Prevalence of hypertension determined by 24-hours ambulatory blood pressure monitoring among MUHAS employees	
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MMed (Internal Medicine) Dissertation Muhimbili University of Health and Allied Sciences October, 2019	

# MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES SCHOOL OF MEDICINE

# DEPARTMENT OF INTERNAL MEDICINE



# PREVALENCEOF HYPERTENSIONDETERMINEDBY24-HOURS AMBULATORYBLOODPRESSUREMONITORING AMONG MUHASEMPLOYEES

By,

**Godfrey Chuwa, MD** 

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Masters of Medicine (Internal Medicine) of

Muhimbili University of Health and Allied Sciences October, 2019

## **CERTIFICATION**

The undersigned certify that, she has read and hereby recommend for examination by Muhimbili University of Health and Allied Sciences a dissertation entitled, "Prevalence of hypertension determined by 24-hours ambulatory blood pressure monitoring among MUHASemployees" in (partial) fulfillment of the requirement for the degree of Master of Medicine (Internal Medicine) of the Muhimbili University of Health and Allied Sciences.

Dr. Pilly Chillo (MD, MMED, PhD)
(Supervisor)

Date

## **DECLARATION AND COPYRIGHT**

I, Dr. Godfrey Chuwa, declare that this dissertationis my own original work and that it has
not been presented to any other University for a similar or any other degree award.

<b>C</b> . 4	D (
Signature	Date

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## **ACKNOWLEDGEMENTS**

I would first like to thank almighty God for his unconditional love and countless blessings through all these years that have been studying.

Secondly,I would also like to extend my sincere gratitude to Dr.Pilly Chillo,my supervisor for her teachings,guidance and time throughout this research work. She has endured a lot of sacrifices to make sure this research work will be completed in a timely manner. Her unwavering support is unprecedented and unmatched and I'm so proud ofher being my teacher. I would also like to thank all the MUHAS employees who participated in this research work. Your contribution will never gounnoticed.

I would also like to thank my family for their love, prayers and support during all this time that have been busy with studies. Special thanks to my wife Lilian and my daughter Natalie.

Lastly,I would also like to thank all my colleagues for their inputs and support.

# **DEDICATION**

To my beloved family for all their sacrifices for me

#### **ABSTRACT**

**Background:** Prevalence of hypertension is rising in sub-Saharan Africa, especially among urban middle-class individuals. In Tanzania, majority of previous studies on hypertension were based on elevation of clinic blood pressure (BP). However, clinic BP may over-estimate the true prevalence of hypertension due to white coat effect, may miss to diagnose individuals with masked hypertension and miss to identify nocturnal non-dippers. There is also increasing evidence that Ambulatory Blood Pressure Monitoring (ABPM) is more accurate than clinic BP in predicting cardiovascular risk.

**Objective**: To determine the prevalence of hypertension using ABPM and to explore the relationship between ABPM profiles with cardiovascular risk factors among MUHAS employees.

**Methods**: A descriptive cross-sectional study was conducted from October 2018 to February 2019. A structured questionnaire was used to gather information on socio-demographic characteristics and cardiovascular risk history. Anthropometric measurements were taken and blood samples were collected and analyzed for glucose, creatinine, cholesterol and uric acid. Two sets of BP were taken; one at the clinic and another using 24hrs ABPM. Data analysis was done using SPSS Version 20, and a p-value of <0.05 was considered statistically significant.

**Results:** This study had 390 participants. Their mean (SD) age was 40.5(8.9) years, and 53.6% were men. Prevalence of hypertension was found to be 23.1%. Prevalence for white coat, masked and nocturnal non dippers were 16.2%, 11.6% and 66.7% respectively. The mean 24-hours BP showed the best correlations with traditional cardiovascular risk factors. In multivariate analysis, independent factors associated with hypertension were male gender (OR=7.96), age  $\geq 40$  years (OR=3.94), family history of hypertension (OR=5.6), central obesity (OR=8.98), hypercholesterolemia (OR=3.84) and hyperuricemia (OR=7.9), all p<0.01.

**Conclusion and Recommendation:** The prevalence of hypertension among MUHAS employees is 23.1% and is associated with traditional cardiovascular risk factors. Ambulatory BP correlates better with cardiovascular risk factors and is the best measure of one's true BP Work-based hypertension control programs are recommended at MUHAS.

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## LIST OF ABBREVIATIONS

ABPM Ambulatory Blood Pressure Monitoring

BMI Body Mass Index

BP Blood Pressure

BUN Blood Urea Nitrogen

BWT Body Weight

CKD Chronic Kidney Disease

DBP Diastolic Blood Pressure

DM Diabetes Mellitus

eGFR Estimated Glomerular Filtration Rate

FBG Fast Blood Glucose

HDL-C High Density Lipoprotein-c

LDL-C Low Density Lipoprotein-c

MAMC Muhimbili Academic Medical Centre

MAP Mean Arterial Pressure

MUHAS Muhimbili University of Health and Allied Sciences

#### **DEFINITION OF KEY TERMS**

**Ambulatory Blood Pressure Monitoring:** Is measurement of BP performed with the individual wearing a portable BP measuring device, usually on the non-dominant arm for a 24-hour period so that it gives information on BP during daily activities and at night during sleep.

Clinic Blood Pressure: Also known as Office BP - is the BP measurement taken within a hospital/clinic environment. The BP measurements at the hospital environment are subject to variations due to a white coat effect, which can over-estimate the BP readings as a result of the nervousness caused of being in a hospital environment.

White Coat Hypertension: Is defined as BP that is consistently elevated by clinic readings but does not meet diagnostic criteria for hypertension based upon ABPM or out of clinic BP measurements.

**Masked Hypertension:** Is defined as BP that is consistently elevated by ABPM and out-of-clinic measurements but does not meet the criteria for hypertension based upon clinic readings.

**Nocturnal Non-Dipping:** Is a phenomenon whereby BP fails to drop by at least 10 percent during sleep.

**Cardiovascular Risk Factors:** These are habits, environmental conditions or genetic factors that predispose an individual to develop a cardiovascular disease

## **CHAPTER ONE**

## 1.1 Introduction

Hypertension is an important public-health challenge worldwide. It is the most important cardiovascular risk factor and according to World Health Organization (WHO) data, worldwide hypertension is responsible for 51% of stroke, 39 – 59% of heart failure and 45% of ischemic heart disease(1). Furthermore, hypertension is responsible for 28.4% of chronic kidney disease in the United States of America (2), and 21% of chronic kidney disease in South Africa (3). The relationship between BP and risk of cardiovascular disease is continuous, consistent, and independent of other risk factors. In people with hypertension, the risk of cardiovascular diseases doubles for each incremental increase of 20/10mmHg of BP, starting as low as 115/75mmHg(4). Furthermore, the presence of any additional cardiovascular risk factor augments the health risk brought about by hypertension(5).

Globally, the overall prevalence of hypertension is reported to be around 40% among people aged ≥25 years(6). Hypertension prevalence is highest in Africa(46%) as compared to other parts of the world, with Americahaving the least prevalence (35%)(6). Due to the epidemiological transition currently taking place in the African region, population estimates indicate that hypertension will continue to rise, and by 2030 non-communicable diseases including hypertension will by-pass communicable diseases as the most important cause of Disability Adjusted Life Years (DALYs) in Africa(7). In Africa, the prevalence of hypertension is characteristically higher among urban dwellers(8-10) and those in middle and high socio-economic ranks(11, 12), posing a great economic challenge by causing premature morbidity and mortality(13)among the continent's educated workforce especially needed to effectively work in the different developmental areas like education, health, industries, etc.

In African population studies, hypertension has been found to be associated with other cardiovascular risk factors including older age, overweight and obesity, hypercholesterolemia, hyperuricemia, diabetes mellitus, smoking, sedentary life style, increased salt intake as well as stressful life which is commonly seen among the urban population(10, 14-17). All these factors are attributed to changes in lifestyle and urbanization(13). Apart from environmental

and lifestyle factors, genetics also play a major role in facilitating hypertension occurrence (18) and according to family and twin studies, the heritability of hypertension ranges from 24% to 50%(19, 20).

The present study aimed at determining the prevalence of hypertension among MUHAS employees, who can be regarded as urban and middle class working population in Tanzania. Furthermore, the study determined the prevalence of hypertension by use of 24-hours ABPM which has been considered superior in diagnosing true hypertension(21, 22). This is because, as opposed to the classical hypertension diagnosisby screening or clinic BP measurements, diagnosing hypertension by use of 24-hours ABPM provides a detailed understanding of one's BP changes with daily activities and during sleep. ABPMalso excludes people with white coat hypertension which is hypertension that is seen in the clinic orduringscreening but that normalizes while at home. White coat hypertension has been reported to be presentin as high as 30% in the general population(23). ABPM also gives an added advantage of identifying people with normal BP measurements when these are taken at the clinic but raised BP while at home; known as masked hypertension. In addition, 24-hours ABPM has the ability to identify individuals with persistent high BP levels during sleep (non-dippers), itself an independent predictor of cardiovascular events including stroke(24, 25). Among known hypertensives on treatment, ABPM has an advantage of monitoring the level of BP control over a 24-hours' period.

## **1.2 Literature Review**

## 1.2.1 Prevalence of hypertension

According to WHO, the prevalence of hypertension was estimated to be 40% among adults aged 25 years or more in the general population globally in 2008(6). These estimates reported a modest fall of proportion of the population with hypertension between 1980 and 2008, however because of population growth and ageing, the absolute number of people with hypertension rose from 600 million in 1980 to nearly 1 billion in 2008(6). According to WHO, Africa has the highest prevalence of hypertension 46% compared to other part of the worldand America has the least prevalence 35%(6). In all WHO regions, men had slightly higher prevalence of hypertension than women, and across income groups of countries, the prevalence of hypertension were consistently high, with low, lower middle and upper middle countries all having rates of around 40%, while the prevalence in high income countries was lower at 35%(6).

In a recent publication that examined the global disparities of hypertension prevalence by world regions with different levels of economic development in 2010, involving 90 countries worldwide, it was found that 31.1% of the world's adults aged 20 years or older had hypertension(26). The prevalence of hypertension was higher in low and middle income countries at 31.5% while it was 28.5% in high income countries. An estimated absolute number of 1.39 billion people had hypertension in the same year (2010) and of these 349 million were in high income countries while 1.04 billion were in low and middle income countries(26). **Furthermore**, from 2000 to 2010, the age-standardized prevalence of hypertension decreased by 2.6% in high income countries but increased by 7.7% in low and middle income countries(26).

In Africa, particularly sub Saharan Africa where hypertension prevalence was rare until few decades ago (27, 28) and was very low until recently(29-31)marked increase in the prevalence of hypertension has been seen over time(32). For instance, studies on hypertension prevalence in the 80s consistently reported lower rates of hypertension in the sub Saharan Africa communities ranging from as low as 2.6% in men and 3.4% in women of Mara region in

Tanzania (30)to 9.2% of men and 12.9% of women in Cape Peninsula, South African blacks(31). However, to-date hypertension is one of the most common conditions in sub Saharan Africa and it is estimated that out of the approximately 650 million people in sub Saharan Africa, between 10 to 20 million may have hypertension (33). Furthermore, it has been suggested that the prevalence of cardiovascular diseases and hypertension are increasing rapidly in sub Saharan Africa and the current prevalence in many sub Saharan Africa countries, particularly in urban areas, is already as high as that seen in developed countries(34, 35). The number of adults with hypertension in 2025 is predicted to increase by about 60% to a total of 1.56 billion, with disproportionate prevalence in developing countries including sub Saharan Africa(35).

Characteristically, hypertension in sub Saharan Africa affects younger people, is associated with fast progression towards end organ damage and causes severe morbidity and mortality than any other part of the world(36). Furthermore, there is a clear difference in urban and rural settings such that in communities where the traditional lifestyles of eating and engaging in physical activities of farming are practiced, hypertension is still low and on the other hand in urban settings where lifestyles have become Westernized, hypertension is a major public health problem affecting as many as 50% of those aged 55 years or more(9).

In a study done to document the burden of hypertension in four sub Saharan countries involving nurses in Nigeria, primary school teachers in Tanzania and South Africa as well as peri-urban and rural residents in Uganda, the overall age-standardized prevalence of hypertension among the 1,216 participants was 25.9%, while the crude prevalence was 36.9% (32). A study by Hendriks et al that assessed prevalence of hypertension and determinants of raised BP in rural Nigeria, rural Kenya, urban Tanzania and urban Namibia between 2009 – 2011 found crude prevalence of hypertension to range from 19.0% to 32%, with age-standardized prevalences of 19.3%, 21.4%, 23.7% and 38.0% in Nigeria, Kenya, Tanzania and Namibia, respectively(9).In yet a more recent study by Gomez-Olive et al that looked on the prevalence of hypertension among older adults in rural and urban settings located in West (Burkina Faso), East (Kenya) and Southern Africa (South Africa), including 10,696 adults aged 40-60 years found the mean prevalence of hypertension to range from 15.1% in Burkina

Faso to 54.1% in South Africa(37). In the same study, the overall prevalence of hypertension in the entire cohort was 33.3%(37).

Similar high prevalences have been reported in almost all other sub Saharan African countries, and in all studies that looked at the urban-rural hypertension prevalence, consistently the prevalence of hypertension is reported to be higher in urban than rural communities(8). In Angola, available studies on hypertension prevalence estimate the burden to range from 23% to 45.2%(12, 17, 38). A study done in rural and urban Ghana found the age-adjusted prevalence of hypertension among 1,431 participants aged 18 years and above to be 29%(39). Age-adjusted mean systolic BP and diastolic BP levels were lower in rural men and women when compared to urban dwellers and urban dwellers were reported to be more likely to be aware of their hypertension status than their rural counterparts(16). A crude prevalence of hypertension was found to be 34.8% in a population survey involving 1,928 individuals aged ≥25 years old in Lusaka District households in Zambia(15). The overall pooled prevalence of hypertension was found to be 30% in a meta-analysis study to quantify the burden of hypertension in Zimbabwe(40). The analysis included four studies conducted between 1997 − 2000 across five provinces in Zimbabwe and involved 4,829 adults. The authors concluded that the prevalence of hypertension was higher in urban than rural areas in Zimbabwe(40).

A number of studies have looked on the prevalence of hypertension among different workers in sub Saharan Africa and the results show different prevalence of hypertension among different working populations. A systematic review conducted to assess the prevalence of hypertension among formal and informal sector workers in West Africa, involving 30,727 participants aged ≥15 years found the prevalence of hypertension to range from 12.0% among automobile garage workers to 68.9% among traditional Chiefs(11). Furthermore, in 15 of the 45 studies involved in the review, the prevalence exceeded 30% and it was reported that typically sedentary workers such as traders, bankers, civil servants and Chiefs were at high risk of having hypertension(11). In a study that involved nurses, teachers, and general population in Tanzania, South Africa, Uganda and Nigeria, nurses had higher prevalence of hypertension, followed by teachers and lastly the general population(32).

In Tanzania, the prevalence of hypertension and its associated risk factors have been reasonably widely studied and similar to other sub Saharan African countries, the prevalences are consistently high and increasing over time(14, 30, 41-43). Again, similar to studies on hypertension prevalence from the region, majority of the studies on hypertension prevalence have been done using the conventional screening or clinic BP measurements. Only a few studies in sub Saharan Africa(44-46) and only one study in Tanzania(47)have used ABPM to estimate the prevalence of hypertension in the population. The prevalence of hypertension was found to be 25.9% in a nation-wide population survey of adults aged 25 – 65 years in Tanzania involving 5,680 adults as part of the STEPS Survey that was conducted in 2012(14). Other cardiovascular risk factors observed in the STEPS survey include tobacco use 15.9%, alcohol consumption 29.3%, low fruit/vegetable intake 97.2%, overweight and obesity 26%, high cholesterol 26%, high triglycerides 33.8% and diabetes 9.1%(14). A community-based cross sectional study that was conducted in adults of Magu District in 2013 found the prevalence of hypertension and pre-hypertension to be 8.0% and 36.2% respectively(42). In this study, older age, area of residence, overweight and obesity were major risk factors to develop hypertension(42). In the only study that used ABPM in Tanzania, a representative sample of 79 elderly participants (>70 years old) from a baseline cohort of 2,322 people in Hai District in Kilimanjaro was studied(47). The researchers found the prevalence of hypertension to be 55.7%, which was less than the prevalence found in the same group using conventional screening BP measurements (78.4%)(47).

## 1.2.2. Hypertension as the main cardiovascular risk factor

Hypertension remains the leading single contributor to global burden of disease and mortality accounting for approximately 9.4 million deaths annually, more than elevated body mass index (BMI), fasting plasma glucose, and total cholesterol combined(5, 6, 48). As of 2008, almost 1 billion people had uncontrolled hypertension worldwide (49). When not controlled, the effects of hypertension are devastating and include stroke, myocardial infarction, cardiac failure and renal failure among others(5, 8, 35, 50). In contrast with other cardiovascular risks such as high BMI, the burden of hypertension is greater in lower income countries than higher income settings (51). Furthermore, multiple risk factors positively interact with hypertension

to exacerbate cardiovascular disease risks. Hypertension for example, combined with unhealthy diets (high sodium, low potassium, and highalcohol consumption), high BMI and lack of physical activities has a multiplicative negative effect on cardiovascular disease mortality and DALYs(5).

In 2009 the WHO reported the leading global health risks for mortality in the world to be high BP which was responsible for 13% of deaths globally, followed by tobacco use (9%), high blood glucose (6%), physical inactivity (6%), and overweight and obesity which was responsible for 5% of global health risk of mortality(1). These risks are responsible for raising the risk of chronic diseases such as heart disease, diabetes and cancers. They affect countries across all income countries; high, middle and low(1). In a study done to estimate deaths and disability adjusted life years lost (DALY's) attributable to the independent effects of 67 risk factors and clusters of risk factors for 21 regions in 1990 and 2010, it was found that 3 leading risk factors for global disease burden in 2010 were high BP which was responsible for 7.0% of global DALYs, tobacco smoking including second hand smoke accounted for 6.3% and alcohol use 5.5% while in 1990 the leading risks were childhood underweight which accounted for 7.9%, household air pollution from solid fuels (7.0%) and tobacco including second hand smoking which accounted for 6.1% DALYs in 1990(5). The researchers concluded that worldwide, the contribution of different risk factors to disease burden has changed substantially, with a shift away from risks for communicable diseases in children towards those for non-communicable diseases in adults. These changes are related to the ageing population, decreased mortality among children younger than 5 years, changes in cause of death composition, and changes in risk factors exposures(5).

In a study by Hendriks, et al that looked on hypertension and other cardiovascular risk factors prevalence in four sub Saharan African countries namely Tanzania, Nigeria, Namibia and South Africa, hypertension was the most prevalent risk factor for cardiovascular disease in all four populations(9). The crude prevalence was reported to range from 19.0% in Tanzania to 32% in Namibia in that study. The study found the prevalence of other cardiovascular risk factors to be lower compared to the prevalence of hypertension whereby obesity ranged from 6.1% in Nigeria to 17.4% in Tanzania, diabetes was lowest in Namibia (2.1%) and highest in

Tanzania (3.7%), smoking prevalence ranged from 4.1% to 13.5% and hypercholesterolemia from 2.7% in Namibia to 4.8% in Kenya(9).

More efforts should be put in sub Saharan Africa to control hypertension, (52). Nearly 250,000 lives will be saved in sub Saharan Africa if hypertension will be treated effectively.(52).Despite the fact the that prevalence of hypertension in Africa has surpassed that of developed world but still we are lagging behind in terms of detection and proper control of the disease.(53, 54). In the study by Hendriks et al(9), control of hypertension among known hypertensives was found to be alarmingly low, ranging from only 2.6% in Kenya to 17.8% in Namibia in that study. The authors recommended strengthening of health care systems in sub Saharan Africa region in order to contain the emerging epidemic of hypertension and cardiovascular disease(9). As part of the WHO's target in reducing heart attacks and stroke by 2025, the World Heart Federation (WHF) launched a roadmap focusing on hypertension in order to reduce mortality caused by hypertension by 25%(55). The roadmap focused on practical steps towards hypertension control. One of the practical steps identified was reaching out to people who are unaware of their raised BP status through opportunistic screening for awareness of hypertension status so as to allow effective drug treatment for those found to have high BP as well as lifestyle modifications to prevent new onset hypertension among those found to have normal BP(55). The WHFroadmap was largely accepted and adopted by the Pan-African Society of Cardiology (PASCAR)(56).

## 1.3 Risk Factors for Developing Hypertension

Several cardiovascular risk factors have been known to have link with hypertension. They include older age, smoking, diabetes mellitus, overweight and obesity, dyslipidemia, excessive alcohol consumption, sedentary lifestyle, stressful life conditions as well as genetic predisposition (8, 9, 14, 32, 42, 57).

#### 1.3.1 Diabetes Mellitus

Diabetes among metabolic disorders is a well-known independent risk factor for hypertension. According to a study done by de Simone et al, diabetes was the most potent predictor of 8-year incident hypertension in a cohort of 967 American Indians participating in the Strong Heart

Study (57). Diabetes and hypertension are common diseases that coexist at a greater frequency than chance alone would predict. Hypertension in the diabetic individual markedly increases the risks of and accelerates the course of cardiovascular disease, peripheral vascular disease, stroke, retinopathy, and nephropathy(58, 59). Our understanding of the factors that markedly increase the frequency of hypertension in diabetic individuals remains incomplete. Diabetic nephropathy is an important factor involved in the development of hypertension in diabetic patients particularly type 1(60). However, the etiology of hypertension in the majority of diabetic patients cannot be explained by underlying renal disease and remains "essential" in nature. The hallmark of hypertension in type 1 and type 2 diabetic appears to be increased peripheral vascular resistance(61). Increased exchangeable sodium may also play a role in the pathogenesis of hypertension in diabetics. There is also increasing evidence that insulin resistance/hyperinsulinemia may play key role in the pathogenesis of hypertension in both subtle and overt abnormalities of carbohydrate metabolism(62). Population studies suggest that elevated insulin levels, which often occurs in type 2 diabetes mellitus, is an independent risk factor for cardiovascular disease(63, 64).

## 1.3.2 Older Age

As age of an individual increases, the BP tends to increase especially the systolic BP as well as an increased incidence of hypertension(65). Adults above the age of 35 years have an increased risk of having high BP than adults below the age of 35 years (66). This is because the structure and function of the human heart and vasculature change with age(67, 68). Structural changes in the vasculature increase arterial stiffness, which reduces arterial buffering capacity and gives rise to age-associated changes in systolic and diastolic BP(69, 70). On average, systolic BP rises with age, while diastolic BP increases until approximately 50 years and then declines(70). Elevated systolic or diastolic BP is associated with increased risk of cardiovascular disease and death(4). Indeed, isolated systolic hypertension is a major cause of morbidity and mortality in older adults(70).

## 1.3.3 Cigarette Smoking

Both cross sectional and cohort studies have shown an excess of hypertension among smokers(71, 72). In addition, smoking is known to promote increased sympathetic

activity(73), damage to the endothelium (74, 75) and accelerated atherosclerosis(76), processes thought to be important in the pathophysiology of hypertension (77). Loss in body weight due to smoking usually is interpreted to contribute in reducing BP. On the contrary, regular smokers usually develop a stable hypertension mainly due to the toxic effects of carbon monoxide(78). As previously described, endothelial dysfunction, increased arterial stiffness, and platelet function changes caused by smoking exposure contribute to increase chronically BP, but are also factors strongly related to hypertension. These observations undoubtedly show a strong relationship between cigarette smoking and hypertension that, in addition, exponentially potentiate their adverse effects on cardiovascular system when they are associated.

## 1.3.4 Overweight and Obesity

Obesity is a known major risk for essential hypertension, it manifests by increasing tubular reabsorption to impair pressure natriuresis and cause volume expansion via the activation of the sympathetic nervous system and the renin angiotensin aldosterone system(79). Several studies have reported strong association of a rise in BP with increasing body weight(80, 81). It has been shown that obese individuals have a 3.5 fold increased likelihood of having hypertension and that two-thirds of hypertension is attributable to increase in adipose tissue(82). In 1967, a prospective analysis of data from the Framingham Heart Study highlighted the relationship between obesity and hypertension(81). Indeed, the high prevalence of hypertension among patients with obesity (>60%) accounts for 78% of incident hypertension in men and 64% of incident hypertension in women (83-85). The prevalence of hypertension increases in relation to BMI in both men and women (86, 87). Estimates indicate that the increased risk of developing hypertension is 20-30% for every 5% increment in weight gain (88).

## 1.3.5 Alcohol Consumption

Alcohol has been identified as risk factor for hypertension. A study done by Flavio and his colleagues found that alcohol intake that exceeds 210g per week is an independent predictor of hypertension in North American populations(89). They also found out there is an increased risk of hypertension among black men who consume low to moderate alcohol(89). In a similar

study done by Mandlenkosietal among black South Africans who were interviewed and say yes to alcohol intake had 30% likelihood of developing hypertension with the hazard ratio of 1.3(90). Excessive alcohol consumption increases BP significantly in normotensive and hypertensive patients and is strongly linked to hypertension (91). Even in young patients with pre-hypertension or stage 1 hypertension, daytime diastolic BP variability is significantly increased in heavy drinkers, even after adjusting for smoking (92). Studies in humans indicate that the initial direct effect of alcohol is vasodilatation, with secondary stimulation of the sympathetic nervous system and vasoconstriction (93).

## 1.3.6 Dyslipidemia

Hypertension is commonly associated with other cardiovascular factors, including dyslipidemia(94). Dyslipidemia, itself a strong predictor of cardiovascular disease (95) causes endothelial damage (96) and loss of the physiological vasomotor activity that results from endothelial damage may become manifested as increased BP. Cross sectional studies have shown a link between abnormal lipids and hypertension (97-99). Some studies have prospectively examined the relationship between plasma lipids and future development of hypertension, finding that there is an association between plasma lipids and development of hypertension (98, 99). Trials have shown that BP lowering after use of lipid lowering medications (100, 101). Furthermore, elevated lipid levels appear to pre-date the onset of hypertension by years.

# 1.3.7 Hyperuricemia

Asymptomatic hyperuricemia is a risk factor for gout and for higher cardiovascular risk(102). The link between hyperuricemia and hypertension has been reported in several studies (103-106). Among children newly diagnosed with hypertension, serum uric acid is highly correlated with both systolic and diastolic BP (105). In the Framingham cohort, subjects with hypertension have been noted to have a 3-fold increased risk of gout (107). Among people with pre-hypertension, those in the highest uric acid quartile are at >2 times greater risk for microalbuminuria than those in the lowest quartile, but this relationship is not found among normotensive subjects (103). A study by Krishnan et al investigating hyperuricemia and incidence of hypertension among men without metabolic syndrome in the United States of

America demonstrated that hyperuricemia increases the risk of developing hypertension by 80%, independent of baseline BP measurements, renal function, serum lipid levels, BMI, proteinuria, alcohol use and age(108). A 4-year longitudinal follow-up of 3329 Framingham participants showed that hyperuricemia preceded onset of hypertension (109). Furthermore, evidence shows that for every unit rise in serum uric acid, the systolic and diastolic BP increases by a magnitude of 28mmHg and 15mmHg (110).

## 1.3.8Family history of hypertension

Family history is an important non-modifiable risk factor for hypertension. The hereditary nature of hypertension is well established by numerous family studies demonstrating associations of BP among siblings and between parents and children(111, 112). About 30 - 40% of the BP variance can be attributed to genetic factors(112, 113). Hypertension was found to vary from 25% in pedigree studies to 65% in twin studies. Among various mechanisms proposed to explain the relation between hypertension and positive family history of hypertension are increased renal proximal sodium reabsorption, genetic traits related to high BP such as high sodium-lithium counter-transport, low urinary kallikrein excretion, elevated uric acid level, high density LDL sub-fractions, fat pattern index, oxidative stress and BMI, as well as shared environmental factors such as sodium intake and heavy metal exposure(114-118). In screening programs, hypertension is more likely to be diagnosed if family history of hypertension is positive (119). Multiple studies have shown increased risk of having hypertension when a family history of hypertension was positive whereby compared to those without family history people with a positive family history have 2-4 times more likely to develop hypertension (113, 119).

## 1.4 Use of ABPM to Confirm Hypertension

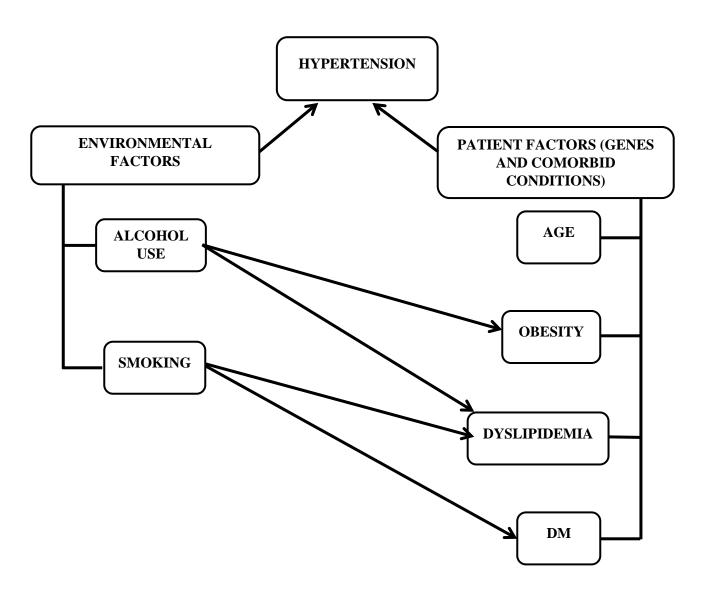
The use of 24-hours ABPM was introduced in the 1970s. In the beginning, the devices were large, heavy and cumbersome but nowadays the devices are light-weight and nearly all of them use an oscillometric measurement method to compute BP levels. This method eliminates observer bias and provides information on BP levels and heart rate throughout the day. The large numbers of readings obtained during the patient's daily activities provide a superior assessment of the true BP and can be used for the diagnosis of hypertension. In addition, 24-

hours ABPM provides information on BP variability, circadian changes, and the effects of environmental and emotional conditions on BP levels. Several studies that compared ABPM with intra-arterial measurements and mercury column sphygmomanometer demonstrated the accuracy of ABPM(22). It is recommended to perform the 24-hours ABPM on a typical working week-day and to obtain a diary or log of activities, wake and sleep times, time of medication administration, meals and any occurrence of symptoms. Excessive heavy physical activities during measurements are to be avoided.

The classic definition of hypertension is based on office BP measurements and most data relating hypertension to cardiovascular morbidity and mortality are derived from office measurements (4). Yet, the measurements in the office may not reflect the true BP levels. They may be elevated when the true BP is normal (white coat hypertension) or they may be normal when the true BP is elevated (masked hypertension). Office measurements also do not reflect the diurnal variation and nocturnal BP levels. 24-hours ABPM is a precise method to quantify BP levels and diagnose hypertension.

Moreover, recent studies have shown that 24-hours ABPM is more accurate than office BP measurements in predicting cardiovascular morbidity and mortality(120-123) as well as better correlation with cardiovascular outcome than clinic BP levels(122, 123). In a sub-study of the systolic hypertension in Europe (Syst-Eur) Trial, Staessen et al showed that in elderly subjects with untreated isolated systolic hypertension, ambulatory systolic BP was a significant predictor of cardiovascular risk over and above clinical BP values(123). In a prospective cohort study that included 1,464 subjects who were followed for 6.4 years, Ohkubo et al showed that ambulatory BPs were significantly better related to stroke risk than were screening office BP levels(122). Hara et al showed in 1,007 subjects that 24 hours daytime and night time ABP values were closely associated with the risk of silent cerebrovascular lesions detected by brain magnetic resonance imaging, whereas the clinic BP values were not associated with subclinical cerebrovascular events(120). Of the ambulatory BP values, night time BP was the strongest predictor of silent cerebrovascular events(120).

# 1.5. The Conceptual Framework



## **CHAPTER TWO**

#### 2.0 PROBLEM STATEMENT

The prevalence of hypertension is high in sub Saharan Africa, including Tanzania with trends showing that there will be even more increase of hypertension over time(6, 26). In sub Saharan Africa, hypertension affects middle aged-working class due to the increased likelihood of hypertension precursors in this population, including overweight and obesity, unhealthy diet, work-related stress as well as lack of physical activities(8, 9). As recommended, opportunistic screening for hypertension and associated factors offers a unique opportunity for work-based hypertension control programsthrough promoting healthy lifestyles at work(8). Hypertension screening also increase awareness of hypertension and offers opportunity for treating those already with hypertension - in a way overcoming one of the roadblocks for hypertension management in Africa as mentioned in the PASCAR roadmap(56).

Study by Hendriks, et al that looked on hypertension and other cardiovascular risk factors prevalence in four sub Saharan African countries namely Tanzania, Nigeria, Namibia and South Africa, hypertension was the most prevalent risk factor for cardiovascular disease in all four populations(9). The crude prevalence was reported to range from 19.0% in Tanzania to 32% in Namibia in that study. The study found the prevalence of other cardiovascular risk factors to be lower compared to the prevalence of hypertension whereby obesity ranged from 6.1% in Nigeria to 17.4% in Tanzania, diabetes was lowest in Namibia (2.1%) and highest in Tanzania (3.7%), smoking prevalence ranged from 4.1% to 13.5% and hypercholesterolemia from 2.7% in Namibia to 4.8% in Kenya (9).

In total, the prevention, detection, management and control of hypertension in sub Saharan Africa should now be regarded as a high priority (52). It is estimated that if the 10-20 million people who are believed to have hypertension in sub Saharan Africa were treated effectively, about 250,000 deaths would be prevented annually(52). Sadly, repeated reports over the years indicate that although the prevalence of hypertension has reached – and in some parts of Africa overcome that seen in the developed world, the prevention, detection, management and control of high BP are haphazard and insufficient (53, 54).

However, these studies done to determine the prevalence of hypertension in Tanzania and sub Saharan Africa region have used the conventional screening or clinic BP measurements to determine the prevalence of hypertension. However, clinic BP measurements over-estimate the true prevalence of hypertension by including individuals with white coat hypertension, fails to diagnose a proportion of individuals with masked hypertension and cannot identify individuals whose BP do not fall during sleep - a condition that has been linked to cardiovascular morbidity including stroke(122). In addition, severallongitudinal population studies have confirmed that 24-hours ABPM is stronger than clinic or screening BP in predicting cardiovascular morbidity(121).

## 2.1. Rationale of the Study

Studies have shown that ABPM is a better predictor of hypertension than office or home BP measurement and facilitates better prescription among established hypertensive patients as well as identify patients with masked hypertension(121). Accurately diagnosing and managing hypertension as well as identifying cardiovascular risk factors is essential in order to reduce morbidity and mortality related to cardiovascular diseases and its complications(8). Furthermore, identification of individuals with white coat hypertension may cut down the pill burden and cost of anti-hypertensive medications.

In Tanzania, only one study has been done to determine the prevalence of hypertension using 24-hours ABPM among 79 elderly population aged ≥70 years in Hai District(47), and it is unclear how well 24-hours ABPM measurements relate to cardiovascular risk profile of an individual. Furthermore, the prevalence of masked hypertension, white coat hypertension and nocturnal non-dippers as well as the relationship between 24-hours ambulatory BP profiles and cardiovascular risk factors among middle class working population in Tanzania are not known. The present study was conducted to address this knowledge gap.

Resultsfrom this study will provide a guidance to decide on appropriate method of correctly diagnosing hypertension as well as identifying cardiovascular risk factors linked to hypertension as diagnosed using office and ABPM. The study will also form a baseline for a work-based hypertension control program at MUHAS.

# 2.2. Research Questions

- 1. What is the prevalence of hypertension among MUHAS employees?
- 2. What proportion of MUHAS employees has white coat hypertension?
- 3. What proportion of hypertensive individuals has masked hypertension among MUHAS employees?
- 4. What is the prevalence of nocturnal non-dippers among MUHAS employees?
- 5. What is the relationship between 24-hours ambulatory BP profiles and cardiovascular risk factors among MUHAS employees?

# 2.3. StudyHypothesis

Do hypertension and its associated risk factors prevalent among MUHAS employees?.

# 2.4. Study Objectives

# 2.4.1 Broad Objective

To determine the prevalence of hypertension using 24-hours ABPM and to explore the relationship between 24-hours ABPM profiles with cardiovascular risk factors among MUHAS employees.

# 2.4.2 Specific Objectives

- 1. To determine the prevalence of hypertension using 24-hours ABPM among MUHAS employees.
- 2. To determine the prevalence of white coat hypertension among MUHAS employees
- 3. To determine the prevalence of masked hypertension among MUHAS employees
- 4. To determine the prevalence of nocturnal non-dippers among MUHAS employees
- 5. To assessthe relationship between 24-hours ambulatory BPprofilesand cardiovascular risk factors among MUHAS employees.

## **CHAPTER THREE**

#### 3.0 METHODOLOGY

## 3.1. Study Design

A descriptive cross-sectional study

# 3.2. Study Duration

This study was conducted over a period of 5 months from October 2018 to February 2019

## 3.3. Study Area

Muhimbili University of Health and Allied Sciences (MUHAS) is located in Dar es Salaam. It has two campuses; Muhimbili Campus and Mloganzila Campus, which incorporates the MUHAS Academic Medical Center (the teaching hospital for MUHAS). The University has a total of 648 staff of whom 306 are academic staff and 342 are administrative and technical staff. The overall female proportion of University staff is 40.7%.

## 3.4. Study Population

All active MUHAS employees at the time of data collection

## 3.5. Inclusion and Exclusion Criteria

**Inclusion Criteria:** 

MUHAS employees who consented to participate in the study

**Exclusion Criteria:** 

Foreign MUHAS employees

**Expectant mothers** 

## 3.6 Sample Size Calculation

The sample size was calculated using the Kish-Leslie formula as shown below:

$$n = \frac{Z^2 P(100 - P)}{\epsilon^2}$$

Whereby:

n = minimum sample size required

Z = standard normal deviation set at 1.96 (corresponding to confidence level of 95%)

P = Prevalence of hypertension among general population. A prevalence of 24.5% was used based on the Tanzania STEPS survey report.

 $\varepsilon$  = marginal error used of 4.5%

$$n = \frac{1.96^2 \, X \, 24.5 \, (100 - 24.5)}{4.5^2}$$

$$n = 351$$

Therefore, the minimum sample size was 351 subjects.

The power of this study is 80% with a non-response rate (f) of 10%.

With the inclusion of an error due to non-response, the final sample size was calculated using the formula:  $N = (n \times 100)/(100 - f)$ .

Therefore, the sample size used was 390 employees

## 3.7 Sampling Method

The sampling frame included all MUHAS employees. A simple random sampling method was used toobtain the sample size. The list of employees was entered into computer software which randomly allocated numbers and the sample size was obtained using table of random numbers.

## 3.8 Variables

*Continuous variables* includedoffice SBP, office DBP, ambulatory SBP, ambulatory DBP, serum uric acid levels, total cholesterol, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol, waist circumference, height, body weight and age.

*Categorical variables* includedage groups, gender, smoking status, overweight and obesity status, alcohol consumption, diabetes mellitus as well as family history of hypertension and other cardiovascular disease.

*Independent variables* include age of the patient, gender, height, weight, smoking status, diabetes status as well as family history of hypertension and cardiovascular disease.

**Dependent variables** are office and ambulatory blood pressure levels, lipid profile and serum uric acid levels.

### 3.9 Data Collection Methods

## 3.9.1 Questionnaire

A structured questionnaire (Appendix I) was used to collect information on socio-demographic characteristics of the participants, previous and family history of cardiovascular disease, as well as information on smoking, alcohol consumption, level of physical activities, etc.

## 3.9.2 Anthropometric Measurements

All participants underwent anthropometric measurements.

<u>Height</u> was measured using a stadiometer. This consists of a rigid vertical surface with an attached scale in centimeters and a horizontal mobile surface at right angles, which slides freely vertically along the scale. The inferior surface lies on the floor at the zero of the measuring scale. The participant was asked to stand with his/her back to the vertical scale so that the heels, buttocks and head are in contact with it. The heels were kept together and shoulders relaxed. The participant looked forward at eye level. The mobile horizontal surface was then slid downwards to the top of the head of the participant. The reading was taken from the vertical scale. Measurement was then recorded to the nearest 1 cm.

<u>Body weight</u> was measured using a weighing scale (*Momert*®, China), with participants wearing light clothing and without shoes. Itwasmeasured in kilograms (kg)and averaged to the nearest tenth of a kilogram.

Body mass index (BMI) was calculated by dividing the participant's weight in kg by the participant's height in meter squared (m<sup>2</sup>) as shown: BMI = Weight(kg)/Height (m)<sup>2</sup>. BMI levels was used to categorize participants as normal weight ( $\leq$ 24.9kg/m<sup>2</sup>), overweight (25.0 – 29.9kg/m<sup>2</sup>) and obese ( $\geq$ 30kg/m<sup>2</sup>)(124).

<u>Waist circumference</u> was measured using a tape measure at the level of the umbilicus and wasrecorded to the nearest centimeter. Abdominal obesity was considered present when the waist circumference is >102cm and 88cm in men and women respectively(125).

# 3.9.3 Blood Pressure Measurements

Office BP was measured on the participant's left arm using an automated BP machine (heur). Measurements were taken in a quiet room after the participant has had a 5 minutes' rest and seated comfortably in a chair with the back and left arm supported, legs uncrossed and the upper arm at the level of the right atrium. A proper cuff size wasused. Three measurements weretaken and the average of the last two wasrecorded as the participant's office BP. Office Hypertensionwasdefined as a BP of more than or equal to 140mmHg systolic and/or 90mmHg diastolic(126).

Ambulatory BP measurement was initiated immediately after recording of office BP. Measurements were done using an automated ABPM device (*Jawon Medical FA48*<sup>®</sup>, Korea). BP recordings were done every 15 minutes between 07:00 and 22:00 hours, and every 30minutes between 22:00 and 7:00 hours. Participants weregiven a diary to record the time they go to sleep at night and the time they wake up in the morning. Participants were asked to avoid vigorous physical activities on the day of the test.Ambulatory hypertension was defined as average daytime ABPM of ≥135mmHg systolic and/or 85mmHg diastolic(22). White Coat Hypertension was defined as clinic BP of ≥140/90mmHg with average daytime ABPM of <135/85mmHg, while Masked hypertension was defined as clinic BP of <140/90mmHg with average daytime ABPM ≥135/85mmHg, and Nocturnal non-dipping was defined as a decrease in systolic and/or diastolic BP of less than 10% from day to night(22).

## 3.9.4 Laboratory Investigations

All participants underwent blood tests. The tests included serum glucose levels, serum lipid profile (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and triglycerides levels), serum creatinine, urea anduric acid levels. Participants were asked to fast overnight before blood collection the next morning. Four milliliters of blood was collected in a red-top tube and all blood tests, except fasting blood glucose were analyzed

at MAMC laboratoryusing the Architect Plus ci410<sup>®</sup> analyzer. Fasting blood glucose was analyzed on-spot using a portable glucometer(Accucheck Performa<sup>®</sup>, Roche).

<u>Diabetes</u> was defined as fasting blood glucose of 7.0mmol/l or previous diagnosis of diabetes mellitus with use of medications(127).

<u>Hypercholesterolemia</u> was defined as total cholesterol level  $\geq$  5.2mmol/l, while elevated low density lipoprotein cholesterol was defined as serum low density lipoprotein cholesterol  $\geq$ 3.4mmol/l, and High density lipoprotein cholesterol was considered low when it was < 1mmol/l(128).

<u>Serum creatinine</u> wasused to calculate estimated glomerular filtration rate (eGFR), using the Modification of Diet in the Renal Disease (MDRD) equation(129), and the eGFR was considered low when it is <60ml/min/1.73m<sup>2</sup>.

<u>Serum uric acid</u> level was considered elevated when it was >7.0mg/dl(130).

## 3.10 Data Handling

The data obtained from the questionnaires were entered manually in the computerized software program, statistical package of social sciences (SPSS) version 20 for Windows for data analysis. There was a cross checking of filled questionnaires after data collection for quality control of data.

## 3.11 Data Analysis

The main outcome variable was ambulatory BP. Continuous variables are expressed as the mean  $\pm SD$  or as median and interquartile ranges. Comparisons of categorical variables between groups were performed using chi-square test or Fisher's exact t-test. Multivariate logistic regression analysis was used to determine association between hypertension and binary variables. Bivariate linear correlations were assessedusing Pearson's correlation coefficient to determine the association between ABPM profiles and continuous variables. All tests were two sided and a p-value of less than 0.05 was considered statistically significant.

## 3.12 Ethical Considerations

Ethical clearance was obtained from the directorate of research and publication at MUHAS and the permission to conduct the study was obtained from MUHASadministration(Appendix II). All participants signed an informed consent form (Appendix III) after they well understood the information and agreed to participate in the study. Confidentiality and privacy was assured, and participants were free to refuse participating in the study without any consequences.

# 3.13 Disposal of participants with hypertension and other cardiovascular risk factors

Participants found to have hypertension and other cardiovascular risk factors were counseled and treated according to National guideline and given follow up clinic dates. Those with normal BP and nocturnal non-dippers were counseled also on modifiable traditional cardiovascular risk factors and monitoring of their blood pressure regularly.

## **CHAPTER FOUR**

## 4.0 RESULTS

## 4.1 Demographic and clinical characteristics of the study participants

In total 390 MUHAS employees participated and they constitute the present study population. Of the 390 participants, 305(78.2%) were recruited from Muhimbili campus whereas the remaining 85(21.8%) were from Mloganzila campus. There were 209(53.6%) men and 181(46.4%) women. The mean  $\pm$ SD age of the total study population was  $40.5\pm8.9$  years (range 24-59 years). Positive family history of hypertension was found in 138(35.4%) participants while 15.6% and 2.1% were known hypertensives and diabetics, respectively, Table 1. Alcohol intake was present in 44.1% of the total population, with men significantly having more proportion of alcohol drinkers (66%) when compared to women (18.8%), p <0.0001. Only 13 (3.3%) participants (all men) had ever smoked cigarette. Table 1 summarizes demographic and clinical characteristics in the total population and according to gender.

Table 1: Demographic and clinical characteristic of study participants

	Total population	Men	Women	p-value*
Characteristic	$(\mathbf{N} = 390)$	(n = 209)	(n = 181)	
Mean ±SD Age (years)	40.5±8.9	41.0±8.0	39.9±9.9	0.216
Age groups (years); n (%)				
<30	67 (17.2)	32 (15.3)	35 (19.3)	0.000
31 - 40	138(35.4)	68(32.5)	70(38.7)	
41 - 50	126(32.3)	90(43.0)	36(19.9)	
>50	59(15.1)	19(9.1)	40(22.1)	
Work station; n(%)				
Mloganzila campus	85(21.8)	35(16.7)	50(27.6)	0.009
Muhimbili campus	305(78.2)	174(83.3)	131(72.4)	
Education level; n (%)				
Primary	15(3.8)	5(2.4)	10(5.5)	0.001
Secondary	59(15.1)	19(9.1)	40(22.1)	
College	208(53.3)	118(56.5)	90(49.7)	
University	108(27.7)	67(32.1)	41(22.7)	
Occupation; n (%)				
Doctors/Lecturers	66(16.9)	30(14.4)	36(19.9)	0.243
Nurses	54(13.8)	27(12.9)	27(14.9)	
Other professions	270(69.2)	152(72.7)	118(65.2)	
Marital status; n (%)				
Single	94(24.1)	48(23.0)	46(25.4)	0.323
Married	280(71.8)	155(74.2)	125(69.1)	
Divorced/Widowed	16(4.1)	6(2.9)	10(5.5)	
Weight (kg)	68.9±11.3	$70.5 \pm 11.4$	$67.1 \pm 10.9$	0.003
Height (m)	160 ±6	160±7	$159 \pm 5$	0.011
BMI $(kg/m^2)$	27.1 ±4.6	27.5 ±4.8	26.7 ±4.4	0.071
Obesity status; n (%)				
Normal	125(32.0)	61(29.2)	64(35.3)	0.251
Overweight	140(35.9)	74(35.4)	66(36.5)	
Obese	125(32.1)	74(35.4)	51(28.2)	
Positive family history of	, ,	` ,	` ,	
hypertension; n (%)	138(35.4)	72(34.4)	66(36.5)	0.678
Known hypertensive; n (%)	61(15.6)	36(17.2)	25(13.8)	0.355
Known diabetic; n(%)	8(2.1)	3(1.4)	5(2.8)	0.357
Taking alcohol; n (%)	172(44.1)	138(66.0)	34(18.8)	0.000
Ever smoked; n (%)	13(3.3)	13(6.2)	0 (0.0)	0.001

Results are mean ±SD unless stated otherwise; \*p-value, comparing differences between men and women.

# 4.2 Laboratory results of study participants

Laboratory findings are presented in Table 2. In the total population, the mean fasting blood glucose, total cholesterol, triglycerides and Uric acid levels were all in the normal range. However, there were 13 (3.3%)participants with raised fasting blood glucose, 92 (23.6%) participants with hypercholesterolemia, and 53 (13.6%) with hyperuricemia, Table 2. Likewise, the mean serum creatinine of the total population was normal (86.3µmol/l), but 17 (4.4%) of participants had lower estimated glomerular filtration rate indicating impaired renal function. Participants with impaired renal function were all women, Table 2.

**Table 2: Laboratory findings of study participants** 

	Total			
	population	Men	Women	
Laboratory findings	(N = 390)	(n=209)	$(\mathbf{n} = 181)$	p-value
Fasting blood glucose (mmol/l)	4.5±0.6	4.6±0.7	4.4±0.5	0.015
Proportions with diabetes mellitus n				
(%)	13 (3.3)	8 (3.8)	5(2.8)	0.559
Total cholesterol(mmol/l)	$4.5 \pm 0.9$	$4.6 \pm 0.9$	$4.4 \pm 0.7$	0.010
Proportions with raised total				
cholesterol n (%)	92(23.6)	56(26.8)	36(19.9)	0.109
HDL-Cholesterol (mmol/l)	$1.19 \pm 0.13$	$1.19 \pm 0.13$	$1.19 \pm 0.12$	0.862
LDL-Cholesterol (mmol/l)	$2.4\pm0.4$	$2.46 \pm 0.41$	$2.37 \pm 0.28$	0.018
Proportion with raised LDL-C, n (%)	18 (4.6)	12 (5.7)	6 (3.3)	0.255
Triglycerides(mmol/l)	$1.53 \pm 0.27$	$1.57 \pm 0.28$	$1.48 \pm 0.23$	0.001
Proportion with <b>raised</b>				
Triglycerides, n (%)	87 (22.3)	54 (25.8)	33 (18.2)	0.072
Uric acid (mmol/l)	$0.36\pm0.12$	$0.38\pm0.13$	$0.34\pm0.11$	0.006
Proportions with hyperuricemia, n				
(%)	53(13.6)	36(17.2)	17(9.4)	0.024
Blood Urea Nitrogen(mmol/l)	$3.96 \pm 0.93$	$3.98 \pm 1.04$	$3.94 \pm 0.79$	0.700
Serum Creatinine(mmol/l)	$86.3 \pm 17.4$	$88.0 \pm 18.8$	$84.4 \pm 15.3$	0.042
Estimated GFR(ml/min/1.73m <sup>2</sup> )	$94.9 \pm 23.3$	$105.9 \pm 23.0$	82.1±16.1	0.000
Proportions with <b>eGFR</b> < <b>60</b> , <b>n</b> (%)	17(4.4%)	0 (0.0)	17(9.4)	0.000

Results are mean ±SD unless stated otherwise; \*p-value, comparing differences between men and women

## 4.3 Blood Pressure findings of study participants

Table 3 shows office and ambulatory BP findings in the total population and in men and women separately. Men had significantly higher mean values for all systolic and diastolic BP measurements. In the total population, 82 (21%) participants were found to have office hypertension (i.e. Office BP  $\geq$ 140/90mmHg). Office hypertension did not differ between men (23.4%) and women (18.2%), p = 0.208. Sixty-four (16.4%)participants were found to have hypertensionby ambulatory day time BP (i.e. mean ambulatory day-time BP  $\geq$ 135/85mmHg). Ambulatory day-time hypertension was significantly more in men (22%) than in women (9.9%), p = 0.001, Table 3.

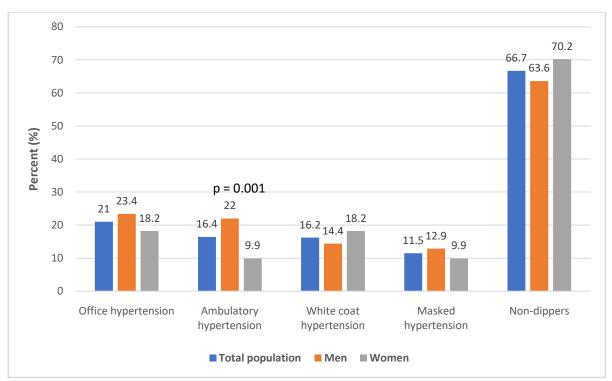
Table 3: Office and 24-hours Ambulatory Blood Pressure findings of study participants

	Total population	Men	Women	
BP Measurements	(N = 390)	(n = 209)	(n = 181)	<i>p</i> -value
Office Systolic BP(mmHg)	126±12	128 ±12	124 ±13	0.001
Office Diastolic BP(mmHg)	78 ±13	$80 \pm 14$	76 ±11	0.004
Daytime Systolic BP(mmHg)	126±12	$129 \pm 12$	123 ±11	0.000
Daytime Diastolic BP(mmHg)	77 ±10	$79 \pm 10$	75 ±10	0.000
Sleep time Systolic BP(mmHg)	118±18	$121 \pm 17$	$116 \pm 17$	0.006
Sleep time Diastolic BP(mmHg)	72 ±12	74 ±11	$70 \pm 13$	0.002
24-hours Systolic BP(mmHg)	122±14	$125 \pm 13$	$119 \pm 14$	0.000
24-hours Diastolic BP(mmHg)	75 ±10	$77 \pm 10$	$72 \pm 11$	0.000
Proportion with Office hypertension, n (%)	82 (21.0)	49 (23.4)	33 (18.2)	0.208
Proportion with Ambulatory Daytime Hypertension, n (%) Proportion with white coat	64 (16.4)	46 (22.0)	18 (9.9)	0.001
Hypertension, n (%)	63 (16.2)	30 (14.4)	33 (18.2)	0.299
Proportion with masked hypertension, n (%)	45 (11.5)	27 (12.9)	18 (9.9)	0.359
Proportion with nocturnal non-dipping, n (%)	260 (66.7)	133 (63.6)	127 (70.2)	0.173

Results are mean ±SD unless stated otherwise; \*p-value, comparing differences between men and women

In the total population, 63 (16.2%) participants were found to have white coat hypertension (i.e. raised office BP with normal ambulatory daytime BP), while 45 (11.5%) were having masked (i.e. normal office BP with raised ambulatory daytime BP) hypertension, Table 3. Both white coat and masked hypertension did not differ between men and women, p >0.05 for both. Of note, only 19 (23.2%) of the 82 participants with office hypertension had true ambulatory daytime hypertension (all of them were men). As many as two thirds(66.7%) of study participants were found to have nocturnal non-dipping (i.e. less than 10% mean drop of BP during sleep). Figure 1 summarizes the prevalence of hypertension using office and ambulatory BP profiles in the total population, and in men and women.

Figure 1: Prevalence of hypertension, white coat effect, masked hypertension and nocturnal non-dipping in the total population and in men and women



## 4.4 Association between BP and other cardiovascular risk factors in study participants

Univariate correlations of meansystolic and diastolic BP with continuous variables are shown in Table 4 and in figures 2A and 2B. Using ambulatory daytime mean systolic BP, significant positive correlation were found with all tested known risk factors for hypertension, Table 4. As expected, estimated GFR showed a negative correlation, r = -0.362, p < 0.0001. Similar correlations were found with diastolic BP, except that waist circumference did not significantly correlate with mean ambulatory daytime diastolic BP, Table 4.

Table 4: Correlates of Ambulatory Day-time mean systolic and diastolic blood pressure

Factors -	Mean Da	ytime SBP	Mean Dayti	me DBP
ractors	r	p-value	r	p-value
Age(years)	0.323	0.000	0.247	0.000
Weight(kg)	0.256	0.000	0.206	0.000
Height(cm)	-0.094	0.065	-0.078	0.126
$BMI(kg/m^2)$	0.286	0.000	0.219	0.000
Waist circumference	0.139	0.006	0.088	0.081
Fasting Blood Sugar(mmol/l)	0.446	0.000	0.316	0.000
Total Cholesterol(mmol/l)	0.707	0.000	0.49	0.000
HDL-Cholesterol(mmol/l)	0.733	0.000	0.306	0.000
LDL-Cholesterol(mmol/l)	0.439	0.000	0.351	0.000
Triglyceride(mmol/l)	0.49	0.000	0.285	0.000
Uric acid(mmol/l)	0.652	0.000	0.496	0.000
Serum Creatinine(µmol/l)	0.67	0.000	0.439	0.000
$eGFR(ml/min/1.73m^2)$	-0.362	0.000	-0.208	0.000
Urea Nitrogen (µmol/l)	0.465	0.000	0.284	0.000

Figure 2 compares the strengths of correlations between continuous variables age, body mass index, waist circumference, fasting blood sugar, total cholesterol, triglycerides, uric acid, serum creatinine and estimated GFR, with mean systolic (figure 2A) and diastolic (figure 2B) BP for office and ambulatory BP profiles. By comparison, the mean 24-hours BP performed best when compared to other ambulatory BP profiles as well as compared to office BP in terms of best overall correlations with the variables tested. Of note, office BP performed least in correlating with the tested variables.

Figure 2A: Univariate correlates of Office, Day-time, Sleep and 24-hours mean Systolic BP

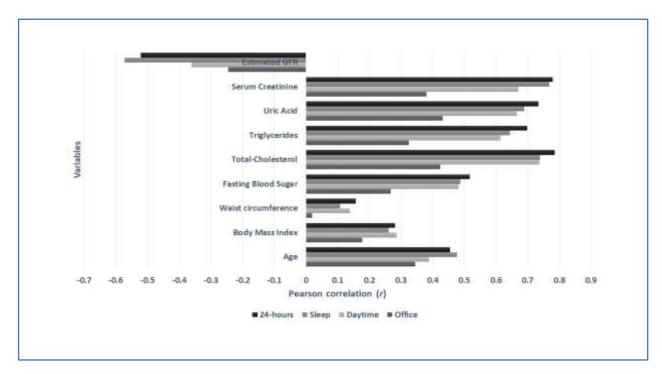
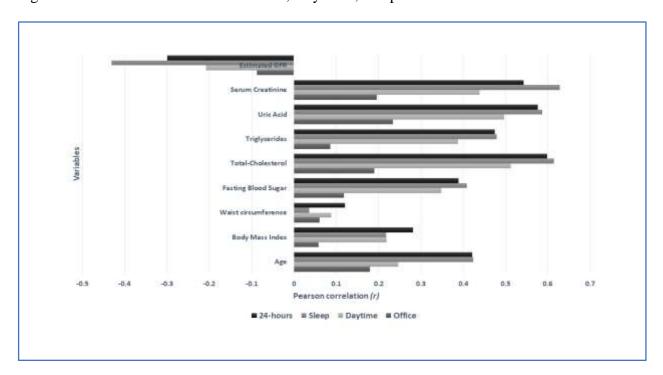


Figure 2B: Univariate correlates of Office, Day-time, Sleep and 24-hours mean Diastolic BP



# 4.5 Prevalence and factors associated with hypertension among study participants

In this study, overall hypertension was defined as raised ambulatory daytime BP of  $\geq$ 135/85 or known hypertensive. In the **total population** therefore, 90 (23.1%) participants fulfilled the definition of hypertension while the remaining 300 (76.9%) did not have hypertension. Among the hypertensives, 61 (67.8%) were previously known and 29 (32.2%) were newly diagnosed in the current study. Of the previously known hypertensives, 35 (57.4%) were having uncontrolled hypertension as evidenced by elevated mean ambulatory day time BP during this screening. Compared to patients without hypertension, patients with hypertension were less likely to be women, were older and were more likely to be from Muhimbili campus, all p < 0.05, Table 5. Furthermore, hypertensive participants were more likely to have had smoked cigarette as well as more likely to have positive family history of hypertension, Table 5.

Table 5: Socio-demographic and clinical findings in study participants with and without hypertension

hypertension			
Characteristic	Non-hypertensive	Hypertensive	p-value
	(n = 300)	(n = 90)	
Women, n (%)	150 (50)	31 (34.4)	0.009
Mean(±SD) Age (years)	38.5±8.6	$47.2 \pm 6.1$	0.000
Age groups (years)			
≤30	67 (22.3)	0 (0)	0.000
31 - 40	129 (43.1)	9 (10)	
41 - 50	70 (23.3)	56 (62.2)	
>50	34 (11.3)	25 (27.8)	
Age groups (years)			
<40	168 (56)	9 (10)	0.000
≥40	132 (44)	81(90)	
Profession, n (%)			
Nurses	47 (15.7)	7 (7.8)	0.02
Doctors	56 (18.7)	10 (11.1)	
Other professions	197 (65.7)	73 (81.1)	
Muhimbili staff, n (%)	221 (73.7)	84 (93.3)	0.000
Marital status, n (%)			
Single	82 (27.3)	12 (13.3)	0.000
Married	214 (71.3)	66 (73.3)	
Divorced/widowed	4 (1.3)	12 (13.3)	
Ever screened for hypertension, n (%)	216 (72.0)	76 (84.4)	0.017
Family history of hypertension, n (%)	79 (26.3)	59 (65.6)	0.000
Smokers, n (%)	5 (1.7)	8 (8.9)	0.001
Taking alcohol, n (%)	127 (42.3)	45 (50.0)	0.199
Mean(±SD) BMI (kg/m <sup>2</sup> )	$26 \pm 4.2$	$31 \pm 3.8$	0.000
Obesity class, n (%)			
Normal weight	125 (41.7)	0 (0.0)	0.000
Overweight	114 (38.0)	26 (28.9)	
Obese	61 (20.3)	64 (71.1)	
Mean(±SD) Waist circumference	88 ± 8	$92 \pm 7$	0.000
Raised waist circumference, n (%)	51 (17.0)	27 (30.0)	0.007

Results are Mean ±SD, unless stated otherwise

Table 6 shows laboratory findings' comparisons between hypertensive and non-hypertensive study participants. Hypertensive participants differed significantly with non-hypertensive participants in all laboratory parameters, withhypertensive participants having significantly higher mean values for fasting blood glucose, total cholesterol, triglycerides, uric acid as well as serum creatinine levels. Consequently, hypertensive participants were more likely to have elevated blood glucose with hypercholesterolemia, as well as more likely to have impaired renal function, all p<0.05, Table 6.

Table 6: Laboratory findings in study participants with and without hypertension

Characteristic	Non-hypertensive	Hypertensive	p-value
	(n = 300)	(n = 90)	
Fasting Blood Glucose (mmol/l)	$4.4 \pm 0.3$	$4.9 \pm 1.0$	0.000
Proportion with raised glucose, n (%)	0 (0.0)	13 (14.4)	0.000
Total Cholesterol (mmol/l)	$4.3 \pm 0.5$	$5.4 \pm 1.1$	0.000
Proportion with raised Total Cholesterol, n (%)	31 (10.3)	61 (67.8)	0.000
Triglycerides (mmol/l)	1.46 (0.17)	1.76 (0.37)	0.000
Proportion with raised Triglycerides level, n (%)	43 (14.3)	44 (48.9)	0.000
Uric Acid level (mmol/l)	$0.32 \pm 0.08$	$0.48 \pm 0.16$	0.000
Proportion with raised Uric Acid, n (%)	12 (4.0)	41 (45.6)	0.000
Serum Creatinine (µmol/l)	$82 \pm 12$	$102 \pm 23$	0.000
eGFR (ml/min/1.73m²)	$99 \pm 21$	$80 \pm 26$	0.000
Proportion with eGFR<60, n (%)	6 (2.0)	11 (12.2)	0.000

Results are Mean±SD, unless stated otherwise

Socio-demographic, clinical and laboratory parameters that showed significant association with being hypertensive among MUHAS employees were entered in a multivariate logistic regression analysis model. After removal of variables with high interactions (e.g. weight and BMI), the final best fitting model included gender, age, family history of hypertension, central adiposity, hypercholesterolemia and hyperuricemia. In this multivariate logistic regression analysis, the independent factors associated to having hypertension among MUHAS employees were being male (OR = 7.9), having age  $\geq 40$  years (OR = 3.9), having positive family history of hypertension (OR = 5.6), having central obesity (OR = 9.0), as well as having high total cholesterol (OR = 3.8) and elevated uric acid levels (OR = 7.9), p < 0.05 for all, Table 7.

Table 7: Independent factors associated with hypertension among MUHAS employees obtained in multivariate logistic regression analysis

Variable	Odds Ratio	95% CI	p-value
Male gender	7.96	2.50 - 25.30	0.000
Age ≥40 years	3.94	1.66 - 9.33	0.002
Positive family history of hypertension	5.60	2.61 – 12.04	0.000
Central obesity	8.98	2.72 - 29.70	0.000
Ever smoked	0.42	0.08 - 2.20	0.301
Elevated total cholesterol	3.84	1.56 - 9.50	0.004
Elevated uric acid	7.90	2.55 – 24.43	0.000

## **CHAPTER FIVE**

#### 5.0 DISCUSSION

This study was conducted to determine the prevalence of hypertension as well as association of hypertension with cardiovascular risk factors among MUHAS employees by using ambulatory BPmonitoring. The study is among the few studies done in subSaharan Africacomparing office BP with ambulatory BP as screening tools for hypertension. The study has found many interesting findings and add to existing literature in hypertension in Tanzania and sub Saharan Africa.

The prevalence of hypertension of 23.1% found in the present study is almost similar to the nation-wide population survey of Tanzanianadults aged 25 – 65 years as part of the STEPS survey(14). In the STEPS survey the prevalence of hypertension was found to be 25.9%, with the slight higher prevalence likely due to inclusion of people with white coat effect, since only one-time screening BP was used to determine the prevalence of hypertension in the STEPS study. Our prevalence is however remarkably similar to the prevalence of hypertension among adult Kenyans in Kilifi, where hypertension was found to be 23.1% by screening and 16% when the same individuals underwent ambulatory BP measurements(46). In the only previous ambulatory BP study from Tanzania (among elders >70 years), the prevalence of hypertension was 55.7%, with the difference from our study most likely due the age differences between the two studies(47). Of note, in the present study increasing age was independently associated with hypertension, underscoring age as a risk factor for hypertension(65).

The prevalence of White Coat Hypertension found in this study (16.2%) was higher than that found in the Kilifi study (3.8%)(46),but lower than that found among African Americans (29.3%)(131). The differences could be explained by differences in study settings, operational definitions, as well as the populations studied. This group of people with white coat hypertension had a risk of being placed unnecessarily on lifelong treatment that would have inflated their medical bills as well as unwanteddrug's side effects. Furthermore, 11.5% of the participants in the present study had masked hypertension. This group would have been missed if office BP measurementswere to be used to define hypertension, putting them at risk of

stroke,heart failure and ischemic heart disease(1, 123).Two-thirds(67%) of participants in the present study had nocturnal non-dipping very similar to the findings from African Americans by Thomas et al(131), which found 69.6% of participants in the large Jackson Heart Study to have nocturnal non-dipping - itself a risk of stroke(122). Of note, the prevalence of nocturnal non-dipping was very low in the Kilifi study (9%), again showing differences from our study, likely from the differences in study set up and maybe methods and/or definitions applied(46).However, nocturnal non-dipping is a recognized phenomenon that seem to occur more common among individuals of African descent (132, 133)and may be responsible for the increased stroke rate among these populations (134).

This study has confirmed the general understanding that cardiovascular risk factors tend to aggregate among individuals(10, 14-17). As noted in the univariate analysis, participants with hypertension were also more likely to have all other cardiovascular risk factors either modifiable (like obesity, high blood glucose,high cholesterol as well as cigarette smoking) or non-modifiable risk factors (like age, gender and family history of hypertension) when compared to participants without hypertension. This finding is similar to many other previous studies in literature(14-19). Importantly, most of the risk factors were independently associated with hypertension in multivariate analysis, therefore underscoring the importance of addressing a total cardiovascular risk profile of an individualwhile managing hypertension.

The finding that ambulatory BP correlated better with cardiovascular risk factors is in agreement with previous studies in literature, explaining the fact that multiple measurements of BP during 24-hour period is superior to a few measurements in a doctor's office for estimating the cardiovascular risk of an individual(21, 25, 123, 135). Our finding that the best correlated cardiovascular risk factors were total cholesterol, serum creatinine, and uric acid is an interesting one. High total cholesterol can be regarded as part of the increased cardiovascular risk profile of an individual and this is well documented in literature(97-99), while increased serum creatinine can be regarded as an outcome of the effect of hypertension(3, 4, 8). Previous studies have also documented the relationship between uric acid level and hypertension(103-106), and debate still continue as to whether raised uric acid is a

risk or an outcome of hypertension (136). More research is therefore needed to confirm this association, especially among Africans in sub Saharan Africa. Furthermore, by comparison, systolic BP was overall better correlated with the cardiovascular risk factors in our study when compared to diastolic BP, this finding issimilar to other previous studies (8-10).

We found many other known factors associated with hypertension to be independently associated with hypertension in the present study, therefore confirming findings from previous studies in literature (6, 65, 66, 80, 81, 113, 119), and adding that these associations are stronger when ambulatory BP measurements are used. In all WHO regions, men had slightly higher prevalence of hypertension than women(6). Similar to the present study male gender was independently associated with hypertension among MUHAS employees(OR=7.9). In this study, having age>40 years, was also independently associated with hypertension(OR=3.9). These findings are in line with other studies which showed as age of an individual increases, the BP tends to increase especially the systolic BP as well as an increased incidence of hypertension(65)(66). The finding that positive family history of hypertension independently predicts hypertension(OR=5.6) is similar to other multiple studies that concluded increased risk of having hypertension to be 2-4times likely, when family history of hypertension was present compared to families without history of hypertension(113, 119).In this current study the Odds Ratio for the association between central obesity and hypertension is 9, which is similar to several studies that have demonstrated strong association between obesity and increased in BP(80, 81).

## **CHAPTER SIX**

## 6.0 CONCLUSION AND RECOMMENDATIONS

Hypertensionis the leading cause of stroke,heart failure and ischemic heart disease. The prevalence of hypertension among MUHAS employeesas determined using Ambulatory Blood Pressure Monitoring (ABPM) is 23.1% and is associated with known traditional cardiovascular risk factors. In addition, ambulatory BP correlates better with cardiovascular risk factors, which means it may be the best measure of one's true BP when compared to office BP. Timely diagnosis and treatment are the cornerstone in reducing morbidity and mortality among patients with hypertension. Office BP has for decades been used as a screeningtool todiagnose hypertension. However, we have seen that it fell short of diagnosing participants with masked hypertension as well as overestimating true prevalence of hypertension in participants with white coat effect. ABPM is breakthrough in diagnosing individuals with hypertension. This will cut down the pill burden and unnecessary medical bills in low resource setting country like Tanzania as well as identifying and treating individuals with masked hypertension. This will enable more resources to be allocated in this group. Not only have that but ABPM also showed best prediction of hypertension in individuals with modifiable cardiovascular risk factors such as elevated cholesterol.

Before we recommend the use of ABPM as a screening tool for hypertension, affordability, availability of equipment, technicians as well as patient awareness on ABPM has to be factored in. It's high time for different stakeholders to sit down and see how best we can ensure our patients receive best treatment after correctly been diagnosed with hypertension. Altogether, work-based hypertension control programs are recommended at MUHAS in order to manage staff with hypertension, but also to educate those without hypertension on the way to prevent hypertension development. Secondly, results from this study suggest that Ambulatory BP measurements can be used to confirm true hypertension, especially in individuals found to have raised office BP but with low cardiovascular risk profile.

# **6.1 Study Limitations**

Since the study is a cross-sectional, single centered and done on relatively affluent group, apotential for bias exists. These results cannot therefore be generalized to the general public in Tanzania, but rather to selected work-based populations.

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# **APPENDICES**

# Appendix IA: Questionnaire- ABPM among MUHAS Employees Study

A.	SOCIO-DEMOGRAPHIC CHARAC
1.	Study ID No:
2.	Date of interview:
3.	Participant's initials:
4.	Contact number:
5.	Main work station
	a.Muhimbili
	b.Mloganzila
6.	Gender
	a.Male
	b.Female
7.	Date of Birth:
8.	Occupation cadre
	a.Nurse
	b.Doctor
	c.Others profession
9.	Highest level of education
	a.Primary
	b.Secondary
	c.College
	d.University
10.	Marital status
	a.Single
	b.Married

c.Divorced/Widowed

R	CA	DI	M	<b>T/ A</b>	CCIII	A D	DICK	<b>PROFI</b>	LE
D.	· A		,,,,	VA		$A \mathbf{R}$	$\mathbf{n}_{1}$	FRUIT	, r

11. Have you ever been screened/ checked for any of	the following conditions?
a.Hypertension (yes/no)	
b.Diabetes mellitus (yes/no)	
c.Hypercholesterolemia (yes/no)	
12. Have you ever been diagnosed to have any of the	following conditions?
a. Hypertension (yes/no); if yes are you on an	ntihypertensive medications? (yes/no)
b.Diabetes mellitus (yes/no); if yes are you o	on diabetic medications? (yes/no)
c.Hypercholesterolemia (yes/no); if yes ar	re you on anti-cholesterol medications?
(yes/no)	
13. Do any of your first-degree relatives (parents, sibl	ings or children) have hypertension?
a.Yes	
b.No	
c.I don't know	
14. Have you ever smoked cigarette?	
a.Yes	
b.No	
15. Have you ever taken alcohol?	
a.Yes	
b.No	
C.ANTHROPOMETRIC MEASUREMENTS	
16. Height cm	
17. Weight kg	
18. Waist circumference cm	
19. Office Blood pressure measurements	
a.Office SBP 1: (mmHg)	Office SBP 2: (mmHg)
b.Office DBP 1: (mmHg)	Office DBP 2: (mmHg)
c.Pulse rate 1:beats/min	Pulse rate 2:
beats/min	

	(mmHg)
	(mmHg)
	(mmHg)
	(mmHg)
	(mmHg)
	_
_ (mmol/l)	
_ (mmol/l)	
_ (mmol/l)	
(µmol/l)	
	_ (mmol/l)

# **Appendix IB: Dodoso**

# Dodosokuhusuupimajiwashinikizo la damu (ABPM) kwawafanyakaziwa MUHAS

B.	TAARIF	A BINAFSI ZA MUHUSIKA	
1.	Nambayaı	ıtafiti:	
2.	Tareheyau	ısahili:	
3.	Herufizaa	walizajina la muhusika:	
4.	Nambayas	simu:	
5.	Unapofan	yiakazi;	
	a.	Muhimbili	
	b.	Mloganzila	
6.	Jinsia		
	a.	Me:	
	b.	Ke:	
7.	Tareheyak	zuzaliwa:	_
8.	Kazikada:		
	a.	Muuguzi	
	b.	Daktari	
	c.	Kadanyingine	
9.	Kiwango	cha juu cha elimu	
	a.	Msingi	
	b.	Sekondari	
	c.	Diploma	
	d.	Digrii	
10.	Hali yand	oa	
	a.	Hajaoa/Hajaolewa	
	b.	Ameoa/kuolewa	

c. Mtalaka/Mtaliki/Mjane/Mgane

## B. VIASHIRIA HATARISHI VYA UGONJWA WA MOYO NA MISHIPA YA DAMU

11.	Ulishaw	ahikufan	viwauc	hunguziwa	ahivivifuat	atavvo?
	CIIDIIa	aiiii ai ai	j i waac	mangazi ,, ,	<i></i> , . ,	aca i j o .

- a. Shinikizo la juu la damu(ndio/hapana)
- b. Kisukari(ndio/hapana)
- c. Lehemu/Cholesterol yajuu(ndio/hapana)

## 12. Ulishawahikugundulikanamagonjwahayayafuatayo?

- a. Shinikizo la damu la juu(ndio/hapana); kamajibunindio, je unatumiadawazakupunguzashinikizo la damu? (ndio/hapana)
- b. Kisukari(ndio/hapana); kamajibunindio, je unatumiadawazakisukari? (ndio/hapana).
- c. Lehemu/Cholesterol yajuu(ndio/hapana);kamajibunindio, je unatumiadawazakupunguzalehemu/cholesterol? (ndio/hapana)
- 13. Je unanduguwakaribu (wazazi, kaka/dada au watoto) wenyeshinikizo la juu la damu?
  - a. Ndio
  - b. Hapana
  - c. Sijui
- 14. Ulishawahikuvutasigara?
  - a. Ndio
  - b. Hapana
- 15. Ulishawahikunywapombe?
  - a. Ndio
  - b. Hapana

C.VIPIMO V	YA MWILI			
16. Urefu	cm			
17. Uzito	kg			
18. Mzingowa	akiuno	cm		
19. Shinikizo	la damuofisi			
a.	Ofisi SBP 1:	(mmHg)	Ofisi SBP 2:	(mmHg)
b.	Ofisi DBP 1:	(mmHg)	Ofisi DBP 2:	(mmHg)
c.	Idadiyamapigomoyo	o 1:bea	ts/min	
	idadiyamapi	gomoyo 2:	beats/min	
20. Shinikizo	la damukwamasaa 24	l.		
21. Wastaniw	aupimajiwakatiwamc	hana		
a.	Wastanishinikizo la	damu la juumchana	SBP	(mmHg)
b.	Wastanishinikizo la	damu la chinimcha	na DBP	(mmHg)
22. Wastaniw	aupimajiwakatiwausi	ku		
a.	Wastanishinikizo la	damu la juuusiku S	BP	_(mmHg)
b.	Wastanishinikizo la	damu la chiniusiku	DBP	(mmHg)
23. Wastaniw	asaa 24 zaupimaji			
a.	Wastanisaa 24 shini	kizo la juu la damu	SBP	_(mmHg)
b.	Wastanisaa 24 shini	kizo la chini la dam	u DBP	(mmHg)
D. MAJII	BU KUTOKA MAA	BARA VIPIMO V	YA DAMU	
24. Fasting R	BG	(mmol/l)		
25. Total Cho	lesterol	(mmol/l)		
26. HDL-C _		(mmol/l)		
27. LDL-C		(mmol/l)		
28. Triglyceri	des	(mmol/l)		
29. Uric acid		(mmol/l)		
30. Serum cre	atinine	(µmol/l)		
31. Urea Nitro	ogen	(µmol/l)		

## **Appendix II: Letter of Ethical Clearance**

# MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES OFFICE OF THE DIRECTOR OF POSTGRADUATE STUDIES

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Ref. No. DA.287/298/01A/

2nd August, 2018

Dr. Godfrey Chuwa MMed. Internal Medicine MUHAS

RE: APPROVAL OF ETHICAL CLEARANCE FOR A STUDY TITLED: "PREVALENCE OF HYPERTENSION DETERMINED BY 24-HOURS AMBULATORY BLOOD PRESSURE MONITORING AMONG MUHAS EMPLOYEES"

Reference is made to the above heading.

I am pleased to inform you that, the Chairman has, on behalf of the Senate, approved ethical clearance for the above-mentioned study. Hence you may proceed with the planned study.

The ethical clearance is valid for one year only, from 1st August, 2018 to 30th August, 2019. In case you do not complete data analysis and dissertation report writing by 30th August, 2019, you will have to apply for renewal of ethical clearance prior to the expiry date.

Dr. Emmanuel Balandya

ACTING: DIRECTOR OF POSTGRADUATE STUDIES

cc: Director of Research and Publications

cc: Dean, School of Medicine



## Appendix III: FomuyaridhaakushirikikwenyeUtafiti

JinalanguniDr. Godfrey B. Chuwa.

Ninatokea ChuoKikuu cha AfyanaSayansiShirikishi Muhimbili katikaIdarayaTiba.

Ninafanyautafitikuhusushinikizo la juu la
damunaviashiriahatarishivyamagonjwayamoyonamishipayadamukwawafanyakaziwa MUHAS
(kampasiyaMloganzilana Muhimbili).

Ikiwahuuniutafitiwakisayansiunapaswakufahamuyafuatayokablayakushiriki;

Kushirikikwakonikwahiari. Unawezakukataakushiriki au kusitishamahojianoaumajadilianowakatiwowote.

Utafitihuuutahusishakujazadodosokuhusumasualayanayohusiananaafyayako, kupimashinikizo la mzungukowatumbonakiuno, damu, urefu, uzito, pamojanavipimovyadamukwaajiliyakujuawingiwalehemu/cholesterol, utendajikaziwafigonakiasi sukarimwilinimwako. Vile vileutawekewakipimo cha cha la kupimashinkizo damukwasaa 24, ambapoutahitajikakwendanakipimonyumbaninabaadayekukirudishakeshoyakekwaajiliyakuso mwanadaktarimatafiti.

Taarifazakoutakazozitoahazitawekwahadharanikwanamnayeyoteilekwahiyoushirikiwakohauta fahamika. Jinalako au taarifazozotezinazokutambulishahazitaambatanishwanataarifazakoutakazozitoa. Utapatiwamajibukuhusumatokeoyauchunguzina pia ushaurinasahakuhusunini cha kufanyakamakutakuwanatatizololotelililogundulika.

Usisitekuulizaswalilolote pale unapoonakunasababu.

Kama unamaswalizaidiambayoungependakuulizakuhusiananautafitihuu, tafadhali:

Wasiliananamtafitimkuu0756 699100idarayamagonjwayatiba Chuo Kikuu cha afya Muhimbili S.L.P. 65001 Dar es salaam, Tanzania.

 $Nimesoma\ au\ nimeambiwakuhusuyaliyo mohumundani. Maswaliyanguya mejibiwa.$ 

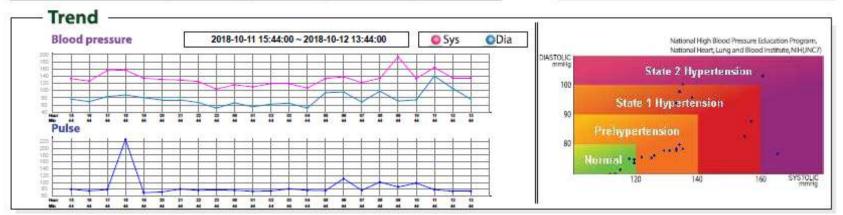
Nakubalikushirikikatikautafitihuu.

SAHIHI	TAREHE		
MTAFITI	TAREHE		

# **Appendix IV: Sample of ABPM results**



Name /ID				
Date	Height	Weight	Age	Gender
2018-10-12 14:23:58	157.0 cm	64.0 kg	57 yrs	FEMALE



# Summary -

Total time: 2018-10-11 15:44:00 ~ 2018-10-12 13:44:00 [ Valid sample : 23 Invalid sample : 0 ]

Awake / Asleep : Dipper

#### Total

	Min	Mean	Max	SD
SYS	104	133	193	19
DIA	51	78	140	19
pp	24	55	122	18
MAP	68	96	148	18
Pulse	70	87	226	31

#### Awake

ļ	Min	Mean	Max	SD
SYS	122	140	193	18
DIA	67	83	140	19
PP	24	57	122	22
MAP	86	102	148	16
Pulse	70	90	226	37

## Asleep

J	Min	Mean	Max	SD
SYS	104	118	138	11
DIA	51	68	96	17
PP	39	51	57	6
MAP	68	84	110	15
Pulse	74	81	111	11



Name /ID				
Date	Height	Weight	Age	Gender
2018-10-12 14:23:58	157.0 cm	64.0 kg	57 yıs	FEMALE

