The spectrum and the effects of cardiovascular risk factors on the cardiac structure and function at Madadeni Provincial Hospital (Internal Medicine Department)

By

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DECLARATION

I, Nokwanda A. Gambushe, hereby declare that this thesis is my own original work and that it has not been previously submitted in any form for another degree at any university or higher learning institution. All information used from published or unpublished work of others has been acknowledged in the text.

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DEDICATION

I would like to dedicate this thesis to:

1. My heavenly father; I am grateful to have been blessed with an opportunity to make something of my life. Thank you Lord for never leaving my side and providing me with everything I needed to complete this chapter of my life.

2. To my grandmother and siblings, your constant queries as to when will I be completing my research was annoying yet very motivating and encouraging. Thank you for your constant love, words of wisdom and encouragement throughout my studies.

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ABSTRACT

Background: Cardiovascular disease (CVD) is now a leading cause of death worldwide. According to the United Nations (UN), people who are above 60 years of age have a greater chance of developing CVD, and are projected to double in South Africa by 2050. There is clear evidence that patients with cardiovascular risk factors present with cardiac structural and functional abnormalities. Left ventricular remodelling is a major complication in patients with cardiovascular risk factors, particularly hypertension. Very few studies evaluating the structural and functional changes in response to CVD, and none, to the knowledge of the researcher, have been conducted on black Africans in South Africa in urban and rural areas

Aim: The aim of the study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department).

Materials and method: The researcher systematically sampled 200 participants in the northern KwaZulu-Natal (KZN) region, presenting to Madadeni Provincial Hospital (Internal Medicine Department) and administered a questionnaire that collected their information on socio-demographic and cardiovascular risk factors. Other measurements included blood pressure, blood glucose, biochemical analysis and transthoracic echocardiography. The statistical analysis included descriptive statistics (frequency tables, bar and pie charts) and chi-squared and T tests to present the study findings and comparison between variables. A multinomial logistic regression analysis was performed to determine independent predictors for left ventricular geometry.

Results: The mean age was 50.10 ± 16.188 (range: 18 – 79), with 114 (57.0%) females and 86 (43.0%) males. Black patients had the highest prevalence of cardiovascular risk factors (56, 87.0%), followed by Indians (27, 13.5%), whites (12, 6.0%) and mixed ancestry (5.4.0%).

There was a high prevalence of modifiable cardiovascular risk factors with hypertension (HPT) being the leading factor (31.5%), followed by diabetes mellitus

(17.0%). The prevalence of hypertension together with diabetes mellitus was 24.5%. Dyslipedemia was the lowest with 2.0%.

The prevalence of left ventricular systolic function was normal in 174 (87.0%) participants and abnormal in 26 (13.0%). The prevalence of left ventricular diastolic dysfunction was 70.5% in women and 29.4% in men. There was a high prevalence of normal left ventricular geometry with 103 (51.5%), followed by concentric hypertrophy 72 (36.0%), eccentric remodelling 13 (6.5%), concentric remodelling 11 (5.5%), and eccentric hypertrophy with 0.5%. Metabolic syndrome was documented in 58.3% blacks, followed by 33.3% Indians and 8.3% whites. A multinomial logistic regression model was fitted with the dependent variable, which was left ventricle geometry. The independent variables were age, gender, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), HPT, diabetes mellitus and diastolic function. When comparing risk factors for concentric hypertrophy to the category 'normal', the significant variable was diastolic function (*p*-value = 0.015). The risk factors for concentric remodelling was found to be SBP (*p* = 0.057) and BMI category morbid obese (*p* = 0.015). The risk factors for eccentric remodelling were found to be BMI category morbid obese (*p* = 0.002) and HPT (*p* = 0.016).

Conclusion: The study has shown that there is a high prevalence of modifiable cardiovascular risk factors in the northern KZN region and many patients present with metabolic syndrome during the course of the disease. The study also revealed a high prevalence of left ventricular diastolic dysfunction and left ventricular geometric patterns in the studied population. The risk factors for left ventricular geometry were HPT, morbid obesity, diastolic function and SBP.

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LIST OF ABBREVIATIONS

2D:	Two-Dimensional
ARIC:	Atherosclerosis risk in communities
ARIRANG:	Atherosclerosis Risk of Rural Areas in the Korean General population
ASE:	American Society of Echocardiography
BMI:	Body Mass Index
CARDIA:	Coronary Artery Risk Development in young Adults
Cm:	Centimetre
CVD:	Cardiovascular Disease
CW:	Continuous Wave
DBP:	Diastolic Blood Pressure
DM:	Diabetes Mellitus
DUT:	Durban University of Technology
DT:	Deceleration Time
E/A ratio:	Ratio of mitral peak velocity of early and late diastolic filling
HCHS/SOL:	Hispanic Community Health Study/Study of Latinos
HDL:	High-Density Lipoprotein
HIV:	Human Immunodeficiency Virus
HPT:	Hypertension
IREC:	Institutional Research Ethics Committee
Kg:	Kilogram
Kg/m^2:	Kilogram per metre square

KZN:	KwaZulu-Natal
LA:	Left atrium
LDL:	Low Density Lipoprotein
LVEDD:	Left Ventricular End Diastolic Dimension
LVEF:	Left Ventricular Ejection Fraction
MASHAD:	Mashhad Stroke and Heart Atherosclerotic Disorder
MHSc:	Master of Health Sciences
ms:	metre second
MmHg:	Millimetre Mercury
Mmol/L:	Millimoles per litre
NHANES:	National Health And Nutrition Examination Survey
NIAAA:	National Institute of Alcohol Abuse and Alcoholism
PW:	Pulsed Wave
PWT:	Posterior Wall Thickness
RAAS:	Renin-Angiotensin-Aldosterone System
RWT:	Relative Wall Thickness
SA:	South Africa
SABRE:	Southall And Brent Revisited
SAHA:	South African Heart Association
SBP:	Systolic Blood Pressure
SSA:	Sub-Saharan Africa
SPSS:	Statistical Package for the Social Sciences

TC:	Total Cholesterol
THUSA:	Transition and Health during Urbanisation in South Africa
TTE:	Transthoracic Echocardiography
UN:	United Nations
USA:	United States of America
vs.	Versus
WHO:	World Health Organization
WHTR:	Waist-To-Height Ratio

LIST OF SYMBOLS

%:	Percentage
/:	Or
<:	Less than
>:	Greater than
≤:	Lesser than or equal to
≥:	Greater than or equal to
+:	Plus
-:	Minus
×:	Times by
=:	Equal to
(:	Opening bracket
):	Closing bracket
↑:	Increased
↓:	Decreased

CHAPTER 1: INTRODUCTION

1.1 A GLOBAL OVERVIEW BURDEN OF CARDIOVASCULAR DISEASE

Cardiovascular disease (CVD) is a general term used to describe a range of disorders that affect the heart and blood vessels including coronary artery disease, cerebrovascular disease, rheumatic heart disease and other conditions (WHO 2017a). According to Joseph (2013), cardiovascular disease is now the most common cause of death worldwide. Most of these deaths occurred in low and middle-income countries, with sub-Saharan Africa (SSA) being the leading contributor to the global burden of CVD (Keates *et al*. 2017). Gersh *et al*. (2010) reveal that the magnitude of the high prevalence of CVD in developing countries has detrimental consequences for their inhabitants. According to the report from the global burden of disease study, CVD accounted for 43% of the global mortality rate in 2010 (Oduniaya 2016).

Maredza, Hofman and Tollman (2011) report that, according to the United Nations (UN), people who are above 60 years of age, have a greater chance of developing CVD, and that between 1999 and 2050, the risk will double in South Africa. Mensah and Brown (2007) state that in the United States of America (USA), CVD was responsible for 24 million (34.4%) deaths in 2003. Abshire (2014) states that in 2010 the death rate in the USA was 235.5 per 100.000, and that 84 million American adults at the time of writing were living with cardiovascular disease.

Khine and Marais (2016) indicate that according to the American Heart Association, the CVD prevalence was predicted to be 38.7% for USA in 2020. A study conducted by Leeder *et al*. (2004) in five lower income and middle-income countries found that Russia had the highest rate of CVD while South Africa had the lowest. Brazil, India and China were at the highest risk of facing a CVD epidemic.

1.2 CARDIOVASCULAR DISEASE BURDEN IN SOUTH AFRICA

South Africa is in a health transition phase that is characterised by a high burden of non-communicable diseases such as CVD (Mayosi *et al.* 2016). According to Sliwa *et al.* (2012), historically South Africa had low levels of cardiovascular disease; however, this seems to be changing as the population is undergoing epidemiological transition. CVD is now the leading cause of death in South Africa after HIV/AIDS (Sliwa *et al.* 2012).

According to the World Health Organization (WHO 2002), CVD was responsible for 9.2% of total deaths in the African region in 2001. The exact South African prevalence remains unknown, although Byrne, Eksteen and Crickmore (2016) suggest that CVD is responsible for 17.3% of deaths in South Africa. Approximately 195 people die daily from CVD in South Africa (Maredza, Hofman and Tollman 2011).

The cardiovascular burden in South Africa is increasing in all age groups and the prevalence rises with advancing age (Maredza, Hofman and Tollman 2011). This burden has detrimental effects to the South African public health care system and the economy, therefore aggressive intervention measures must be implemented (Maredza, Hofman and Tollman 2011). A recent retrospective study conducted in Dora Nginza hospital in Port Elizabeth, South Africa, reported that the prevalence of cardiovascular risk factors was 27.0% (Mkoko *et al.* 2021). According to the authors, the leading cause of CVD was systemic hypertension (HPT) (95.0%) and heart failure (33.0%). A study conducted by Ntuli *et al.* (2015) aimed at determining and assessing the prevalence of cardiovascular risk factors associated with HPT among adults in a rural community in Limpopo enrolled a total of 1407 participants. Blood pressure, weight and height measurements were taken. Information about marital status, level of education and income was gathered using the WHO stepwise questionnaire (WHO 2010). The overall cardiovascular risk factor prevalence was 41.1% (Ntuli *et al.* 2015).

This global rise in CVD is thought to be due to epidemiologic transition driven by urbanisation, industrialisation, poverty and associated lifestyle changes (Belue *et al*.

2009). According to Belue *et al*. (2009), sub-Saharan African (SSA) countries are currently experiencing the most epidemiological transition. Malan *et al*. (2008) state that the impact of urbanisation is closely linked with an increased risk of cardiovascular disease. Yusuf *et al*. (2001) report that in South Africa the increased migration of blacks to urban areas has resulted to increased poverty, obesity, HPT and elevated cholesterol. The psychological stress of living in townships where there are poor living conditions, overcrowding, poor diet, alcoholism, poor access to healthcare and physical inactivity contributes to CVD (Lindhorst *et al.* 2007).

Hypertension is the most threatening and a leading risk factor for deaths from CVD and accounts for 13% of deaths globally (Byrne, Eksteen and Crickmore 2016). According to Maredza, Hofman and Tollman (2011), the other cardiovascular risk factors such as obesity, diabetes mellitus, smoking, heavy alcohol consumption, physical inactivity and unhealthy diets have increased. This burden poses a serious threat to the health care system.

1.3 THE IMPACT OF CARDIOVASCULAR RISK FACTORS ON THE CARDIAC STRUCTURE AND FUNCTION

Studying cardiac structure and function at any symptomatic stage may predict cardiovascular events and mortality (Zhang *et al*. 2012). Aje *et al*. (2006) report that left ventricular remodelling is a major complication in patients with cardiovascular risk factors, particularly HPT, and this increases the burden of morbidity and mortality.

Prakaschandra and Naidoo (2019) conducted a study with 902 Indian participants who were part of the Phoenix Lifestyle Project (PLP). The researchers aimed to detect the geometrical changes that occur in the left ventricular structure in patients with metabolic syndrome. The left ventricle was normal in 80.8% of the patients and left ventricular hypertrophy was reported in 19.2% with the prevalence rising from 2.7% to 29.5%. Concentric remodelling was detected in 0.5%, eccentric hypertrophy in 15.9% and concentric hypertrophy in 3.2%.

According to Opie *et al*. (2006), ventricular remodelling refers to the structural changes that occur in the left ventricle in response to loading conditions. Joseph (2013)

suggests that these changes occur at the cellular and molecular level and they include alteration in myocyte biology, myocardial changes, alterations in extracellular matrix, and alterations in the left ventricular geometry. Opie *et al*. (2006) describe three patterns that occur in the left ventricle namely:

- Concentric LV remodelling: This pattern develops when there is increased pressure leading to growth in myocyte thickness. This increases the relative wall thickness (RWT) and the ventricular mass remains normal.
- Eccentric LV hypertrophy: This pattern develops when there are increases in volume leading to myocardial lengthening. This increases left ventricular mass (LVM) and the relative wall thickness remains normal.
- Concentric LV hypertrophy: This pattern occurs when there is pressure and volume overload leading to stretching and dilatation of the infarcted tissue. This increases both relative wall thickness and left ventricular mass.

Joseph (2013) states that left ventricular remodelling may occur in response to many different complex events in the left ventricle. Zheng *et al.* (2019) comment that most of the time the left ventricle remodels as a response to cardiovascular and myocardial injury. According to Cokkinos and Belogianneas (2016), when the left ventricle is strained it undergoes functional and structural changes and enlarges, resulting in the activation of a cascade of events leading to cardiac remodelling. This leads to compensatory mechanisms where the left ventricle over-functions to compensate for the decrease in cardiac function. Studying the left ventricular geometrical patterns in clinical practice is very important as they are a major complication in patients with cardiovascular risk factors and they increase morbidity and mortality (Aje *et al*. 2006). The detection of preclinical changes in left ventricular (LV) structure in those with CV disease has not been adequately studied, and may offer valuable early risk stratification opportunities before the development of advanced disease and clinical symptoms.

There is a paucity of research in SSA, South Africa and KwaZulu-Natal on this particular topic. Furthermore, data on the spectrum of cardiovascular risk factors and clustering is scarce in SSA. It has been established that prevention, early diagnosis and management of CV risk conditions are imperative given the growing burden of

CVD in SSA (Hamid *et al.* 2019). Therefore, the findings of this study will have the potential to inform clinical practice and public health directives.

1.4 RATIONALE FOR THIS STUDY

This project was initiated by the observation that cardiovascular risk factors appeared to be on the rise at Madadeni Provincial Hospital (Internal Medicine Department) in northern KZN region; however, there was no clinical data available to support this observation (Thembela 2016). In a conversation on 17 July 2016, the head of internal medicine at that time, Dr Thembela highlighted that while various studies have been conducted on cardiovascular risk factors in South Africa and internationally, the lack of the northern KZN demographics and the effects of cardiovascular risk factors on cardiac structure and function warranted an investigation in that population.

No studies in the northern KZN have been conducted on cardiovascular diseases, as most studies of that type are conducted in urban areas such as Durban (Thembela 2016). Grace and Semple (2012) conducted a study in 143 male executives living in Mpumalanga and Gauteng. The study aimed at determining the prevalence of cardiovascular risk factors. Participants with HPT had high prevalence of cluster of cardiovascular risk factors. Most studies have focussed on the single cardiovascular risk factor such as the study conducted in City of Tshwane on adolescent obesity. According to Ngwenya and Ramukumba (2017), the study reported an obesity prevalence of 8.57%.

This study therefore focused on all cardiovascular risk factors and included patients in both rural and urban northern KZN regions. Although it is generally known that there is an increase in the prevalence of cardiovascular risk factors in the black population, the effects of cardiovascular risk factors on the cardiac structure and function has not been adequately studied. The limited literature in this area shows that CV risk factors are clearly associated with changes to cardiac structure and function. The data on the degree of change to the heart in this ethnic group in relation to CV risk factor presence is novel, especially since these changes are known to occur sub-clinically, in the presence of CVD. The findings will add greatly to

the current body of knowledge, and have the potential to inform clinical practice and public health directives. The data has important clinical benefits.

1.5 AIM

The aim of the study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department).

1.5.1 OBJECTIVES

- To determine the prevalence of modifiable and non-modifiable cardiovascular risk factors among northern KZN adults.
- To determine the cardiovascular risk factors prevalence when stratified for age, gender, race and socio-economic status.
- To determine the prevalence of risk factor clustering and metabolic syndrome in this population group.
- To determine the changes which occur in cardiac structure and function using echocardiography.

1.6 STRUCTURE OF THE THESIS CHAPTERS

Chapter 1: Introduction to the research study and back ground

This chapter has provided background information about the study.

Chapter 2: Literature review on previous related studies

In this section, existing literature on the spectrum and the effects of cardiovascular risk factors on the cardiac structure and function will be reviewed.

Chapter 3: Research methodology

This chapter focuses on methods, materials, and equipment to be used. Study design, sampling strategy, data collection and statistical analysis to be used are presented.

Chapter 4: Presentation of results

This chapter presents the research results and findings.

Chapter 5: Discussion of results

This chapter focuses on the summary and discussion of the results, and compares the research findings with previous research studies.

Chapter 6: Conclusion and recommendations

This chapter presents the conclusion of the study and recommendations arising therefrom.

CHAPTER 2: STUDY BACKGROUND AND LITERATURE REVIEW

2.1 DEFINITION OF CARDIOVASCULAR DISEASE

According to the World Health Association (2017b), CVDs are a group of disorders that affect the heart and blood vessels. CVDs are the most common cause of morbidity and mortality worldwide (Leeder *et al.* 2004).

2.2 THE PREVALENCE OF CARDIOVASCULAR RISK FACTORS

Sarnak and Weiner (2019) state that cardiovascular risk factors are defined as habits, behaviours or conditions that increase a person's risk of developing CVD, and are classified into modifiable and non-modifiable cardiovascular risk factors. Many individuals generally have one or more cardiovascular risk factor (Wilson 2018).

Major modifiable cardiovascular risk factors are HPT, diabetes mellitus, dyslipidemia, obesity, physical inactivity, smoking, heavy alcohol consumption, and unhealthy diets. Major non-modifiable risk factors include age, genetic predisposition, ethnicity and gender (Leeder *et al*. 2004). Duran (2005) indicates that other modifiable risk factors include low-economic status, depression, and psychological stress. Maredza, Hofman and Tollman (2011) report that all cardiovascular risk factors are highly prevalent in SSA, in both rural and urban areas.

2.2.1 MODIFIABLE CARDIOVASCULAR RISK FACTORS

2.2.1.1 HYPERTENSION

Hypertension is defined as a systolic blood pressure (SBP) of > 140 mmHg and diastolic blood pressure (DBP) of > 90 mmHg (Seedat and Rayner 2012). Hypertension is the most threatening risk factor in relation to deaths from CVD, and accounts for 13% of deaths globally (Byrne, Eksteen and Crickmore 2016).

According to Sliwa *et al*. (2014), HPT in the black population has become a biological entity and black Africans are more hypertensive when compared to other ethnic groups. Lindhorst *et al.* (2007) reports that HPT is more severe in urban black populations when compared to whites, based on studies published by the Centers for Disease Control from1999 to 2002, with the prevalence of HPT in blacks being 40.5% and whites being 27.4%. Stewart *et al*. (2011), in the Heart of Soweto cohort study, found a large number of hypertensive cases who were at risk of developing CVD, reporting a 33% prevalence of HPT. Prakaschandra *et al.* (2016) reported that the prevalence of HPT in South African Indians was 47.5% at the time of writing, and was higher in women when compared to men and increased with age. In the cardiovascular risk in black South African (CRIBSA) study, which was conducted in 2008, the HPT prevalence was 35.6% (Peer *et al.* 2015). Hypertension increased with age and was more prevalent in women compared to men as shown in the graph in Figure 2.1. According to Braunwald (2019), the coronary artery risk development in young adults (CARDIA) study reported an association of HPT with systolic and diastolic dysfunction. Santos and Shah (2014) state that HPT has long been associated with left ventricular diastolic dysfunction.

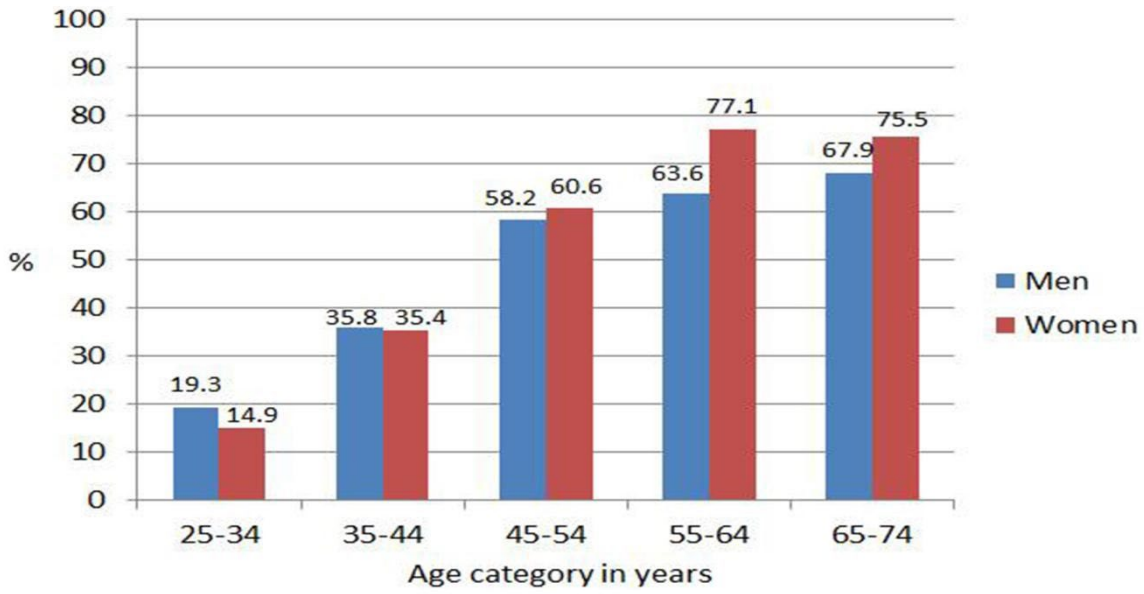

Figure 2.1: Prevalence of hypertension in 25-74- year old men and women in 2008/09 presented by age category in the cardiovascular risk in black South African (CRIBSA) study Source: Peer *et al*. 2013: 22).

2.2.1.2 DIABETES MELLITUS

The World Health Organization (WHO) (2017a) defines diabetes mellitus as a chronic disease that is characterised by high blood glucose. Reports from the Framingham study and other studies have long reported that diabetes mellitus is a risk factor for the development of ischaemic heart disease and stroke whether type or type 2 (Walsh, Fang and Fuster 2013). According to Sahedew and Singaram (2019), the International Diabetes Federation reported that 1.8 million South Africans are affected by diabetes and 69% of the population remain undiagnosed. Duran (2005) states that CVD mortality due to diabetes mellitus is about 75% in Europe. According to the WHO (2017a), diabetes mellitus rate was expected to rise from 135 million in 1995 globally to 300 million by 2025 (Braunwald 2001).

The diabetes mellitus prevalence appears to be higher in developed countries than in developing countries (Keates *et al*. 2017). The authors report that the International Diabetes Federation world atlas indicates that the age-adjusted impaired glucose tolerance African prevalence is 9.1%. The Strong Heart Study was a populationbased study of cardiovascular risk factors reported in diabetic patients (Devereux *et al*. 2000). The authors state that diabetes mellitus affects the left ventricular structure and function independently of any cardiovascular risk factor. Pillay, Lugte and Aldous (2016) conducted a retrospective study on diabetes from all KZN municipalities over a 5-year period. Figure 2.2 shows new patients that were initiated on treatment.

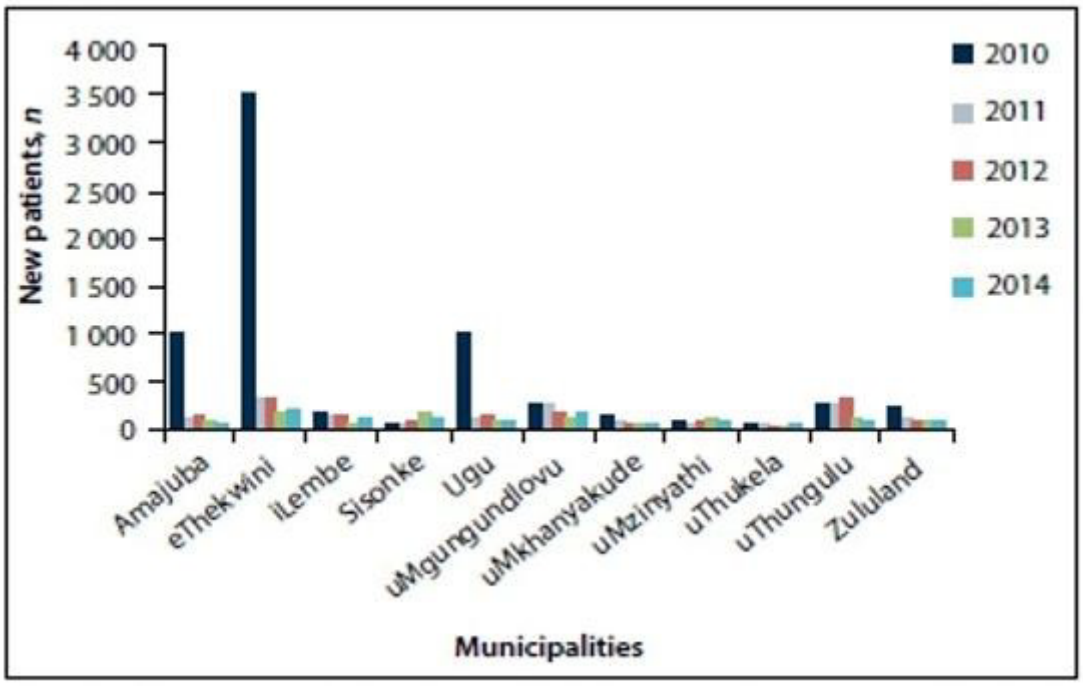

Figure 2.2: The number of new diabetic patients initiated on treatment in each of the KZN municipalities
Source: Pillay, Lugte and Aldous (2016)

2.2.1.3 DYSLIPIDEMIA

Dyslipidemia is defined as an abnormally elevated concentration of lipids in the blood (Stein, Ferari and Scolari 2019). According to Stein, Ferari and Scolari (2019), this includes high levels of LDL cholesterol, total cholesterol and triglycerides and low levels of HDL cholesterol. Cholesterol is a waxy substance found in the blood stream (Global heart journal 2017).

Dyslipidemia has long been established as a strong risk factor and a primary driver for CVD (Ntusi 2018). High levels of cholesterol in the body are estimated to cause 56% of ischaemic heart disease and account for 4.4 million deaths annually (Walsh, Fang and Fuster 2013). Low levels of high-density lipoprotein (HDL), high lowdensity lipoprotein (LDL) and increased triglycerides contribute to CVD. Wilson (2018) reveal that in the INTERHEART study, 49% of the studied population were reported to have dyslipidaemia so were at risk of developing myocardial infarction. According to Byrne, Eksteen and Crickmore (2016), one in four adults have increased levels of cholesterol.

The WHO reports that over the past 25 years there has been a global rise in hypercholesterolemia with men at 40% and women at 37% (Keates *et al*. 2017). A longitudinal study of a rural population in South Africa (Health and Ageing in South Africa) found that 67% of the rural population have dyslipidemia and 30% knew their condition, but only 0.7% were on treatment (Ntusi 2018). According to Walsh, Fang and Fuster (2013), diabetes mellitus has negative adverse cardiac effects which include increased left ventricular mass, increased wall thicknesses, reduced left ventricular systolic function and diastolic dysfunction.

2.2.1.4 OBESITY

Duran (2005) defines obesity as an excessive amount of adipose tissue in relation to lean body mass, with a body mass index (BMI) of 30 or greater. Obesity is rapidly reaching epidemic status worldwide and is associated with increased risk of CVD in both adults and children (Gaal *et al*. 2006). According to Keates *et al*. (2017), the prevalence of obesity is higher in African women when compared to men. Approximately two out of three women and one in three men are obese in South Africa (Byrne, Eksteen and Crickmore 2016). However, the WHO reports that in 31 African countries, obesity affects less than 15% of the adult population (Keates *et al*. 2017). Cois and Day (2015) conducted a research study on the obesity trends in the South African population, and reported a significantly higher BMI in females compared to males, and participants who resided in urban areas had a higher baseline BMI when compared to rural dwellers.

Sengwayo *et al*. (2012) reveal that it is estimated that by 2005, 1.6 billion and 400 million people above the age of 15 would be overweight and obese. According to Braunwald (2001), obesity is often accompanied by other cardiovascular risk factors, which include HPT, diabetes mellitus and lipid profile imbalance. Son *et al*. (2016) report that obesity increases metabolic demands resulting in an increased cardiac output and increased volume overload, thus left ventricular remodelling. In the ARIRANG study, both waist circumference (WC) and increased BMI was associated with diastolic dysfunction, reduced LVEF and dilated LV (Son *et al*. 2016).

2.2.1.5 PHYSICAL INACTIVITY

Physical activity is defined as any body movement by skeletal muscles that produce energy (WHO 2017b). Physical inactivity is an independent cardiovascular risk factor (Walsh, Fang and Fuster 2013). Moderate physical exercise is beneficial in preventing CVD because it increases HDL cholesterol, reduces blood pressure, lessens insulin resistance and promotes weight loss (Wilson 2018). According to the Global heart journal (2017), 150 minutes of physical activity per week reduces the risk of CVD by about 30%. In the INTERHEART study, 12% of the population were reported to have a lack of physical activity (Wilson 2018). In South Africa, a quarter of men and half of women are physically inactive (Byrne, Eksteen and Crickmore 2016). According to Byrne, Eksteen and Crickmore (2016), 29% of the adult population spend three hours lying in bed or watching television.

According to World Health Organization, lack of physical activity accounts for approximately 3.2 million deaths annually and more than 60% of the global population is not physically active (Tennakoon 2012). Oyeyemi and Adeyemi (2013) found that there was a low level of physical activity in a working population in Nigerian adults and this was associated with cardiovascular risk factors. According to Braunwald (2019), active individuals are at lower risk of developing CVD compared to inactive people.

2.2.1.6 UNHEALTHY DIETS

Diet plays a crucial role in the development of cardiovascular disease (Tennakoon 2012). The Western diet is the driving factor for obesity and other cardiovascular risk factors because these diets are rich in unbalanced fats, high in carbohydrates and high calorie-dense foods (Ferron *et al.* 2018). Tennakoon (2012: 123) states: “High saturated to poly unsaturated fat ratios, trans-fatty acids, high salt consumption and low consumption of fruits and vegetables are risk factors for cardiovascular disease”. According to the Global heart journal (2017), a diet that is high in saturated fat increases the risk of developing CVD. Byrne, Eksteen and Crickmore (2016), estimates that one out of five South African adults have a high fat score of 18.3% and high sugar score of 19.7%.

According to Byrne, Eksteen and Crickmore (2016), 55% of the South African population's salt consumption comes from processed foods, and 40% of consumption is salt is added during preparation of meals and at the dining table. Byrne, Eksteen and Crickmore (2016) report that South Africans consume 6 to 11 g of salt daily which is more than the recommended dose. Seedat and Rayner (2012) report that high sodium levels are also found in gravies, stock cubes, packets of soups, processed cheese, breakfast cereals, breads, salty snacks and tinned foods. Duran (2005) reports that low fruit and vegetable consumption is associated with 31% of coronary artery disease and 11% of stroke worldwide.

2.2.1.7 TOBACCO USE

Smoking is a well-known, modifiable and preventable risk factor for CVD (Leigh *et al*. 2017). Keates *et al*. (2017) state that tobacco smoking is the second leading contributor to the global rise of cardiovascular disease. 1.2 billion people were smokers in 2000 worldwide and the numbers are expected to increase to 1.6 billion by 2030 (Walsh, Fang and Fuster 2013). Walsh, Fang and Fuster (2013) point out that tobacco smoking is estimated to cause 10% of all CVD deaths and 6 million smokers die daily due to CVD and other causes. Byrne, Eksteen and Crickmore (2016) report that in South Africa, 1 in 5 adults are tobacco smokers and 1 in 5 are being exposed to secondary smoking. Smoking has always been associated with structural heart changes including abnormal left ventricular geometry and there is paucity of literature review on the impact of cardiac structure and function (Leigh *et al*. 2017).

2.2.1.8 ALCOHOL CONSUMPTION

Tennakoon (2012) reports that heavy alcohol consumption is a major risk factor and is associated with sudden cardiac death and arrhythmias. The National Institute of Alcohol Abuse and Alcoholism (NIAAA) diagnoses alcohol abuse as a risk when a woman drinks more than three drinks per day and seven drinks per week. For men, it is more than four drinks per day and 14 drinks per week (Rodrigues *et al*. 2018).

Various epidemiological studies have reported a link between alcohol consumption, CVD and death (Djousse *et al*. 2009). In mild intake, alcohol has protective effects on the cardiovascular health (Djousse *et al*. 2009). According to Yusuf *et al*. (2004), 7% of the studied population in the INTERHEART study consumed alcohol moderately and mostly were women. More than 100 research studies have reported a link between moderate drinking and risk of ischaemic heart disease, peripheral vascular disease, and sudden cardiac death from all cardiovascular causes (Braunwald 2019). The long term impact of alcohol consumption results in cardiac remodelling (Rodrigues *et al.* 2018). Rodrigues *et al.* (2018) reports that there is a lack of research on the long-term impact of alcohol abuse in relation to cardiac changes in adults globally.

2.2.1.9 PSYCHOLOGICAL STRESS

Psychological stress is a psychiatric condition that a person can experience in their life time (Song and Fang 2019). According to the global heart journal (2017), mental stress has been shown to be a risk factor for CVD and its association with cardiac events has long been recognised. De Hert, Detraux and Vancampfort (2018) state that depression is higher in coronary artery disease patients. The major reason for this is that living a hard, stressful life can cause unhealthy lifestyle behaviours like smoking, engaging in unhealthy dietary habits, lack of physical activity and increased alcohol intake (Giannoglou and Koskinas 2015).

More than 300 million people worldwide of all ages are depressed and numbers are expected to rise by 2030 (De Hert, Detraux and Vancampfort 2018). Esch *et al. (*2002) state that mental stress exerts effects on the cardiovascular pathophysiology, which brings about change in the circulatory system. The INTERHEART study reported a two-fold increase in the incidence of myocardial infarction in relation to chronic mental stressors (Yusuf *et al*. 2004).

2.2.2 NON-MODIFIABLE CARDIOVASCULAR RISK FACTORS

2.2.2.1 FAMILY HISTORY

Family history is the health information of family members (Imes and Lewis 2014). According to Imes and Lewis (2014), the first and second generation of family members are important because a person shares 50% and 25% of their genes.

Family history is regarded as a cardiovascular risk factor especially in younger people with a family history of CVD (Wilson *et al*. 2018). Using studied data from the Danish family relations, persons from family members with two or more premature cardiovascular deaths among first-degree relatives had higher risk of developing CVD before they reach the age of 50 (Wilson *et al*. 2018).

According to Walsh, Fang and Fuster (2013), “more than 35 case-control and prospective studies have identified an association between CVD and history of firstdegree relatives with early onset of cardiovascular risk factors”.

2.2.2.2 AGE

Age is an independent cardiovascular risk factor and plays a huge role in the development of CVD (Rodgers *et al.* 2019). Gao *et al*. (2019) report that CVD have a significantly high correlation with age. From the previous research, the prevalence of CVD appears to increase sharply with age (Walsh, Fang and Fuster 2013). According to Walsh, Fang and Fuster (2013), age appears to be one of the potent cardiovascular risk factors. The authors state that the incidence rates in men are the same to those in women. Persons who are over 75 years of age are at a higher risk of developing CVD (Walsh, Fang and Fuster 2013). The American Heart Association reported a high prevalence of CVD in the United States of America in both men and women with 40% in the range 40-59 years, 75% in 60 to 79 years and 86% in those above (Rodgers *et al*. 2019).

According to Anderson and Vasan (2018), young individuals who are between the ages of 18 to 45 years are increasingly developing cardiovascular risk factors in developed countries. A study conducted in the City of Tshwane on obesity in high

school scholars revealed the prevalence of 8.57% (Ngwenya and Ramukumba 2017). Ngwenya and Ramukumba (2017) state that their results reported an increase in obesity in adolescents. The World Heart Federation (2016) reports that one in four girls and one in five boys who are aged 2-14 are overweight or obese. Anderson and Vasan (2018) state that the research on CVD in young adults is concerning and more research needs to be done.

2.2.2.3 GENDER

According to Regitz-Zagrosek, Lehmkuhl and Mahmoodzadeh (2007), women may be diagnosed with CVD 10-20 years later than men. Hypertension and diabetes are major risk factors in women when compared to men (Regitz-Zagrosek, Lehmkuhl and Mahmoodzadeh 2007). This statement was also reported in the INTERHEART study (Yusuf *et al.* 2004). Gao *et al.* (2019) reveal that most attention is on the hypothesis that oestrogen is cardio-protective in women. The sharp increase of coronary artery disease in women after menopause is evidence that endogenous oestrogen may prevent CVD (Gao *et al.* 2019). Gao *et al.* (2019) report that women have a low risk of CVD but have higher mortality and worse prognosis of CVD events. For the last three decades of adult life, LDL cholesterol levels are lower in women when compared to men and this may delay the onset of CVD in women (Gao *et al*. 2019). This leads to women being neglected in treatment strategies.

2.2.2.4 ETHNICITY

According to Cooper (2001), ethnicity is when a person is categorised with a certain group whereby they share similar origin, social background, distinctive culture, and language that makes them unique from other individuals. Rayner (2010) reports that black South Africans are more prone to HPT. The first demographic and health survey on HPT was conducted in 1998 in Cape Town. A random sample of 13 802 participants were included in the study. The black population had a prevalence of 76%, followed by mixed ancestry with 13%, white with 8% and 3% with Indians.

Similar results were reported in a study in Durban where 25% of black Africans had HPT, 17% of whites , and14% of Indians (Rayner 2010). Rayner (2010) reveals that

the high prevalence of HPT in the black community suggests a strong racial origin. In a Phoenix lifestyle project, 1 428 Indians reported a high prevalence of cardiovascular risk factors with 47.5% of HPT and 20.1% of diabetes
(Prakaschandra *et al.* 2016).

According to Saab *et al*. (2015), African Americans are more prone to diabetes and obesity when compared to whites in the United States. They also have increased risk of metabolic disease which includes HPT, diabetes mellitus, chronic kidney disease, coronary artery disease and cerebrovascular attack (Saab *et al.* 2015). This ethnic difference was not present previously; for example, diabetes was less common in African Americans than Whites in early 1920s (Saab *et al.* 2015). There is a strong association between African Black ethnicity and increased LV mass and relative wall thickness (Nardi *et al*. 2017). Nardi *et al*. (2017) report that in a large cohort of black studies, LVH has always been the significant predictor of mortality. When compared with whites, blacks have always presented with a cluster of cardiovascular risk factors mainly HPT, and this may explain the increased prevalence of LVH seen in blacks (Nardi *et al*. 2017).

2.3 CARDIOVASCULAR RISK FACTORS AND ITS IMPACT ON THE CARDIAC STRUCTURE AND FUNCTION

Cardiovascular risk factors are defined as habits, behaviours or conditions that increase a person's risk of developing CVD and they include HPT, diabetes mellitus, dyslipidemia, obesity, physical inactivity, unhealthy diets, age, ethnicity, gender and family history (Sarnak and Weiner 2019). According to Sarnak and Weiner (2019), a person can have more than one cardiovascular risk factor. There is strong evidence that most patients with HPT also have diabetes mellitus or dyslipedemia (Sarnak and Weiner 2019). Gasparyan (2014) states that the understanding of cardiovascular complications and implications on the cardiac structure has improved over the years. Cardiac structure and function have been thoroughly studied and examined in the Western population by means of studies such as the Framingham Heart Study, the Rotterdam Study and the Atherosclerotic Risk in Communities (ARIC) study. In the Framingham Heart Study, 3 220 participants above the age of 40 years were

enrolled. The aim of the study was to determine the link between cardiac abnormalities and cardiovascular risk factors. Cardiovascular diseases were determined by full medical and physical examination, 12 lead ECG and full echocardiography. During the course of the study and a 4-year follow period, 208 participants presented with cardiac abnormalities, and 37 participants died from CVD. Left ventricular hypertrophy was noted in 73% of the studied population and was associated with deaths.

Zhang *et al*. (2012) conducted a clinical study in China on cardiac structure and function in the population (Zhang *et al*. 2012). A total of 843 hypertensive participants who were on antihypertensive treatment were enrolled and underwent full echocardiography to evaluate the heart, including left atrial volume, left ventricular hypertrophy, and left ventricular diastolic dysfunction. The authors reported that left ventricular concentric remodelling, left ventricular hypertrophy and left atrial enlargement was 12.7%, 5.0% and 2.4% respectively. The prevalence of mild and moderate to severe left ventricular diastolic dysfunction was 14.2% and 3.3%.

The prevalence of structural and functional cardiac abnormalities usually increases with age (Zhang *et al.* 2012). Zhang *et al.* (2012) found that the prevalence of left ventricular concentric remodelling increased from 8.3% in participants above the age of 40 years to 20.9% in participants above the age of 60 years and 15% to 46.6% for left ventricular diastolic dysfunction. According to the authors, there is clear evidence that patients with cardiovascular risk factors do present with cardiac structural and functional abnormalities.

There is a cascade of destructive events that take place at a cellular and molecular level that give rise to cardiovascular risk factors such as HPT and diabetes mellitus (Van der Velde *et al*. 2014). Any injury to the myocytes and extracellular matrix leads to activation of the sympathetic nervous system (SNS) and the renin-angiotensinaldosterone system (RAAS) (Van der Velde *et al*. 2014). Cohn, Ferrari and Sharpe (2000) reports that in a cellular level, angiotensin II which is a mediator for necrosis and fibrosis is activated, causing myocyte death. As the myocytes

decrease, the other surviving cells become stretched or hypertrophied as part of the adaptive mechanism to maintain the cardiac output (Cohn, Ferrari and Sharpe 2000).

Endothelins, cytokines, nitric oxide (NO) production and oxidative stress all contribute to cardiac remodelling (Cohn, Ferrari and Sharpe 2000). Endothelins are vasoconstrictors peptides which play a huge role in the development of heart failure. Cytokines (TNF-alpha) are cell proteins that increase in response to stimuli. Cytokine activation and elevation in myocytes induces the RAAS. Cohn, Ferrari and Sharpe (2000) state that cytokine secretion causes cell death and increases fibroblasts, contributing to increased mitochondria dysfunction, apoptosis, necrosis, progressive loss of myocytes, and inflammation, causing further deterioration of cardiac function.

This vicious cycle causes increased cardiac output and a prolonged compensatory mechanism, which impairs cardiac function (Van der Velde *et al.* 2014). Increased wall thickness plays a major role in provoking the expression of hypertrophyassociated genes, which is seen in hypertensive individuals. Van der Velde *et al*. (2014) reveal that the increased circulation of metalloproteinase increases signalling pathways that cause hypertrophy and this changes the ventricular cavity, resulting in cardiac remodelling and later heart failure.

2.4 METABOLIC SYNDROME

Metabolic syndrome is defined as a cluster of metabolic abnormalities that increase the risk of CVD and diabetes (Joseph 2013). Kaur (2014) states that the metabolic syndrome is becoming a major health challenge worldwide. According to Joseph (2013), the criteria for the metabolic syndrome have evolved over the years since the first definition by the World Health Organization in 1998. Fuster *et al*. (2017) report that the major components for the metabolic syndrome include obesity (measured by waist-hip ratio or BMI), HPT, dyslipidemia (increase triglycerides levels and low highdensity lipoprotein cholesterol levels) and hyperglycaemia (impaired fasting glucose tolerance). Tune *et al.* (2017) state that each component is a cardiovascular risk factor and the combination of these risk factors increases the severity of cardiovascular disease. The prevalence of metabolic syndrome differs worldwide and usually increases with age (Joseph 2013). The prevalence of metabolic syndrome

worldwide is estimated at 20% to 25% in adults and 10% to 19.2% in children (Belete *et al*. 2021). Kaur (2014) states that the International Diabetes

Federation proposes that one-quarter of the world's population has metabolic syndrome. The prevalence is also reported to be higher in Hispanic women when compared with Hispanic men (Rochlani *et al*. 2017). Rochlani *et al.* (2017) states that metabolic syndrome affects a quarter of the United States of American population. The prevalence in African-American women is 57% while the prevalence is 26% in males (Rochlani *et al.* 2017). Table 2.1 shows the prevalence of metabolic syndrome in African countries and South Africa.

Motala, Mbanya and Ramaiya (2009) suggest that its pathology is related to abdominal obesity and insulin resistance. Insulin resistance occurs when an increase in insulin fails to reduce glucose levels normally (Han and Lean 2016). Metabolic syndrome is strongly associated with unhealthy diets, psychological stress, physical inactivity and genetic composition (Han and Lean 2016). Okafor (2012) reveals that metabolic syndrome is becoming prevalent in Africa with a prevalence of 34.3 % in Nigeria. According to a literature review by Motala, Mbanya and Ramaiya (2009), metabolic syndrome in SSA is minimal, particularly in South Africa.

Table 2.1: Prevalence of metabolic syndrome in African countries and South Africa

Table 3. Prevalence of metabolic syndrome in African countries

Reference	Criteria used	Study design	N	Age[a], years	Country	MetS prevalence	
						Male %	Female %
Akintunde et al[23]	IDF	Cross-sectional (urban)	140	55.1 ± 10.8	Osogbo, Nigeria	23.0	37.0
Tran et al[24]	IDF	Cross-sectional (urban)	1,935	≥24	Addis Ababa, Ethiopia	14.0	24.0
Assah et al[25]	Modified NCEP	Cross-sectional (urban/rural)	552	25-55	Cameroon	7.0[b]	25.0[b]
Kengne et al[26]	IDF	Cross-sectional (urban; type 2 diabetics)	308	55.8 ± 10.5	Cameroon	55.7[b]	72.1[b]
Adeoye et al[27]	IDF	Cross-sectional (urban)	256	42.0 ± 9.4	Nigeria	17.0	14.0
Labhardt et al[28]	IDF	Cross-sectional (rural)	1,026	≥25	Lesotho	9.8	22.9
Mogre et al[29]	IDF	Cross-sectional (urban)	200	≥30	Ghana	13.0	27.3

a. Age range or mean age of participants as provided by the respective journal articles.
b. Age-adjusted MetS prevalence used.
National Cholesterol Education Program; IDF, International Diabetes Federation.

Table 4. Prevalence of metabolic syndrome in South Africa

Reference	Criteria used	Study design	N	Age[a], years	Country	MetS prevalence	
						Male %	Female %
Crowther and Norris[3]	Harmonized	Cross-sectional (urban)	1,251	18-84	Soweto, Johannesburg (black South African)	–	42.1
Schutte and Olckers[30]	IDF	Cross-sectional (urban)	102	31.3 ± 8.6	Potchefstroom (black South African)	–	24.8
Jennings et al[31]	IDF	Cross-sectional (urban)	225	27.0 ± 7.0	Cape Town (black South African)	–	9.9
Motala et al[2]	Harmonized	Cross-sectional (rural)	947	>15	Kwa–Zulu Natal (black South African)	11.6	30.2
Kalk and Joffe[32]	IDF	Cross-sectional (urban; type 2 diabetics)	500	>30	Johannesburg, South Africa		
					African	46.5	85.2
					White	74.1	86.6
Peer et al[4]	Harmonized	Cross-sectional (urban)	1,099	25-74	Cape Town (Black South African)	16.5	43.0
George et al[33]	Harmonized	Cross-sectional (urban)	724	18-70	Soweto, Johannesburg		
					Black South African	19.3	39.2
					Indian South African	55.8	37.4
Erasmus et al.[34]	IDF	Cross-sectional (urban)	563	50.9 ±9.1	Bellville, Cape Town (mixed ethnic background)	30.6	67.8

Source: Gradidge and Crowther (2017: 27)

Gradidge and Crowther (2017) state that most SSA countries reported a higher prevalence of metabolic syndrome in women than men. The prevalence of metabolic syndrome is higher in black South African women with 42% and United Kingdom women with 23% (Gradidge and Crowther 2017).

According to Gradidge and Crowther (2017), when comparing the prevalence of metabolic syndrome across the studies, some differences are due to the difference in

age ranges used in each study and the diagnostic criteria used. McGuire and Marx (2015) report that the prevalence of metabolic syndrome is affected by many factors, which include age, gender, diet, lifestyle habits, ethnicity, family history and socioeconomic conditions.

The criterion that is mostly commonly and widely used to diagnose metabolic syndrome are the harmonised criteria that were first introduced in 2009 (Gradidge and Crowther 2017). Gradidge and Crowther (2017) state that studies conducted in various African countries show that some features of metabolic syndrome are more prominent in the African population such as high WC and low HDL levels.

The Cardiovascular Risk in Black South Africans (CRIBSA) study reported a high prevalence of metabolic syndrome in the black women population in Cape Town (Peer *et al.* 2015). They used the harmonised Joint Interim Statement (JIS) criteria. They reported a higher prevalence in women (43.5%) than men (16.5%) which is similar to what has been reported in other SSA countries and among African Americans (Peer *et al*. 2015).

2.4.1 METABOLIC SYNDROME AND CARDIAC STRUCTURE AND FUNCTION

The prevalence of diastolic and systolic dysfunction on metabolic syndrome is increasing globally (Aseri *et al.* 2016). According to Aseri *et al*. (2016), diastolic dysfunction is the main pathophysiological mechanism leading to changes in the cardiac structure and function. Hispanics/Latinos in America have a high prevalence of metabolic syndrome and this was the reason why this population was chosen for a research study by Pena *et al*. (2018). The authors assessed the cardiac structure of the participants using echocardiography, and found that normal weight participants with metabolic syndrome had reduced left ventricular function and increased left ventricular diastolic volume.

The Multi-Ethnic study of Atherosclerosis (MESA) reports a direct link between left ventricular end diastolic volume and increased BMI in both males and females (Tune *et al*. 2017). According to Tune *et al.* (2017), this population-based study also reported that obesity resulted in left ventricular eccentric hypertrophy. The authors found that

more recent research studies are reporting a direct association of obese individuals with concentric left ventricular hypertrophy, diastolic or systolic dysfunction with normal or reduced left ventricular ejection fraction.

Aseri *et al.* (2016) conducted a study on 100 metabolic syndrome individuals to assess the prevalence of left ventricular dysfunction using echocardiography. The study was conducted in Guru Gohind Singh medical college and hospital in India. According to Aseri *et al*. (2016), the prevalence of left ventricular diastolic dysfunction was 60% in the 30-40 years age group, 24% in 41-50 years age group and 28% in participants in 50-60 years of age. This high prevalence of diastolic dysfunction calls for prevention interventions to be implemented (Aseri *et al*. 2016).

A random sample of 2 042 participants in Minnesota residents in the United States of America took part in a population-based study. The authors, Aijaz *et al.* (2008), used the National Cholesterol Education Program Adults Treatment panel III criteria for metabolic syndrome. According to the authors, the prevalence of metabolic syndrome on the studied population was 21.7% in men and 16.7% in women. Left ventricular diastolic dysfunction was present in 28.2% of women with metabolic syndrome and in 14.9% of women without metabolic syndrome (Aijaz *et al.* 2008).

2.5 BACKGROUND TO LEFT VENTRICULAR STRUCTURE AND FUNCTION

Whiteman *et al.* (2021) state that the left ventricle has always been subjected to research due to its important function in the cardiovascular system. According to Sengupta *et al*. (2006), normal left ventricular geometry has long been described as an ellipsoid, cone shape with the right ventricle next to it. Whiteman *et al.* (2021) state that its apex is conical and is situated in the 5^{th} intercostal space, closer to the mid-clavicular line. Because the left ventricle is a major pumping chamber for the systemic circulation, any loading condition may result in modelling its structure and affecting its function (Betts *et al*. 2017).

The myocardium is striated like skeletal muscle (Waugh and Grant 2014). Every cell has a centrally located nucleus and one or more branches (Waugh and Grant 2014). According to Curran and Sheppard (2011), the myocytes are short, branched fibres that measure 10-20 micrometre in diameter and 50-100 micrometre in length. The

ends of the myocytes and branches are in close contact by means of intercalated discs that give the cardiac muscle the appearance of being a laminated sheet of muscle. According to Berman, Tupper and Bhardwaj (2019), these connections allow cardiac myofibrils to synchronously contract. Crumbie (2020) states that the cytoplasm between myofibrils contains cells, mitochondria, and the intracellular membrane system, which is known as the sarcoplasmic reticulum.

Berman, Tupper and Bhardwaj (2019) state that the contractile unit I, the myocyte, is linked to the T-tubule system on both ends. The T-tubules are part of the sarcolemma where calcium channels are stored. The amount of calcium determines the degree of cardiac systole. The mitochondria in cardiac myocytes are large and supply the ATP needed for the repeated cardiac contractions (Curran and Sheppard 2011). According to Crumbie (2020), the sarcomere, which is the structural and functional unit of cardiac contraction, has Z lines. The distance between the Z lines determines the force of cardiac contraction or relaxation. Berman, Tupper and Bhardwaj (2019) reveal that when the left ventricle faces any increase in cardiac demand such as increased preload or afterload, it undergoes a cardiac remodelling process, resulting in left ventricular failure.

2.6 HISTORICAL PERSPECTIVE OF VENTRICULAR REMODELLING

The term “remodelling” was for the first used in 1982 by Hockman and Buckery in a myocardial infarction model (Azevedo *et al*. 2016). Azevedo *et al*. (2016) suggest that this term was used to describe the characteristics involved in the replacement of infarcted tissue with scar tissue. The term “ventricular remodelling” was used for the first time in 1985 by the researcher Janice Pfeffer (Opie *et al*. 2006). Pfeffer and Braunwald (1990) report that their research was aimed to study the patterns of increased left ventricular dilation and poor function after myocardial infarction in a rat model.

Since 1990, the term “left ventricular remodelling” has been used to describe many patterns due to the mechanical stress of CVD (Opie *et al*. 2006). According to Azevedo *et al*. (2016), this was the major reason why in 2000 a review from the international forum on cardiac remodelling was published. The review described the

term “left ventricular remodelling” as relating to molecular, cellular and interstitial changes that cause changes in size, shape and function of the heart due to cardiac injury. The early studies in conscious human beings were by Linzbach 50 years ago, and today the types of remodelling are known as concentric remodelling, eccentric left ventricular hypertrophy and concentric left ventricular hypertrophy (Gaasch and Zile 2011).

2.7 PATHOPHYSIOLOGY OF LEFT VENTRICULAR REMODELLING

Azevedo *et al.* (2016) report that even though left ventricular remodelling leads to left ventricular failure, its pathological mechanism is not fully understood. The first stimulus for left ventricular dilation is stretching of the myocardium which results from myocyte loss causing an affected area to be akinetic or dyskynetic (Cokkinos and Belogianneas 2016). Cellular and molecular changes include myocyte hypertrophy, necrosis, apoptosis, fibrosis, increased fibrillar collages, changes in the extracellular matrix, sarcolemma, sarcoplasmic reticulum, myofibrils, mitochondria, nucleus and protein abnormalities (Cohn, Ferrari and Sharpe 2000) (Table 2.2).

Table 2.2: Pathophysiology of ventricular dysfunction in cardiac remodelling

Mechanism	Main changes	Consequence
Cell death	↑ apoptoxis, ↑ necrosis ↓ Autophagy	Progressive myocyte loss
Energy metabolism	B oxidation, Triglyceride accumulation, ↑ glycolysis Mitochondrial dysfunction Mitochondrial atrophy	Lipotoxicity ↓ Energy ↓ Oxidative stress
Oxidative stress	↑ NADPH oxidase ↑ Catecholamine degradation ↑ Xanthine oxidase mitochondrial dysfunction ↓ Antioxidant systems	Lipid peroxidation, Protein oxidation DNA damage, Cell dysfunction Fibroblast proliferation Metalloproteinase activation, ↑ Apoptosis ↑ Signalling pathways to hypertrophy
Inflammation	Innate response	↑ Inflammatory cytokines
	Adaptive response dysfunction	Macrophage,T cell and B cell dysfunction
Collagen	Fibroblast proliferation ↑ Metalloproteinases	Degradation of normal collagen Fibrosis

Contractile proteins	B-myosin ↓ a-myosin ↑ Troponin T type 2, ↓ Troponin I phosphorylation	↓ Contractility
Calcium transport	↓ L-type calcium channels ↓ Rynodine, ↓ Calsequestrin ↓ Calmodulin, ↓ Phospholamban phosphorylation, ↓ SERCA 2a	↓ Calcium in systole ↑ Calcium in diastole
Geometry	LV cavity, ↓ Wall thickness Elliptical shape→ spherical shape	↑ Parietal stress of the LV
Neurohormonal activation	↑ RAAS ↑ Sympathetic	↑ Cell death,↑ Oxidative stress, ↑ Inflammation,↑ Metalloproteinase.

Source: Azevedo *et al.* (2016)

The potential factors involved in this process are cell death, inflammation, and structural molecular changes, leading to left ventricular remodelling (Azevedo *et al*. 2016). This process is influenced by hemodynamic load and neurohormonal activation (Cohn, Ferrari and Sharpe 2000). According to Burchfield, Xie and Hill (2013), the cellular elements in the ventricle include fibroblasts (promoting fibrosis), vascular smooth muscle cells (promoting vascular stiffness), vascular endothelial cells (promoting endothelial dysfunction and leukocytes (promoting inflammation). The cellular changes in the myocyte are coupled by changes within the extracellular matrix (Braunwald *et al.* 2001). Braunwald *et al.* (2001) state that both myocyte dysfunction and extracellular matrix changes result in myocardial remodelling.

Azevedo *et al*. (2016) reveal that, as previously described, cardiac remodelling is associated with changes in the wall thickness and cavity diameter due to pressure and volume overload. According to Cohn, Ferrari and Sharpe (2000), as the heart remodels, its geometry become less elliptical and more spherical.

2.7.1 PATHOPHYSIOLOGICAL MECHANISMS OF CARDIAC REMODELLING

2.7.1.1 MYOCYTE DEATH

Myocytes play a huge role in the remodelling process (Cohn, Ferrari and Sharpe 2000). According to Burchfield, Xie and Hill *(*2013), cardiac myocytes are responsible for myocardial contractile functions and are able to replicate. Azevedo *et al.* (2016) reveal that there are three pathological mechanisms that are involved in myocyte injury, namely, increased apoptosis, increased necrosis, and autophagy. Apoptosis usually occurs at a faster rate after myocardial infarction and is linked to cardiac hypertrophy. Autophagy is an intracellular process that is characterised by dysfunctional cytoplasmatic components (Azevedo *et al*. 2016). This disturbance causes cell death and further progressive loss of myocytes plays a major role in cardiac remodelling.

As a result of cell death, myocyte numbers decrease progressively and surviving myocytes are stretched or hypertrophied in the course of the initial heart failure compensatory process (Azevedo *et al*. 2016).

2.7.1.2 THE ROLE OF COLLAGEN DEGRADATION

The heart is composed of a collagen network in the interstitium (Azevedo *et al.* 2016). Collagen plays a huge role in maintaining the cardiac structure and function, and its main functions are to regulate apoptosis, restore pathological deformations and maintain the alignment of structures. Collagen also regulates the distensibility of the heart muscle and transmission of strength during fibre shortening, and expresses cytokines and growth factors (Azevedo *et al*. 2016).

According to Opie *et al.* (2006), when collagen synthesis increases, it causes fibrosis when induced by angiotensin II, aldosterone and transforming growth factor beta (TGFB). metalloproteinases are enzymes that degrade collagen and tissue inhibitors of matrix metalloproteinases (TIMPs) prohibit them. Once this enzyme increases, it causes the hypertrophied left ventricle to dilate and the process of cardiac remodelling begins (Opie *et al.* 2006). According to Azevedo *et al*. (2016), the

abnormal increase in collagen III and I have been detected in various models of cardiac injury.

2.7.1.3 NEUROHORMONAL ACTIVATION

According to Hung *et al*. (2005), neurohormonal activation is a major cause of pathological ventricular remodelling. The sympathetic system and the RAAS are both involved in cardiac remodelling when activated (Azevedo *et al.* 2016). Hung *et al*. (2005) report that during neurohormonal activation there is a gradual increase in the release of renin, norepinephrine, angiotensin II and antidiuretic hormone. According to Azevedo *et al*. (2016), stimulation of both systems causes synthesis of metalloproteinases, cellular hypertrophy, causing fibrosis leading to cellular death by necrosis and apoptosis. Neurohormonal activation may occur in response to reduced cardiac output but also plays a major role in the remodelling process (Cohn, Ferrari and Sharpe 2000).

The RAAS plays a major role in the pathophysiology of ventricular remodelling (Pattern and Konstam 2000). Rousseau *et al*. (1994) report that heart failure patients with left ventricular dysfunction have increased levels of angiotensin II despite initiation of an angiotensin converting inzyme (ACE) inhibitor. Pattern and Konstam (2000) suggest that the role of angiotensin II is to act as a growth factor in the myocardium and this has been supported by the researchers Sadoshima and Izumo in 1993). The researchers discovered angiotensin II levels in myocytes granules, 30 mm after the myocardial stretch (Pattern and Konstam 2000).

2.7.2 IDENTIFICATION OF LEFT VENTRICULAR GEOMETRIC PATTERNS

Left ventricular remodelling is defined a complex process involving molecular, cellular, interstitial changes that are characterised by alterations in the left ventricular size, mass, geometry and function after a myocardial stimulus (Pontes *et al.* 2016).

In order to clearly understand ventricular remodelling, the geometry of the changes has been classified. The classification of the geometrical pattern must consider left ventricular volume, left ventricular mass and relative wall thickness, which are terms

which are used by the American Society of Echocardiography (Gaasch and Zile 2011). Marwick *et al.* (2015) state that left ventricular geometry is classified according to whether the left ventricular mass or relative wall thickness is normal or increased. Relative wall thickness (RWT) is 2 X PWT/ LVEDD and has a value of 0.42 while the left ventricular mass index (LVMI) varies in male and females and has value of 95-115 (Marwick *et al*. 2015). A normal left ventricular geometry is defined as a normal chamber size and wall mass, and normal RWT (Zile *et al.* 2014).

2.7.2.1 CONCENTRIC LEFT VENTRICULAR HYPERTROPHY

Concentric hypertrophy is mostly associated with HPT and is a compensatory mechanism due to increased systemic pressure (Marwick *et al.* 2015) (Figure 2.3). In concentric LV hypertrophy there is increased left ventricle wall, increased relative wall thickness, and increased left ventricular mass. The left ventricular end diastolic dimension (LVEDD) is normal. According to Marwick *et al*. (2015), concentric LV hypertrophy affects both men and women regardless of age and race.

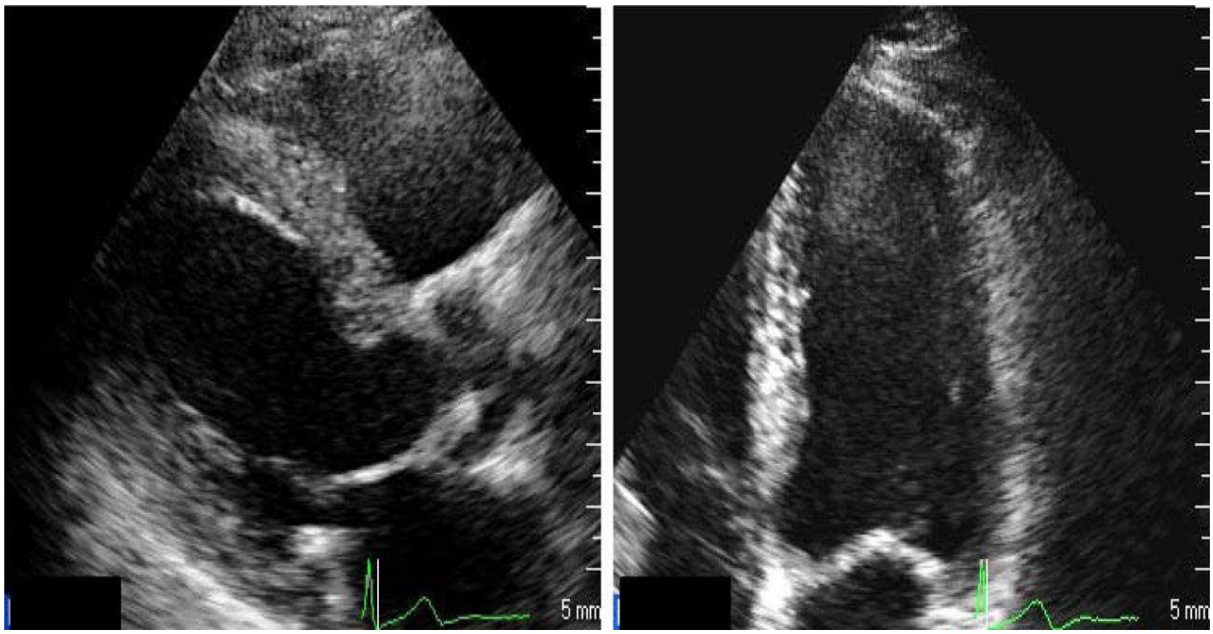

Figure 2.3: A parasternal long-axis (left) and apical four-chamber views (right) from a 55-yearold showing concentric left ventricular hypertrophy
Source: Marwick *et al.* (2015)

2.7.2.2 ECCENTRIC LEFT VENTRICULAR HYPERTROPHY

A dilated chamber indicates eccentric left ventricular geometry (Marwick *et al*. 2015) (Figure 2.4). Zile *et al*. (2014) define eccentric LV hypertrophy as an increase in left ventricular end diastolic dimension (chamber size), increase in left ventricular mass, and normal relative wall thickness (RWT). According to Marwick *et al*. (2015), eccentric LV hypertrophy is associated with increased volumes and these patients usually have low and mildly reduced systolic function.

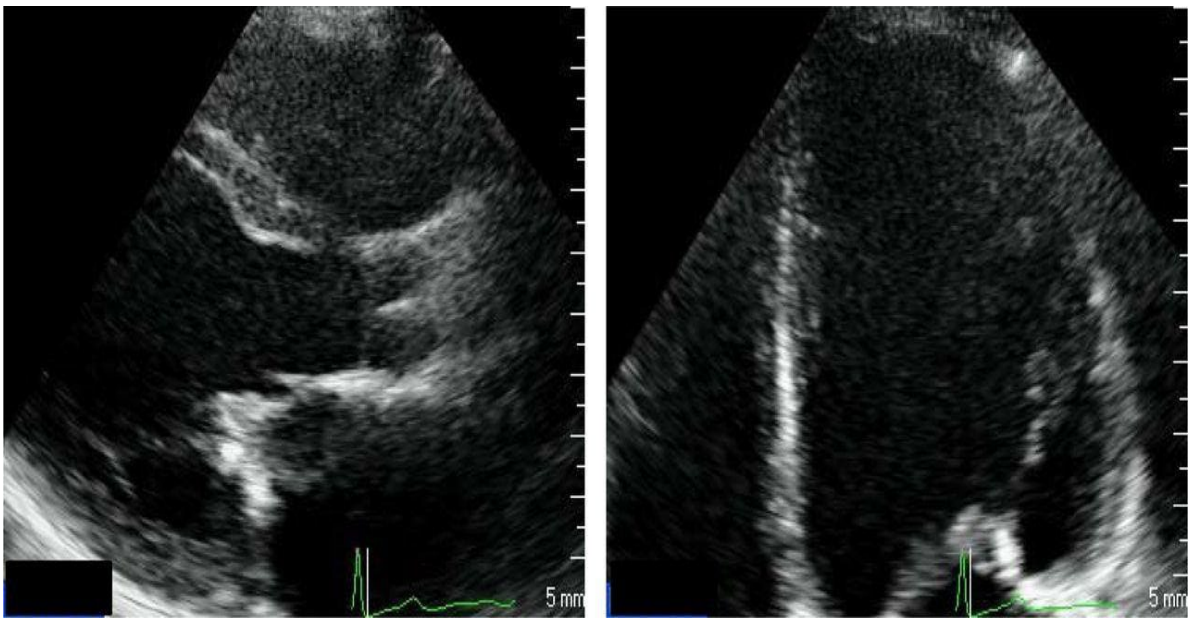

Figure 2.4: A parasternal long-axis view (left) and an apical four-chamber view (right) of a 28year-old female showing eccentric left ventricular hypertrophy Source: Marwick *et al.* (2015)

2.7.2.3 CONCENTRIC LEFT VENTRICULAR REMODELLING

Concentric left ventricular remodelling is usually the late stage of an adaptive response and can be caused by both chronic pressure and volume overload (Marwick *et al*. (2015) (Figure 2.5). Marwick *et al*. (2015) state that patients with concentric remodelling usually present with left ventricular systolic dysfunction. Echocardiographic features of concentric left ventricular remodelling are a normal LV size, normal left ventricular mass, and increased relative wall thickness (Marwick *et al*. 2015).

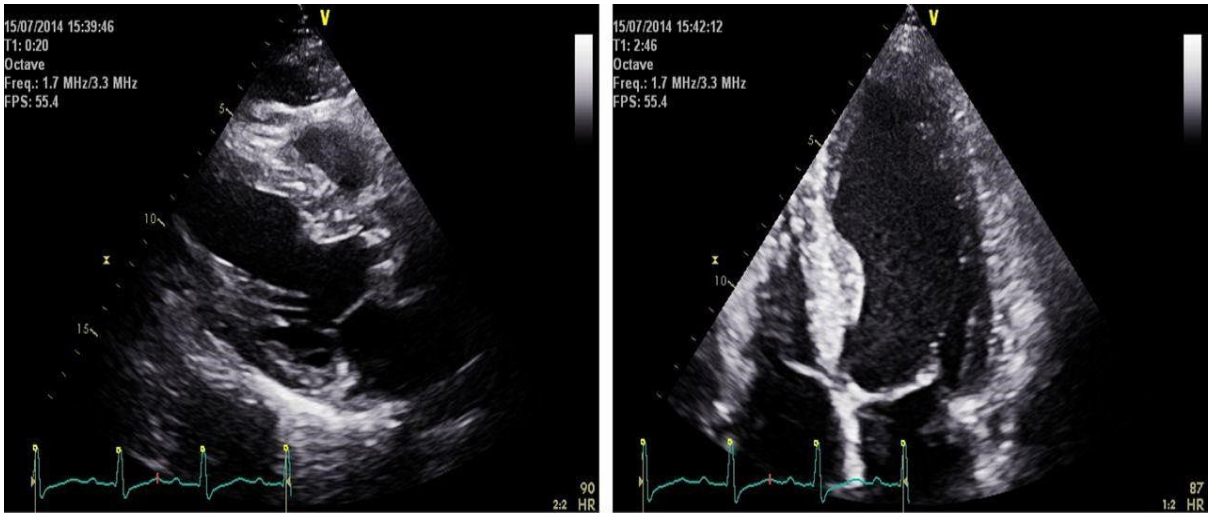

Figure 2.5: Concentric LV remodelling in a 59-year-old male patient Source: Marwick *et al.* (2015)

2.8 HEART FAILURE

Cardiac remodelling involves adaptive and maladaptive responses (Pontes *et al.* 2016). During the adaptive phase, the heart is able to maintain the cardiac output and stroke volume by undergoing cellular and molecular changes. According to Braunwald (2019), the heart depends on the adaptive mechanisms to continue maintaining perfusion to major organs, which includes the Frank-Starling phenomenon, activation of the neurohormonal system, and activation of the RAAS. When these mechanisms fail due to chronic haemodynamic overload, maladaptive events occur and heart failure begins (Braunwald 2019). This usually takes a long period before congestive heart failure occurs.

Heart failure is a major complication of cardiac remodelling (Roever and Chagas 2017). Heart failure is a stage whereby the heart fails to pump enough blood to meet the metabolic demands of the body. According to Toback (2016), heart failure is a worldwide health problem with a high prevalence of approximately 23 million and has a high mortality rate. Galli and Lombardi (2016) reveal that heart failure with reduced ejection (HFrEF) accounts for 50% to 70% of cases of death while heart failure with preserved ejection fraction (HFpEF) has up to 40%.

According to McMurray *et al*. (2015), any damage to the myocyte and extracellular matrix results in changes in the ventricular geometry leading to remodelling. The cycle, which involves a wide range of events over time, leads to heart failure. After any initial injury to the myocytes and extracellular matrix, the process of cardiac failure may begin. The authors point out that there is a wide range of pathological mechanisms that are activated. These include neurohormonal activation, increases cytokine, apoptosis, oxidative stress and inflammatory changes. Such mechanisms may cause progression from an asymptomatic to a symptomatic state (McMurray *et al*. 2015). According to Schirone *et al.* (2017), the best way to fully understand heart failure is to know the pathological process leading to cardiac remodelling. All these activations change the cardiac geometry causing increased volume overload, reducing contractile wall stress, causing increased left ventricular mass leading to reduced left ventricular ejection fraction and heart failure (Schirone *et al*. 2017).

2.9 PREVENTATIVE STRATEGIES TO REDUCE THE IMPACT OF CV RISK FACTORS

CVD is the biggest cause of global deaths, with the majority occurring in low- and middle-income countries (Schwalm *et al*. 2016). According to Piepoli *et al*. (2016: 2319), “Cardiovascular disease prevention is defined as a coordinated set of actions at the population level or targeted at an individual that are aimed at eliminating or minimising the impact of CVDs and their related disabilities”. Van der Sande *et al*. (2001) state that SSA is burdened by this public health problem and the response is inadequate. According to Shah (2014), government should focus on cardiovascular risk reduction and prevention. Schwalm *et al.* (2016) reveal that preventative strategies and management of CVD should include: (1) Evidence of benefit and cost effectiveness, (2) Feasibility of implementing such strategies, (3) The ability to sustain the preventative strategies and (4) Socio-political acceptability.

According to the World Health Organization (2007), there are two types of preventative intervention, namely, population-wide approach where the whole community is targeted particularly those who are at high risk, and an individual approach. The combinations of the two approaches are best in changing the risk of the population at large (Gersh *et al.* 2010).Two other approaches include public

health and high risk clinic-based strategies. According to Schutte (2018), individuallevel preventative strategies together with the primary health care facilities should work together for the detection, treatment and control of cardiovascular risk factors.

2.9.1 STRENGTHENING COMMITMENT OF POLICY MAKERS AND HEALTH EDUCATION

Van der Sande *et al.* (2001) report that generating the commitment of policy makers to understand that CVD is a major problem. Educating the community that there is a high prevalence of cardiovascular risk factors in the community can be motivational to get everyone involved (Van der Sande *et al.* 2001). Government departments, non-government organisations, and the private sector should be involved in establishing CVD units by the health ministry to develop prevention and control strategies (Van der Sande *et al*. 2001). According to Van der Sande *et al*. (2001), such units can tackle CVD issues, give guidance, stimulate extensive planning and offer support to affected individuals and their families. These CVD units can develop intensive programmes which include local and national populations, wide approach through legislation, health policies, practical guidelines and protocols in assisting people adjust their lifestyles. The authors propose that such programmes should also include school health, whereby scholars are educated at a young age about cardiovascular disease.

Media should be utilised with visible posters in the area, advertisements, and engaging talks on radio and television on cardiovascular disease. Work, community, and church support groups should be prioritised (Maredza, Hofman and Tollman 2011).

CVD programmes should explain clearly how to understand and modify prevalent risk factors in targeted populations (Van der Sande *et al*. 2001). Multi-sectional commitment is needed in order for these programmes to be successful. Van der Sande *et al.* (2001) suggests that healthcare workers must be highly trained in both urban and rural populations, as well as the elderly population and first-degree family members of known CVD patients. Regular screening of asymptomatic individuals should be included in the interventions (Van der Sande *et al*. 2001).

According to Beleu *et al.* (2009), involving the government on local and national level is necessary to reduce CVD in SSA. Lack of awareness and misconceptions on cardiovascular risk factors also contribute to the rise of CVD, therefore health education and national campaigns are required (Beleu *et al.* 2009).

2.9.2 LIFESTYLE MODIFICATION

Rrisk factor modification must begin with a change in lifestyle behaviour. Lifestyle modifications require a good doctor-patient relationship, structured programmes that involve multi-disciplinary teams and regular visits to the clinic. According to the WHO (2007), lifestyle modification has been reported to lower blood pressure in clinical trials. These modifications include cessation of smoking, weight loss, physical activity, reducing alcohol intake, eating a well-balanced meal, reduced saturated fat, reduction of sodium intake, increase in potassium intake and maintaining mental health (WHO 2007).

2.9.2.1 SMOKING

According to Maredza, Hofman and Tollman (2011), tobacco control programmes have been implemented in South Africa which include passing of the Tobacco Products Control Act in 1993. This Act restricts smoking in public places and the advertisement of tobacco products (Maredza, Hofman and Tollman 2011). Snuff should also be avoided and nicotine replacement therapy should be used in hypertensive patients under medical supervision (Seedat and Rayner 2012). The most effective way to reduce tobacco use is to assist the users to stop smoking because morbidity and mortality in the following two to three decades increases in those above the age of 30 years (Schwalm *et al*. 2001). Yusuf *et al*. (2001) recommend that the government must increase the price of cigarettes and host antitobacco campaigns every now and then.

2.9.2.2 UNHEALTHY DIET

The research data from high-income countries shows that increasing fruit and vegetable consumption, reducing saturated fats, and avoiding trans-fats in the diet are associated with lower risk of coronary artery disease (Schwalm *et al*. 2016).

Maredza, Hofman and Tollman (2011) report that the Department of Health is proposing a trans-fatty acid bill to reduce trans-fats in processed and prepared meals. Schwalm *et al*. (2016) state that a healthy diet should always be emphasised at all times even at scholarly level. Fruit and vegetable consumption promotes cardiovascular health.

Reducing salt intake reduces blood pressure and improves the quality of life (Shah 2014). The Food Standard Agency (FSA) recommends a maximum consumption of 6 g per day (one teaspoon) for normal individuals and for hypertensive patients as little as possible (Maredza, Hofman and Tollman 2011). According to Maredza, Hofman and Tollman (2011), Woolworths has, over the years, tried to reduce salt in their products. Eating healthily also reduces the chances of being obese (Shah 2014).

2.9.2.3 PHYSICAL INACTIVITY AND OBESITY

Research data from the INTERHEART study showed a decrease of myocardial infarction incidence among participants who engaged in moderate exercise such as walking, cycling, and jogging (Shah 2014). Catepano *et al*. (2016) state that the target should be 2.5 to 5 hours of moderate exercise per week. The government must build recreation parks to promote physical activity in urban and rural areas; in that way, obesity will be prevented (Yusuf *et al.* 2001).

The BMI that should be aimed for is that of 20 - 25 kg/ m^2 and a waist-to-hip ratio (WTHR) of less than 0.90 cm. Maredza, Hofman and Tollman *(*2011) report that selfscreening for weight control is highly recommendable. According to Yusuf *et al.* (2001), regular physical activity also prevents glucose intolerance and HPT.

2.9.2.4 ALCOHOL CONSUMPTION

Catepano *et al.* (2016) report that moderate beverage consumption of 20 g/day (two units) is recommended for men and 10 g/day (one unit) for women. A standard drink is approximately 10 g of ethanol, which is 12 ml of wine and 340 ml of beer (Seedat and Rayner 2012).

2.9.2.5 PSYCHOLOGICAL STRESS

The South African Stress and Health Survey (SASH) in 1999 was the first study to look at mental health in South African adults (Mayosi *et al.* 2009). The study revealed that 8.1% had an anxiety disorder, 4.9% had mood disorder, and 1.8% had explosive disorder. Mayosi *et al.* (2009) states that no other national study has researched the prevalence of psychiatric disorders.

2.9.3 PHARMACOLOGICAL INTERVENTION

Pharmacological intervention must always be accompanied with lifestyle modification (Shah 2014). According to Shah (2014), antihypertensive treatment, lipid-lowering drugs and antiplatelet medication can help.

Blood pressure lowering drugs: According to the National Vascular Disease Prevention Alliance (NVDPA) (2012), blood pressure lowering drugs play a huge role in reducing both morbidity and mortality. These drugs include calcium channel blockers, angiotensin receptor blockers, angiotensin converting enzyme (ACE) inhibitors and thiazide diuretics. Rayner (2010) states that the South African HPT guidelines recommend low doses of diuretics as first-line drugs, which can then be combined with an ACE inhibitor or a calcium channel blocker if necessary.

Lipid-lowering therapy: Catepano *et al.* (2016) report that the 2011 European Atherosclerosis Society (EAS) / European Society of Cardiology (ESD) guidelines for dyslipidaemia management emphasise the importance of LDL cholesterol lowering drugs to prevent CVD. Statins should be used as a first-line therapy. Piepoli *et al*. (2016) report that in the Heart Protection Study (HPS), 40 mg of simvastatin

decreased the risk of CVD. The Collaborative Atorvastatin Diabetes Study (CARDS) also supported this (Piepoli *et al.* 2016).

Antiplatelet therapy: Aspirin should be used as a first-line therapy. Patients with diabetes mellitus have a tendency for developing thromboembolic events (Piepoli *et al*.2016). Piepoli *et al*. (2016) report that the Antiplatelet Trialists Collaboration metaanalysis illustrated the benefits of aspirin in such patients.

Glycaemic control drugs: The UK Prospective Diabetes Study (UKPDS) emphasised the importance of glucose lowering drugs, metformin being the first-line therapy (Piepoli *et al*. 2016).

A few research studies have been conducted to determine or identify the prevalence of CVD in SSA and globally. A research study by Raal *et al*. (2018) was conducted to evaluate the prevalence of cardiovascular risk factors in Africa. The Africa Middle East Cardiovascular Epidemiological (ACE) study was conducted in 14 African countries, 94 general practices, out-patients rural and urban clinics between July 2011 and April 2012. The clustering of cardiovascular risk factors was by national income group that were classified according to the World Bank atlas method. Raal *et al*. (2018) found that dyslipidemia was the most prevalent cardiovascular risk factor reported in 70% of each population across the four national income countries. Hypertension prevalence was low in lower income countries and by gender, and was similar in all African countries. Obesity was reported to be more than twice as common in high-income countries compared with low-income countries and by gender, higher prevalence was reported in females than in males (Raal *et al*. 2018). Tobacco use was the least prevalent cardiovascular risk factor and was more common in upper-middle-income and high-income countries than in low-income and lower-middle-income countries.

CVD has accounted for 43% of the global mortality rate (Oduniaya 2016). There is evidence that CVD is on the rise in African patients in South Africa (Sliwa *et al.* 2008); however, these studies have been largely conducted in peri-urban areas. According to Stewart *et al*. (2011, the rate of CVD has accelerated in the rural areas,

mostly due to the epidemiological transition, but herein is the gap in the knowledge, since the prevalence of CVD risk factors in the rural areas remains unexplored.

Effective management and prevention of intermediate CVD risk factors are possible if they are identified early. This makes early screening for modifiable CVD risk factors an important prevention strategy because of its simplicity and cost effectiveness (Hamid *et al*. 2019).

However, in spite of the plethora of studies which demonstrate the effectiveness of prevention strategies in combating CVDs, there is poor awareness and, unavailability, of these services in low- and middle-income countries, especially among young people (Gakidou *et al.* 2017).

2.10 AIM

The aim of the study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department).

2.10.1 OBJECTIVES

- To determine the prevalence of modifiable and non-modifiable cardiovascular risk factors among northern KwaZulu-Natal adults.
- To determine the cardiovascular risk factor prevalence when stratified for age, gender, race and socio-economic status.
- To determine the prevalence of risk factor clustering and metabolic syndrome in this population group.
- To determine the changes which occur in cardiac structure and function using echocardiography.

The researcher systematically sampled participants in the northern KwaZulu-Natal region, presenting to Madadeni Provincial Hospital (Internal Medicine Department) and administered a questionnaire which collected their information on sociodemography, the presence of diabetes and/or HPT, family history of hypercholesterolaemia, family history of fatal cardiovascular events and engagement

in physical activity. Other measurements included blood pressure, weight, height, WC, hip circumference, waist-to-hip ratio and transthoracic echocardiography (2D, M-mode, colour flow Doppler, CW Doppler). All information in this study was strictly confidential and will remain anonymous at all times. A unique research study number was assigned to each patient who signed the consent form and all the details were recorded under that number.

CHAPTER 3: RESEARCH DESIGN AND METHODOLOGY

3.1 RESEARCH STUDY DESIGN

According to Bist (2014), research is a scientific activity to acquire solutions to a specific question or a problem. This research was a quantitative study with a descriptive design. A research design is a plan to systematically solve a research problem (Kothari 2004). A descriptive study approach makes use of surveys or questionnaires to collect data (Bist 2014).

This study used prospective data of patients with cardiovascular risk factor (s) presenting to Madadeni Provincial Hospital (Internal Medicine Department) in 2021.

3.2 SAMPLING STRATEGY

The researcher systematically sampled participants in the northern KZN region, presenting to Madadeni Provincial Hospital (Internal Medicine Department) and administered a World Health Organization (WHO) stepwise approach to chronic disease risk factor questionnaire (Appendix H) which collected their information on socio-demography, alcohol consumption, cigarette smoking, family history of fatal cardiovascular events, presence of diabetes, HPT, dyslipidemia and engagement in physical activity.

Other measurements included blood pressure, blood glucose, weight, height, BMI, WC, hip circumference, waist-to-hip ratio and transthoracic echocardiography (2D, M-mode, colour flow Doppler, pulsed wave (PW) Doppler, continuous wave (CW) Doppler and tissue Doppler imaging).

3.3 SAMPLE SIZE CALCULATION

The sample size is the number of participants included in a study (Noordzij *et al*. 2010). According to Noordzij *et al*. (2010), there is a wide variety of formulas that can be used to calculate the sample size. The sample size was calculated by a

professional statistician. In observing measurements on blood pressure or cholesterol which are regarded as risk factors for CVD, if one considers an effect size of 0.5 (which is regarded as a medium effect size), a 5% level of significance and a power of 80% then a sample size of 200 was adequate.

3.4 PURPOSE OF THE STUDY

The purpose of the study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department).

3.5 OBJECTIVES OF THE STUDY

- To determine the prevalence of modifiable and non-modifiable cardiovascular risk factors among northern KwaZulu-Natal adults.
- To determine the cardiovascular risk factor prevalence when stratified for age, gender, race and socio-economic status.
- To determine the prevalence of risk factor clustering and metabolic syndrome in this population group.
- To determine the changes which occur in cardiac structure and function using transthoracic echocardiography

3.6 STUDY POPULATION

The target population consisted of individuals who had one or more cardiovascular risk factor (s) and who presented to Madadeni Provincial Hospital (Internal Medicine Department) from January 2021 to November 2021.

3.7 STUDY LOCATION

This study was conducted at Madadeni Provincial Hospital which is situated in the Amajuba district in northern KwaZulu-Natal (Figure 3.1). According to the Amajuba district spatial development framework (2019/2020) (Amajuba District Municipality 2020), Amajuba is one of the 10 districts that make up KZN and has a geographical

size of 6 910 km^2. Amajuba district comprises Newcastle, Emadlangeni and Danhauser local municipalities. It has a total population estimated at 531 327 people who are accommodated in 110 963 households. Newcastle has the highest population which is estimated at 389 117 people, followed by Danhauser with 10 541 and lastly Emadlangeni with 36 869.

Figure 3.1: Amajuba district map
Source: Adapted from www.kznonline.gov.za (2 February 2020).

Table 3.1 provides information on the gender profile for the Amajuba district. There has been a growth in a female gender, dominating by 52%; the major reason is that women are economically active, so they migrate to seek employment for better wages.

Table 3.1: A gender profile of the Amajuba district

	Population by gender				
Gender	**Year**	**Newcastle**	**Emadlangeni**	**Danhauser**	**Amajuba**
Male	2001	157171	18323	48661	224155
	2011	172846	17486	48380	238712
	2016	161899	17748	43349	222996
Female	2001	175810	13954	54118	243882
	2011	190390	16956	53781	261127

	2016	227217	19121	6199	308331

Source: Adapted from www.amajuba.gov.za (2 February 2020).

3.8 SELECTION CRITERIA

Inclusion criteria

-Patients who have signed consent form

-Patients who are 18 years and older of any race, gender or ethnic group

-Patients with one or more cardiovascular risk factor (s)

Exclusion criteria

-Patients who do not issue consent

-Patients below the age of 18 years

-Patients with a known history of stroke, coronary heart disease, chronic kidney disease, congestive heart failure, pregnancy, Covid 19 and epilepsy

Withdrawal criteria

Participants were informed in writing in the information letter (Appendix A, A1) and the informed consent form (Appendix B, B1) in the language of their choice that participation in the study was voluntary and that they could, at any given time, withdraw from the study. Their withdrawal from the study would not affect their management and treatment in any way.

3.9 COVID 19 PANDEMIC

Coronavirus disease (Covid 19) is a communicable respiratory illness in humans caused by SARS-Cov 2 virus (WHO 2021). According to the Department of Health (South Africa, Department of Health 2021), this virus outbreak began in Wuhan, China, in December 2019 and it is now a pandemic, affecting all countries worldwide. The National Institute for Communicable Disease (2020) confirmed the first case in South Africa, March 2020 in a 38-year old male who travelled back from Italy with his

wife. Since then, South Africa has been subject to the Covid 19 pandemic with different variants and lockdown restrictions introduced (National Institute for Communicable Disease 2020).

The government introduced lockdown level 5 in March 2020 to control the pandemic, however there was a negative impact on the economy and so was forced to ease the restrictions to level 1 in 2021 (Kollamparambil and Oyenubi 2021). The World Health Organization (2021) reported that Covid 19 protocols must be thoroughly followed and these include:

- Physical distancing: Practicing social distancing at all times, avoiding crowded places like malls, clubs, churches and social gatherings.
- Sanitising hands soap and water, sanitising with an alcohol-based hand rub. Keeping a good hygiene by opening windows, disinfecting surfaces frequently.
- Covering the mouth or nose with a bent elbow when coughing or sneezing.
- Always wear protective clothing like fitted masks. They must cover the nose, mouth and chin.
- If Covid 19-like symptoms develop or you test positive, isolate for 10 days and call the healthcare provider for advice.
- Keep up to date with the latest information on Covid 19 from trusted sources.

3.10 SAFETY PRECAUTIONS TAKEN DURING THE RECRUITMENT PROCESS AND DATA COLLECTION

Safety precautions were followed throughout the research from the time each patient entered the clinic to recruitment process and data collection. This was done to prevent cross infections and infectious agents from spreading among patients and health care workers.

Hand Hygiene: Before attending the patient and after, the healthcare worker washed their hands and the patient sanitised with an alcohol-based hand rub or a sanitiser. All participants complied with infection control practices in the clinic.

The use of personal protective equipment: Healthcare workers involved in this research wore full personal protective equipment (PPE), which included: clean

nonsterile gloves, clean non-sterile fluid-resistant gown, mask and goggles or a face shield.

Sharps safety: All sharp instruments e.g. needles were disposed of in the correct bin after being used.

3.11 THE RECRUITMENT PROCESS

Participants who met the inclusion criteria were selected in 2021; the recruitment process was fair and was based on scientific and ethical principles. No person was excluded on basis of race, gender, sexual orientation, disability, religious beliefs, marital status, ethnic or social origin.

Upon arrival of patients in the morning at 7.30, the study team was introduced by the principal researcher. All screening and advanced measurements information was explained.

Patients were informed that of their decision to participate in the study was entirely voluntary and that they were entitled to withdraw at any point without affecting their medical treatment rendered to them. They were also informed that all information used in the study was strictly confidential and that any data reported in scientific journals or published would not include information identifying them as a patient in the study. Patients were encouraged to ask questions if they did not understand.

Once the informed consent form was signed and issued, a professional nurse who was recruited to be part of the study, performed blood sampling, blood pressure measurement, anthropometric measurements (weight, height, BMI, WC, hip circumference and waist-to-hip ratio). A questionnaire was conducted by the principal investigator (Nokwanda Gambushe).

Transthoracic echocardiography was performed using a Logic V5 expert GE healthcare echocardiography machine with the patients in the left lateral decubitus position. Patients' data was collected to be analysed.

3.12 STORAGE AND DISPOSAL OF HARD AND ELECTRONIC COPIES OF DATA

Research data results were securely stored in a locked cupboard in the Internal Medicine Department. Only the researcher and the supervisor were allowed to have access to the cupboard. Electronic data was stored in computer where it is password-protect. Only the researcher has access to the computer in the Internal Medicine Department. The research data will be stored for five years and will be disposed thereafter. Hard copies will be discarded in the disposable bin.

3.13 ETHICAL CONSIDERATIONS AND INFORMED CONSENT

The study was ethically approved both by the Durban University of Technology ethics committee (Appendix E) (reference number: DUT 170/20) and the Department of Health, KwaZulu-Natal (Appendix D). The study was also approved by the Madadeni Provincial Hospital CEO (Appendix C) and Department of Health: KZN (reference number: KZN-202102-035).

Informed consent was obtained whereby participants wilfully agreed to participate in the study after an explanation in the language of their choice about the study was given. A letter of information (Appendix A, A1) was used to explain the details of the study and participants were informed that their participation was voluntary. Participants were given a consent form (Appendix B, B1) to sign if they wanted to be part of the study. Participants were also informed that they could withdraw at any given stage of the research. Participant's identities were kept strictly confidential and only identified by a unique reference study number.

3.14 CONFIDENTIALITY AND ANONYMITY

All information in this study was strictly confidential and will remain anonymous at all times. A unique research study number was assigned to each patient who signed a consent form and all the details were recorded under that number. The data and research results are stored in a secured place at Madadeni Provincial Hospital, which only the researcher and co-supervisors have access to and will not be shared with any other person.

3.15 RESEARCH PROCEDURES

3.15.1 QUESTIONNAIRE DATA

In order to acquire information of the participant's age, gender, ethnicity, socioeconomic status, vital signs, smoking and alcohol consumption, a World Health Organization (WHO) stepwise approach to chronic disease risk factor questionnaire (Appendix: H) was used. This questionnaire also collects data on the level of physical activity, nutritional status and family history of cardiovascular disease.
Physical activity levels and smoking status was determined based on self-report.

3.15.2 ANTHROPOMETRIC MEASUREMENTS

Anthropometric measurements are usually conducted to assess a patient's nutritional status and normally include body weight, height, BMI and WTHR (Kankana 2017).

3.15.2.1 BODY MASS INDEX

Body weight was measured using a portable, mechanical digital SECA scale and height was measured using a portable stadiometer with participants wearing no shoes and light clothing. Patient's arms were placed sideways, and feet placed together (WHO 2008).

BMI was calculated as weight in kg divided by height in metres squared.

$$BMI = \frac{\text{Weight (kg)}}{\text{Height } (m^2)}$$

A patient is considered obese when the BMI is above 30.0; such a person is at greater risk of developing cardiovascular diseases (WHO 2008) (see Table 3.2).

Table 3.2: Combined recommendations of BMI and WC cut-off points made for overweight or obesity, and association with disease risk

	Body mass index	**Obesity class**	**Disease risk (relative to normal weight and waist circumference)**	
			Men < 102 cm Women < 88 cm	Men > 102 cm Women >88 cm
Underweight	< 18.5			

Normal	18.5–24.9			
Overweight	25.0–29.9		Increased	High
Obesity	30.0–34.9 35.0–39.9	I II	High Very high	Very high Very high
Extreme Obesity	> 40.0	III	Extremely high	Extremely high

Source: WHO (2008).

3.15.2.2 WAIST-TO-HIP RATIO

Waist circumference is a measurement for obesity and is taken at the narrowest point of the waist (Son *et al*. 2016. The patient is considered to be at risk for CVD if the WC for females is > 80cm and for males at > 94 cm (Kankana 2017).

The hip circumference (HC) is taken at the widest lateral extensions of the hips (WHO 2008). The tape is snug without compressing the skin. Measurements are taken at the end of normal expiration. The WC and HC are measured using a tape measure.

The WTHR is the ratio of the waist to the hips circumference to assess obesity A patient is considered obese when the WTHR is above 0.90 for males and above 0.85 for females (Kankana 2017) (Table 3.3).

$$\text{WTHR} = \frac{\text{waist circumference (cm)}}{\text{hip circumference (cm)}}$$

Table 3.3: World Health Organization cut-off points and risk of CVD

Indicator	**Cut-off points**		**Risk of cardiovascular disease**
	Male	Female	
Waist circumference	> 94 cm	> 80 cm	Increased
Waist-to-Hip ratio	≥ 0.90 cm	≥ 0.85 cm	Substantially Increased

Source: (WHO 2008)

3.15.3 BLOOD PRESSURE MEASUREMENT

Williams, Brown and Conlin (2009) point out that blood pressure measurement is usually recommended in any situation that requires assessment of cardiovascular

health, particularly HPT. Blood pressure measurement is accurate when measured in the seated position with the arm at the level of the heart (Joseph 2013). Joseph (2013) further explains that the accuracy of the results depends on body posture, arm size, time, placement of the cuff, device used, technique and the operator.

In the current study a normal cuff size (15-30 cm) was used for a normal arm and a large cuff size (35-45 cm) was used for large arms as recommended by the 2011 South African HPT guideline. A Mindray patient monitor was used to record systolic and diastolic blood pressure. This monitor uses both Korotkoff and oscillometric methods and the combination of these two gives a more accurate reading (Shenzhen Mindray Bio-Medical Electronics 2011)

Blood pressure was measured in the participant's arms, supported at the heart level, after the participant had been seated quietly for at least five minutes in a chair and with both feet on the floor. Blood pressure measurement was taken in both arms twice with three minutes apart. The test was performed in a quiet examination room where the patient was correctly seated and rested. The cuff was placed over a bare arm approximately 2 cm above the elbow crease.

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean blood pressure (MBP) were recorded where the accepted measurement of a systolic blood pressure is < 120 mmHg and a diastolic blood pressure is < 80 mmHg (American Heart Association 2018). Any value above these measurements is an accepted determination of HPT. Heart rate was also recorded.

Table 3.4: Blood pressure classification

Blood pressure category	**Systolic (mmHg)**		**Diastolic (mmHg)**
Normal	Less than 120	and	Less than 80
Elevated	120-129	and	Less than 80
Hypertension stage 1	130-139	or	80-89
Hypertension stage 2	140 or higher	or	90 or higher
Hypertensive crisis	Higher than 180	and/or	Higher than 120

Source: American Heart Association (2018).

3.15.4 BLOOD GLUCOSE MEASUREMENT

Kiechle and Main (2000) state that a blood glucose test is usually performed to determine the concentration of blood glucose. In this study setting, a glucometer ACCU-Check active device was usually used to assess the levels of glucose. This method of measuring blood glucose was chosen because it is fast and easy, with only a single blood sample being required (Goldenberg and Punthakee 2013).

Patients were in a seated position and using a pressure activated safety lancet device, a finger was pricked and a blood sample was placed on a test strip which was already inserted in a glucometer. Diabetic Care Report (2015) reports that there should be enough blood sample on the test strip. The blood glucose levels were recorded in mmol/L.

According to the Diabetic Care Report (2015), the recommended fasting plasma glucose is 7.0 mmol/L or 126 mg/dL. Any value above the recommended value is recorded as diabetic mellitus or impaired fasting glycaemia (Diabetic Care Report 2015). The WHO criteria were used to classify glycaemic categories based on fasting plasma glucose (FPG) (Table 3.5).

Table 3.5: Blood sugar classification

Fasting	**Normal**	**Impaired fasting glycemia (IFG)**	**Diabetes Mellitus**
mmol/L	< 6.1	> 6.1 and < 7.0	> 7.0
Oral glucose tolerance test (OGTT) -2 hour			
mmol/L	< 8.7	> 7.8 and 11.1	> 11.1

Source: World Health Organization (2005)

3.15.5 BIOCHEMICAL ANALYSIS

In patients who have had blood samples taken for the past three months, biochemical analysis was not performed. Their previous results were used in the study. A professional nurse was recruited to perform blood sampling with a sterile syringe from the brachial vein. All samples were immediately taken to the onsite laboratory.

Basic blood analysis included total cholesterol (TC) and triglycerides using a Unicel DxC 660i synchron access clinical system. Blood sampling was performed between 8:00 and 10:00 to avoid delaying the clinic. Blood parameters were performed according to the standard laboratory protocol in order to comply with SASAS accreditation requirement by the principal investigator Mr Mthembu with the assistance of qualified biomedical technologists at Madadeni Hospital.

This study used ranges that are recommended by the Lipid and Atherosclerosis Society of South Africa and South African Heart Association (Table 3.6).

Table 3.6: Lipid profile

Lipid profile	Values
High TC	> 4.5 mmol/L
High triglycerides	> 1.7 mmol/L
Low HDL cholesterol	< 1.0 mmol/L for males < 1.2 mmol/L for females
LDL cholesterol	> 2.5 mmol/L

Source: Daley (2020).

3.15.6 TRANSTHORACIC ECHOCARDIOGRAPHY

To evaluate the parameters of the cardiac structure and function, a transthoracic echocardiography machine was used. Echocardiography is a non-invasive diagnostic test that utilises high frequency ultrasound to evaluate the structural, functional and hemodynamic status of the cardiovascular system. Mocumbi (2012) reveals that the diagnosis and confirmation of CVD in SSA relies on echocardiography, which is rarely available in rural centres. According to Kardys *et al*. (2010), transthoracic echocardiography provides measurements of the left ventricular dimensions (interventricular septum, posterior wall, end diastolic dimension and end systolic dimension), mass, volume, systolic and diastolic function. In view of the lack of evidence in this critical area, the aim of this study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function at Madadeni Provincial Hospital (Internal Medicine Department).

Transthoracic echocardiography was performed according to the standardised protocol (Appendix G) using a Logiq V5 Expert GE healthcare echocardiography

machine. Patients were asked to lie in the left lateral decubitus position with the left arm behind the head and the right arm by the side. This position brings the heart forward to the chest wall (Kaddoura 2009). The adult transducer probe was placed on the patient's anterior chest wall and the ultrasound gel was placed on the transducer probe to ensure good images.

Marwick *et al.* (2015) state that as the heart is the target organ for cardiovascular risk factors, echocardiography measurements of structure and function are very important. All the measurements were measured according to the American Society of Echocardiography by a qualified cardiac technologist (principal investigator).

Relative wall thickness (RWT), left atrium (LA), right atrium (RA), end diastolic dimension (EDD), end systolic dimension (ESD) and left ventricular ejection fraction (LVEF) and diastolic function were measured and assessed.

3.15.6.1 LEFT VENTRICULAR SYSTOLIC FUNCTION

Kaddoura (2009) defines the LVEF as the percentage change in left ventricular volumes in systole and diastole. It is a major prognostic factor in heart failure (Kaddoura 2009). Left ventricular ejection fraction was measured using a leading edge method in a parasternal short axis view at the level of the left ventricular papillary muscles where the normal range is 55% to 70% (Otto 2013). According to Rimington and Chambers (2007), mildly impaired LVEF is 45–54%, while moderately impaired LVEF is 30–44% and severely impaired is < 30%. According to Tissot *et al*. (2018), the M-mode curser is placed perpendicular to the interventricular septum and posterior wall. The posterior wall (PW) and interventricular septum (IVS) were measured. PW and IVS above 11 mm indicate ventricular hypertrophy (Otto 2013).

3.15.6.2 LEFT ATRIAL VOLUME MEASUREMENT

The left atrial volume was calculated using the area-length method. According to Lang *et al*. (2015), the length must be measured from the shorter of the two long axes in the apical 4 chamber or apical 2 chamber (Table 3.7).

Table 3.7: Normal ranges for left atrial area and volume

Standard LA Anterior-Posterior Dimensions		
Parameter	Male	Female
LAd (cm)	3.0-4.0	2.7-3.8
LAd indexed by BSA (cm/m^2)	1.5-2.3	1.5-2.3
LAd, left atrium diameter; *BSA*,body surface area		

Normal ranges and severity cutoff values for LA Area and LA Volume				
Parameter	Normal	Mildly enlarged	Moderately enlarged	Severely enlarged
LA Area (cm^2)	≤ 20	21-30	31-40	≥41
LA Volume indexed by BSA (mL/m^2)	16-34	35-41	42-48	>48
LA, left atrium; *BSA*, body surface area				

Source: Lang *et al.* (2015: 28)

3.15.6.3 RELATIVE WALL THICKNESS

The relative wall thickness (RWT) is defined as the ratio of twice the posterior wall thickness (PWT) and the left ventricular end diastolic dimension (LVEDD) (Marwick *et al*. 2015).

$$RWT=\frac{2xPWTd}{LVEDD}$$

According to Marwick *et al*. (2015), M-mode is used in parasternal short axis view in the left ventricular papillary muscles to measure left ventricular end diastolic dimension (LVEDD), posterior wall thickness (PWT) and interventricular septum (IVS). Increased RWT is said to be present when it is more than 0.42.

Concentric left ventricular hypertrophy is diagnosed when the RWT is > 0.42 and eccentric left ventricular hypertrophy when the RWT is < 0.42 (Marwick *et al*. 2015) (Table 3.8).

Table 3.8: Normal ranges for left ventricular wall thickness

Normal Ranges and Severity Cutoff Values for LV Wall Thickness and Mass Using Linear Method

	Male				Female			
	Normal range	Mildly abnormal	Moderately abnormal	Severely abnormal	Normal range	Mildly abnormal	Moderately abnormal	Severely abnormal
LV mass by linear method								
Septal wall thickness (cm)	0.6–1.0	1.1–1.3	1.4–1.6	>1.6	0.6–0.9	1.0–1.2	1.3–1.5	>1.5
Posterior wall thickness (cm)	0.6–1.0	1.1–1.3	1.4–1.6	>1.6	0.6–0.9	1.0–1.2	1.3–1.5	>1.5
LV mass (g)	88–224	225–258	259–292	>292	67–162	163–186	187–210	>210
LV mass/BSA (g/m^2)	49–115	116–131	132–148	>148	43–95	96–108	109–121	>121

Source: Lang *et al*. (2015: 28)

3.15.6.4 LEFT VENTRICULAR DIASTOLIC FUNCTION

Otto (2013) reports that diastolic function plays a huge role in patients with clinical heart failure. Diastolic dysfunction may be an early sign in patients with CVD (Otto 2013). According to Otto (2013), the normal ventricular inflow pattern is identified by the time interval between an aortic valve closure and beginning of ventricular filling known as the isovolumetric time (IVRT). When the mitral valve opens, blood flows to the ventricles with an early peak velocity (E) of 0.6 to 0.8 m/s. After this maximum velocity, blood flow decreases rapidly with a steep slope known as a deceleration time (DT) and it ranges from 150 to 200 ms (Otto 2013). It is measured from the peak E wave to the baseline (Kaddoura 2019) (Figure 3.2).

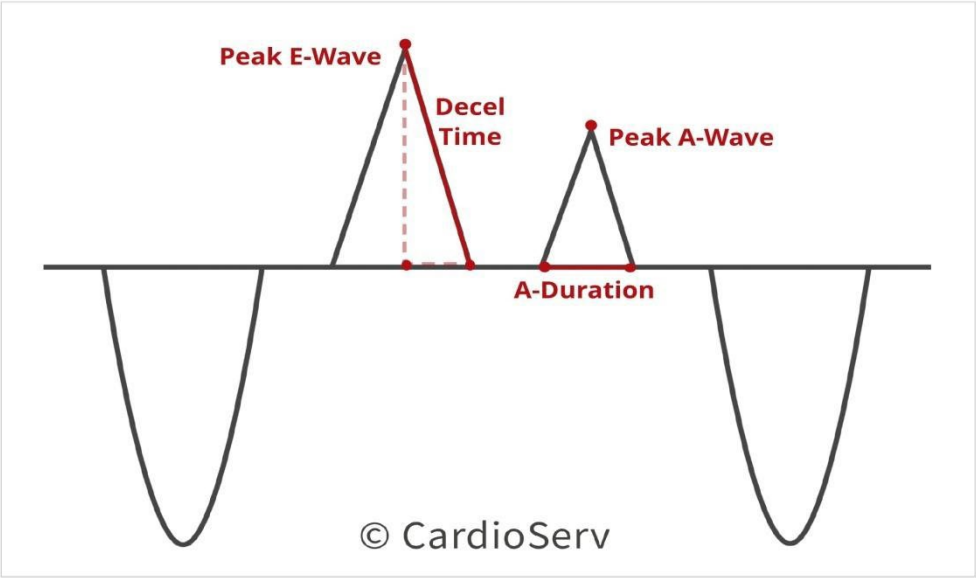

Figure 3.2: Mitral valve inflow pattern
Source: Fields (2020)

Otto (2013) states that as the atrial systole pressure increases and exceeds the ventricular pressure, the late peak A wave is produced. As the ventricular pressure increases during the isovolumetric contraction time (IVCT), the aortic valve is forced to open and ventricular ejection occurs (Otto 2013).

To assess the left ventricular filling, a pulsed wave (PW) Doppler was used in the apical 4-chamber view. A 1-3 mm sample volume was placed between the mitral valve leaflets tips with parallel alignment to inflow. Parameters of left ventricular diastolic function included peak velocity of early (E) and late (A) diastolic filling E/A ratio and the mitral valve deceleration time (DT) (Table 3.9).

Table 3.9: Guideline diagnosis of diastolic dysfunction

LV diastole	E/A ratio	E Deceleration Time (DT)
Normal	0.7-1.5	150-250
Mild dysfunction	< 0.7	> 250
Moderate dysfunction	0.7-1.5	150-250
Severe dysfunction	> 1.5	< 150

Source: Rimington and Chambers (2007)

3.15.6.5 TISSUE DOPPLER IMAGING

Kadappu and Thomas (2015) state that tissue doppler imaging (TDI) is an echocardiographic technique that is used to evaluate myocardial diastolic and systolic function throughout the cardiac cycle (Figure 3.3). The myocardial velocity curve is the same as the transmitral inflow pattern but inverted (Otto 2013). According to Tissot *et al.* (2018), pulsed wave doppler is placed on either basal septal or lateral region and myocardial velocities are calculated.

The first early diastolic velocity is abbreviated as E' (E-prime) following a second peak end diastolic velocity known as the A' (A-prime). The peak systolic annular velocity (S') measures the left ventricular function and has a normal value of > 10 cm/s (Tissot *et al*. 2018).

The normal E'/A' ratio is greater than 1.0. A decreased E'/A' ratio is regarded as left ventricular diastolic dysfunction (Otto 2013).

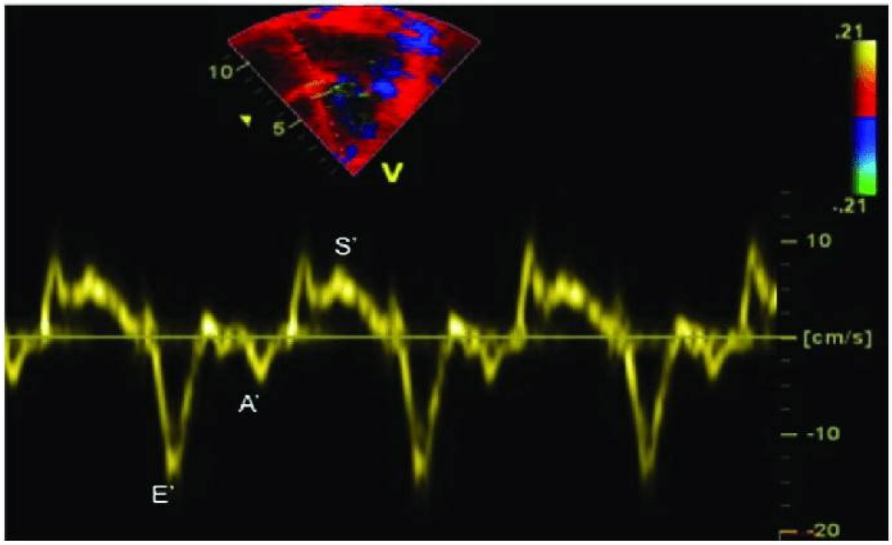

Figure 3.3: Tissue doppler imaging
Source: Singh *et al*. (2018).

In this study, tissue doppler signals were recorded using a pulse wave in an apical 4chamber view, positioned in the basal ventricular septal and at the basal ventricular lateral region (Otto 2013).

E', A', S', E'/A ratio, IVRT and IVCT were all calculated.

3.15.7 METABOLIC SYNDROME

This study used the JIS (2009) criteria to determine the prevalence of metabolic syndrome. Patients with similar cardiovascular risk factors were clustered together, cardiac structure and function findings were identified and reported.

Table 3.10: JIS criteria 2009 for metabolic syndrome

Components	JIS criteria (2009)
Central obesity	Waist circumference (WC) Males > 94 cm and Females >80 cm
Hypertension	SBP > 130 mmHg or DBP > 85 mmHg
Hyperglycaemia	Fasting plasma glucose (FPG) of > 5.6 mmol/L
Dyslipidemia	Triglycerides (TG) of > 1.7 mmol/L

Source: (Motala *et al*. 2011).

3.16 STATISTICAL ANALYSIS

The WHO stepwise approach to chronic disease risk factor questionnaire (Appendix H) was used to record the demographic information (e.g. age, gender, race, education level) and cardiovascular risk factor(s).

All data collected were first entered into Microsoft excel and then transferred to IBM statistical package for social science (SPSS) version 26 for data analysis. The statistical analysis included descriptive statistics, chi-squared test, multinomial logistic regression analysis, frequency tables, bar and pie charts to present the study findings for the demographic and other variables. A *p*-value of less than 0.05 was considered statistically significant. A professional statistician assisted with data analysis.

CHAPTER 4: RESULTS

4.1 INTRODUCTION

The prevalence of cardiovascular risk factor research in a rural setting is scarce, hence a need to contribute to the current body of knowledge. Over the years there have been a number of patients that have presented with cardiovascular risk factors at Madadeni Provincial Hospital Internal Medicine Department (Thembela 2016). To date, there has been no formal account of the actual prevalence of CV risk factors and risk clustering patterns in this district. Furthermore, the effect of CV risk factors on cardiac structure and function has not been studied in this ethnic group in South Africa. With the increased preponderance for cardiomyopathy observed in patients with advanced CV disease, it will be useful to know the features which manifest early, in the subclinical phase.

4.2 AIM

The aim of the study was to determine the spectrum and the effects of cardiovascular risk factors on cardiac structure and function in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department).

4.3 OBJECTIVES

- To determine the prevalence of modifiable and non-modifiable cardiovascular risk factors among northern KwaZulu-Natal adults.
- To determine the cardiovascular risk factor prevalence when stratified for age, gender, race and socio-economic status.
- To determine the prevalence of risk factor clustering and metabolic syndrome in this population group.
- To determine the changes which occur in cardiac structure and function using transthoracic echocardiography

4.4 DEMOGRAPHIC INFORMATION

A total of 202 participants were recruited; however, during the study, two patients were excluded from the study due to chronic kidney failure on investigation, leaving a total of 200 patients for data analysis. The data was captured on an Excel spread sheet and analysed using the IBM statistical package for social science (SPSS) statistics version 26. All patients' data were coded for recording, analysis and reporting, thereby keeping thereby information confidential and anonymous.

Table 4.1 shows that the mean age of the studied group was 50.1 years, with a standard deviation of 16.19 years and a range from 18 to 79 years. There is a reasonable spread of subjects across the arbitrarily assigned age group ranges of 18-25, 26-35, 36-45, 46-55, 56-65, 66-75 and 76-80. The age group 56-65 presented the most with cardiovascular risk factors with 24.0% (p = 0.302), although this was not statistically significant, while the older group of 76-80 had lowest cardiovascular risk factors with 4.0%. There was a high prevalence of black patients (p = 0.606) when compared to other races but was not considered statistically significant.

Table 4.1: Demographic data of participants

Demographic characteristics	Female N = 114	Male N = 86	Total N = 200
Age (Years) 18-25	6	8	14 (7.0%)
26-35	20	14	34 (17.0%)
36-45	16	12	28 (14.0%)
46-55	21	18	39 (19.5%)
56-65	29	19	48 (24.0%)
66-75	18	11	29 (14.5%)
76-80	4	4	8 (4.0%)
Ethnicity Black	85	71	156 (87.0%)
Indian	20	7	27 (13.5%)
White	7	5	12 (6.0%)
Mixed Ancestry	2	3	5 (5.0%)
Marital status Single	54	51	105 (52.5%)
Currently Married	39	29	68 (34.0%)

Widowed	15	5	20 (10.0%)
Divorced	6	1	7 (3.5%)
Level Of Education Completed High School	30	33	63 (31.5%)
Completed Primary School	41	20	61 (30.5%)
Completed University	28	23	51 (25.5%)
No Formal Schooling	15	10	25 (12.5%)
Employment status Employed	24	27	51 (25.5%)
Unemployed	67	42	109 (54.5%)
Self-employed	17	13	30 (15.0%)
Pension	35	23	58 (29.0%
Retired	4	4	8 (4.0%
R350	12	9	21 (10.5%)
Scholar	1	0	1 (0.5%)

4.4.1 GEOGRAPHICAL AREA

The majority of participants resided in Newcastle with 144 (72.0%) followed by Emadlangeni 41 (20.5%), and Danhauser 15 (7.5%). Figure 4.1 shows the geographical area distribution.

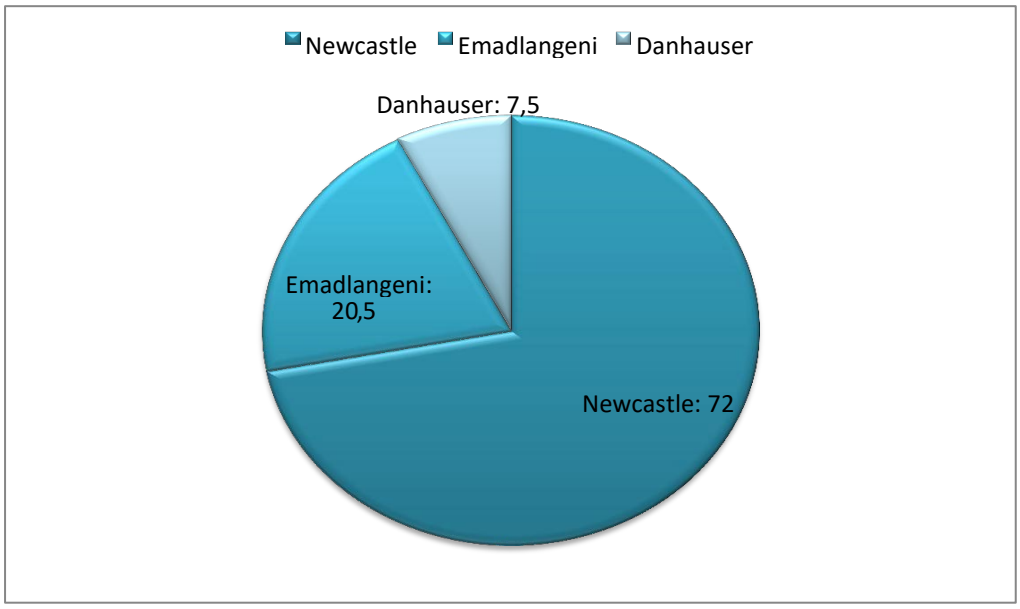

Figure 4.1: Geographical distribution of participants

4.5 BEHAVIOURAL MEASUREMENTS

Table 4.2: Behavioural measurements

	Female	Male	Total	18-25	26-35	36-45	46-55	56-65	66-75	76-80	*P*-value*
Tobacco Use (n)											<0.001/ 0.57
Smoker	30.5%	69.4%	18.0%	2.8%	16.7%	11.1%	30.6%	22.2%	11.1%	5.6%	
Non-smoker	62.8%	37.1%	82.0%	7.9%	17.1%	14.6%	17.1%	24.4%	15.2%	3.7%	
Alcohol use (n)											<0.001/ 0.16
Drinker	24.0%	75.9%	27.0%	11.1%	25.9%	14.8%	20.4%	14.8%	9.3%	3.7%	
Non-drinker	69.1%	30.8%	73.0%	5.5%	13.7%	13.7%	19.2%	27.4%	16.4%	4.1%	
Fruit consumption (n)											0.16/0.58
None	33.3%	66.6%	9.0%	5.6%	16.7%	11.1%	5.6%	38.9%	22.2%	0.0%	
Once in a week	53.6%	46.3%	41.5%	6.1%	15.9%	13.4%	17.1%	24.4%	17.1%	6.1%	
Two times	69.2%	30.7%	32.0%	7.7%	16.9%	23.1%	24.6%	16.9%	9.2%	1.5%	
Three times	52.1%	47.8%	11.5%	8.7%	21.7%	0.0%	21.7%	30.4%	13.0%	4.3%	
Four times	55.5%	44.4%	4.5%	0.0%	22.2%	0.0%	22.2%	22.2%	22.2%	11.1%	
Six times	100.0%	0.0%	0.5%	0.0%	0.0%	0.0%	0.0%	50.0%	0.0%	0.0%	
Seven times	50.0%	50.0%	1.0%	50.0%	0.0%	0.0%	50.0%	0.0%	0.0%	0.0%	
Vegetable consumption (n)											0.31/0.52
None	37.5%	62.5%	4.0%	0.0%	25.0%	0.0%	12.5%	37.5%	25.0%	0.0%	
Once in a week	56.0%	43.9%	41.5%	13.4%	17.1%	15.9%	18.3%	19.5%	12.2%	3.7%	
Two times	53.7%	46.2%	27.0%	3.7%	13.0%	18.3%	22.2%	22.2%	14.8%	5.6%	
Three times	75.0%	25.0%	29.0%	0.0%	25.0%	10.7%	17.9%	28.6%	17.9%	0.0%	
Four times	44.4%	55.5%	8.5%	0.0%	16.7%	11.1%	0.0%	33.3%	11.1%	5.6%	
Five times	66.6%	33.3%	3.5%	0.0%	0.0%	0.0%	50.0%	50.0%	33.3%	16.7%	
Seven times	75.0%	25.0%	2.0%	25.0%	25.0%	0.0%	50.0%	0.0%	0.0%	0.0%	
Physical activity (n) Not physically active	58.3%	41.6%	90.0%	6.1%	15.0%	13.3%	20.6%	25.6%	15.6%	3.9%	0.25/0.09
Physically active	45.0%	55.0%	10.0%	15.0%	35.0%	20.0%	10.0%	10.0%	5.0%	5.0%	
Family history (n)											0.87/0.69
No family history	55.2%	44.7%	71.5%	5.6%	16.8%	13.3%	20.3%	23.8%	16.8%	3.5%	
Family history	61.4%	38.5%	28.5%	10.5%	17.5%	15.8%	17.5%	24.6%	8.8%	5.3%	
Mental health (n)											0.13/0.94
Not depressed	61.9%	38.0%	63.0%	7.1%	15.1%	14.3%	18.3%	25.4%	15.9%	4.0%	
Depressed	48.6%	51.3%	37.0%	6.8%	20.3%	13.5%	21.6%	21.6%	12.2%	4.1%	

There were 164 (82.0%) of the enrolled participants who were non-smokers. Table 4.2 shows that, among 200 participants, more men were current smokers than women (p = 0.129) although this was not statistically significant. There were 164 (82.0%) of the enrolled participants who were non-smoker. The rate of alcohol consumption was highest in the young adult age group 26-35 (p < 0.042) with 25.9% surveyed in both males and females and this was statistically significant. The older age group (76-80) consumed the least alcohol with 3.7%. Most men and women did not engage in any level of physical activity while 20 (10.0%) did engage in physical activity (p = 0.065) but this was not statistically significant.

The prevalence of family history of cardiovascular risk factors in their first and second-degree relative revealed that 143 (71.5%) participants had no family history of CVD (p < 0.001) and this was statistically significant. A family history of CVD was reported in 57 (28.5%) participants.

Regarding their mental health, this research shows that 74 (37.0%) of the participants were stressed and depressed while 126 (63.0%) had a normal mental status. Self-reported depression was mostly reported in the age groups 46-55 and 56-65 (p < 0.001) and this was statistically significant. The older age group of 76-80 were less depressed with 4.0%.

4.6 ANTHROPOMETRY, BLOOD PRESSURE AND BIOCHEMICAL MEASUREMENTS

Table 4.3: Anthropometry, blood pressure and biochemical measurements.

Characteristics	Female	Male	Total	18-25	26-35	36-45	46-55	56-65	66-75	76-80	p-value*
Mean HR	82.17	82.5	82.3	81.7	84.3	79.9	82.6	85.1	79.5	75.1	0.86/0.61
Normal BMI (kg/m^2)	34	42	76	28.1	28.6	29.0	30.3	30.5	28.7	30.0	0.02
Overweight (kg/m^2)	33	20	53	3	10	5	17	4	11	3	
Obese (kg/m^2)	31	14	45	10	16	2	7	3	6	1	
Morbid obesity (kg/m^2)	17	9	26	9	5	2	6	2	1	1	
Mean waist circ (cm)	40.0	40.8	40.4	34.4	38.5	40.8	40.8	42.2	40.8	42.6	0.64/0.34

Mean hip circ (cm)	42.9	43.2	43.0	37.2	42.0	43.1	42.9	44.8	43.4	46.6	0.84/0.84
Mean W-H ratio (cm)	0.91	0.92	0.91	0.91	0.90	0.92	0.93	0.92	0.92	0.91	0.38/0.65
Mean systolic BP (mmHg)	148.6	156.7	152.6	149.2	149.6	158.6	149.4	150.0	150.4	176.0	0.07/0.37
Mean diastolic BP (mmHg)	87.5	92.8	90.1	86.5	89.3	92.7	91.0	88.4	88.7	93.8	0.06/0.94
Hypertension (n)	32	31	63	3	14	9	17	8	10	2	0.23
Diabetes mellitus (n)	21	13	34	5	8	5	4	3	8	1	0.54
Mean TC (mmol/L)	6.8 n=17	6.7 n=14	6.7	6.1	6.3	6.6	6.6	7.4	6.1	6.8	0.91
Mean triglycerides (mmol/L)	3.2 n=17	3.0 n=14	3.1	3.3	2.8	4.0	3.0	2.8	2.9	2.5	0.55
Dyslipidemia (n)	1	3	4	1	0	1	0	1	1	0	0.34

The mean heart rate for both men and women was within normal limits and the variation among the different age groups was minimal. Among the participants, females presented with greater BMI numbers compared to males ($p < 0.020$) and this was statistically significant. The overall prevalence of HPT in the study was 63 (31.5%) and was more prevalent in women ($p = 0.340$) but this was not statistically significant.

Dyslipidemia was detected with other cardiovascular risk factors. The prevalence of dyslipidemia with diabetes mellitus, HPT and obesity was the most prevalent with 12 (6.0%), and this was statistically significant ($p < 0.001$) (Figure 4.2).

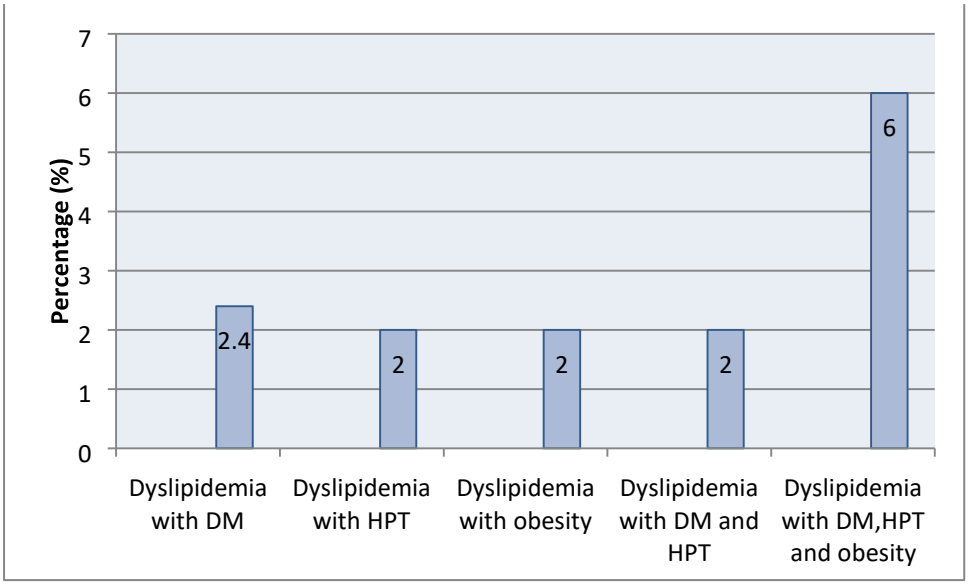

Figure 4.2: Dyslipidemia with other cardiovascular risk factors

4.7 BACKGROUND KNOWLEDGE ON HYPERTENSION AND DIABETES

All 200 participants were well informed and have received lifestyle advice before from a health care worker on reducing salt intake, losing weight, stopping smoking and increasing physical activity. All participants reported to have been checked previously by a health care worker for blood pressure and blood sugar. All participants had a good knowledge about HPT and diabetes.

Among the participants, 3 (1.5%) had consulted traditional healers about their HPT or diabetes. Six (6) (3.0%) had taken traditional remedies to try and manage HPT and diabetes and all were blacks, with 1.5 % being males and 1.5% being females, all being over the age of 40 years.

4.7.1 TRANSTHORACIC ECHOCARDIOGRAPHY

Table 4.4 shows the baseline demographic and echocardiographic clinical characteristics of participants.

Table 4.4: Baseline demographic and echocardiographic clinical characteristics of participants

WOMEN						
26-35	36-45	46-55	56-65	66-75	76-80	*P*-value*
86.2 (12.6)	76.0 (11.2)	81.2 (17.8)	82.3 (17.9)	82.2 (15.5)	72.5 (10.6)	<0.001
39.9 (17.6)	40.0 (7.0)	41.7 (8.0)	39.7 (4.3)	39.7 (4.2)	44.0 (7.8)	<0.001
43.3 (14.9)	42.5 (5.5)	44.2 (6.0)	42.9 (4.6)	42.2 (3.8)	47.2 (8.0)	<0.001
29.0 (8.4)	30.9 (8.7)	32.4 (8.4)	30.8 (6.6)	30.7 (6.5	28.9 (8.4)	<0.001
148.6 (27.9)	161.7 (37.9)	142.9 (24.7)	143.9 (31.3)	142.5 (23.5)	169.0 (39.7)	<0.001
88.16.2	93.0 (20.0)	86.8 (17.6)	88.0 (18.9)	79.6 (15.1)	94.5 (10.2)	<0.001
32.8 (4.6)	30.5 (4.5)	33.3 (4.8)	34.1 (5.1)	32.6 (7.0)	31.2 (5.7)	0.480
28.7 (4.0)	27.8 (4.9)	30.2 (4.5)	29.2 (3.7)	28.1 (6.0)	29.5 (2.0)	0.466
10.3 (1.8)	10.2 (2.3)	10.9 (2.8)	9.6 (2.1)	9.5 (2.1)	10.0 (1.8)	0.126
9.9 (1.8)	9.8 (2.2)	10.2 (2.5)	9.2 (1.8)	9.3 (2.3)	9.5 (2.6)	0.268
14.0 (34.0)	10.0 (28.0)	22.0 (39.0)	11.0 (48.0)	8.0 (29.0)	4.0 (8.0)	0.916
0.45(0.09)	0.42 (0.10)	0.43 (0.12)	0.37 (0.10)	0.43 (0.16)	0.50 (0.33)	0.314
44.4 (0.8)	0.46 (5.2)	47.4 (0.8)	49.6 (8.2)	44.8 (7.5)	42.7 (13.7)	0.232
29.6 (6.5)	30.8 (5.6)	32.9 (8.4)	33.9 (9.5)	29.9 (6.1)	29.2 (7.5)	0.187
33.0 (4.6)	32.6 (3.7)	31.3 (7.5)	31.6 (7.7)	33.1 (5.1)	36.7 (2.9)	0.175
61.9(7.1)	61.4 (5.3)	58.4 (12.0)	59.1 (12.8)	61.7 (7.2)	66.5 (4.2)	0.070
0.8 (0.2)	1.0 (0.3)	1.1 (0.4)	0.7 (0.23)	0.8 (0.3)	0.8 (0.2)	0.098
0.08 (0.02)	0.1 (0.02)	0.11 (0.03)	0.08 (0.02)	0.09 (0.02)	0.08 (0.02)	<0.000
0.09 (0.02)	0.08 (0.02)	0.10 (0.03)	0.1 (0.02)	0.09 (0.02)	0.09 (0.02)	<0.000
0.08 (0.01)	0.07 (0.01)	0.08 (0.01)	0.08 (0.01)	0.07 (0.01)	0.07 (0.00)	0.145
0.8 (0.12)	0.9 (0.4)	1.1 (0.4)	0.7 (0.2)	0.8 (0.2)	0.6 (0.4)	<0.000
93.5 (24.7)	91.8 (32.1)	78.4 (25.1)	96.3 (26.9)	83.2 (17.6)	96.7 (41.1)	<0.000
80.1 (11.9)	79.0 (16.8)	74.3 (20.0)	78.7 (11.3)	79.1 (9.6)	85 (8.4)	0.401
0.1 (0.02)	0.10 (0.03)	0.12 (0.04)	0.09 (0.02)	0.09 (0.02)	0.09(0.02)	<0.001
0.1 (0.02)	0.09 (0.02)	0.11 (0.32)	0.11 (0.03)	0.10 (0.02)	0.10 (0.02)	<0.000
0.08 (0.01)	0.08 (0.00)	0.10 (0.26)	0.09 (0.02)	0.8 (0.01)	0.08 (0.01)	0.073
0.8 (0.2)	1.0 (0.3)	1.1 (0.4)	0.7 (0.2)	0.9 (0.2)	0.9 (0.2)	<0.001
93.8 (24.20)	85.5 (27.9)	79.1 (25.1)	93 0 (25.3)	88.0 (20.9)	91.5 (37.5)	<0.000
80.9 (11.8)	79.0 (14.9)	73 (17.4)	81 1 (13.1)	79.8 (10.8)	81.2 (15.6)	0.061
30.9 (6.4)	27.7 (6.7)	29.9 (6.2)	32.4 (8.9)	29.5 (7.0)	29.2 (12.6)	<0.000
15 (75.0)	12 (75.0)	16 (76.2)	16 (55.2)	12 (66.7)	3 (75.0)	<0.001

(23.8) 13 (44.8) 6 (33.3) 5 (25.0) (25.0) <0.001

MEN				
46-55	56-65	66-75	76-80	P-value
81.2 (17.8)	82.3 (17.9)	82.2 (15.5)	77.7 (11.3)	<0.001
41.7 (8.0)	39.7 (4.3)	39.7 (4.2)	41.2 (5.9)	<0.001
44.2 (6.0)	42.9 (4.6)	42.2 (3.8)	46.0 (9.7)	<0.001
32.4 (8.4)	30.8 (6.6)	30.7 (6.5)	31.0 (8.3)	<0.001
142.9 (24.7)	143 (31.3)	142.5 (23.5)	182.0 (47.9)	<0.001
86.8 (17.6)	88.0 (18.9)	79.6 (15.1)	93.2 (18.9)	<0.001
33.7 (5.2)	35.4 (4.7)	32.0 (7.8)	33.5 (8.1)	0.464
32.2 (5.0)	29.7 (4.4)	27.6 (5.1)	29.5 (3.3)	0.435
12.6 (3.0)	9.6 (2.0)	10.2 (3.6)	12.0 (2.1)	0.109
11.8 (3.6)	9.1 (2.1)	10.6 (4.1)	11.5 (3.3)	0.268
17.0 (22.0)	37.0 (11.0)	21.0 (29.0)	4.0 (4.0)	0.919
0.5 (0.2)	0.3 (0.1)	0.4 (0.2)	0.5 (0.1)	0.355
46.3 (6.3)	48.7 (9.7)	45.9 (8.9)	44.0 (6.3)	0.247
31.6 (6.0)	34.2 (9.8)	30.7 (8.1)	28.7 (4.1)	0.164
30.8 (5.5)	31.1 (7.6)	30.5 97.4)	33.2 (1.7)	0.193
58.1 (8.7)	57.9 (11.7)	57.9 (12.7)	62.0 (2.4)	0.099
0.9 (0.4)	0.9 (0.2)	0.8 (0.2)	0.7 (0.3)	0.139
0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	<0.000
0.0 90.0)	0.0 (0.0)	0.0 (0.0)	0.1 (0.0)	<0.000
0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.215
0.9 (0.5)	0.9 (0.2)	0.8 (0.20	0.6 (0.2)	<0.001
82.7 (28.0)	82.0 (20.8)	81.8 (16.8)	96.2 (41.0)	<0.000
74.8 (16.3)	74.8 (12.6)	80.5 (10.9)	76.7 (10.8)	0.486
0.1 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	<0.000
0.0 (0.0)	0.0 (0.00	0.0 (0.0)	0.1 (0.0)	<0.000
0.0 (0.0)	0.0 (0.00	0.0 (0.0)	0.0 (0.0)	<0.018
0.9 (0.4)	0.9 (0.3)	0.7 (0.4)	0.7 (0.1)	<0.000
85.3 (27.0)	82.9 (21.0)	80.5 (17.6)	90.5 (33.6)	<0.000
71.2 (15.3)	80.1 (13.0)	76.7 (11.4)	78.7 (18.0)	<0.024
30.5 (8.1)	27.2 (6.1)	26.7 (5.9)	34.0 (5.9)	<0.000
14 (77.8)	17 (89.5)	10 (90.9)	2 (50.0)	<0.001
4 (22.2)	2 (10.5)	1 (9.1)	2 (50.0)	<0.001

Mean ,(SD)	
	18-25
Age (years)	
HR	93.6 (26.0)
Waist circ	35.0 (7.45)
Hip circ	37.6 (8.0)
BMI	29.0 (6.72)
SBP	160.8 (30.9)
DBP	89.5 (13.3)
LA	31.5 (4.5)
AO	26.0 (4.2)
IVSD	10.1 (2.3)
PW	9.6 (2.7)
LVH	5.0 (14.0)
RWT	0.44 (0.1)
LVEDD	4.1 (5.6)
LVESD	27.3 (3.7)
FS	35.3 (4.0)
EF	65.1 (5.0)
E/A ratio	0. 9 (0.3)
Medial e	0.09 (0.03)
Medial a	0.1 (0.01)
Medial s	0.07 (0.01)
Medial e/a ratio	0.9 (0.4)
Medal IVRT	94.8 (27.4)
Medial IVCT	83.8 (10.3)
Lateral e	0.9 (0.02)
Lateral a	0.1 (0.02
Lateral s	0.8 (0.00)
Lateral e/a ratio	1.0 (0.38)
Lateral IVRT	97.1 (22.7)
Lateral IVCT	87.0(8.0)
LA volume	29.1 (7.7)
Normal D F n (%)	4 (66.7)

Mean (SD)			
	18-25	26-35	36-45
Age (years)			
HR	72.9 (19.1)	86.2 (12.6)	76.0 (11.2)
Waist circ	34.0 (10.2)	39.8 (17.6)	40.0 (7.0)
Hip circ	36.8 (9.3)	43.3(14.9)	42.5 (5.5)
BMI	27.4 (12.7)	29.0 (8.4)	30.9 (8.7)
SBP	140.6 (31.1)	148 (27.9)	161.7 (37.9)
DBP	84.2 (14.1)	88.3 (16.6)	93.0 (20.0)
LA	33.1 (4.5)	32.0 (6.3)	34.6 (7.8)
AO	29.7 (4.5)	30.2 (4.7)	30.7 (3.0)
IVSD	9.8 (3.0)	11.3 (2.5)	10.1 (3.3)
PW	10.3 (3.1)	10.8 (2.6)	10.1 (3.2)
LVH	9.0 (14.0)	20.0 (34.0)	18.0 (10.0)
RWT	0.4 (0.2)	0.4 (0.1)	0.4. (0.1)
LVEDD	47.5 (11.0)	48.0 (8.4)	48.3 (5.8)
LVESD	32.1 (10.3)	32.5 (9.6)	30.2 (5.8)
FS	33.1 (7.9)	31.0 (8.0)	33.1 (4.4)
EF	61.2 (12.30	58.0 (12.8)	61.5 (6.4)
E/A ratio	1.1 (0.2)	1.0 (0.2)	1.2 (0.50
Medial e	0.1 (0.0)	0.0 (0.0)	0.0 (0.00
Medial a	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
Medial s	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
Medial e/a ratio	1.0 (0.2)	1.0 (0.2)	1.1 (0.4)
Medal IVRT	75.5 (10.60	77.3 (18.3)	81.9 (22.3)
Medial IVCT	73.3 (10.9)	74.2 (7.3)	73.0 (13.8)
Lateral e	0.1 (0.0)	0.1 (0.0)	1.1 (0.0)
Lateral a	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
Lateral s	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
Lateral e/a ratio	1.1 (0.2)	1.0 (0.2)	1.1 (0.3)
Lateral IVRT	80.0 (11.7)	78.0 (18.6)	82.0 (19.7)
Lateral IVCT	77.8 (5.9)	75.8 (10.7)	74.3 (12.1)
LA volume	26.1 (4.9)	27.8 (5.0)	28.9 (7.0)
Normal D F n (%)	8 (100)	12 (85.7)	8 (66.7)
Diastolic dysfunc	0 (0)	2 (14.3)	4 (33.3)

As can be seen from Table 4.4, the systolic ventricular function was normal in 87.0% of the participants. Eccentric remodelling was more prevalent in patients with left ventricular systolic dysfunction with 42.3%, followed by concentric left ventricular hypertrophy with 26.9% and eccentric left ventricular hypertrophy with 11.5%. The prevalence of diastolic dysfunction was 25.5% with women a statistically significantly higher proportion (70.5%) than men (29.4%). Black Africans presented more with diastolic dysfunction when compared with other races. Diastolic dysfunction was present in 22.2% of hypertensive participants and 8.8% of diabetes mellitus patients. The prevalence of a normal left ventricular geometrical pattern was 51.5% and this was the highest of all LV geometrical patterns. Concentric hypertrophy was the second highest with 36.6%, followed by eccentric remodelling with 6.5%, concentric remodelling (5.5%), and lastly eccentric hypertrophy (0.5%). Concentric hypertrophy was statistically significantly higher in males with 43.0%. Decreased E/A ratio, prolonged IVRT and increased LA volume were noted in 25.5% of the studied population and this was higher in women compared to men.

4.7.2 VENTRICULAR SYSTOLIC FUNCTION

The systolic ventricular function was normal in 174 (87.0%) participants and abnormal in 26 (13.0%), (p = 0.009), and this was statistically significant. Of the 26 participants with impaired left ventricular function, 42.3% were hypertensive, 19.2% had both HPT and diabetes mellitus, 15.3% had HPT, diabetes mellitus and obesity, 7.6% had diabetes mellitus and obesity, and 3.8% had diabetes mellitus, HPT, and obesity, and 3.8% had diabetes mellitus and dyslipidemia (Figure 4.3 and Table 4.5).

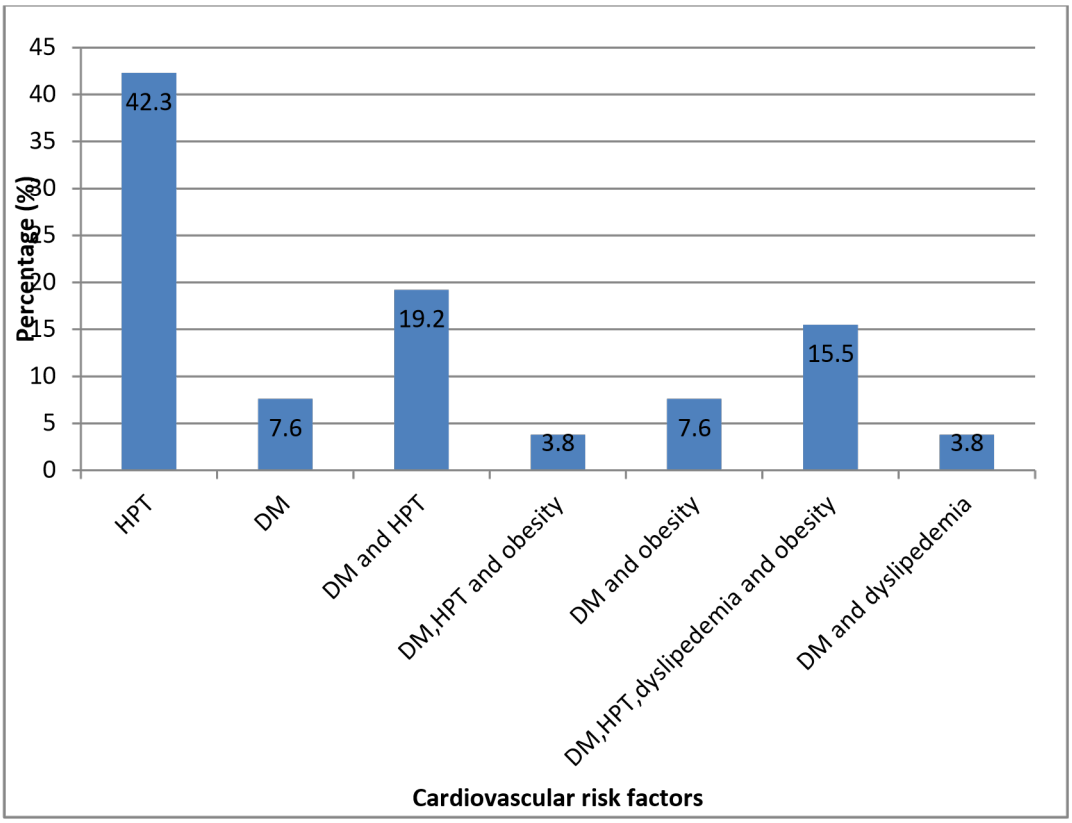

Figure 4.3: Left ventricular systolic dysfunction by cardiovascular risk factors

Table 4.5: Left ventricular systolic dysfunction with left ventricular geometry

	Normal N= 200	Concentric remodelling	Concentric hypertrophy	Eccentric hypertrophy	Eccentric remodelling	*P*-value
LV systolic dysfunction Abnormal (row)	20 39.2%	1 2%	25 49%	0 0%	5 9.8%	0.08

From Figure 4.4 one can note that 65.3% had normal diastolic function, 23.0% presented with mild diastolic dysfunction while 11.5% presented with severe left ventricular diastolic dysfunction.

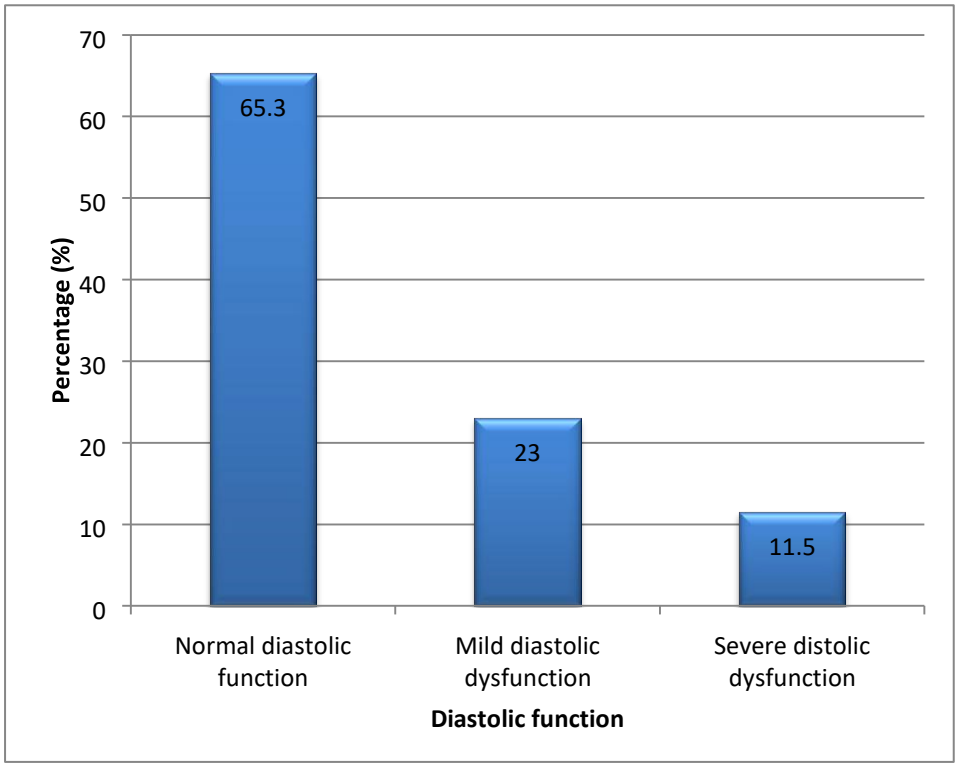

Figure 4.4: Diastolic function in left ventricular systolic dysfunction participants

4.7.3 LEFT VENTRICULAR DIASTOLIC FUNCTION

The prevalence of diastolic dysfunction in this study was 51 (25.5%), with women presenting with a statistically significantly higher proportion (36, 70.5%) than men (15, 29.4%) ($p < 0.023$). The majority (149, 74.5%) had normal diastolic function. The 56-65 age group was mostly affected with diastolic dysfunction with a prevalence of 15 (29.4%) ($p = 0.798$), but this was not statistically significant.

Blacks presented more with diastolic dysfunction when compared with other races with 19.0%, followed by Indians with 3.5% and whites with 2.5%. The lowest were mixed ancestry with 0.5% ($p = 0.606$). This was not statistically significant.

Table 4.6 shows the cardiovascular risk factors associated with left ventricular diastolic function

Table 4.6: Cardiovascular risk factors by left ventricular diastolic function

	Normal diastolic function	**Diastolic dysfunction**	**Total**	***P*-value***

Tobacco smoking (n)	25	11	36	0.129
Alcohol user (n)	42	12	54	0.30
Physical inactivity (n)	133	47	180	0.657
Family history (n)	42	15	57	0.86
Psychologically stressed (n)	54	20	74	0.70
BMI				0.298
Normal (n)	61	15	76	
Overweight (n)	39	14	53	
Obese (n)	33	12	45	
Morbid obesity (n) Risk factors	16	10	26	0.028
Hypertension (%)	49	14	63	
Diabetes mellitus (%)	32	3	35	
Other combinations (DM, HPT, Dyslipidemia and Obesity)	37	17	54	

4.7.4 LEFT VENTRICULAR GEOMETRY

The prevalence of a normal left ventricular geometry was 103 (51.5%), which was the highest among all the LV geometrical patterns (p = 0.139). Concentric hypertrophy was the second highest with 72 (36.0%), followed by eccentric remodelling with 13 (6.5%), concentric remodelling with 11 (5.5%) and eccentric hypertrophy 1 (0.5%). Concentric hypertrophy was more prevalent in the age group 46-55 with 19 (48.7%). Eccentric remodelling was more prevalent in the age group 56-65 with 14.5%, (p < 0.042) and this was statistically significant.

Table 4.7: Left ventricular geometry by race

		Race				Total	*P*-value*
		African	Indian	Mixed ancestry	White		
LV geometry	Concentric hypertrophy (n)	58	9	3	2	72	0.077
	Concentric remodelling (n)	8	3	0	0	11	
	Eccentric hypertrophy (n)	1	0	0	0	1	

	Eccentric remodelling (n)	8	1	0	4	13	
	Normal (n)	81	14	2	6	103	
Total		156	27	5	12	200	

The race versus LV geometry association (Table 4.7) is not significant at a 5% level (*p*-value = 0.077). Within the Africans, 58 out of 156 (37%) presented with concentric hypertrophy. Within the category white, 2 out of 12 (16.7%) presented with concentric hypertrophy. Eight out of 156 Africans (5.1%) presented with concentric remodelling and eccentric remodelling. Within the Indian race group, concentric hypertrophy had the highest prevalence with 9 out of 27 (33.3%).

Table 4.8: Cardiovascular risk factors by left ventricular geometry

Cardiovascular risk factors	Normal	Concentric hypertrophy	Concentric remodelling	Eccentric hypertrophy	Eccentric remodelling	*P*-value*
Tobacco smokers (n)	16	15	0	0	5	0.13
Alcohol users (n)	25	23	2	1	3	0.34
Family history (n)	23	24	5	0	5	0.26
Psychologically stressed (n)	35	32	5	0	2	0.23
BMI category (col%)	Normal	Concentric hypertrophy	Concentric remodelling	Eccentric hypertrophy	Eccentric remodelling	*P*-value*
Normal	39 51.3%	29 38.2%	3 3.9%	1 1.3%	4 5.3%	0.02*
Overweight	31 58.5%	18 34%	2 3.8%	0 0%	2 3.8%	
Obesity	26 57.8%	16 35.6%	2 4.4%	0 0%	1 2.2%	
Morbid obesity	7 26.9%	9 34.6%	4 15.4%	0 0%	6 23.1%	
Hypertension (col%)	23 36.5%	31 49.2%	5 7.9%	1 1.6%	3 4.8%	0.026*
Diabetes Mellitus (%)	25 73.5%	6 17.6%	3 5.9%	0 0.0%	1 2.9%	0.069
Dyslipidemia (%)	75.0%	25.0%	0.0%	0.0%	0.0%	0.306

Table 4.8 shows that the association between BMI category and LV geometry is significant (*p*-value = 0.02).

Within the morbid obese category, 7 (26.9%) have normal LV geometry, 9 (34.6%) have concentric hypertrophy, 4 (15.4%) have concentric remodelling, while 6 (23.1%) have eccentric remodelling.

The association between Hypertension and LV geometry is also significant (p -value = 0.026). Of the 63 with HPT, 31 (49.2%) present with concentric hypertrophy.

4.7.5 TISSUE DOPPLER IMAGING

Decreased E'/A' ratio, prolonged IVRT and increased LA volume was noted in 51 (25.5%) of the studied population and this was more prevalent in females than males (p < 0.023), which was statistically significant.

4.7.5.1 MEDIAL MEASUREMENTS

The mean medial e' was 0.9, the mean a' medial was 0.9, the mean medial s' was 0.7 and the mean medial e'/a' was 0.9.

The IVRT medial mean was 86.19 and IVCT medial mean was 77.2.

4.7.5.2 LATERAL MEASUREMENTS

The mean lateral e' was 0.10, the mean lateral a' was 0.10, the mean lateral s' was 0.8 and the mean lateral e'/a' was 0.9.

The IVRT lateral was 86.0 and IVCT lateral mean was 77.9 and was greater in women when compared to men (p < 0.023) which was statistically significant. The age group 56-65 presented more with prolonged IVRT when compared to the other group ages (p = 0.798) but this was not statistically significant.

4.8 METABOLIC SYNDROME

This study used the JIS (2009) criteria to determine the prevalence of metabolic syndrome where central obesity, HPT, hyperglycaemia and dyslipidemia were clustered. The overall prevalence of metabolic syndrome on the studied population was 12 (6.0%). Males were more prevalent with 58.3% and females with 41.6%, $p < 0.041$ which was statistically significant (Table 4.9).

Table 4.9: Metabolic syndrome by left ventricular geometry

Left ventricular geometry	Normal	Concentric hypertrophy	Eccentric remodelling	Eccentric hypertrophy	Concentric remodelling	*P*-value*
Men n = 86	40	35	7	1	2	0.139
Women n = 114	63	37	6	0	9	
Total (n)	103	72	13	1	11	
Metabolic syndrome (%)	16.6%	33.3%	3.0%	0.0%	25.0	

Metabolic syndrome participants had mostly normal diastolic function with 75.0% ($p < 0.001$) and this was statistically significant. Only 25.0% of metabolic syndrome presented with diastolic dysfunction.

Table 4.10: Metabolic syndrome by left ventricular diastolic function

Diastolic function	Normal	Abnormal	*P*-values
Metabolic syndrome (%)	75.0%	25.0%	<0.001

4.9 MULTINOMIAL LOGISTIC REGRESSION ANALYSIS

A multinomial logistic regression model was fitted with the dependent variable being the left ventricle geometry. This variable had five categories namely concentric hypertrophy, concentric remodelling, eccentric remodelling, eccentric hypertrophy and normal. Since eccentric hypertrophy only had one observation, this category was excluded in this model. The reference category for the LV geometry was the category “normal”. The independent variables were age, gender, BMI category, SBP, DBP, risk factors (HPT, DM and HPT/DM, combinations of HPT, DM, obesity and dyslipidemia) and diastolic function. The category for the variable risk factors was

defined as follows: risk factor = 1 is HPT, risk factor = 2 is diabetes mellitus (DM), risk factor = 3 is HPT and diabetes mellitus (HPT/DM) and risk factor = 4 is any combination of dyslipidemia, HPT, DM and obesity. The results for the multinomial logistic regression model are given in Table 4.11 below.

When comparing risk factors for LV geometry concentric hypertrophy to the category normal, the significant variable was diastolic function (*p*-value = 0.015). (Note that the categories for diastolic function were: diastolic function = 1 is normal and diastolic function = 0 is abnormal.) Furthermore, keeping the other variables in the model constant, the odds of concentric hypertrophy rather than the category normal among those whose diastolic function was abnormal was 2.6 times the odds of being concentric hypertrophy rather than normal, for those whose diastolic function was normal.

When comparing risk factors for concentric remodelling to the category normal, the significant variable was SBP (systolic blood pressure), (*p*-value = 0.057) and BMI category (*p*-value = 0.015). Keeping other variables in the model constant, being in the category morbid obesity led to the odds of concentric remodelling compared to normal being 23 times that for those having BMI category normal.

When comparing the risk of eccentric remodelling to the category normal, the significant variables were BMI category (*p*-value = 0.002) and HPT (*p*-value = 0.016). Keeping other variables in the model fixed, the presence of HPT led to the odds of eccentric modelling rather than normal being 14.5 times the category with combinations of HPT, DM, dyslipidemia and obesity (the reference category).

Table 4.11: Parameter estimates (B), odds ratio (OR) in the multinomial logistic regression model.

lv_geometry[a]		B	Std. Error	Wald	df	Sig.	OR = Exp(B)
concentric hypertrophy	Intercept	.752	1.186	.401	1	.526	
	age	-.014	.011	1.797	1	.180	.986
	SBP	-.007	.009	.577	1	.447	.993
	DBP	.008	.013	.369	1	.543	1.008
	[BMI category=morbid obesity]	.626	.668	.880	1	.348	1.870
	[BMI category=obese]	-.136	.461	.086	1	.769	.873
	[BMI category=overweight]	-.261	.426	.375	1	.540	.771
	[BMI category=znormal]	0[b]	.	.	0	.	.
	[diastolic_function=0]	.965	.395	5.964	1	.015	2.625
	[diastolic_function=1]	0[b]	.	.	0	.	.
	[gender=female]	-.624	.351	3.168	1	.075	.536
	[gender=male]	0[b]	.	.	0	.	.
	[risk_factors=1,00]	.794	.502	2.504	1	.114	2.213
	[risk_factors=2,00]	-.985	.632	2.434	1	.119	.373
	[risk_factors=3,00]	-.243	.507	.231	1	.631	.784
	[risk_factors=4,00]	0[b]	.	.	0	.	.

	lv_geometry[a]	B	Std. Error	Wald	df	Sig.	OR = Exp(B)
concentric remodelling	Intercept	.144	2.723	.003	1	.958	
	age	-.016	.023	.443	1	.506	.984
	SBP	-.046	.024	3.614	1	.057	.955
	DBP	.038	.029	1.722	1	.189	1.039
	[BMI category=morbid obesity]	3.142	1.293	5.900	1	.015	23.139
	[BMI category=obese]	.155	1.019	.023	1	.879	1.168
	[BMI category=overweight]	-.110	1.012	.012	1	.913	.896
	[BMI category=znormal]	0[b]	.	.	0	.	.
	[diastolic_function=0]	-.938	1.226	.586	1	.444	.391
	[diastolic_function=1]	0[b]	.	.	0	.	.
	[gender=female]	.899	.854	1.108	1	.293	2.456
	[gender=male]	0[b]	.	.	0	.	.
	[risk_factors=1,00]	1.526	1.198	1.623	1	.203	4.600
	[risk_factors=2,00]	.308	1.141	.073	1	.787	1.361
	[risk_factors=3,00]	.154	1.407	.012	1	.913	1.166
	[risk_factors=4,00]	0[b]	.	.	0	.	.

	lv_geometry[a]	B	Std. Error	Wald	df	Sig.	OR = Exp(B)
eccentric remodelling	Intercept	-1.965	2.573	.584	1	.445	
	age	.030	.024	1.645	1	.200	1.031
	SBP	.003	.018	.025	1	.875	1.003
	DBP	-.042	.032	1.747	1	.186	.959
	[BMI category=morbid obesity]	3.259	1.059	9.472	1	.002	26.033
	[BMI category=obese]	-.944	1.213	.606	1	.436	.389
	[BMI category=overweight]	-.768	.983	.610	1	.435	.464
	[BMI category=znormal]	0[b]	.	.	0	.	.
	[diastolic_function=0]	.850	.759	1.253	1	.263	2.339
	[diastolic_function=1]	0[b]	.	.	0	.	.
	[gender=female]	-.452	.712	.404	1	.525	.636
	[gender=male]	0[b]	.	.	0	.	.
	[risk_factors=1,00]	2.676	1.109	5.828	1	.016	14.533
	[risk_factors=2,00]	-.018	1.429	.000	1	.990	.982
	[risk_factors=3,00]	.724	1.025	.499	1	.480	2.062
	[risk_factors=4,00]	0[b]	.	.	0	.	.

Notes:
B = is used to predict the dependent variable from the independent variables and presented in log odds units.
Wald statistic = is used to assess the significance of the coefficients and contributions of individual predictors in a given model.
BMI = Body mass index; DBP = Diastolic blood pressure; SBP = Systolic blood pressure; HPT = Hypertension; DM = Diabetes mellitus.

CHAPTER 5: DISCUSSION

The limited literature in this area shows that CV risk factors are associated with the changes to cardiac structure and function. The data on the degree of change to the heart in this ethnic group in relation to CV risk factor presence is limited, especially since these changes are known to occur sub-clinically, in the presence of CVD. As far as evaluating the cardiac structural and functional abnormalities in response to cardiovascular risk factors, there have been very few studies in South Africa or KwaZulu-Natal in particular. The findings of this study will add greatly to the current body of knowledge, and have the potential to inform clinical practice and public health directives.

According to Dwane, Wabir and Manda (2020), there were 56 million deaths worldwide. Of these, 17.5 million of these deaths were due to CVD and they are causing a huge burden to the health care system. An alarming statistic was that three-quarters of these deaths took place in low- and middle-incoming countries, such as those in SSA, which also contributed 80% of the global burden of the disease. Though the pattern, magnitude, and trends of CVD deaths in SSA are not completely understood, emerging data indicate the rising prevalence and mortality in this region (Mensah *et al.* 2015).

This quantitative study represents the first compilation of clinical data on the spectrum of cardiovascular risk factors and their effects on the cardiac structure and function in the Northern KZN at Madadeni provincial hospital. This was done using the World Health Organization criteria in diagnosing every cardiovascular risk factor. Patients with known stroke, coronary heart disease, chronic kidney disease, congestive heart failure, pregnancy, Covid 19 and epilepsy were excluded.

The main objective of the study was to determine the prevalence of modifiable and non-modifiable cardiovascular risk factors in patients presenting to Madadeni Provincial Hospital (Internal Medicine Department). The second objective was to determine the effects of cardiovascular risk factors on the cardiac structure and

function using echocardiography. The third objective was to determine the prevalence of metabolic syndrome on the studied population.

All participants were screened and tested for Covid 19. All results were negative.

There was a high prevalence of cardiovascular risk factors at Madadeni Provincial Hospital, with 86 males (43.0%) and 114 females (57.0%).

In this study, HPT was more prevalent with 31.5% (31.5%) with mostly women and in blacks (33.3%). Diabetes mellitus was the second highest cardiovascular risk factor with 17.0%. The unemployment rate in this study was the highest with 54.5%. 90% of the participants reported they do not engage in any physical activity, more so women. The main prevalence of left ventricular geometry was normal (51.5%) followed by concentric hypertrophy with 36.0% which was more prevalent in males. Black Africans presented more with metabolic syndrome (58.3%) than any other race.

5.1 THE PREVALENCE OF MODIFIABLE CARDIOVASCULAR RISK FACTORS

5.1.1 THE PREVALENCE OF HYPERTENSION

In this study, HPT was more prevalent with 31.5% of the participants. According to Keates *et al.* (2017), in a systematic review of 33 studies from 15 SSA countries, conducted from a period of 1999 to 2013, the prevalence range was 15-70%. Participants in the current study who presented with HPT and diabetes were 24.5%. Given the findings and the increase in the prevalence of both HPT and diabetes in South Africa, this trend may continue to worsen if it is not quickly addressed. Sliwa *et al*. (2008) report that the prevalence of HPT was high in the Heart of Soweto study with 44% and those hypertensive patients were obese.

In this study, HPT was more prevalent in women, with 32 (28.0%), compared to men with 31 (27.1%). This data is in keeping with findings from Jongen *et al*. (2019) that 27.4% women and 26.1% of men in South Africa are hypertensive. Despite the gender differences, the treatment guidelines remain the same (Gillis and Sullivan 2016).

Previous family studies have proven than HPT is hereditary and genetic factors play a huge role (Padmanabhan, Caulfield and Dominiczak 2015). According to Padmanabhan, Caulfield and Dominiczak (2015: 116, it is evident that HPT exists as a polygenic trait, that “From an epidemiological perspective a majority of individuals with HPT have essential or primary HPT, which is thought to result from a genetic predisposition underlying the cumulative effects of various lifestyle factors over years”.

In this study, concentric left ventricular hypertrophy was more prevalent in hypertensive individuals with 49.2%, followed by eccentric left ventricular remodelling 7.9% and concentric left ventricular remodelling 4.7%. Eccentric left ventricular hypertrophy was the least with 1.5%. This finding was also supported by the study conducted by Zheng *et al.* (2019). The presence of left ventricular hypertrophy has always been a complication and causes mortality in hypertensive individuals (Dobrowolski *et al*. 2015). Left ventricular hypertrophy occurs as a result of physiological adaptation due to increased cardiac function. Various events are activated with include mitochondrial dysfunction, cell death, oxidative stress, neurohormonal activation, inflammation, and changes in the cardiac geometry leading to cardiac remodelling.

Concentric hypertrophy has always been associated with hypertensive patients (Dobrowolski *et al*. 2015). In the RESISTOL-Pol study, 155 patients were enrolled in Poland between the year 2009 and 2011 to evaluate factors associated with HPT. Patients were then divided into four groups based on their left ventricular geometrical patterns. According to the researchers, Group 1-Normal (24.4%), group 2-concentric remodelling (25.8%), group 3-eccentric hypertrophy (16.8%) and group 4-concentric hypertrophy (33.0%). Concentric hypertrophy was the most prevalent and the most common type of left ventricular geometrical pattern in hypertensive patients (Dobrowolski *et al*. 2015).

In the current study, concentric hypertrophy was the most left ventricular geometrical pattern associated with a high prevalence of cardiovascular risk factors. The prevalence of normal left ventricular geometry was 49.7% and of concentric remodelling was 31.1%.

Diastolic dysfunction in hypertensive individuals was detected in 22.2% patients. Diastolic function was normal in 77.7% participants. According to Santos and Shah (2014), HPT has long been associated with diastolic dysfunction. Left ventricular diastolic dysfunction is an independent predictor for heart failure and can cause structural and functional cardiac abnormalities through many events and mechanisms which happen at molecular and cellular level (Tran *et al.* 2020). A study of 925 newly diagnosed hypertensive patients without cardiac disease were enrolled in Portugal (Ladeiras-Lopes *et al*. 2018). Their study reported a strong relationship between HPT and early diastolic dysfunction (62.7%).

Most patients with HPT have a history of diastolic dysfunction (Lalande and Johnson 2008). Lalande and Johnson (2008) state that HPT is provoked by several pathological mechanisms that happen at a cellular level. This includes activation of RAAS, SNS, inflammation, altered fibrinolysis, oxidative stress, secretion of cytokines, apoptosis and production of endothelins (Cohn, Ferrari and Sharpe 2000). According to Cohn, Ferrari and Sharpe (2000), this leads to thickening of the ventricular walls to maintain the cardiac output and afterload, resulting in ventricular hypertrophy. Increased wall mass induces impaired ventricular stiffness and filling, causing increased left ventricular end diastolic pressure and in due course diastolic dysfunction occurs (Lalande and Johnson 2008).

Findings from the current study were lower than those reported by Adamu *et al.* (2010), who reported a prevalence of 62.0% of diastolic dysfunction in hypertensive participants in a study conducted in Nigeria.

5.1.2 THE PREVALENCE OF DIABETES MELLITUS

This study showed that type 2 diabetes mellitus was the second highest cardiovascular risk factor, after HPT, with 17.0%. According to Belue *et al*. (2009), IDF estimated that the prevalence of diabetes mellitus type 2 in Africa at the time of study was 2.8% with Malawi and Ethiopia under 2% and South Africa over 3%, so the findings from the current study are higher than the reported prevalence.

Diabetes mellitus was more prevalent in women with 21 (18.0%) compared to men 13 (15.1%). Keates *et al.* (2017) found the prevalence of diabetes to be higher among African women aged 20-39 years and African men up to 79 years. This is because diabetes mellitus advances with age (Keates *et al.* 2017). According to Keates *et al*. (2017), deficiency of insulin secretion develops with age and this is induced by many factors which include ageing, obesity, psychological stress, and economic transition, to name a few.

A recent study by Sahadew and Singram (2019) conducted in Eastern Cape province revealed a higher burden and increase in the prevalence of diabetes compared to 2014. This community-based study was conducted to determine the burden of diabetes mellitus during the year 2014 and 2016 in the eight districts of the province (Sahadew and Singram 2019). According to the researchers, there was a diabetes mellitus prevalence of 17.0% and this was higher in rural areas compared to the urban population. The results found in this study are in keeping with the results discovered in Eastern Cape.

In this study, diabetes mellitus was associated with concentric hypertrophy with 17.6%, followed by concentric remodelling with 5.8%. Most (73.5%) participants had normal left ventricular geometry. In the HyperGen population-based study, diabetes was associated with left ventricular hypertrophy and concentric hypertrophy regardless of age, gender, BMI or blood pressure (Santos and Shah 2014). Diabetes mellitus is closely associated with left ventricular hypertrophy, left ventricular systolic and diastolic dysfunction (Mohan *et al.* 2021). Half (50.0%) of type 2 diabetes patients develop left ventricular hypertrophy (Mohan *et al.* 2021). Left ventricular hypertrophy is a strong predictor of cardiac outcomes and heart failure. According to Mohan *et al*. (2021), the pathology of left ventricular hypertrophy in type 2 diabetes is

still unclear, however previous studies have explained the association of cellular alterations, changes in the extracellular matrix, molecular abnormalities, fibrosis inflammation, oxidative stress, AMP-activated kinase and insulin resistance in the initial stages. In later stages, this leads to ventricular stiffness and impaired relaxation results.

In this study, diastolic function in diabetic individuals was normal in 91.1% participants and abnormal in 8.8% participants. In the study by Cassidy *et al*. (2015), conducted in Newcastle Upon Tyne hospital, despite concentric remodelling which was detected in type 2 diabetic participants, there was a high prevalence of left ventricular diastolic dysfunction. Their study revealed a greater relationship between diabetes mellitus and cardiac changes. These disparities were also explained by Bergerot *et al.* (2018) in a 3-year follow study in France. The researchers conducted a prospective study of 310 diabetic patients which was aimed at evaluating cardiac changes (Bergerot *et al.* 2018). All participants underwent full clinical and physical examination, echocardiography and biochemical analysis. The prevalence of diastolic dysfunction increased from 49.0% to 67.0%. According to the researcher (2018), only 32.0% had normal diastolic function. The overall diastolic dysfunction prevalence in this study was 27.0% and this was independently related to their age and increased blood pressure during follow up (Bergerot *et al.* 2018).

5.1.3 THE PREVALENCE OF DYSLIPIDEMIA

The overall prevalence of dyslipidemia in the study was 4 (2.0%). According to Keates *et al*. (2017), the prevalence of dyslipidemia in Africa ranges from 20% to 35% with women having higher total cholesterol levels than men. In a research study conducted in Nigeria on healthy individuals, 5% of the studied population had dyslipidemia with 23.0% of increased total cholesterol and 51.0% increased LDL cholesterol.

In this study, dyslipidemia was more prevalent in men with 3 (3.4%) compared to women with 1 (1.1%). The observation regarding ethnic differences in lipid profiles is similar to other previous studies. In the Soweto Heart Study, blacks had low prevalence of dyslipidemia, with the highest being Indians (Sliwa *et al*. 2017).

In this study, participants with dyslipidemia only presented with 75.0% of the normal left ventricular geometry, and 25.0% presented with concentric hypertrophy. There were no other left ventricular geometry patterns detected in dyslipidemia participants.

All participants with dyslipidemia only had normal diastolic function. No diastolic dysfunction was reported.

A prospective study was conducted on 8 465 participants in Mashhad in northern Iran. Participants were from the Mashhad Stroke and Heart Atherosclerotic Disorder (MASHAD) cohort study (Hadeyatnia *et al.* 2020). The study investigate the association of dyslipidemia with cardiac events in that population after six years of being followed up. Their study revealed that abnormally high levels of LDL cholesterol were associated with the atherosclerosis process and coronary artery disease in middle-aged men (Hedayatnia *et al.* 2020). Hedayatnia *et al*. (2020) found no association between HDL-C and any cardiac events.

5.1.4 THE PREVALENCE OF OBESITY

According to Son *et al.* (2016), the atherosclerosis Risk of Rural Areas in the Korean General (ARIRANG) population-based study conducted research on 1 460 healthy males between the years 2005 and 2008. Participants underwent full echocardiography and tissue doppler imaging. WC was associated with increased left ventricular end diastolic dimension and left ventricular end systolic dimension. Relative wall thickness did not increase. Increased WC was linked with reduced left ventricular failure. Increased WC was also associated with diastolic dysfunction, longer deceleration time and decreased e/a ratio (Son *et al*. 2016).

In this study, 53 (26.5%) were overweight, 45 (22.5%) were obese, 26 (13.0%) were morbidly obese and 76 (38.0%) had a normal BMI. Morbid obesity was noted in 65.3% of females and 34.6% in males. Obesity was noted in 68.8% of females and 31.1% of males. Overweight was noted in 62.2% of females and 37.7% of males. It is evident from the study results, that females had high body mass indexes when compared with males. A WHO criteria was used to assess the levels of BMI.

According to Keates *et al.* (2017), from the previous studies, obesity has always been higher in African women. Obesity was associated with left ventricular diastolic dysfunction in females (Keates *et al*. 2017). Santos and Shah (2014) state that these data findings prove that central obesity causes left ventricular diastolic abnormalities leading to heart failure. Obesity causes 11% of cardiac failure cases in men and 14% cases in women in the United States of America (Carbone *et al*. 2017).

In this study the association between BMI category and LV geometry was significant (*p*-value = 0.02). There were 53 (26.5%) patients who were overweight. Of these 53 patients, 18 (34%) presented with concentric hypertrophy. There were 45 (23.5%) patients who were obese. Of these patients who were overweight, 26 (57.8%) had normal LV geometry, while 16 (35.6%) presented with concentric hypertrophy. There were 26 (13%) patients in the category morbid obese. Of these, 9 (34.6%) presented with concentric hypertrophy, while 4 (15.4%) presented with concentric remodelling and 6 (23.1%) presented with eccentric remodelling.

This study is comparable with a study that was conducted in Johannesburg, South Africa conducted by Woodiwiss *et al.* (2008), involving 309 African participants with obesity . These authors reported that 16.0% presented with concentric LV remodelling, 15.0% with eccentric hypertrophy and 7.0% with concentric hypertrophy.

At a cellular level, the human body adapts to increased caloric intake (Parto and Lavie 2017). According to Parto and Lavie (2017), there is a link between obesity and increased adipocyte size, which alters the adipose tissue function. Parto and Lavie (2017) state that adipocyte hyperplasia through weigh gain is induced by adipogenic progenitors and growth factors such as cytokines (TNF-alpha), angiotensin II and macrophage stimulating factor. Parto and Lavie (2017) found that as obesity increased, adipocytes undergo activation of RAAS and SNS, apoptosis, cell necrosis, and fibrosis, which precipitates an inflammatory state causing adipocyte dysfunction. Once the inflammatory cytokines are secreted in high numbers, a whole process of cardiac remodelling begins (Carbone *et al.* 2017). All this leads to an increased cardiac output and stroke volume to maintain the cardiac function; with time, cardiac structural and functional abnormalities start appearing (Carbone *et al.* 2017).

5.1.5 THE PREVALENCE OF PHYSICAL INACTIVITY

Keates *et al.* (2017) report on a survey of 22 African countries with 57 000 participants aged 25-64 from 2003 to 2009. The study revealed that males and females were no longer active. Mozambique and Malawi were the only African countries that had high levels of physical activity with > 90% of WHO recommended activity levels (Keates *et al.* 2017).

The current study indicates that 180 (90.0%) of participants reported that they do not engage in any level of physical activity while 20 (10.0%) do engage in physical activity. 95 (47.5%) of the participants reported walking and cycling while 105 (52.5%) do not engage in exercise at all. Keates *et al*. (2017) report that in studies on young African adults, physical inactivity is linked with increased BMI and HPT, which in turn, contributes to cardiac remodelling as discussed above.

In this study, 26.1% physically inactive participants presented with diastolic dysfunction. Physically active individuals mostly presented with normal diastolic function.

A systematic study and meta-analysis review was conducted on the previous prospective studies to investigate the relationship between physical inactivity and cardiac abnormalities (Aune *et al.* 2021). A total of 27 prospective studies with 34 publications were included. All these studies were from USA, Canada and Europe. Aune *et al.* (2021) state that the findings prove that physical activity is associated with reduced risk of any cardiac abnormalities and better outcomes.

5.1.6 THE PREVALENCE OF UNHEALTHY DIET

Keates *et al*. (2017) report that a study conducted in 187 countries in 2015, which examined the food intake of individuals from the year 1990 to 2010, reported a high consumption of unhealthy foods especially in central African countries.

The current study reveals that the studied population consume less fruit and vegetables. This is similar to a study by Miller *et al*. (2016) in the PURE study, which

included 18 countries, who found that participants in low-income countries consumed less fruit and vegetables than the recommended five daily servings.

The implications of Western fast food contribute to 30% of the cardiovascular risk worldwide (Casas *et al.* 2018). According to Casas *et al*. (2018) there is evidence that a Western diet is associated with the production of inflammatory cytokines when compared to a healthy diet. The intake of healthy products such as fruit, vegetables, whole grains and avocado to name a few, are associated with lower risk of inflammation (Casas *et al.* 2018). According to Casas *et al*. (2018), once cytokines are secreted, there is a wide range of activations that happen at a cellular level. This include activation of RAAS, SNS, collagen degradation, nitric oxide (NO) production, mitochondria dysfunction, necrosis, and apoptosis, leading to the development of cardiovascular risk factors (Casas *et al*. 2018). Casas *et al*. (2018) state that the Western diet is linked to obesity, dyslipidemia, HPT and diabetes mellitus.

Ferron *et al.* (2018) conducted in Brazil to evaluate the association of Western diets and cardiac dysfunction on rats (Ferron *et al*. 2018). Full transthoracic echocardiography was performed according to the American Society of Echocardiography (ASE). After 20 weeks of Western diet consumption, 63% of the rats had systolic and diastolic dysfunction and remodelling (Ferron *et al*. 2018).

5.1.7 THE PREVALENCE OF TOBACCO USE

Keates *et al*. (2017) reports that according to the WHO data of 29 African countries, the prevalence of tobacco use was 45-50% in Morocco and Egypt , 43-44% in Cameroon and the Republic of the Congo, and 44-60% in Mauritius and Sierra Leone. The prevalence of smokers in SSA will increase by 1.5-fold to > 200 million people by 2030 (Keates *et al*. (2017).

This study found that 36 (18.0%) of all participants were current smokers of which 23 (11.5%) were men and 11 (5.5%) women, with 164 (82.0%) of the enrolled participants being non-smokers.

In this study, tobacco use was associated with concentric hypertrophy with 93.4% followed by eccentric remodelling with 31.2%. There was a high prevalence of

nonsmokers when compared with smokers. In the ECHO-Sol study, current smokers presented with worse diastolic dysfunction, worse left ventricular geometry and increased left ventricular mass (Leigh *et al*. 2017). According to Leigh *et al.* (2017), in animal models, tobacco use was closely associated with left ventricular hypertrophy and left ventricular remodelling.

Tobacco use was associated with diastolic dysfunction in 21.5% of individuals, while 16.7% smokers had normal diastolic function. There was a high prevalence of nonsmokers with 82.0%.

5.1.8 THE PREVALENCE OF ALCOHOL CONSUMPTION

According to Djouse *et al.* (2009), the Nurse and Health Follow-up Study, alcohol intake three to four days per week reduces the risk of developing coronary artery disease in both males and females. Alcohol intake had a beneficial effect on moderate drinkers (Djousee *et al*. 2009).

In this study, alcohol consumption was self-reported. The proportion of participants who consumed alcohol during the past month (current drinkers) was 54 (27.0%), with 146 (73%) reported never having drunk any alcohol. Of the participants who had consumed alcohol, 41 (75.0%) were males and 13 (24.0) were females.

In this study, alcohol consumption was associated with concentric hypertrophy in 31.9% of participants. Non-drinkers were also associated with concentric hypertrophy. There was high prevalence of non-drinkers when compared with drinkers. In this study, 28.1% drinkers had normal diastolic function while 23.5% had diastolic dysfunction.

The toxicity of heavy alcohol consumption deteriorates the myocardium, resulting in the activation of many pathological mechanisms (Kawano 2010). These include increased activation of RAAS, SNS, inflammatory markers, interstitial fibrosis, apoptosis, increase in oxidative stress, and myocardial injury, which disrupts protein synthesis leading to contractive dysfunction (Kawano 2010). All these activations change the cardiac geometry causing increased volume overload leading to cardiac

changes (Schirone *et al*. 2017). Kawano (2010) states that heavy alcohol consumption is linked to HPT, obesity and heart failure.

The CARDIA (Coronary Artery Risk Development in Young Adults) cohort study was a long on-going study in North America. A total of 2 368 participants who consumed alcohol were enrolled and followed up for 20 years to assess the role of alcohol in the cardiac structure and function (Rodrigues *et al.* 2018). The findings were that 76 (3.2%) of the participants had LVEF < 50% at the end of the 20-year follow up. There was an association of enlarged LA size with alcohol consumption. According to Rodrigues *et al*. (2018), there was also a significant change in LV mass and LVED volume which was related to alcohol intake. This cohort study proved that alcohol abuse does cause cardiac abnormalities (Rodrigues *et al.* 2018).

Alcohol consumption has received too little attention regarding its effects on cardiac structure and function, according to Keates *et al.* (2017).

5.1.9 THE PREVALENCE OF PSYCHOLOGICAL STRESS

Song and Fang (2019) report that they studied the Swedish population to assess the association between stress and cardiovascular disease. Stress was associated with CVD, mostly as early as < 50 years. 63.0% of the studied group were women. Individuals with severe mental disorder including bipolar disorder and depression have increased risk of developing coronary artery disease (De Hert, Detraux Vancampfort 2018). Hamad *et al*. (2008) conducted a study in a KZN village in 2001 to evaluate the prevalence of psychiatric morbidity. The researchers found that increased stress was associated with females and African race. The overall prevalence was 27.0%.

Stress related disorders tend to be high in lower income families, divorced or widowed, when compared to unexposed individuals. This was also supported by Hamad *et al.* (2008). They conducted a cohort study in 2007 on 257 individuals in Durban, Cape Town, and Port Elizabeth to evaluate the level of stressors in low income families. The study indicated that increased levels of stress were associated with many family members all living under one roof, and low education (Hamad *et*

al.2008). Poverty in South Africa is associated with unemployment, poor housing, lack of clean water, low levels of education and depression (Morgan 2013).

In this study, 44.4% of the psychologically stressed individuals had concentric hypertrophy and 39.2% presented with diastolic dysfunction. Normal mental health was associated with normal left ventricular geometry. According to the American Heart Association (2020), chronic stress increases blood pressure, cholesterol and blood sugar which all have negative effects on the cardiovascular system and can cause coronary artery disease. Chronic mental stress is associated with increased angiotensin II production and activation of the SNS (Hamad *et al*. 2008). Hamad *et al*. (2008) state that this increases norepinephrine and epinephrine secretion, increasing oxidative stress, inflammatory cytokines, apoptosis, necrosis, which induces cardiac remodelling which causes structural and functional cardiac abnormalities.

5.1.10 THE PREVALENCE OF PSYCHOSOCIAL ISSUES

5.1.10.1 MARITAL STATUS

Unemployment is a major problem in South Africa and has a negative impact on our communities (Morgan 2013). In this study, 105 (52.5%) of the participants were single, 68 (34%) were currently married, 20 (10.0%) were widowed and 7 (3.5%) were divorced. Of the 20 widowed individuals, 19 (95.0%) were unemployed, 13 (65.0) were pensioners and living in a household with 3 to 12 adults above the age of 18 years old. Of the 7 divorced individuals, 4 (57.1%) were unemployed, 2 (28.5%) were employed and 1 (14.2%) was self-employed. Zagozdzan *et al*. (2014) conducted a study in Poland between 2009 and 2010 to assess how unemployment can impact the cardiovascular system. They recruited 3 052 unemployed participants. There was a high prevalence of HPT, obesity, smoking and coronary artery disease in these participants (Zagozdzan *et al*. 2014). Their results are in keeping with this study's results, where there are high levels of unemployment in participants with cardiovascular risk factors.

5.1.10.2 LEVEL OF EDUCATION

In this study, 63 (31.5%) of the participants completed high school, 61 (30.5%) of the participants completed primary school, 51 (25.5%) completed university and 25 (12.5%) did not have any formal schooling. The age group 26-35 achieved a higher level of education than those in the older age groups. Participants over the age of 45 years had no formal education and less completed high school or university when compared with those less than 45 years as shown in the age group 56-65.

5.1.10.3 EMPLOYMENT STATUS

Pillay, Lugte and Aldous (2016) state that with the high unemployment rate in South Africa, poverty is mostly the reason why individuals cannot afford to prepare healthy meals.

It is seen that employed persons accounted for 52 (26.0%) of participants while unemployed participants accounted for 109 (54.5%). Self-employed participants accounted for 30 (15.0%). Participants who are on pension were 58 (29.0%), participants receiving a R350 grant were 21 (10.5%) and there was 1 (0.5%) scholar. According to the 2017 Heart Failure congress and the 4th World congress on acute heart failure, unemployment was associated with 50% of cardiac events (Morgan 2013).

5.2 THE PREVALENCE OF NON-MODIFIABLE CARDIOVASCULAR RISK FACTORS

5.2.1 THE PREVALENCE OF FAMILY HISTORY

In the SABRE (Southall And Brent Revisited) population-based study, 974 Europeans and 734 South Asians were enrolled where they completed a follow up questionnaire on family history data (Wang *et al*. 2020). The results of the study showed a strong link between family history and CVD in South Asians (Wang *et al.* 2020).

Imes and Lewis (2014) state that the Framingham study reported that CVD in one parent doubles the 8-year risk of CVD among men and women. A retrospective

cross-sectional study was performed on 6 399 patients with confirmed coronary artery studies in Tehran Heart Centre, Iran.

In this study, 143 (71.5%) of the studied group had no family history of cardiovascular disease. Family history was reported in 57 (28.5%) participants.

5.2.2 THE ASSOCIATION OF AGE AND CV RISK FACTORS

In this study, the age group 56-65 presented more with cardiovascular risk factors when compared with other age groups. The age group 46-55 followed with 19.5%. The young adults presented more with cardiovascular risk factors when compared with the older generation. The older generation of the age group 76-80 were the lowest with 4.0%. This is because Amajuba district, where the study was conducted, is a rural place where the older generation still practices farming to eradicate poverty (Amajuba District Special Development Framework [2019/2020]). According to Raidmi (2014), women contribute to 50% to 80% of agricultural labour in South Africa and they eat food from their gardens. However, it is concerning to note that dietary patterns have since altered, with the move to consuming lower quantities of fresh food in this community sample.

5.2.3 THE PREVALENCE OF GENDER

The data results from the Framingham study reported that women were likely to develop cardiovascular risk factors (Santos and Shah 2014). This pattern was reflected in the current study, as 57.0% were females and 43.0% were males, revealing a higher prevalence in females.

Santos and Shah (2014) reported that that women developed concentric LV hypertrophy and men with eccentric LV hypertrophy. In this study, normal left ventricular geometry was more prevalent in both males and females, followed by concentric hypertrophy and eccentric remodelling. Concentric hypertrophy was mostly detected in males with 18.5%, while females had 17.5%. Normal left ventricular geometry was mostly prevalent in females with 31.5% while males had 20.0%. This is because the hormone oestrogen in women has cardio-protective

effects (Woodward 2019). This statement was also reported in the INTERHEART study (Yusuf *et al.* 2004). The sharp increase of coronary artery disease in women after menopause is evidence that endogenous oestrogen may prevent CVD (Gao *et al*. 2019). Gao *et al*. (2019) report that women have a lower risk of CVD than males but have a higher mortality and worse prognosis of CVD events.

5.2.4 THE PREVALENCE OF ETHNICITY

China and Vietnam have lower levels of cardiovascular risk factors while South Asians and sub-Saharan Africans have higher risks, especially regarding HPT (Santos and Shah 2014).

There was a high prevalence of black patients 156 (78%), the remainder being made up of Indians 27 (13.5%) followed by whites 12(6.0%) and mixed ancestry 5 (2.5%).

In the Cardiovascular Health Study, there was a lower prevalence of LVH in Whites hypertensive participants when compared to blacks (Santos and Shah 2014). According to Lindhorst *et al.* (2007), HPT is more prevalent in the black population. In the CARDIA study, African blacks reported higher diastolic dysfunction when compared with whites (Nardi *et al*. 2017). According to Nardi *et al*. (2017), such results collated with the ones in Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) where hypertensive individuals without heart failure were echocardiographically assessed by utilising tissue doppler imaging. The early diastolic velocity e` was lower in African-Caribbean patients and this proves that Blacks are highly susceptible to structural and functional cardiac abnormalities (Nardi *et al*. 2017). This is the case because there is a strong association between African black ethnicity and increased LV mass and relative wall thickness (Nardi *et al*. 2017). Nardi *et al*. (2017) reported that in a large cohort of black studies, LVH has always been a significant predictor of mortality. When compared with whites, blacks have always presented with a cluster of cardiovascular risk factors (mainly HPT) and this may explain the increased prevalence of LVH seen in Blacks (Nardi *et al*. 2017).

5.3 THE EFFECTS OF CARDIOVASCULAR RISK FACTORS ON CARDIAC STRUCTURE AND FUNCTION

Concentric hypertrophy was one of the left ventricular geometries that was associated with highest prevalence of all cardiovascular risk factors with 36.0%, followed by eccentric remodelling with 6.5%, concentric remodelling with 5.5%, and eccentric hypertrophy with 0.5%. It must be noted that 51.5% of the studied population demonstrated a normal left ventricular geometry. According to Santos and Shah (2014), most previous studies have reported a high prevalence of the normal left ventricular geometry. This study had similar results.

Of those suffering from HPT, 49.2% presented with concentric hypertrophy. Of those in the category dyslipidemia, 25.0% presented with concentric hypertrophy, and, of those with diabetes mellitus, 17.6% had concentric hypertrophy. Eccentric remodelling was mostly associated with diabetes mellitus (5.8%) followed by HPT (4.7%).

A high prevalence of concentric hypertrophy was also detected by Zheng *et al.* (2019). A total of 76 142 participants were included in the study where 49.7% had normal left ventricular geometry while concentric remodelling was 31.1%. A similar study was conducted on 843 participants in China where they examined healthy individuals. The prevalence of left atrial enlargement, left ventricular hypertrophy and concentric remodelling was 2.4%, 5.0% and 12.7% respectively (Zhang *et al.* 2012). The prevalence of mild and moderate left ventricular diastolic dysfunction was 14.0% and 3.3% respectively (Zhang *et al*. 2012).

Left ventricular hypertrophy is one of the visible cardiac features in patients with HPT (Santos and Shah 2014). Our study revealed a 37.0% prevalence of left ventricular hypertrophy, which was mostly prevalent in hypertensive individuals. Wu *et al*. (2017) examined 474 Chinese women above the age of 65 years, 10.3% of the studied population presented with left ventricular hypertrophy while 11.6% had left ventricular enlargement (Wu *et al.* 2017). Wu *et al.* (2017) state that data results from the Framingham Heart Study show that women are prone to left ventricular hypertrophy.

In this study, there was a higher prevalence of diastolic dysfunction than systolic dysfunction. Left ventricular diastolic dysfunction prevalence was 25.5% while left ventricular systolic dysfunction was noted in 26 (13.0%). Diastolic dysfunction was mostly associated with HPT with 22.2%, followed by diabetes mellitus with 8.8%.

5.4 THE PREVALENCE OF METABOLIC SYNDROME

Salim *et al.* (2021) state that the NHANES (National Health and Nutrition Examination Survey 2007 to 2014) analysis found that the overall prevalence of metabolic syndrome was 34.3%, with 35.3% in males and 33.3% in females, and it increased with age.

For investigation of metabolic syndrome prevalence in the current study, the JIS (2009) criteria were used. Parameters that were used included central obesity, (WC of > 94 cm in male subjects and > 80 cm in female subjects), HPT (systolic blood pressure of > 130 mmHg, and diastolic blood pressure of > 85 mmHg), dyslipidaemia (triglyceride levels > 1.7 mmol/L), and diabetes mellitus (blood glucose > 5.6 mmol/L). The overall prevalence of metabolic syndrome on the studied population was 12 (6.0%). Males were more prevalent with 58.3% and females with 41.6%.

It is evident that blacks presented more with metabolic syndrome when compared to other races with 58.3%, followed by Indians with 33.3% and whites with 8.3%. None of the mixed ancestry presented with metabolic syndrome. This is similar to a study that was conducted by Peer *et al*. (2015) in Cape Town, South Africa, where 1 099 participants were enrolled between the year 2008 and 2009 with the aim of determining the prevalence of metabolic syndrome using the 2009 harmonised JIS criteria. The study found the prevalence to be 30.7% and was significantly higher in the black population, particularly women. Motala, Mbanya and Ramaiya (2009) reported a prevalence of 22.1% in rural South Africans.

Another study was conducted in a psychiatric unit at King Edward VIII hospital (KEH) in Durban where 276 psychiatric participants were enrolled (Saloojee *et al*. 2016).The aim was to assess the prevalence and cardiovascular risk factors for metabolic syndrome in psychiatric patients. The prevalence of metabolic syndrome

was 23.3% with females presenting more (38.3%) than males (15.4%). The majority in terms of ethnicity were black African with 84.1% in this study (Saloojee *et al*. 2016).

In the current study, metabolic syndrome was associated mostly with concentric hypertrophy with 33.3%. Metabolic syndrome participants had mostly normal diastolic function with 75.0%. Only 25.0% of metabolic syndrome presented with diastolic dysfunction. This is similar to a study by Zheng *et al*. (2019) where the researchers conducted a community-based study on 1 965 middle-aged Chinese in 2014 to 2016. Their aim was to investigate the association of metabolic syndrome with left ventricular diastolic function, as per transthoracic echocardiography, full physical and medical examination (Zheng *et al*. 2019). A questionnaire was used to collect demographic information. The metabolic risk factors were defined according to the revised National Cholesterol Education Programme ATP III 2004 criteria (Zheng *et al*. 2019). Zheng *et al*. (2019) reported that the prevalence of left ventricular diastolic dysfunction was higher in males (37.05%) than female participants (32.53%). The overall prevalence of diastolic dysfunction among these participants was 17.76% (Zheng *et al*. 2019).

5.5 INDEPENDENT PREDICTORS OF LEFT VENTRICULAR REMODELLING

Wu *et al.* (2017) state that left ventricular remodelling is the initial stage of cardiac failure. Identifying the predictors of left ventricular remodelling must be fully understood.

The independent predictors of left ventricular remodelling in this study were age, gender, BMI, SPB, DBP (risk factors HPT, diabetes mellitus and dyslipidemia) and diastolic dysfunction.

In a community-based study conducted by Wu *et al*. (2017), a total of 474 Asian women underwent transthoracic echocardiography and were enrolled in a study from the year 2009 to 2014. Over a 5-year follow-up, 10.3% developed left ventricular hypertrophy while 12.6% developed left ventricular enlargement. A multivariate model was developed to predict the events of LVH and LVE and was entered into a

Cox regression model for evaluation of independent risk factors. A *p*-value < 0.10 was set as a criterion for independent variables to enter in a model. Independent predictors of left ventricular remodelling in the study were central BP (CBP), BMI, LDL Cholesterol and e'/e' ratio (Wu *et al*. 2017).

Prakaschandra *et al.* (2016) conducted a study on South African Indians in Durban which aimed to determine the prevalence of cardiovascular risk factors in that population. The authors found that age, total cholesterol, triglycerides, HDL cholesterol and WC were significant independent risk factors for diabetes mellitus and HPT.

5.6 STUDY LIMITATIONS

To the best of the researcher's knowledge, this is the first study on the prevalence of cardiovascular risk factors and their impact on cardiac structure and function to be conducted in the rural geographical KZN setting. While this study has important findings, it has several limitations, namely:

- The study was limited to individuals with cardiovascular risk factor (s) only
- Time to adequately enrol patients was another constraint
- Inadequate budgetary allocations
- Variables such as a smoking status, alcohol consumption, diet and physical activity were self-reported, which may have affected the reliability of the data
- A fingerprick method for blood glucose was used
- This study did not include LDL and HDL cholesterol values due to the unavailability in the hospital facility.
- Transthoracic echocardiography (TTE) is a user dependent diagnostic test; as a result some measurements may be overestimated or underestimated but with diastolic dysfunction, it is not common to give the same diagnosis due to the striking features that can be seen
- The study assessed all echocardiographic measurements non-invasively, although they were compared with invasive reference standards. However, doppler echocardiography has been reported to determine all measurements accurately

- Unavailability of some information, resulting in some participants needing to return to the clinic.

CHAPTER 6: CONCLUSION AND RECOMMENDATIONS

6.1 RECOMMENDATIONS FOR FUTURE RESEARCH STUDIES

The increasing burden of cardiovascular risk factors in SSA is a major concern. The clinical findings of this study set the stage for future investigations.

- Further research on this topic needs to be conducted on a wider scale. This research study was limited in its generalisation due to a small sample size. Therefore, it is suggested that a larger study be conducted in South Africa or SSA.
- Clinical trials could be performed on the studied population to identify the major contribution of individual cardiovascular risk factor(s).
- Large population-based studies can also include ethnic differences.
- There are other psychological and social factors that are associated with cardiovascular risk factors, such as level of education, psychological stress, and marital status. As these were not the primary focus of the study, I recommend future studies evaluate psychosocial factors in the development of cardiovascular risk factors .

6.2 CONCLUSION

This study highlighted the major issues faced by individuals living in northern KwaZulu-Natal. The young adults were more likely to present with more cardiovascular risk factors compared to the older generation. This is thought to be because the older generation still mostly eat from their gardens and still practice farming. The middle-age population were more likely to present with cardiac structural and functional abnormalities. This study also highlighted the issue of unemployment and poverty as a major obstacle to supporting healthy lifestyle changes.

The study has shown that there is a high prevalence of modifiable cardiovascular risk factors in the northern KZN region and many patients present with metabolic syndrome. All modifiable cardiovascular risk factors had significant effects on the cardiac structure and function. In a multiple logistic regression model with normal as the reference category for LV geometry, diastolic function was a significant risk factor leading to concentric hypertrophy (p = 0.015). The risk factor for concentric remodelling was BMI category and morbid obesity (p = 0.015). The risk factors for eccentric remodelling was BMI category, morbid obesity (p = 0.002) and HPT (p = 0.016).

In order to control cardiovascular risk factors, affected individuals need to attend their local clinics regularly, and modify their lifestyles (Dwane, Wabir and Manda 2020). According to Dwane, Wabir and Manda (2020), the high unemployment rate in KZN makes it challenging to achieve good treatment strategies and prevent complications. Schutte (2018) reports that more aggressive preventative approaches are needed to manage CVD in SSA and the rest of the world. Comprehensive approaches to fully understand cardiovascular risk factors are required. This study therefore raises important data on the increasing prevalence of CV risk factors in this community and calls for urgent intervention strategies for lifestyle modification.

Studying and understanding cardiovascular risk factors in association with cardiac structure and function remains an important aspect in medicine that warrants more research because it is the key step in preventing heart failure.

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APPENDICES

APPENDIX A: INSTITUTIONAL RESEARCH ETHICS COMMITTEE: LETTER OF INFORMATION

Dear participant

Thank you for taking the time to read this letter and for considering participating in this research study.

Title of the research study

The spectrum and the effects of cardiovascular risk factors on the cardiac structure and function at Madadeni provincial hospital (Internal medicine department).

Principal investigator/ Researcher

Full names and Surname : Miss Nokwanda Annatoria Gambushe

Qualification(s) : N Dip: Clinical Technology (Cardiology)
: B-Tech: Clinical Technology (Cardiology)

Co-investigator (s)/ Supervisor (s)

Full names and Surname: Dr F.A Mohamed
Qualification(s) : BSc
: MBchB
: FCP (SA)
: FRCP (London)
: Cert Endocrinology

Full names and Surname: Dr Rosaley Prakaschandra
Qualification : PhD (Cardiology) (UKZN)

Brief introduction and purpose of the study

Cardiovascular disease (CVD) is a general term used to describe a range of disorders that affect the heart and blood vessels and this includes coronary heart disease, cerebrovascular disease, rheumatic heart disease and other conditions (WHO:2017).

Keates *et al*. (2017) state that cardiovascular disease is the significant cause of mortality worldwide in 2008 and 80% of these deaths occur in low- and middle-income countries. According to the World Health Organization (WHO) report (2002), cardiovascular disease was responsible for 9.2% of total deaths in the African region in 2001. The exact South African prevalence remains unknown, however Byrne, Eksteen, and Crickmore (2016), reveals that cardiovascular disease is responsible

for 17.3% of deaths in South Africa and Maredza *et al*. (2011) reports that approximately 195 people die every day due to cardiovascular disease in South Africa.

This project was initiated by the observation that cardiovascular risk factors appeared to be on the rise at Madadeni Provincial Hospital (Internal Medicine Department) in the Northern Kwazulu-Natal (KZN) region; however, there was no clinical data available to support this observation (Thembela: 2016). In a conversation on 17 July 2016, the head of internal medicine at that time, Dr. Thembela highlighted that while various studies have been conducted on cardiovascular risk factors in South Africa and internationally, the lack of the Northern KwaZulu-Natal (KZN) demographics and the effects on the cardiac structure and function warrants an investigation in the Northern KwaZulu-Natal (KZN) population.

No studies in the Northern KwaZulu-Natal (KZN) have been conducted on cardiovascular diseases, as most studies are conducted in urban areas such as Durban (Thembela: 2016). This study will focus on all cardiovascular risk factors and will take place in a both rural and urban Northern Kwazulu-Natal (KZN) region. In addition, although it is generally known that there is an increase in the prevalence of cardiovascular risk factors in the black population, the effects of cardiovascular risk factors on the cardiac structure and function has not been adequately studied. The study aims to evaluate the spectrum of cardiovascular risk factors and its effects on the cardiac structure and function in patients presenting to Madadeni Provincial hospital (Internal Medicine Department).

Inclusion criteria

-Patients who have signed consent form

-Patients who are 18 years and older of any race, gender or ethnic group

-Patients with 1 or more cardiovascular risk factor (s)

Exclusion criteria

- Patients who do not issue consent

-Patients below the age of 18 years

- Patients with a known history of stroke, Coronary heart disease, Chronic kidney disease, Congestive heart failure Pregnancy, Covid 19 and epilepsy were excluded.

Outline of the procedure (s)

A researcher systematically sampled participants Northern KwaZulu-Natal region, presenting to Madadeni provincial hospital (internal medicine department) and administered a questionnaire, which collected their information on sociodemographic, presence of diabetes, hypertension, family history of hypercholesterolaemia, family history of fatal cardiovascular events and engagement in physical activity. Other measurements will include blood pressure, weight, height, waist circumference, hip circumference, waist-to-hip ratio and transthoracic echocardiography (2D, M mode, colour flow Doppler, CW Doppler).

Echocardiographic measurements were performed using a Logiq V5 Expert echocardiography machine with patients in the left lateral decubitus position. Left ventricular mass (LVM), Left ventricular mass index (LVMI), Relative wall thickness (RWT), left atrium (LA), Right atrium (RA), End diastolic dimension (EDD), End systolic dimension (ESD) and left ventricular ejection fraction (LVEF) were measured. Left ventricular ejection fraction (LVEF) was measured using a lead edge method. Concentric left ventricular hypertrophy was diagnosed when the relative wall thickness is > 0.42 and eccentric when relative wall thickness is <0.42. Concentric remodelling was diagnosed when the left ventricular mass was <134g/m2 in men and <110 g/m2 in woman. Transmitral inflow velocities were obtained using pulsed Wave Doppler and transmitral E and A velocities will be measured to calculate the E/A ratio.

Risks, discomforts or inconvenience arise from the study

Blood samples were taken on their follow up date, other than that; there was no foreseeable emotional, spiritual hard or risks to the participants.

Potential benefits that may arise from the study

As a participant, the study will directly benefit you on the correct cardiovascular risk factor (s) and any complications will be discovered early which will help specialists to treat you appropriately. The prevalence of cardiovascular risk factor (s) will be established from this study. By identifying the cardiovascular risk factors, information regarding prevention programmes implementation should be addressed to lower the risk for developing cardiovascular disease. Benefits for the researchers may include a publication at a seminar, congress or academic journal.

Reasons why the participant may be withdrawn from the study

Your participation in this study is completely voluntary. As a participant, you have the right to withdraw from the study at any given time, without penalties. Withdrawing will not affect your relationship with the Department of Health staff as a patient.

Remuneration

You will not receive any form of remuneration for your participation in this study.

Cost of the study

You will not be liable for any financial contribution (s) towards this research. You will be liable for the normal hospital cost.

Confidentiality

The information provided by you will be treated highly confidential and will remain anonymous at all times. You will not be required to include your name or any identifiable details when completing the questionnaire. You will be allocated a unique research study number and all your details will be recorded under that number. The data and research results will be stored in a secured place at Madadeni Provincial Hospital, where only the researcher and co-supervisors have access to and will not be shared with any other person.

Research-related injuries

There will not be any research-related injuries.

Persons to contact in the event of any problems or queries related to the study: You may contact the researcher, Miss N. A Gambushe (034 328 8327) or the study supervisors, Dr F.A Mohamed (082 377 8072) and Dr. R. Prakaschandra (031 373 6885). You may also contact the Institutional Research Ethics administrator on 031-373 2900.

Yours faithfully

Miss N. A Gambushe (Principal researcher).

APPENDIX A1: INSTITUTIONAL RESEARCH ETHICS COMMITTEE: INCWADI YOLWAZI NGOCWANINGO

Mngeneleli othandekayo

Ngiyabonga ngokuthatha isikhathi sakho sokuthi ufunde incwadi yolwazi nanokuba yingxenye yalolucwaningo

Isihloko sesifundo ngocwaningo

Lesi 135abantu sizoqxila ekubhekeni ukudlanga kwezinto ezigcina zikubeke engcupheni ukuthi ugcine usunesifo senhliziyo eMadadeni Provincial hospital.

Umcwaningi omkhulu

Amagama aphelele nesibongo : Nokwanda Annatoria Gambushe:

Iziqu : Ndip: Clinical Technology

: B-Tech: clinical Technology (Cardiology)

Abaqhaphi bocwaningo

Amagama aphelele nesibongo : Dr F.A Mohamed

Iziqu : BSc

: MBchB

: FCP (SA)

: FRCP (London)

: Cert Endocrinology

Amagama aphelele nesibongo : Dr Rosaley Prakaschandra

Iziqu : PhD (Cardiology) (UKZN)

Isingeniso kanye nenhloso yocwaningo ngamafuphi

Isifo senhliziyo igama elisetshenziswayo ukuchaza izifo eziningi ezithinta inhliziyo kanye nemithambo yegazi okubandakanya ukuvaleka kwemithambo yenhliziyo,isifo sohlangothi kanye nesifo esihlasela izivalo zenhliziyo nokunye.

Uncwaningi Keates nabanye (2017) ngonyaka ka 2008 bathola ukuthi isifo senhliziyo sibulala abantu jikelele. Ngokombiko womnyango wezempilo emhl;abeni (2017) isifo senhliziyo sabulala 9.2% emazweni abantu abansundu ngo 2001.Umncwangingi

Hofman nabanye (2011) baveza ukuthi sicishe sifinyelele kubantu abawu 195 ababulawa isifo senhliziyo ngelanga eMzansi Africa.

Isizathu esenza sisungule lolucwaningo ukuthi ngokuka (Dokotela Thembela:2016),isibalo sabantu abasengcupheni yokuphathwa isifo senhliziyo siya ngokwanda entshonalanga yakwaZulu,nasesibhedlela eImadada.Ngokwazi kwaDokotela Thembela,akekho noyedwa oseke wabheka ukuthi lokhu kuyilimaza kanjani inhliziyo,yingakho ngasungula lolucwaningo.

Uncwaningo luhlose ukubheka iningi 136abantu abahlaselwe ukudlanga kwezinto ezigcina zikubeke engcupheni yokuthi ugcine usunesifo senhliziyo enyakatho neKwazulu-Natal.

Abangangenela isifundo

-Abasayine isivumelwano

-Abangaphezulu kweminyaka engamashumi nesishagalolunye kuya phezulu, akukhethe ngabala nangabulili.

-Osengcupheni yokuthola isifo senhliziyo.

Abangeke bakwazi ukungenela isifundo

-Ongasayinile isivumelwano

-Ongaphansi kweminyaka eyishumi nesishagalolunye

- Onesifo sohlangothi kanye nesifo senhliziyo, onesifo sezinso, nokhulelwe angeke angenelele esifundweni.

Uhlelo locwaningo

Umncwaningi uzohambela imitholampilo yeNyakatho neKwazulu Natal. Zonke iziguli zizohlangana nomncwaningi, azinikeze incwadi yolwazi ngocwaningo.Umngeneleli kuyomele avume aphinde asiyine ngaphambi kokuba siqale isifundo. Zonke iziguli ezisengcupheni zokuthola isifo senhliziyo ziyocwaningwa kuthathwe negazi,baphinde basitshene umlando ngempilo yabo,Lapho ke bazokalwa

ubude,isisindo,ukhalo selilonke ngokwabaka World Health Organization (WHO).Kuphinde kukalwe umfutho wegazi nokushaya kwenhliziyo, amfutha ayingozi kanye noshukela. Iphepha elinemibuzo liyobe seligcwaliswa umngeleli kanye nomcwaningi.Umcwaningi uyobe eseqoqa yonke imiphumela ngezivivinyo.

Ubungozi nokungaphatheki kahle komngeneli

Isampula legazi lizothathwa emtholampilo ngosuku obuze ngalo okudlula loko abukho obunye ubungozi noma ukungaphatheki kahle okuzo kwenzeka ngokungenela kwakho.

Okungazuzwa

Wonke umuntu ozobamba iqhaza kucwaningo kuzotholakala isifo sakhe ngokufanele kuphinde kusizakale odokotela ekulapheni isifo ngokufanele. Ukudlanga kwalezifo ezibekana engcupheni ukuthi ugcine unesifo senhliziyo kuzotholakala, kuphinde kutholakale izindlela zokuvimba noma ukwehlisa izinga lokuthola isifo senhliziyo. Umncwaningi uzohlomula ngokuthi isifundo siyoshicilelwa emabhukwini noma sithulwe emihlanganweni yokufunda.

Okungenza ungathandi ukubamba iqhaza kucwaningo

Ukungenela lolucwaningo akuphoqelekile. Isiguli sinelungelo lokuphuma ocwaningweni noma yinini.Ukuphuma kwakho ocwaningweni akuzoba namthelelo omubi kwisiguli, futhi angeke kuqede ubudlelwane phakathi kwesiguli kanye nabasebenzi basesithombeni senhliziyo esibhedlela eMadadeni.

Ukukhokhelwa

Akukho mvuzo wemali ozotholwa isiguli ngokubamba iqhaza kulo cwaningo.

Izindleko zocwaningo

Akukhomali ezokhokhwa isiguli ngokubamba iqhaza kulesisifundo. Kuzoba imali yesibhedlela ejwayelekile.

Imfihlakalo

Imininingwane yakho yonke yalolucwaningo ngeke ludalulwe kumuntu izogcinwa iyimfihlo.Kuzoba khona inombolo ozonikwa yona ekuzofakwa kuyo imininingwane yakho.Imiphumela etholwe emva kwalolucwaningo izofakwa ezincwadini zochwepheshe. Imiphumela izobekwa endaweni ephephile lapho umcwaningi omkhulu nenduna bazokwazi ukuyibona.

Ukulimala ngesikhathi socwaningo

Angeke kube khona ukulimala ngesikhathi socwaningo.

Abantu ongaxhumana nabo uma unenkhinga noma imibuzo mayelana ngocwaningo

Ungathintana nomcwaningi omkhulu: Nokwanda Gambushe (034 328 8327) noma abaqhaphi bocwaningo udokotela F.A Mohamed (082 377 8072) no dokotela R. Prakaschandra (031 373 6885). Ungaxhumana futhi nomnyango wezocwaningo nobulungiswa, kulenombolo 031-373 2375.

Ozithobayo
N. A Gambushe
Umcwaningi omkhulu

APPENDIX B: **INSTITUTIONAL RESEARCH ETHICS COMMITTEE (IREC): CONSENT LETTER**

Statement of agreement to participate in the research study:

- I hereby confirm that I have been informed by the researcher, Miss N. A Gambushe, about the nature, conduct, benefits and risks of this study.
- I have also received, read and understood the above written information (Participant Letter of Information) regarding the study.

- I am aware that the results of the study, including personal details regarding my sex, age, date of birth and initials will be anonymously processed into a study report.
- In view of the requirements of research, I agree that the data collected during this study can be processed in a computerised system by the researcher. In any stage, without prejudice, withdraw my consent and participation in the study.
- I have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the study.
- I understand that significant new findings developed during the course of this research, which may relate to my participation will be made available to me.

_____ _____ _____ _____ Full name of participant Date Time Signature

I, Miss Nokwanda Annatoria Gambushe, herewith confirm that the above participant has been fully informed about the nature, conduct and risks of the proposed research study.

_____ _____ _____
Full name of research Date Signature

_____ _____ _____
Full name of witness Date Signature

APPENDIX B1: INSTITUTIONAL RESEARCH ETHICS COMMITTEE (IREC): ISIFUNGO SOKUZIVUMELA

Isitatimende sesivumelwano sokuba yingxenye yocwaningo:

- Ngiyaqinisekisa ukuthi ngazisiwe ngumcwaningi, Nkosazana N.A Gambushe ngobunjalo ukuphathwa, inzuzo nokungaba yingozi ngalolucwaningo.
- Ngiyitholile, ngayifunda ngaqonda ngokubhalwe ngenhla (Encwadini yolwazi) maqondana nocwaningo.
- Ngiyazi ukuthi imiphumela yocwaningo nemininingwane yami mayelana nobulili, ubudala, usuku lokuzalwa, iziqalo zamagama ami nesifo esingiphethe angeke kuvezwe kumbiko wocwaningo.
- Ngenxa yezidingo zocwaningo, ngiyavuma ukuthi ulwazi oluqoqwe ngumcwaningi kulolucwaningo angalusebenzisa nge computer.
- Ngingayihoxisa imvume nokuba yingxenye yokuba yingxenye yalolucwaningo ngaphandle kokucwaswa.
- Ngibe nethuba elanele ukubuza imibuzo ngakho ke ngiyavuma ukuthi ngikulungele ukuba yingxenye yalolucwaningo.
- Ngiyaqonda ukuthi ngiyokwaziswa ngokusha okutholakele kulolucwaningo ngenxa yokuzimbandakanya kwami nalo.

_____ _____ _____ _____

Igama laloyingxenye Usuku Isikhathi Sayina

Mina Nokwanda Annatoria Gambushe, ngiyaqinisekisa ukuthi lona obhalwe ngasenhla oyingxenye yocwaningo wazisiwe ngobunjalo, ukuphatha nokungaba yingozi obuphathelene nalolu- cwaningo.

_____ _____ _____

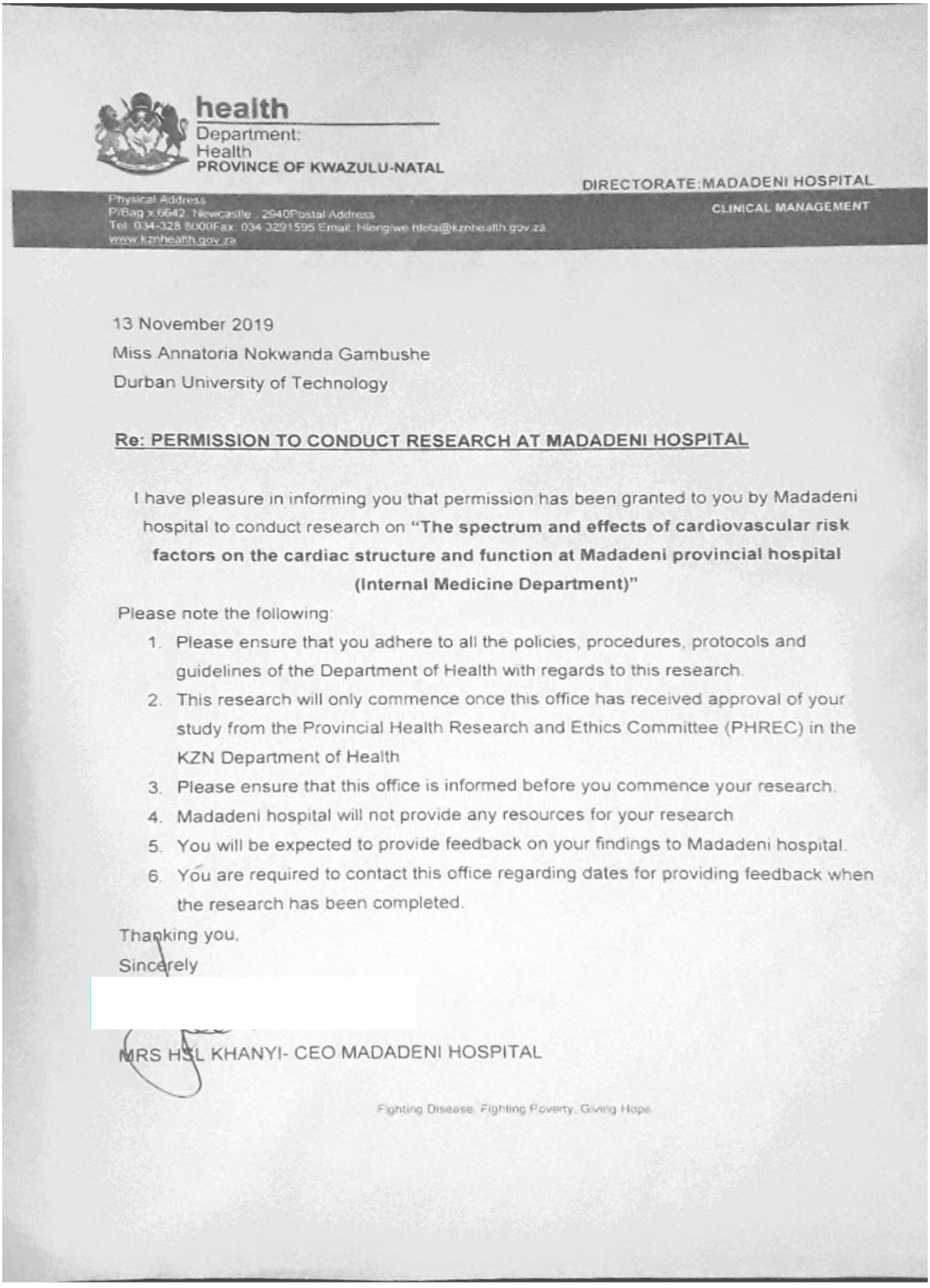
Igama lomcwaningi Usuku Sayina

_____ _____ _____

Igama lomfakazi Usuku Sayina

APPENDIX

C: MADADENI PROVINCIAL HOSPITAL APPROVAL LETTER



health
Department:
Health
PROVINCE OF KWAZULU-NATAL

DIRECTORATE:MADADENI HOSPITAL
CLINICAL MANAGEMENT

Physical Address
P/Bag x 6642 Newcastle , 2940Postal Address
Tel: 034-328 8000Fax: 034 3291595 Email: Hlengiwe.hlela@kznhealth.gov.za
www.kznhealth.gov.za

13 November 2019

Miss Annatoria Nokwanda Gambushe

Durban University of Technology

Re: PERMISSION TO CONDUCT RESEARCH AT MADADENI HOSPITAL

I have pleasure in informing you that permission has been granted to you by Madadeni hospital to conduct research on **"The spectrum and effects of cardiovascular risk factors on the cardiac structure and function at Madadeni provincial hospital (Internal Medicine Department)"**

Please note the following:

1. Please ensure that you adhere to all the policies, procedures, protocols and guidelines of the Department of Health with regards to this research.
2. This research will only commence once this office has received approval of your study from the Provincial Health Research and Ethics Committee (PHREC) in the KZN Department of Health
3. Please ensure that this office is informed before you commence your research.
4. Madadeni hospital will not provide any resources for your research
5. You will be expected to provide feedback on your findings to Madadeni hospital.
6. You are required to contact this office regarding dates for providing feedback when the research has been completed.

Thanking you,

Sincerely

MRS HSL KHANYI- CEO MADADENI HOSPITAL

Fighting Disease, Fighting Poverty, Giving Hope

APPENDIX

D: KZN HEALTH APPROVAL LETTER

KWAZULU-NATAL PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

Postal Address: Private Bag X9050
Physical Address: 330 Langalibalele Str; PM Burg; 3201
Tel: 0333953189/3123/2805 Fax: 033-3943782
Email address: hrkm@kznhealth.gov.za
www.kznhealth.gov.za

DIRECTORATE:
Health Research & Knowledge Management Unit

NHRD Ref: KZ_202102_035

Dear Ms N A Gambushe
(DUT)

Approval of research

1. The research proposal titled **'The spectrum and the effects of cardiovascular risk factors on the cardiac structure and function at Madadeni Provincial Hospital (Internal Medicine Department)'** was reviewed by the KwaZulu-Natal Department of Health (KZN-DoH).

   The proposal is hereby **approved** for research to be undertaken at Madadeni hospital.

2. You are requested to take note of the following:

   a. *All research conducted in KwaZulu-Natal must comply with government regulations relating to Covid-19. These include but are not limited to: regulations concerning social distancing, the wearing of personal protective equipment, and limitations on meetings and social gatherings.*
   b. *Kindly liaise with the facility manager BEFORE your research begins in order to ensure that conditions in the facility are conducive to the conduct of your research. These include, but are not limited to, an assurance that the numbers of patients attending the facility are sufficient to support your sample size requirements, and that the space and physical infrastructure of the facility can accommodate the research team and any additional equipment required for the research.*
   c. *Please ensure that you provide your letter of ethics re-certification to this unit, when the current approval expires.*
   d. *Provide an interim progress report and final report (electronic and hard copies) when your research is complete to **HEALTH RESEARCH AND KNOWLEDGE MANAGEMENT, 10-102, PRIVATE BAG X9051, PIETERMARITZBURG, 3200** and e-mail an electronic copy to hrkm@kznhealth.gov.za*
   e. *Please note that the Department of Health shall not be held liable for any injury that occurs as a result of this study.*

For any additional information please contact Ms G Khumalo on 033-395 3189.

Yours Sincerely

Dr E Lutge
Chairperson, Health Research Committee
Date: 24/03/2021

GROWING KWAZULU-NATAL TOGETHER

APPENDIX

E: DUT ETHICAL APPROVAL LETTER

Institutional Research Ethics Committee Research and Postgraduate Support Directorate 2nd Floor, Berwyn Court
Gate 1, Steve Biko Campus Durban University of Technology

P O Box 1334, Durban, South Africa, 4001 Tel: 031 373 2375
Email: lavishad@dut.ac.za
http://www.dut.ac.za/research/institutional_research_ethics

www.dut.ac.za

1 April 2021

Ms N A Gambushe
10 Highlands Estate Madadeni
Section 4
2951

Dear Ms Gambushe

The spectrum and the effects of cardiovascular risk factors on the cardiac structure and function at Madadeni Provincial Hospital (Internal Medicine Department) Ethical Clearance number IREC 170/20

The Institutional Research Ethics Committee acknowledges receipt of your gatekeeper permission letter.

Please note that FULL APPROVAL is granted to your research proposal. You may proceed with data collection.

Any adverse events [serious or minor] which occur in connection with this study and/or which may alter its ethical consideration must be reported to the IREC according to the IREC Standard Operating Procedures (SOP's).

Please note that any deviations from the approved proposal require the approval of the IREC as outlined in the IREC SOP's.

APPENDIX

Sincerely

Dr K Padayachy
Deputy Chairperson: IREC

F: TRAINING AND RESOURCES IN RESEARCH ETHICS EVALUATION (TRREE) CERTIFICATES

Zertifikat
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Certificado
Certificate

TRREE

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CID : znGDVrVHOL
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Coordinateur TRREE Coordinator
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CID : K7GCwtVki1

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Coordinateur TRREE Coordinator

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[REV : 20170310]

APPENDIX G: IMAGING PROTOCOL FOR ECHOCARDIOGRAPHY LAB AT MADADENI PROVINCIAL HOSPITAL

health

Department:
Health
PROVINCE OF KWAZULU-NATAL

INTERNAL MEDICINE DEPT
Madadeni Provincial Hospital
Cnr Nkosi Albert Luthuli & Isilo udinuzulu Str
Box 6642, Newcastle, 2940
Tel.034 328 8327, Fax.034 329 1595

ECHOCARDIOGRAPHY

Aim for this procedure: To assess the cardiac function and structures.

EQUIPMENTS USED IN THIS PROCEDURE:

- Disposable electrodes

- Shaving blades
- Swabs
- Acetone solution
- Echocardiography machine
- Ultrasound gel

PROCEDURE

- Read the patient's history so that you will know and have a better understanding of the indication for the procedure. Reading a patient's history gives you an indication of what to expect during the test.
- Welcome and greet the patient.
- Explain the procedure to the patient and how the patient should lie.
- Once that is done, connect the ECG (3 leads).
- A patient should lie in a Left Lateral decubitus position where a patient should recline at 45 degrees, lying on the left hand side with the right arm by the side and the left arm behind the head. This brings the heart to the chest wall and more to the left of the sternum which improves the ultrasound window.
- Apply an ultrasound gel to the transducer probe so that you can be able to see the image of the heart.
- Place the transducer probe according to which view you want.
- Report and record everything that is seen as you are scanning.
- Once you are satisfied with the results, inform the patient that you are done and offer a tissue to the patient to wipe away the ultrasound gel.

- Dismiss the patient.

STANDARD VIEWS

Parasternal Long axis view

The transducer probe is placed at the left parasternal edge in the 2^{nd}-4^{th} intercostal space. The transducer should be aligned so that the marker is pointing towards the right shoulder. This view is very useful to estimate the size and contractility of the right and left ventricle (septum and posterior wall), to assess the morphology and function of the mitral and aortic valves. With colour Doppler you can look for an aortic or mitral regurgitation and try to determine their mechanism. Echo studies begin with this view.

If you angle down, the tricuspid valve together with the right ventricle may be visualised.

If angling up, both the left and right pulmonary arteries may be seen.

Parasternal Short axis view

It is obtained by rotating the transducer 90 degrees towards the left shoulder.

This view has 3 levels

1. Aortic valve level.
2. Papillary muscle level: Is used usually for the purposes of describing abnormal LV wall motion and measuring the End Diastolic Dimension and End Systolic Dimension to determine Ejection Fraction. LV wall thickness can also be assessed.
3. Mitral valve level: This is used for measuring the mitral valve area.

Apical views

Views the apex of the heart and the transducer probe is directed to the left side of the patient's hand.

Apical 4 chamber

Transducer position: apex of heart.

Marker dot direction: points towards the bed on the left shoulder side .

The view is obtained from this view by slight anterior angulation of the transducer towards the chest wall.

Apical 5 chamber: This view incorporates the left ventricular outflow tract as a fifth chamber. It can be used to assess the aortic valve peak gradient.

Apical 2 chamber

This involves rotation of the probe from the standard 4 chamber view 60 degrees anticlockwise so that the marker points towards the left shoulder. Assessment of LV anterior wall and LV inferior wall.

The apical windows are the most useful for:

1) Assessing flow across the aortic, tricuspid and mitral valves with Doppler as flow across them is parallel to the direction of the ultrasound beam. This information can be used directly to assess for degree of stenosis and regurgitation.

2) Assessment of diastolic function, including use of tissue Doppler and pulsing across the pulmonary veins, up to 3 of which may be visible in this position.

3) Assessment of RV size and function including use of tricuspid annular plane systolic excursion (TAPSE).

4) Evaluation of LV segmental wall motion and evaluation of LV thrombus (the apex is in the near field), sometimes with help of an IV echo contrast agent.

5) Assessment for an Atrial Septal Defect in conjunction with the use of saline contrast.

Subcostal view and the suprasternal view can also be used more especially in paediatrics

ECHOCARDIOGRAPHY MODALITIES

2D measurements

This technique is used to "see" the actual structures and motion of the heart structures at work.

M-mode measurements

These measurements are thus very dependent upon lining up your M-mode correctly with the LV. Septal wall and posterior wall thickness may be measured and an LV ejection fraction. Aortic root diameter and LA diameter can also be measured.

Doppler echocardiography is a method for detecting the direction and velocity of moving blood within the heart.

Pulsed Wave (PW) useful for low velocity flow in a small region e.g. Mitral Valve flow.

Continuous Wave (CW) useful for high velocity flow in a general region e.g. aortic stenosis.

Colour Flow Doppler (CFD): Different colours are used to designate the direction of blood flow. **Red** is flow towards, and **Blue** is flow away from the transducer with turbulent flow shown.

Tissue doppler imaging: Kadappu and Thomas (2015) state that tissue doppler imaging (TDI) is an echocardiographic technique that is used to evaluate myocardial diastolic and systolic function throughout the cardiac cycle. The myocardial velocity curve is the same as the transmitral inflow pattern but inverted (Otto: 2013).

According to Tissot *et al.* (2018), pulsed wave doppler is placed on either basal septal or lateral region and myocardial velocities are calculated.

The first early diastolic velocity is abbreviated as E' (E-prime) following a second peak end diastolic velocity known as the A' (A-prime). The peak systolic annular velocity (S') measures the left ventricular function and has a normal value of >10 cm/s (Tissot *et al*. 2018).

APPENDIX H: WHO QUESTIONNAIRE

For Chronic Disease Risk Factor Questionnaire: English

Survey Information

Location and date of the study	Response
1. Cluster/Centre/Village name	
2. Interviewer ID	
3. Date of completion of the questionnaire	
4. Cell phone number	
5. Cardiovascular risk factor (s)	

Consent, Preferred language and Name	Response
1. Consent has been read and obtained	
2. Interview Language	

3. Family Surname and first name	

Step 1: Demographic Information

Demographic Information	Response
Sex	
What is your date of birth?	
age	
What is the highest level of education	
you have completed?	
What is your ethnic group / racial group?	
What is your marital status?	
Which of the following best describes your main work status over the past 12 months?	
How many people older than 18 years, including Number of people yourself, live in your household?	

Behavioural Measurements

Do you currently smoke any tobacco products, such as cigarettes, cigars or pipes?	

Do you currently smoke tobacco products daily?	
How old were you when you first started smoking daily?	
On average, how many of the following do you smoke each day?	

Alcohol Consumption

Have you ever consumed an alcoholic drink such as beer, wine, spirits, fermented cider?	
Have you consumed an alcoholic drink within the past 12 months?	

Diet

In a typical week, on how many days do you eat fruit	
How many servings of fruit do you eat on one of those days?	
In a typical week, on how many days do you eat vegetables?	
What type of oil or fat is most often used for meal preparation in your household?	

On average, how many meals per week do you eat that were not prepared at a home?	

Physical Activity

Does your work involve vigorousintensity activity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work for at least 10 minutes continuously?	
In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	
Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places?	
How much time do you usually spend sitting or reclining on a typical day?	

History of Raised Blood Pressure

Have you ever had your blood pressure measured by a doctor or other health worker?	

Have you ever been told by a doctor or other health worker that you have raised blood pressure or hypertension?	
Have you been told in the past 12 months?	

Are you currently receiving any of the following treatments/advice for high blood pressure prescribed by a doctor or other health worker?

Drugs (medication) that you have taken in the past two weeks	
Advice to reduce salt intake	
Advice or treatment to lose weight	
Advice or treatment to stop smoking	
Advice to start or do more exercise	
Have you ever seen a traditional healer for raised blood pressure or hypertension?	
Are you currently taking any herbal or traditional remedy for your raised blood pressure?	

History of Diabetes

Have you ever had your blood sugar measured by a doctor or other health worker?	

Have you ever been told by a doctor or other health worker that you have raised blood sugar or diabetes?	
Have you been told in the past 12 months?	

Step 2: Physical Measurements

Height	
Weight	
Waist circumference	
Hip circumference	
Waist-to-Hip ratio	
Heart rate: Reading 1	
Reading 2	
Reading 3	
Cuff size used	
Blood Pressure: Reading 1	
Reading 2	
Reading 3	
During the past two weeks, have you been treated for raised blood pressure	
with drugs (medication) prescribed by a doctor or other health worker?	

Fasting blood glucose Choose accordingly: mmol/l or mg/dl	
Today, have you taken insulin or other drugs (medication) that have been prescribed by a doctor or other health worker for raised blood glucose?	
Total cholesterol Choose accordingly: mmol/l or mg/dl	
Triglycerides Choose accordingly: mmol/l or mg/dl	
HDL Cholesterol Choose accordingly: mmol/l or mg/dl	
During the past two weeks, have you been treated for raised cholesterol with drugs (medication) prescribed by a doctor or other health worker?	

APPENDIX H1: WHO QUESTIONNAIRE

For Chronic Disease Risk Factor Questionnaire: IsiZulu

Imbibuzo ngocwaningo

Indawo nosuku lemibuzo	Impendulo
1.Igama lendawo	

2. Umasizi wobambe iqhaza	
3. Usuku lwemibuzo	
4.Inombolo yocingo	
5. kwezinto ezigcina zikubeke engcupheni ukuthi ugcine usunesifo senhliziyo	

Isifungo, Ulimi olukhethiwe kanye negama nesibongo	Impendulo
1. Isifungo sifundiwe savunyelwa	
2. Ulimi locwaningo	
3. Isibongo negama	

Isigaba 1: Ulwazi

Ulwazi ngobambe iqhaza	
ubulili	
Usuku,inyanga nonyaka wokuzalwa	
Iminyaka	
Uqede kuliphi ibanga?	
Ubuzwe buphi?	
Isimo sakho somshado	
Isimo sakho somsebenzi	

Bangaphi ekhaya abangaphezu kweminyaka eyishumi nashagalombili	

Isilinganiso sesimilo

Kukhona okubhemayo	
Uyabhema ngosuku	
Wawuneminyaka emingaphi mawuqala	
Ubhema kangaphi ngosuku?	

Uphuzo oludakayo

Kukhona okudakayo okuphuzayo	
Ezinyangeni eziyishumi nesbili,usuke waphuza isiphuzo esidakayo	

Ukudla

Esontweni,ulidla kangaphi isithelo	
Zisuke zingaphi izithelo	
Esontweni, uyidla kangaphi imifino	
Usebenzisa maphi amafutha mawupheka ukudla	
Esontweni,udla kangaphi ngaphandle	

Ukuzivocavoca

Uyazivocavoca	
Esontweni,uzivocavoca kangaphi	
Uyahamba noma usebenzise ibhayisekili	
Singaphi isikhathi osichithayo ulele osukwini	

Umlando ngehayi hayi

Wake wayihlolela ihayi hayi umhlengikazi	
Wake watshenwa umhlengikazi ukuthi unehayi hayi	
Uthsenwe ezinyangeni eziyishumi nambili	

Usuke wayalwa umhlengikazi mayelana nokuthola ulwazi nge hayi hayi nesigo sashukela

Imithi	
Ukwehlisa usawoti	
Ukuyalwa ngokwehlisa isisindo	
Ukuyalwa ngokuyeka ukubhema	
Ukuyalwa ngokuzivocavoca	
Usuke wayibona inyanga mayelana ne hayi hayi noma ushukela	
Usuke wayithatha amakhambi ukuwelapha ihayi hayi noma ushukela	

Umlando ngesifo sashukela

Wake wawuhlolela ushukela umhlengikazi	
Wake watshenwa umhlengikazi ukuthi ushukela ephezulu?	
Utshenwe ezinyangeni eziyishumi nambili	

Isigaba 2: Isilinganiso somzimba

Ukuphakama	
Isisindo	
Ukujikeleza kwesinqe	
Ukujikeleza kwe hip	
Isinqe ne hip ratio	
Isilinganiso senhliziyo: Ukufunda 1	
Ukufunda 2	
Ukufunda 3	
Usayizi wecuff	
Ukushaya kwegazi: Ukufunda 1	
Ukufunda 2	
Ukufunda 3	

Emasontweni amabili adlule,usuke walashelwa ihayihayi udokotela?	
Ushukela	
Namhlanje,usuke wawathatha amaphilisi ashukela?	
Amafutha omzimba	
Amafutha omzimba	
Amafutha omzimba	
Emasontweni amabili adlule,usuke walashelwa amafutha udokotela?	