>21.80</td>
</tr>
<tr>
<td>Stage 1</td>
<td>233</td>
<td>46.60</td>
</tr>
<tr>
<td>Stage 2</td>
<td>152</td>
<td>30.40</td>
</tr>
<tr>
<td>Error readings</td>
<td>2</td>
<td>0.40</td>
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</table>
Table 3. Demographics on the medical history of the study population

<table>
<thead>
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<th>Background Characteristics</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart attack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>494</td>
<td>98.80</td>
</tr>
<tr>
<td>Yes</td>
<td>6</td>
<td>1.20</td>
</tr>
<tr>
<td>Kidney disease</td>
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<td></td>
</tr>
<tr>
<td>No</td>
<td>496</td>
<td>99.20</td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
<td>0.20</td>
</tr>
<tr>
<td>Don’t Know</td>
<td>2</td>
<td>0.20</td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>491</td>
<td>98.20</td>
</tr>
<tr>
<td>Yes</td>
<td>4</td>
<td>0.80</td>
</tr>
<tr>
<td>Don’t know</td>
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<td>0.40</td>
</tr>
<tr>
<td>No response</td>
<td>3</td>
<td>0.60</td>
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<tr>
<td>Stroke</td>
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<tr>
<td>No</td>
<td>493</td>
<td>98.60</td>
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<tr>
<td>Yes</td>
<td>7</td>
<td>1.40</td>
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### Table 4. Anthropometric, Ultrasound measurements and Clinical characteristics of study participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male Mean ± SD</th>
<th>Female Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong> median, (IQR)</td>
<td>51.65 ± 5.77</td>
<td>53.37 ± 5.47</td>
</tr>
<tr>
<td><strong>Anthropometry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>21.10 ± 3.49</td>
<td>22.67 ± 4.14</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>746.57 ± 84.76</td>
<td>782.00 ± 103.94</td>
</tr>
<tr>
<td>Hip Circumference</td>
<td>855.49 ± 102.13</td>
<td>900.19 ± 95.12</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>0.88 ± .06</td>
<td>0.87 ± .06</td>
</tr>
<tr>
<td>Height</td>
<td>1670.85 ± 78.53</td>
<td>1579.88 ± 68.03</td>
</tr>
<tr>
<td>Weight</td>
<td>59.01 ± 11.13</td>
<td>56.79 ± 11.99</td>
</tr>
<tr>
<td>Waist-Height Ratio</td>
<td>0.45 ± .05</td>
<td>0.50 ± .06</td>
</tr>
<tr>
<td><strong>Blood Pressure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>149.24 ± 15.92</td>
<td>152.97 ± 20.93</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>90.37 ± 10.26</td>
<td>92.31 ± 12.28</td>
</tr>
<tr>
<td>Pulse</td>
<td>72.89 ± 13.39</td>
<td>76.62 ± 14.23</td>
</tr>
<tr>
<td><strong>Ultrasound Measurements</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CIMT right</td>
<td>0.68 ± .15</td>
<td>0.71 ± .14</td>
</tr>
<tr>
<td>CIMT left</td>
<td>0.72 ± .17</td>
<td>0.75 ± .16</td>
</tr>
<tr>
<td>VAT</td>
<td>4.34 ± 1.28</td>
<td>3.71 ± 1.16</td>
</tr>
<tr>
<td>SCAT</td>
<td>0.87 ± .46</td>
<td>1.24 ± .59</td>
</tr>
<tr>
<td><strong>Chemical Tests</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. Cholesterol (mmol/l)</td>
<td>3.26 ± 1.07</td>
<td>3.34 ± 1.05</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>0.66 ± 0.33</td>
<td>0.69 ± 0.39</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/l)</td>
<td>1.28 ± 0.46</td>
<td>1.18 ± 0.41</td>
</tr>
<tr>
<td>LDL-cholesterol (mmol/l)</td>
<td>1.76 ± 0.87</td>
<td>1.79 ± 0.86</td>
</tr>
<tr>
<td>Insulin (mmol/l)</td>
<td>10.30 ± 41.01</td>
<td>5.11 ± 6.70</td>
</tr>
<tr>
<td>Creatinine (mmol/l)</td>
<td>76.17 ± 23.31</td>
<td>66.12 ± 28.28</td>
</tr>
<tr>
<td>Albumin mmol/l</td>
<td>47.30 ± 256.60</td>
<td>50.62 ± 308.65</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>4.57 ± 0.90</td>
<td>4.63 ± 0.66</td>
</tr>
<tr>
<td>Albumin: Creatinine ratio</td>
<td>3.87 ± 21.51</td>
<td>8.98 ± 66.1</td>
</tr>
<tr>
<td>Insulin (mmol/l)</td>
<td>0.15 ± 0.39</td>
<td>0.14 ± 0.58</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>4.57 ± 0.91</td>
<td>4.63 ± 0.66</td>
</tr>
</tbody>
</table>
4.1  RELATIONSHIP BETWEEN CIMT (RIGHT) AND BLOOD PRESSURE

The mean CIMT (right) for participants with normal blood pressure was the highest compared to the other three groups. The boxplot for participants with normal blood pressure levels shows negative skew: the median is pulled to the upper end of the box. This was followed by participants with stage 2 blood pressure. Participants within the pre-hypertensive and stage 1 hypertension group had similar CIMT levels with a few outliers.

Due to the skewed nature of the data, a Kruskal-Wallis test was conducted to determine if the observed difference in CIMT was different for the four groups: Normal blood pressure \([n=4; \text{Median}= 0.77; \text{IQR} = (0.65, 0.77)]\); prehypertension \([n=109; \text{Median}= 0.66; \text{IQR} = (0.59, 0.76)]\); Stage 1 \([n= 233; \text{Median}= 0.67; \text{IQR} = (0.59,0.78)]\) and Stage 2 \([n= 152; \text{Median}= 0.70; \text{IQR} = (0.63, 0.82)]\). The result from the Kruskal-Wallis test showed that there was a statistical significant difference in CIMT between the four groups, \(X^2(3) = 12.65, p \text{– value} = 0.0054\).

\[\text{Figure 3. Box plot of the mean CIMT by blood pressure levels.}\]
4.2 RELATIONSHIP BETWEEN CIMT (RIGHT) AND SMOKING STATUS

Figure 2 shows a similar distribution of CIMT for participants who are current smokers [n=104; Median=0.66; IQR= (0.59, 0.77)] or never smoked [n=334; Median=0.67; IQR= (0.60, 0.79)] with few outlying values. The median CIMT level for former smokers was highest than the other groups [n=61; Median=0.71; IQR= (0.61, 0.80)].

To determine if there is a significance difference in the mean CIMT (right) between the three groups, Kruskal-Wallis test was used. The result from the Kruskal-Wallis test showed that there was no statistical significant difference in CIMT between the four groups, $X^2(2) = 1.383, p - value = 0.5005$.

Figure 4. Box plot of the mean CIMT by participant smoking status.
4.3 RELATIONSHIP BETWEEN CIMT (RIGHT) AND ALCOHOL CONSUMPTION

Participants with current problematic alcohol consumption status have lower CIMT [n=169, Median=0.64; IQR= (0.58, 0.75) compared to those in the other groups suggesting that there is difference in the mean CIMT levels between the alcohol groups. The results from the Kruskal-Wallis further supports that there is a significant difference in the population median of CIMT between the alcohol categories, [\(X^2(3) = 16.000, p \text{-value} = 0.0011\)].

![Box plot of the mean CIMT by participant alcohol consumption status.](image)
4.4 RELATIONSHIP BETWEEN CIMT (RIGHT) AND LDL

Figure 6. suggests that the mean CIMT differ across the five LDL levels. However, the results from the Kruskal-Wallis test \( X^2 (4) = 2.55, p \text{-value} = 0.63 \) shows that there is no evidence of rejecting the null hypothesis that there is difference in the median of the mean CIMT between the five LDL groups.

Figure 6. Box plot of the mean CIMT by participant LDL
4.5 RELATIONSHIP BETWEEN CIMT (RIGHT) AND TOTAL CHOLESTEROL

Figure 7 shows similar distribution of CIMT for participants with cholesterol levels less than 200 [n= 311; Median= 0.68; IQR= (0.60, 0.78)] with few outlying values, between 200 and 239 [n=15; Median=0.62; IQR= (0.56, 0.80)] which is a bit skewed in a positive direction. The median CIMT level for participant with cholesterol levels from 240 and above [n=61; Median= 0.71; IQR= (0.61, 0.80)].

To determine if there is a significance difference in the median CIMT between the three groups, Kruskal-Wallis test was used. The result from the Kruskal-Wallis test further confirmed that there was no statistical significant difference in CIMT (right) between the four groups, $X^2 (2) = 2.82, p-value = 0.24$.

![Box plot of the mean CIMT by participant Cholesterol](http://ugspace.ug.edu.gh)

*Figure 7. Box plot of the mean CIMT by participant Cholesterol*
4.6 RELATIONSHIP BETWEEN CIMT (RIGHT) AND TRIGLYCERIDE

Across the four levels of triglyceride (<150; 150-199; 200-499 ;> 499), the results shows that no participants had triglyceride levels higher than 499. The median CIMT for participants with triglyceride levels less than 150 was lower compared to the other two groups as shown in Figure 8.

According to the results from the Kruskal-Wallis test the median triglyceride levels for participants with triglyceride less than 150 [n=318; Median= 0.67; IQR= (0.60, 0.78)]; between 150 and 199 [n=7; Median=0.77; IQR= (0.68, 0.94)] and between 200 and 499 [n=3; Median= 0.76; IQR= (0.56, 0.94)], statistically insignificant for the three levels of triglyceride [$X^2(2) = 4.75, p-value = 0.9$].

![Figure 8. Box plot of the mean CIMT (right) by Triglyceride](http://ugspace.ug.edu.gh)
4.7 RELATIONSHIP BETWEEN CIMT (RIGHT) AND HDL

Figure 9 shows participants with HDL levels above 40 have relatively higher CIMT [n=231, Median= 0.67; IQR= (0.59, 0.78)] compared to those in with levels less than 40 [n= 97, Median= 0.70; IQR= (0.64, 0.79)].

To determine if there is a significance difference in the mean CIMT (left) between the two groups, a Wilcoxon rank sum test was used. The result from the test showed that the difference in the median of the mean CIMT between the two groups was statistical significant [$Z= 2.00, p − value = 0.045$]

Figure 9. Box plot of the mean CIMT (right) by HDL
4.8 RELATIONSHIP BETWEEN CIMT (RIGHT) AND ACR, WH RATIO, VAT, SCAT, WHt RATIO, BMI

Figure 10 shows a combined scatter plots of CIMT and albumin-creatinine ratio (first on the first row), visceral fat (middle on the first row), subcutaneous fat (last on the first row), WH ratio (first on the second row), WHt ratio (middle on the second row) and BMI (last on the second row). The plots show no relationship between these sets of variables and mean CIMT. This is because no pattern could be discerned from the plots to suggest any form of relationship or association.

Spearman correlation was further used to examine whether there was any form of association, the strength of the linear relationship and determine if the observed relationship is statistically significant. According to the results from the spearman correlation, there is a significant positive relationship between mean CIMT right and WH \( r_{ho} = 0.240, p\text{ – value} < 0.001 \).
Figure 10. Combined scatter plots of CIMT and other variables
4.9 RELATIONSHIP BETWEEN CIMT (LEFT) AND BLOOD PRESSURE

The median CIMT left for participants with Stage 2 blood pressure was the highest compared to the other three groups with few outliers. The boxplot for participants with prehypertension blood pressure levels shows positive skew: the median is pulled to the lower end of the box. The normal BP and prehypertension participants was negatively skewed. Participants within the pre-hypertensive and stage 1 hypertension group had similar median CIMT left levels with a few outliers.

Due to the skewed nature of the data, a Kruskal-Wallis test was conducted to determine if the observed difference in CIMT left was different for the four groups: Normal blood pressure \( [n=4; \text{Median}=0.70; \text{IQR}=(0.61, 0.78)] \); prehypertension \( [n=109; \text{Median}=0.71; \text{IQR}=(0.65, 0.80)] \); Stage 1 \( [n=233; \text{Median}=0.71; \text{IQR}=(0.61, 0.80)] \) and Stage 2 \( [n=152; \text{Median}=0.74; \text{IQR}=(0.63, 0.86)] \). The result from the Kruskal-Wallis test showed that there was a statistical significant difference in CIMT between the four groups, \( X^2(3) = 4.39, p-value = 0.022 \)
Figure 11. Box plot of the mean CIMT left by blood pressure levels
4.10 RELATIONSHIP BETWEEN CIMT (LEFT) AND SMOKING STATUS

Figure 12 shows a similar distribution of CIMT for participants who are current smokers \([n=104; \text{Median}=0.71; \text{IQR}=(0.61, 0.80)]\) with few outlying values. The median of mean CIMT (left) level for never smoked \([n=334; \text{Median}=0.72; \text{IQR}=(0.64, 0.82)]\) was highest followed by former smokers among the groups \([n=61; \text{Median}=0.70; \text{IQR}=(0.61, 0.84)]\).

To determine if there is a significance difference in the mean CIMT left between the three groups, Kruskal-Wallis test was used. The result from the Kruskal-Wallis test showed that there was no statistical significant difference in mean CIMT between the four groups, \(X^2(2) = 1.325, p-value = 0.5157\)

*Figure 12.Box plot of the mean CIMT by participant’s smoking status*
4.11 RELATIONSHIP BETWEEN CIMT LEFT AND ALCOHOL CONSUMPTION STATUS

Figure 13 shows that participants with current problematic alcohol consumption status have lower CIMT \([n= 169, \text{Median}= 0.69; \text{IQR}= (0.61, 0.79)\] as may be expected compared to those in the other groups suggesting that there is difference in the mean CIMT levels between the alcohol groups. Non-alcohol consumers; \([n= 72, \text{median}=0.77 \text{ IQR}= 0.66, 0.86]\] had the highest median that was negatively skewed followed. Interestingly, the median for former alcohol consumers was relatively highest among the groups apart from non-alcohol consumers and positively skewed with two outliers \([n=95, \text{median}= 0.75, \text{IQR}= 0.65, 0.87]\] followed by current non-problematic consumers \([n=163, \text{median}=0.71, \text{IQR}= 0.63, 0.81]\]

The results from the Kruskal-Wallis further supports that there is a significant difference in the population median of mean CIMT (left) between the alcohol categories \([X^2(3) = 13.754, p–value = 0.0033]\).
Figure 13. Box plot of the mean CIMT left by participant’s alcohol consumption status
4.12 RELATIONSHIP BETWEEN CIMT LEFT AND LDL

Figure 14 suggests that the mean CIMT left differ across the five LDL levels. However, the results from the Kruskal-Wallis test [$X^2(4) = 4.842, p-value = 0.305$] shows no statistical significant difference in the population median of mean CIMT (left) between the LDL levels.

![Figure 14. Box plot of the mean CIMT left by participant’s LDL levels](image)

Figure 14. Box plot of the mean CIMT left by participant’s LDL levels
4.13 RELATIONSHIP BETWEEN CIMT (LEFT) AND TOTAL CHOLESTEROL

Figure 15 shows differences in the distribution of mean CIMT (left) for participants with cholesterol levels less than 200 [n= 311; Median=0.72; IQR= (0.63, 0.84)] with few outlying values, between 200 and 239 [n=15; Median= 0.62; IQR= (0.58, 0.82)] which is a skewed in a positive direction. The highest median level was that of participants with cholesterol levels from 240 and above [n=2; Median= 0.78; IQR= (0.64, 0.97)].

To determine if there is a significance difference in the median of the mean CIMT (left) between the three groups, Kruskal-Wallis test was used. The result from the Kruskal-Wallis test further showed that there was no statistical significant difference between the mean CIMT (left) between the four groups, $X^2 (2) = 1.325, p - value = 0.515$.

Figure 15. Box plot of the mean CIMT (left) by participant’s Total Cholesterol levels
4.14 RELATIONSHIP BETWEEN CIMT LEFT AND TRIGLYCERIDE

Across the four levels of triglyceride groupings (<150; 150-199; 200-499; > 499), the results show that no participants had triglyceride levels higher than 499. The median CIMT for participants with triglyceride levels less than 150 was lower compared to the other two groups.

According to the results from the Kruskal-Wallis test the median triglyceride levels for participants with triglyceride less than 150 \([n=318; \text{Median}= 0.71; \text{IQR}= (0.62, 0.83)]\); between 150 and 199 \([n=7; \text{Median}= 0.78; \text{IQR}= (0.62, 0.96)]\) and between 200 and 499 \([n=3; \text{Median}= 0.80; \text{IQR}= (0.68, 0.84)]\), were statistically insignificant for the three levels of triglyceride \([X^2(2) = 2.705, p - value = 0.2585]\) as reported in Figure 16.

![Box plot of the mean CIMT (left) by Triglyceride](http://ugspace.ug.edu.gh)

*Figure 16. Box plot of the mean CIMT (left) by Triglyceride*
4.15 RELATIONSHIP BETWEEN CIMT (LEFT) AND HDL

Figure 17 shows participants with HDL levels above 40 have relatively higher CIMT \([n=231, \text{Median}=0.72; \text{IQR}=(0.62, 0.81)]\) compared to those in with HDL levels less than 40 \([n=97, \text{Median}=0.71; \text{IQR}=(0.62, 0.81)]\).

To determine if there is a significance difference in the median CIMT (left) between the two groups, a Wilcoxon rank sum test was used. The result from the test showed that the difference in the median of the mean CIMT (left) between the two groups was statistical significant \([Z = 0.86, p - value = 0.039]\)

*Figure 17. Box plot of the mean CIMT (left) by HDL*
4.16 RELATIONSHIP BETWEEN CIMT (LEFT) AND ACR, WHR, VAT, SCAT, WHtR, AND BMI

Figure 18 shows a combined scatter plots of CIMT and albumin-creatinine ratio (first on the first row), VAT (middle on the first row), SCAT (last on the first row), WHR (first on the second row), WHtR (middle on the second row) and BMI (last on the second row). The plots show no relationship between these sets of variables and mean CIMT. This is because no pattern could be discerned from the plots to suggest any form of relationship or association.

Spearman correlation was however used to examine the existence of any relationship, the strength of the linear relationship and determine if the observed relationship was statistically significant. According to the results from the spearman correlation, there is a marginal positive significant relationship between mean CIMT (left) and WH $[\text{rho} = 0.0848, \ p - \text{value} = 0.0581]$. 
Figure 18. Combined scatter plots of CIMT (left) and ACR, WH ratio, VAT, SCAT, WHt ratio, BMI
4.17 ASSOCIATION BETWEEN BLOOD PRESSURE AND BMI, WHR, WC AND WHtR

The median BMI across blood pressure levels were similar with some few outliers for prehypertension, stage 1 and stage 2. Similar pattern was observed for WHtR and blood pressure. The median WHtR and WC was higher among participants with normal blood pressure levels compared to the other groups.

Kruskal-Wallis tests was then performed to determine if the observed distribution of these four anthropometric measures across the blood pressure levels were statistically significant.

According to the results of the test shows that the median BMI among participants with normal blood pressure [n=4, median=21.73; IQR= (20.64, 24.73)], prehypertension [n=109, median= 20.81; IQR= (19.00, 23.71)], stage 1 [n= 233, median= 20.90; IQR= (19.26, 23.03)] and stage 2 [n= 152, median= 21.56; IQR= (19.67, 23.93)] were not significant \([X^2(3) = 3.03, p - value = 0.387]\) as presented in Figure 19.

According to the results from the Kruskal-Wallis test \([X^2(3) = 3.87, p - value = 0.276], \ [X^2(3) = 4.01, p - value = 0.261] \) and \([X^2(3) = 3.11, p - value = 0.375]\), no significant association was also observed for WH ratio, WHt ratio and waist circumference and BP respectively.
Figure 19. Combined boxplots of blood pressure levels and WHtR, BMI, WHR, WC
ASSOCIATION BETWEEN VAT AND ANTHROPOMETRIC INDICES (BMI, WHR, WC AND WHtR)

The plot for visceral fat and WH ratio shows no apparent relationship between the two variables. The plots for the other three anthropometric measures (BMI, WHtR and WC) suggest a positive relationship between the visceral fats and BMI, WHtR and WC as presented in Figure 20.

Spearman correlation was used to examine the strength of the linear relationship and determine if the observed relationship is statistically significant. According to the results from the spearman correlation, there is a significant positive relationship between VAT and BMI \([\rho = 0.261, p \text{-value} < 0.001]\). Contrary to the no apparent pattern in the scatter plots for WHR and VAT, the result from the spearman correlation shows a significant relationship between the two variables \([\rho = 0.248, p \text{-value} < 0.001]\). Again the results from the spearman correlation showed that WHtR \([\rho = 0.177, p \text{-value} < 0.001]\) and waist circumference \([\rho = 0.301, p \text{-value} < 0.001]\) had a significant positive relationship with visceral fat.
Figure 20. Combined boxplots of visceral fat and WHtR, BMI, WHR, WC measurements
4.19 ASSOCIATION BETWEEN SCAT AND ANTHROPOMETRIC INDICES

Figure 21 shows similar relationship between subcutaneous fat and the anthropometric measures. The plots for subcutaneous fats and WH ratio show no apparent relationship between the two variables. The plots for the other three anthropometric measures (BMI, WHt ratio and waist circumference) suggest a positive relationship between the subcutaneous fats and BMI, WHt ratio and waist circumference.

Again, spearman correlation was used to examine the relationship between subcutaneous adipose tissue and the anthropometric measures. According to the results from the spearman correlation, there is a significant positive relationship between subcutaneous fat and all the four anthropometric measures: BMI \( [\rho = 0.632, p - value < 0.001] \), WH ratio \( [\rho = 0.176, p - value < 0.001] \), WHt ratio \( [\rho = 0.616, p - value < 0.001] \) and waist circumference \( [\rho = 0.602, p - value < 0.001] \).
Figure 21. Combined boxplots of subcutaneous fat and the four anthropometric
CHAPTER FIVE

DISCUSSION

5.1 ASSOCIATION OF CIMT WITH ANTHROPOMETRIC INDICES OF BODY ADIPOSY

In this study, a differential strength of association between the various anthropometric indices of body adiposity with CIMT, an extensively validated surrogate marker of subclinical atherosclerosis (Ren et al., 2014) was observed. The correlation with CIMT was strongest for WHR for both right and left CIMT and no association with BMI at all. Of these three simple bedside measures of obesity commonly used in contemporary clinical practice and epidemiological studies, WHR consistently demonstrated the strongest and most robust continuous independent relationship with CIMT, beyond most known determinants of subclinical atherosclerosis and least confounded by age, and surpassed waist circumference and in turn BMI as the anthropometric parameter most correlated with CIMT. These findings are concordant with recent observations from large epidemiological studies, which showed stronger associations between indices more specific of abdominal adiposity and particularly WHR to the risk of prevalent (Balkau et al., 2007; Yusuf et al., 2005) and incident (Canoy et al., 2007) CV disease.

These findings suggest better discrimination of atherosclerotic burden afforded by WHR, which reflects its superior prognostic value in the context of CV disease associated with obesity.
5.2 ASSOCIATION OF CIMT WITH CARDIOVASCULAR RISK VARIABLES

CV risk variables measured that were significantly associated with both right and left CCIMT included; BP, HDL, WHR and alcohol intake status which included; current problematic consumers, current non-problematic consumers, former consumers and never consumed individuals. Contrarily never-consumed alcohol and normal BP individuals also showed a strong positive association with CIMT which was due to some other risk factors may be, bearing in mind that the entire study population were hypertension individuals. The normal BP participants were individuals who were part of those on anti-hypertension drugs.

The results also indicated that SCAT, VAT and WHtR were not associated with CIMT; this is in contrast to previous study that SCAT and VAT are positively associated with CVD risk (Abate, Garg, Peshock, Stray-Gundersen., 1995; Lear et al., 2007).

The interaction in the analysis between anthropometric measures and VAT and SCAT were significant. The higher correlations between WC, WHR and SCAT, VAT were expected, as WC and WHR are assessments of both abdominal SCAT and VAT (Camhi et al., 2011).
5.3 CONCLUSION

The study results indicate that the following variables are significantly associated with common carotid intima-media thickness: BP, Smoking, Alcohol intake, WHR and WC.

The findings support the use of WHR over waist circumference and BMI both in clinical practice in the evaluation of CV risk and in obesity-related atherosclerosis research. The association between WHR and the extent of subclinical atherosclerosis may indicate the pathogenic link between excessive abdominal adiposity and the development of CV events.

The findings further showed significant association between CIMT and some biomarkers such as LDL and total cholesterol in the case of the CIMT (left).

The study also established the existence of significant association between VAT, SCAT and BMI, WHtR, WH, and WC.

5.4 RECOMMENDATIONS

From the findings in this study it is recommended that:

- In spite of the fact that CIMT measurements of the common carotid artery are more reliable and less difficult to obtain compared to the commonly used segment such as carotid bifurcation or the internal carotid artery, the later should jointly be considered in future studies.

- Further studies on CIMT be carried out on a wider age range to include children and young adults to enhance comparison among the various age groups and sex.
Further work is needed to identify the genetic and environmental determinants of the variables under study.

Comparison should be made between healthy non-hypertension individuals and hypertension individuals in future studies.

A longitudinal study should be conducted in the future to ascertain the changes that will occur in the study variables.

The confounding effects of the study variables on each other should be investigated in further studies.

Data on participant’s physical activities should be included in future studies.

5.5 LIMITATION

The study analysis never included the confounding effects of other variables on the individual variables.


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APPENDIX I

CONSENT FORM

PROJECT TITLE: RELATIONSHIP BETWEEN CAROTID INTIMA MEDIA THICKNESS AND SELECTED ANTHROPOMETRIC VARIABLES IN GHANAIAN HYPERTENSION

Dear Potential Participant,

I am a postgraduate student from the University of Ghana who is working on the above stated project.

The study intends to help us understand and assess the risk of developing cardiovascular related diseases using ultrasound images of abdominal fat deposits, and the common carotid, blood sample, urine sample and some measurements of the body.

I would like to invite you to take part in the study. In order to take part, you need to agree to provide a sample of blood and urine samples, to have various measurements done on your body and to complete a list of questions related to your health and your life.

Your participation is voluntary, and if you wish not to participate there is no penalty or loss of benefits in any way.

Blood Sample

Once the blood sample (two tablespoons or 20ml) is collected, it will be given a code and this code will be used to identify your sample during the study. The information that links your name to your code will be safely stored and will not be used in the study.
Taking a blood sample from a vein in your arm may cause a little discomfort and a little pain. This procedure will be done by a qualified technician or phlebotomist.

Measurements

Your weight, height, waist and hip circumference will be measured. This will take about 5 minutes.

Ultrasound Scans

An ultrasound scan will be used to measure the internal fat in your abdomen. An ultrasound scan uses high frequency sound waves to take pictures of the body. This is not an invasive procedure, it will cause you no pain and is perfectly safe. The procedure should not take more than 10 minutes to complete.

CIMT (Carotid Intima Media Thickness)

This measurement will be done by a trained technician. It is almost the same as the ultrasound procedure above. You will lie on a flat bed and the technician will gently pass a small hand held instrument up and down your neck. This small instrument will measure if the arteries leading to your brain are healthy. This is not an invasive procedure and the actual procedure should not take more than 20 minutes.

Questionnaires

You will be asked to complete a brief questionnaire. You can skip any question that makes you uncomfortable. The investigator will fill in the answers for you.

Urine sample

You will be requested to donate a small urine sample (20ml) as instructed in a clean toilet facility. This will be used to test if your kidneys are working well.
Benefits

The discoveries that come from the studies will not be of direct benefit to you and will not be communicated back to you. The discoveries may lead to information that will help us in the future to diagnose cardiovascular risk factors.

Risks

There is no known risks associated the entire process of the study of the data collection.

Participant Declaration

I am informed that the nature of procedure such as the method of data collection and its subsequent analysis will not expose me to any serious risk or hazard; neither will it cause undue pain to me.

I will not by my participation in this study derive any personal benefits but help in generating data that will be useful to researchers.

Name of participant  Signature/Mark or Thumbprint

...............................  ........................................

Date and Time

.................

Should you require further information or wish to withdraw from the study, you may contact the following:
1. The Principal Supervisor (Prof. F.K Addai). Department of Anatomy, School of Biomedical and Allied Health Sciences, College of Health Sciences, Korle-Bu.

Tel: 0302672020

2. The Ethical and Protocol Review Committee, College of Health Sciences, University of Ghana, Korle-Bu Campus, Accra.

3. Eric Fato (The Research student), Department of Anatomy, School of Biomedical and Allied Health Sciences, College of Health Sciences, Korle-Bu. Tel: 0249586897
APPENDIX II
RESEARCH QUESTIONNAIRE

DEPARTMENT OF HUMAN ANATOMY
SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES,
COLLEGE OF HEALTH SCIENCES, UNIVERSITY OF GHANA

RELATION BETWEEN CAROTID INTIMA MEDIA THICKNESS AND
SOME SELECTED ANTHROPOMETRY MEASUREMENT IN
HYPERTENSION GHANAIANS

<table>
<thead>
<tr>
<th>Participant’s study ID No.</th>
</tr>
</thead>
</table>

1. GENERAL INFORMATION

1.1 Data collection date

| d | d | m | m | y | y | y | y |

2. DEMOGRAPHIC INFORMATION

2.1 Date of birth known? [If no, skip to Q.2.3] □ Yes □ No

2.2 Date of Birth [eg. 27 SEP 1957] | d | d | m | m | y | y | y | y |

106
### 2.3 Approximate year of birth

| 1 | 5 | J | U | N | y | y | y | y |

### 2.4 Gender

- [ ] Female
- [ ] Male

*please tick the appropriate box*

### 2.5 Country

- Ghana

### 3. PREGNANCY

[If male, please skip to the next section – substance use]

#### 3.1 Are you pregnant?

- [ ] Yes
- [ ] No

### 4. SUBSTANCE USE

#### 4.1 Tobacco use

##### 4.1.1 Have you ever smoked any tobacco products such as cigarettes, cigars or pipes? *If no, skip to Q.4.2*

- [ ] Yes
- [ ] No

##### 4.1.2 Do you *currently* smoke any tobacco products, such as cigarettes, cigars or pipes? *asking if they smoke when they have the opportunity to do so. If no, skip to Q.4.1.6*

- [ ] Yes
- [ ] No

##### 4.1.3 What do you smoke? *please tick the appropriate boxes*

- [ ] Cigarettes
- [ ] Pipe
- [ ] Hand rolled
- [ ] Cigars
- [ ] E-cigarettes

##### 4.1.4 How often do you smoke tobacco products?

- [ ] Daily
- [ ] 5-6 days per week
- [ ] 1-4 days per week
- [ ] 1-3 days per month
- [ ] Less than once per month

##### 4.1.6 How old were you when you first started smoking?

|___|___|___|___|___|___| years old

##### 4.1.7 When did you stop smoking completely?

| y | y | y | y |

---

University of Ghana  http://ugspace.ug.edu.gh
### 4.2. Alcohol use

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
</table>
| 4.2.1 Have you ever consumed an alcoholic drink such as beer, wine, spirits, fermented cider, thothotho, or traditional beer? [If “no” or “don’t know”, skip to Q.5] | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
| 4.2.2 Do you currently (in the last 30 days) consume any alcoholic drink such as beer, wine, spirits, fermented cider, pito, or traditional beer? [If no, skip to Q.4.2.10] | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
| 4.2.3 How often do you have at least one alcoholic drink?                | □ Daily  
□ 5-6 days per week  
□ 1-4 days per week  
□ 1-3 days per month  
□ Less than once per month                                                                 |
| 4.2.4 On the days that you drink alcoholic drinks, how many alcoholic drinks do you have? | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
| 4.2.5 Have you ever felt that you should cut down on your drinking?     | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
| 4.2.6 Have people annoyed you by criticising your drinking?             | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
| 4.2.7 Have you ever felt bad or guilty about your drinking?             | □ Yes  
□ No  
□ Don’t know  
□ Refuse to answer                                                                 |
### 4.2.8
What type of alcoholic beverage do you, or did you, usually drink?

- [ ] Beer
- [ ] Wine
- [ ] Spirits
- [ ] Home brew
- [ ] Other (specify)

### 5. GENERAL HEALTH

#### 5.1. Please indicate if your mother has, or has had, any of the following illnesses

*Please tick the appropriate boxes*

<table>
<thead>
<tr>
<th>5.1.1</th>
<th>Weight problem/obesity</th>
<th>Yes</th>
<th>No</th>
<th>Don’t know</th>
<th>Refuse to answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.1.2</td>
<td>High blood pressure</td>
<td>Yes</td>
<td>No</td>
<td>Don’t know</td>
<td>Refuse to answer</td>
</tr>
<tr>
<td>5.1.3</td>
<td>High cholesterol</td>
<td>Yes</td>
<td>No</td>
<td>Don’t know</td>
<td>Refuse to answer</td>
</tr>
</tbody>
</table>

#### 5.2. Please indicate if your father has, or has had any of the following illnesses

*Please tick the appropriate boxes*

<table>
<thead>
<tr>
<th>5.2.1</th>
<th>Weight problem/obesity</th>
<th>Yes</th>
<th>No</th>
<th>Don’t know</th>
<th>Refuse to answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.2.2</td>
<td>High blood pressure</td>
<td>Yes</td>
<td>No</td>
<td>Don’t know</td>
<td>Refuse to answer</td>
</tr>
<tr>
<td>5.2.3</td>
<td>High cholesterol</td>
<td>Yes</td>
<td>No</td>
<td>Don’t know</td>
<td>Refuse to answer</td>
</tr>
</tbody>
</table>
# 6. CARDIOVASCULAR RISK FACTORS

## 6.1. Diabetes

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes</th>
<th>No</th>
<th>Don’t know</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.1.1 Has a doctor, nurse, or other healthcare worker ever measured your blood or urine for diabetes (sugar in the blood)?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.1.2 Have you ever been told by a doctor or healthcare worker, that you have diabetes or high blood sugar (outside of pregnancy)?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>[The brackets apply only to females] [If “no” or “don’t know”, skip to Q. 6.1.7]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.1.3 Have you ever received treatment for diabetes prescribed by a doctor, nurse, or other healthcare worker?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.1.4 Are you currently receiving treatment for diabetes prescribed by a doctor, nurse, or other healthcare worker?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.1.7 Do you have a family history of diabetes? [If “no” or “don’t know”, skip to Q. 6.2]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 6.2. Stroke*

<table>
<thead>
<tr>
<th>6.2.1</th>
<th>Have you ever been told by a doctor, nurse, or other healthcare worker that you have had a stroke?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[If “no” or “don’t know”, skip to Q. 6.3]</td>
</tr>
<tr>
<td></td>
<td>□ Yes  □ No  □ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.2.2</th>
<th>When was it first diagnosed?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[Please indicate in which year your stroke was diagnosed]</td>
</tr>
<tr>
<td></td>
<td>□ y □ y □ y □ y</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.2.3</th>
<th>Have you ever been told by a doctor, nurse, or other healthcare worker that you have had a ministroke, or transient ischemic attack (TIA)?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ Yes  □ No  □ Don’t know</td>
</tr>
</tbody>
</table>

### 6.3. Hypertension

<table>
<thead>
<tr>
<th>6.3.1</th>
<th>Has a doctor, nurse, or other healthcare worker ever measured your blood pressure?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ Yes  □ No  □ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.3.2</th>
<th>Have you ever been told by a doctor, nurse, or other healthcare worker that you have hypertension (high blood pressure)?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[If “no” or “don’t know”, skip to Q. 6.4]</td>
</tr>
<tr>
<td></td>
<td>□ Yes  □ No  □ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.3.3</th>
<th>Have you ever received treatment for hypertension prescribed by a doctor, nurse or other healthcare worker?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□ Yes  □ No  □ Don’t know</td>
</tr>
</tbody>
</table>
### 6.3.5
Are you currently on treatment for hypertension prescribed by a doctor, nurse or other healthcare worker?

- [ ] Yes
- [ ] No
- [ ] Don’t know

### 6.3.6
What medicine are you taking for this? Please list if possible.

_________________________________

### 6.4. Angina*

*Angina is characterised as an intense chest pain or discomfort. This pain may occur in the shoulders, arms, neck, back and jaw as well. It may also feel like indigestion. It is considered to be the main symptom of Coronary Heart Disease (CHD).*

### 6.4.1
Have you ever been told by a doctor, nurse, or other healthcare worker that you have angina (chest pain due to heart disease)?

[If “no” or “don’t know”, skip to Q. 6.5]

- [ ] Yes
- [ ] No
- [ ] Don’t know

### 6.4.2
Have you ever received treatment for chest pain due to heart disease prescribed by a doctor, nurse or other healthcare worker?

- [ ] Yes
- [ ] No
- [ ] Don’t know

### 6.4.3
Are you currently taking any medication for angina prescribed by a doctor or other healthcare worker for this?

- [ ] Yes
- [ ] No
- [ ] Don’t know
<table>
<thead>
<tr>
<th>6.4.4</th>
<th>What medicine are you taking for this? Please list if possible.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.5. Heart Attack</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.5.1 Have you ever been told by a doctor, nurse, or other healthcare worker that you have had a heart attack?</td>
</tr>
<tr>
<td>[If “no” or “don’t know”, skip to Q. 6.6]</td>
</tr>
<tr>
<td>□ Yes</td>
</tr>
<tr>
<td>□ No</td>
</tr>
<tr>
<td>□ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.5.2 Did you ever receive medical treatment for your heart attack?</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Yes</td>
</tr>
<tr>
<td>□ No</td>
</tr>
<tr>
<td>□ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.6. Congestive Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.6.1 Have you ever been told by a doctor, nurse, or other healthcare worker that you have had heart failure?</td>
</tr>
<tr>
<td>[If “no” or “don’t know”, skip to Q. 6.7]</td>
</tr>
<tr>
<td>□ Yes</td>
</tr>
<tr>
<td>□ No</td>
</tr>
<tr>
<td>□ Don’t know</td>
</tr>
</tbody>
</table>

| 6.6.2 Have you ever received medical treatment for heart failure prescribed by a doctor, nurse, or other healthcare worker? |
| □ Yes |
| □ No |
| □ Don’t know |

| 6.6.3 Are you currently on treatment for heart failure prescribed by a doctor, nurse, or other healthcare worker? |
| □ Yes |
| □ No |
| □ Don’t know |
### 6.7. High Cholesterol

<table>
<thead>
<tr>
<th>6.7.1</th>
<th>Has a doctor, nurse or other healthcare worker ever measured your cholesterol?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>☐ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.7.2</th>
<th>Have you ever been told by your doctor or other healthcare worker that you have high cholesterol?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>☐ Don’t know</td>
</tr>
</tbody>
</table>

[If “no” or “don’t know”, skip to Q. 6.7.5]

<table>
<thead>
<tr>
<th>6.7.3</th>
<th>Have you ever been treated for high cholesterol by a doctor, nurse, or other healthcare worker?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>☐ Don’t know</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.7.4</th>
<th>Are you currently using any of the following to treat your high cholesterol, as prescribed by a doctor, nurse, or other healthcare worker?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Special diet</td>
</tr>
<tr>
<td></td>
<td>☐ Medicine</td>
</tr>
<tr>
<td></td>
<td>[Please tick the appropriate boxes, more than one may be selected]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6.7.5</th>
<th>Are you currently taking any herbal or traditional remedy for high cholesterol?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>☐ Don’t know</td>
</tr>
</tbody>
</table>

### 6.8 KIDNEY DISEASE

<table>
<thead>
<tr>
<th>6.8.1</th>
<th>Has a doctor ever told you that you have kidney disease?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>☐ Don’t know</td>
</tr>
</tbody>
</table>

[If no, skip to Q. 6.8.3]

<table>
<thead>
<tr>
<th>6.8.2</th>
<th>Do you know what type of kidney disease? If yes please specify.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>☐ Yes</td>
</tr>
<tr>
<td></td>
<td>Specify</td>
</tr>
</tbody>
</table>
6.8.3  Has a doctor ever told you that your kidneys have low function?

- Yes
- No
- Don’t know

6.8.4  Has anyone in your family either had kidney disease, or died from it?

[If no, please skip to next section.]

- Yes
- No
- Don’t know

14.5  If yes, who?

[“Other” refers to any other blood relatives eg. Sister, aunt etc]

- Mother
- Father
- Other (specify)

14.6  Do you know what kind of kidney disease he or she had? If yes, please specify.

- Yes  
- No

Specify ______________

**SAMPLE COLLECTION DATA**

**1. ANTHROPOMETRIC MEASUREMENTS**

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Standing height</td>
<td></td>
<td></td>
<td>m</td>
</tr>
<tr>
<td>1.2</td>
<td>Weight</td>
<td></td>
<td></td>
<td>kg</td>
</tr>
<tr>
<td>1.3</td>
<td>Waist circumference</td>
<td></td>
<td></td>
<td>m</td>
</tr>
<tr>
<td>1.4</td>
<td>Hip circumference</td>
<td></td>
<td></td>
<td>m</td>
</tr>
</tbody>
</table>
## 2. BLOOD PRESSURE

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th>mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>Systolic 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.2</td>
<td>Systolic 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.3</td>
<td>Systolic 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.4</td>
<td>Diastolic 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.5</td>
<td>Diastolic 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.6</td>
<td>Diastolic 3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Time blood pressure taken in the first instance

**[Based on a 24 hour clock eg.15:30]**

| h | h | m | m |

## 3. PULSE

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th>beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1</td>
<td>Pulse 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.2</td>
<td>Pulse 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.3</td>
<td>Pulse 3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## 4. ULTRASOUND MEASUREMENTS

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1</td>
<td>Ultrasound</td>
<td>Yes</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>4.2</td>
<td>Visceral (medial) fat</td>
<td></td>
<td></td>
<td>cm</td>
</tr>
<tr>
<td>4.3</td>
<td>Subcutaneous (transverse) fat</td>
<td></td>
<td></td>
<td>cm</td>
</tr>
<tr>
<td>4.4</td>
<td>Date ultrasound taken</td>
<td>[eg. 27 SEP 1957]</td>
<td>d</td>
<td>d</td>
</tr>
<tr>
<td>4.5</td>
<td>CIMT</td>
<td>Yes</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>4.6</td>
<td>Minimum CIMT on the right</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
<tr>
<td>4.7</td>
<td>Maximum CIMT on the right</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
<tr>
<td>4.8</td>
<td>Mean CIMT on the right</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
<tr>
<td>4.9</td>
<td>Minimum CIMT on the left</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
<tr>
<td>4.10</td>
<td>Maximum CIMT on the left</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
<tr>
<td>4.11</td>
<td>Mean CIMT on the left</td>
<td></td>
<td></td>
<td>mm</td>
</tr>
</tbody>
</table>

University of Ghana  http://ugspace.ug.edu.gh
1. BLOOD COLLECTION

1.1 Fasting blood? [If YES, skip to Q1.3] □ Yes □ No

1.2 At what time did you last eat? [Based on a 24 hour clock eg. 15:30] | h | h |:| m | m |

1.4. Please complete the following table

<table>
<thead>
<tr>
<th>5ml Tubes</th>
<th>Number</th>
<th>Reason</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red</td>
<td>2</td>
<td>Yes</td>
<td>lipid</td>
</tr>
<tr>
<td>Grey</td>
<td>1</td>
<td>Yes</td>
<td>glucose</td>
</tr>
</tbody>
</table>

1.5 Phlebotomist Name _________________________________

1.6 Date blood taken [eg. 27 SEP 1957] | d | d | | m | m | m | | y | y | y |

1.7 Time blood taken | h | h |:| m | m |

2. URINE COLLECTION

2.1 Urine sample taken? □ Yes □ No
<table>
<thead>
<tr>
<th></th>
<th>CHECKLIST</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Informed consent</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>2</td>
<td>Questionnaire</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>3</td>
<td>Anthropometric measurements</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>4</td>
<td>Blood pressure</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>5</td>
<td>Pulse</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>6</td>
<td>Blood samples</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>7</td>
<td>Ultrasound</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>8</td>
<td>CIMT</td>
<td>□ Yes □ No</td>
</tr>
<tr>
<td>9</td>
<td>Urine sample</td>
<td>□ Yes □ No</td>
</tr>
</tbody>
</table>
APPENDIX III

1.0 STANDARD OPERATING PROCEDURE FOR TAKING
ANTHROPOMETRIC MEASUREMENTS

1.1 STANDING HEIGHT

1.1.1. Participants should either be barefoot or wearing thin socks

1.1.2. Where possible; heels, buttock, shoulders and the back of the head should be touching the wall or the vertical board of the stadiometer. If this isn’t possible ensure that a straight posture is maintained with at least the buttocks and heels touching the vertical board

1.1.3. Heels must be together with the medial borders of the feet forming a maximum angle of 60

1.1.4. Shoulders should be relaxed but straightened, and arms may hang freely alongside the body with palms facing inwards

1.1.5. The head must be positioned in the Frankfurt Horizontal Plane and the participant should be asked to take a deep breath and maintain an erect posture

1.1.6. Bring the headboard onto the vertex of the head (sufficient pressure must be applied to compress the hair). Take care not to push the subject down, lest you lower his/her height

1.1.7. Slight upward pressure may be applied on the mastoid processes to achieve the maximum level to which the head should be raised

1.1.8. Record standing height in mm as it shows on the Harpenden digital stadiometer
1.2 WEIGHT

1.2.1. Participants must remove shoes, heavy clothing, jewelry and empty their pockets prior to being weighed.

1.2.2. Participants should be standing still in the center of the platform, with a 10cm gap between their heels, and arms hanging loosely at their sides.

1.2.3. Weight is to be recorded in kg with one decimal place.

1.2 WAIST CIRCUMFERENCE

1.3.1. Clothing should be removed in order to position the tape correctly.

1.3.2. The participant should be standing up straight with arms slightly abducted, and breathing normally.

1.3.3. Squat beside the participant and place the tape horizontally around the narrowest part of the torso, about halfway between the iliac crest and the lowest rib.

1.3.4. Measurement should be taken at the end of a normal expiration without compression of the tape.

1.3.5. Waist circumference is recorded in mm.

1.4. HIP CIRCUMFERENCE

1.4.1. Participants should be wearing light, or tight clothing.

1.4.2. Squat beside the subject and place the tape around the most protruding part of the buttocks.

1.4.3. Ensure that the zero mark is always on the participant’s side.

1.4.4. Hip circumference should be recorded in mm.
APPENDIX IV

STANDARD OPERATING PROCEDURE FOR TAKING BLOOD PRESSURE

1.0 BLOOD PRESSURE AND PULSE

Systolic blood pressure (SBP) represents the pressure while the heart contracts to pump blood to the body, and is the higher of the two blood pressure values. Diastolic blood pressure (DBP) represents the pressure when the heart relaxes between beats, and is the lower value.

1.1. Please note that smoking, heavy exercise and alcohol use before assessment could affect the accuracy of measurement. Participants should be notified to abstain at the time of their appointment

1.2. The participant should be seated with their back supported and feet firmly rested on the floor. Please ensure that feet are not crossed and check that the position of the participant remains unchanged during the measurement

1.3. The measurement must be taken from the left arm. The left arm should be resting on either the desk or arm rest; with the antecupital fossa level with the heart, and palm facing upwards. If necessary, use a pillow to elevate the arm

1.4. Place the cuff on the left arm about 2-3cm above the antecupital fossa. Ensure that the top edge of the cuff is not restricted by clothing, and that one finger can fit between the cuff and the arm

1.5. To measure blood pressure follow the instructions on the Omron BP monitor

1.6. Record the SBP, DBP, pulse and time the first reading was taken on the Sample Collection Data sheet

1.7. Repeat measurements twice more, with 2 minutes between each measurement
APPENDIX IV

STANDARD OPERATING PROCEDURE FOR TAKING ULTRASOUND VAT, SCAT AND CIMT MEASUREMENTS

1.1 VISCERAL (MEDIAL) FAT MEASUREMENT

1.1.1. Switch the machine on and select ‘Patient’ (button with 2 human icons) to access the Patient Data menu

1.1.2. Use the trackball to move the cursor to select ‘New Patient’ and enter the five-digit number found on the barcode sticker on the top of the Sample Data Collection sheet (make sure that it is entered correctly)

1.1.3. Select ‘Register’ and then ‘Exit’

1.1.4. You are now ready to take the medial fat measurement. Ask the participant to lie down on the bed.

1.1.5. Set the ‘Depth’ at 15cm, press ‘Comment’ and select ‘MEDIAL’ on the side menu that pops up

1.1.6. Apply gel to the participant’s lower abdomen and position the probe in the midline at a level midway between the lower costal margin and the iliac crest. Use the xiphi-sternum and umbilicus as a guide to accurate positioning.

1.1.7. The spine should be positioned horizontally, and the vertebra centrally in the image. Adjust gain accordingly.

1.1.8. Ask the participant to breath quietly and press ‘Freeze’ at the end of expiration and ‘Calc’
1.1.9. Place the first cursor anterior to the spine (on the fat pad if visible), and the second cursor on the interior of the anterior abdominal muscles (only the peritoneum). Please ensure that this measurement is perpendicular to the surface of the lumbar vertebra.

1.1.10. This measurement is taken between the peritoneum and the spine, when there is a clear space between the vertebra and the aorta.

1.1.11. Press ‘P1’ to save the image. Unfreeze and obtain a second image and repeat the measurements as described above from 5.1.7.

1.1.12. Record readings in the appropriate slots on the Sample Data Collection sheet in cm (2 decimal places).

1.2 SUBCUTANEOUS (TRANSVERSE) FAT MEASUREMENT

1.2.1. Transverse fat measurement is to be performed directly after the medial measurement is taken to ensure that the image is saved under the same unique identifier assigned to the participant.

1.2.2. Rotate the probe 90 degrees and adjust the ‘Depth’ to 9cm.

1.2.3. Press ‘Comment’ and then ‘Clear’ to remove the annotate ‘Medial’ on the monitor.

1.2.4. Use the cursor to select ‘Sub-Cut’ on the side menu.

1.2.5. Visualize both rectus abdominus muscles on either side of the monitor. Please ensure that both muscles are symmetrical and that the linea alba is centrally located.

1.2.6. Press ‘Measure’
1.2.7. Measure the distance between the skin and the outer edge of the *linea alba*.

1.2.8. Press ‘P1’ to save the image. Unfreeze and obtain a second image and measurement, as described above.

1.2.9. Record readings in the appropriate slots on the *Sample Data Collection* sheet in cm (2 decimal places).

### 1.3 CIMT MEASUREMENT

1.3.1. Press ‘Patient’ on the control panel.

1.3.2. Enter participants ID and date of birth using the keyboard.

1.3.3. Press ‘Register’ on the left hand side of the screen.

1.3.4. Press ‘Exit’ and then the ‘Preset’ button on the bottom left of the keyboard.

1.3.5. Select ‘Probe/Transducer’.

1.3.6. Select ‘Vas’ (vascular) and wait for a new platform. Then select ‘Carotid’. The correct settings have now been applied to begin scanning.

1.3.7. Ask the participant to please lie down.

1.3.8. The right carotid is scanned first. Gently turn the participant’s head towards their left, and apply gel to the neck area exposed.

1.3.9. Start scanning in a *longitudinal* plane and find the *common carotid artery* (CCA).

1.3.10. The image above is representative of what should be seen. ‘Freeze’ the image and then press the ‘Calcs’ button which is the top button around the trackball.
1.3.11. A calculation menu will pop up on the left hand side of the screen. Using the cursor, select ‘R’ for the right carotid, followed by ‘CCA IMT’

1.3.12. The menu will update. Select ‘POS IMT’

1.3.13. Using the trackball, move the cursor (1st point) to the top line of the posterior intima. Press ‘ENTER’, then move the cursor along the anterior aspect of the intima to 2nd point (about 2cm) then press ‘ENTER’.

1.3.14. Readings for min, max and average distance will be given in mm (2 decimal places)

1.3.15. Record readings in the appropriate slots on the Sample Data Collection sheet

1.3.16. Repeat the entire process for the left carotid
APPENDIX VI

STANDARD OPERATING PROCEDURES FOR BLOOD SAMPLES

Red, and grey capped tubes will be collected. This protocol applies only to red and grey capped tubes

1.0 RED CAPPED TUBES (No anticoagulant) are for lipid profile assessment

1.1 Two 6ml red capped tubes are to be collected from each participant.

1.2 After collection, tubes should be left to clot at room temperature for 10-15 minutes at 3000rpm.

1.3 After clotting, tubes should be spun at room temperature and the supernatant i.e. serum should be removed from each tube, as described below.

1.4 From each red capped tube a 500µl aliquot should be added to a cryovial for storage/transport (see point 6 and 7). These samples are for lipid profile assessment. The remainder of the supernatants from each tube should be divided equally into 3 aliquots in tubes (cryovials) that can be used for freezing (-80°C).

1.5 If samples are to be transported immediately for lipid measurements, these aliquots can be stored for up to 5 days in a fridge and should be transported in a polystyrene container filled with ice or any other container which can maintain a temperature of 4°C.

1.6 If lipid aliquots need to be stored for longer period than 5 days, they should be put in cryovials that can withstand freezing and stored in a -80°C freezer.

1.7 If blood samples are collected in the field at sites remote from a laboratory, the red capped tubes should be kept at 4°C; either in a polystyrene container filled
with ice or ice blocks*. Do not let tubes touch the blocks as they may freeze. Wrap them in tissue to avoid this. They must be spun within 4 hours of collection, and treated as described above.

2.0 GREY CAPPED TUBES (Potassium oxalate as anticoagulant) are only for glucose measurements

2.1 After collection, tubes should be left to clot at room temperature for 10-15 minutes.

2.2 After clotting, tubes should be spun at room temperature and the supernatant i.e. plasma (grey cap) should be removed from each tube, as described below.

2.3 From each grey capped tube a 500µl aliquot should be added to a cryovial for storage/transport (see point 6 and 7). These samples are for glucose measurement. The remainder of the supernatants from each tube should be divided equally into 3 aliquots in tubes (cryovials) that can be used for freezing (-80°C).

2.4 If samples are to be transported immediately for glucose measurements, these aliquots can be stored for up to 5 days in a fridge and should be transported in a polystyrene container filled with ice or any other container which can maintain a temperature of 4°C.

2.5 If glucose aliquots need to be stored for longer period than 5 days, they should be put in cryovials that can withstand freezing and stored in a -80°C freezer.

2.6 If blood samples are collected in the field at sites remote from a laboratory, the grey capped tubes should be kept at 4°C either in a polystyrene container filled with ice or ice blocks*. Do not let tubes touch the blocks as they may freeze.
Wrap them in tissue to avoid this. They must be spun within 4 hours of collection, and treated as described above.

Table 5. Blood sample and appropriate vials

<table>
<thead>
<tr>
<th>Tube</th>
<th>Tests</th>
<th>Substance stored</th>
<th>Storage Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red</td>
<td>Total cholesterol</td>
<td>serum</td>
<td>4°C temporary</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td></td>
<td>- 80°C long term</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grey</td>
<td>Blood glucose</td>
<td>plasma</td>
<td>4°C temporary</td>
</tr>
<tr>
<td></td>
<td>Glucose tolerance</td>
<td></td>
<td>- 80°C long term</td>
</tr>
</tbody>
</table>