EFFECTS OF STRUCTURED SLEEP EDUCATION ON BLOOD PRESSURE CONTROL AMONG ADULT HYPERTENSIVES ATTENDING JOS UNIVERSITY TEACHING HOSPITAL GENERAL OUT PATIENT CLINIC, JOS.

BY

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BM BCH, JOS (2006)

NOVEMBER, 2016.
DECLARATION

I, Shurkuk, Cletus B. hereby declare that this work is original unless otherwise acknowledged. This work has not been submitted in part or in full to any other examining body for an award or sent to any journal for publication.

Sign------------------------------------------

Dr Shurkuk, Cletus B.

Date-------------------------------------------
CERTIFICATION

This is to certify that this study was carried out by Dr Shurkuk, Cletus B. (BM BCH) at Jos University Teaching Hospital, Jos.

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DEDICATION

This work is dedicated to God Almighty who gave me the strength and wisdom, my wife Nentawe, my sons Rotshak and Rotnen and to all hypertensive patients.
ACKNOWLEDGEMENT

I am grateful to the management of Jos University Teaching Hospital for the assistance I received during my residency training.

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<td>%</td>
<td>Percentage</td>
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<tr>
<td>=</td>
<td>Equals</td>
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<td>&lt;</td>
<td>Less than</td>
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<td>&gt;</td>
<td>Greater than</td>
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<td>≤</td>
<td>Equals or less than</td>
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<tr>
<td>≥</td>
<td>Equals or greater than</td>
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<tr>
<td>±</td>
<td>Plus or minus</td>
</tr>
<tr>
<td>ADA</td>
<td>American Diabetes Association</td>
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<tr>
<td>AHI</td>
<td>Apnoea-hypopnoea index</td>
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<td>BMI</td>
<td>Body mass index</td>
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<tr>
<td>BP</td>
<td>Blood pressure</td>
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<td>CDC</td>
<td>Centre for Disease Control and prevention</td>
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<tr>
<td>CHD</td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
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<tr>
<td>CKD</td>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>CO</td>
<td>Cardiac output</td>
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<tr>
<td>CRP</td>
<td>C-reactive protein</td>
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<tr>
<td>CVD</td>
<td>Cardiovascular diseases</td>
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<tr>
<td>CXRs</td>
<td>Chest X-rays</td>
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<tr>
<td>DBP</td>
<td>Diastolic blood pressure</td>
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<tr>
<td>DF</td>
<td>Degree of freedom</td>
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<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
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<td>--------------</td>
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</tr>
<tr>
<td>ESR</td>
<td>Erythrocyte Sedimentation Rate</td>
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<tr>
<td>GOPC</td>
<td>General outpatient clinic</td>
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<tr>
<td>hr</td>
<td>Hour</td>
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<tr>
<td>HBP</td>
<td>High blood pressure</td>
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<td>HR</td>
<td>Heart rate</td>
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<tr>
<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>IU</td>
<td>International Unit</td>
</tr>
<tr>
<td>JNC-7</td>
<td>The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and treatment of high blood pressure</td>
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<tr>
<td>JUTH</td>
<td>Jos University Teaching Hospital</td>
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<tr>
<td>kcal</td>
<td>Kilo Calorie</td>
</tr>
<tr>
<td>kg</td>
<td>Kilogram</td>
</tr>
<tr>
<td>LGA</td>
<td>Local Government Area</td>
</tr>
<tr>
<td>MDG</td>
<td>Millennium Development Goals</td>
</tr>
<tr>
<td>mm$^3$</td>
<td>Cubic Millimeter</td>
</tr>
<tr>
<td>mg</td>
<td>Milligram</td>
</tr>
<tr>
<td>mmHg</td>
<td>millimeter of mercury</td>
</tr>
<tr>
<td>₦</td>
<td>Naira</td>
</tr>
<tr>
<td>NHANES</td>
<td>The National Health and Nutrition Examination Survey</td>
</tr>
<tr>
<td>NHP</td>
<td>National Health Policy</td>
</tr>
<tr>
<td>NPHCDA</td>
<td>National Primary Healthcare Development Agency</td>
</tr>
<tr>
<td>NREM</td>
<td>Non rapid eye movement</td>
</tr>
<tr>
<td>OR</td>
<td>Odd ratio</td>
</tr>
<tr>
<td>P-Value</td>
<td>The probability of obtaining a result equal to or more</td>
</tr>
<tr>
<td>PHC</td>
<td>Primary Health Care</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>PSQI</td>
<td>Pittsburg Sleep Quality Index</td>
</tr>
<tr>
<td>RCTs</td>
<td>Randomized controlled trials</td>
</tr>
<tr>
<td>REM</td>
<td>Rapid eye movement</td>
</tr>
<tr>
<td>RR</td>
<td>Relative risk</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>SES</td>
<td>Socio-economic status</td>
</tr>
<tr>
<td>SHHS</td>
<td>Sleep Heart Health Study</td>
</tr>
<tr>
<td>t-test</td>
<td>Student t-test</td>
</tr>
<tr>
<td>UNICEF</td>
<td>United Nation Children Education Fund</td>
</tr>
<tr>
<td>USA</td>
<td>United States of America</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
<tr>
<td>$X^2$</td>
<td>Chi square</td>
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</table>
Summary

Background: Sleep is one of the most basic elements of human lives. It improves physical and mental abilities. Sleep related problems are important public health concerns and there exists growing evidence that poor sleep quality is associated with increased cardiovascular complications of which hypertension is one. There is a downward trend in sleep quality; and poor sleep quality has been noticed to be more common among hypertensive patient than the general population. This study was therefore, designed to look at the effect of improving sleep quality on blood pressure.

Objective: To determine the effects of structured sleep education on blood pressure control among adult hypertensive patients.

Study design/Setting: A randomized controlled trial involving 212 patients with hypertension, aged 18 years and above presenting in the General Outpatient Clinic of the Jos University Teaching Hospital.

Method: Participants were randomly allocated to the control and the intervention groups. The intervention groups were offered structured sleep education on sleep hygiene that was delivered in a structured format to the participants. Data collected from the participants include socio-demographic data, medical and family history, sleep quality was assessed using the Pittsburg’ sleep quality index (PSQI) questionnaire. Focused physical examination was done. Participants were followed up monthly over 12 weeks during which measurements were repeated.

Results: Majority (64.6%) of the participants had poor sleep quality with PSQI score of 6.86±3.52. Most (75.9%) of them had short sleep and slept less than seven hours per night. The mean systolic and diastolic blood pressure in the intervention group reduced from
145.19±21.00mmHg to 127.50±16.19mmHg and 91.66±11.66mmHg to 80.00±6.76mmHg respectively after intervention. This was statistically significant. (p=0.001). This study found a positive relationship between sleep quality and hypertension.

**Conclusion:** Structured sleep education administered along with routine medical care for hypertension led to reduction in blood pressure and improvement in the quality of sleep.
CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND INFORMATION

Hypertension (HTN) is the most common cardiovascular disease and one of the reasons patients visit Family Physicians. It is defined as systolic blood pressure greater than or equal to 140 mm Hg or diastolic blood pressure greater than or equal to 90 mmHg. The World Health Organization’s report on non-communicable diseases in 2008 showed that approximately 40% of adults aged 25 and above had been diagnosed with hypertension globally; the number of people with the condition rose from 600 million in 1980 to one billion in 2008. By 2025, the number of people with hypertension (HTN) will increase by about 60% to a total of 1.56 billion as the proportion of elderly people will increase significantly.

Approximately 30% of adult Americans have hypertension and 7% of adults with HTN have not been told by their physician that they have high blood pressure (HBP). The National Health and Nutrition Examination Survey (NHANES) report of 2005 in the United States found that in the population aged 20 years or older, the prevalence of pre-hypertension (Systolic Blood Pressure (HBP), 120-139 mm Hg; Diastolic Blood Pressure (DBP), 80-99 mm Hg), is 41.9 million in men and 27.8 million women. For stage 1 hypertension (SBP, 140-159 mm Hg; DBP, 90-99 mm Hg), it is 12.8 million men and 12.2 million women while in stage 2 hypertension (SBP ≥160 mm Hg; DBP ≥100 mm Hg) the prevalence for men and women are 4.1 million and 6.9 million respectively.

According to the Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7), systolic blood pressure of greater than 140 mm Hg is a more important cardiovascular disease risk factor than diastolic
blood pressure in individuals older than 50 years of age. But current reports by the Eighth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-8) states that systolic blood pressure of greater than 150 mmHg is an important cardiovascular risk factor in persons aged 60 years or older, and DBP of >90 mmHg in those less than 60 years. Black individuals have a higher prevalence and incidence of hypertension than white persons. The World Health Organization (WHO) 2010 Global report on non-communicable diseases showed that the prevalence of hypertension was highest in the African region. They reported a prevalence of 46% for both sexes in those aged 25 years and above, while the lowest prevalence of raised blood pressure was in the Americans, with 35% for both sexes. The reason for the high prevalence in Africa is due to the influence of the low income and socioeconomic status. The report also showed that age is an important risk factor for hypertension. Reports by the National Centre for Health and Statistic in the United State of America(USA), JNC-7, JNC-8 and WHO confirms this with advancing age.

In the West African sub-region, the prevalence of hypertension between 1970 and 2009 in Ghana was in the range of 19% to 48%, and in 2007, hypertension was said to be the second leading cause of outpatient morbidity in the Greater Accra region. In Nigeria and other developing countries there has been an increase in the prevalence of hypertension. A 2010 report by WHO put the prevalence of high blood pressure in Nigeria at 42.8% in both sexes. A study in South-South semi-urban Nigeria showed that 47.0% of the study population had a raised BP >140/90mmHg. In a meta analysis in South-West Nigeria the prevalence of hypertension ranged from a minimum of 12.4% to a maximum of 34.8%. In Jos, North-Central, the prevalence of hypertension was 36.6%. The rising prevalence of hypertension in Nigeria and other developing countries is due to change in diet and lifestyles and
improved access to health facilities for early screening and detection of hypertension and due to adoption of western lifestyle and diet.\textsuperscript{12}

Because of the increasing prevalence of hypertension, the World Health Organization declared April 7, 2013 as a year of hypertension with the theme "silent killer, global public health crisis." with the slogan "healthy heart beat, healthy blood pressure, while the World Health Day for hypertension is celebrated on the 17\textsuperscript{th} of May annually."\textsuperscript{3} Report by WHO, 2010 showed that high-income countries have a lower prevalence of hypertension which was 35\% as compared to that of low-income countries which was 40\%.\textsuperscript{3}

The classification of blood pressure is the same both in JNC-7 and JNC-8 as shown in appendix A. The only difference is that in JNC-8 the definition of pre-hypertension and hypertension were not addressed.\textsuperscript{2, 5}

The risk factors for high blood pressure are classified into modifiable and non-modifiable. The non-modifiable factors include advancing age, genetic predisposition and race, while the modifiable risk factors include obesity, excessive alcohol consumption, psychological stress, short sleep duration, sedentary lifestyle, unhealthy diet.\textsuperscript{2, 3, 6}

Worldwide hypertension has been estimated to cause 7.5 million deaths, about 12.8\% of the total of all annual death in adults.\textsuperscript{3} It is a major risk factor for coronary heart disease and ischaemic as well as haemorrhagic stroke.\textsuperscript{3} The report by JNC-7 showed that in some age groups, the risk of cardiovascular disease doubles for each incremental increase of 20/10 mmHg of blood pressure, starting as low as 115/75 mmHg.\textsuperscript{2}

In treating high blood pressure, a study by Egan showed that maintaining systolic blood pressure and diastolic blood pressure so that they are below 140/90 mmHg is associated with a reduction in cardiovascular complications.\textsuperscript{13} Treatment of high blood pressure using the JNC7 algorithm involves both lifestyle modifications and the use of right combination of drugs.\textsuperscript{2}

This algorithm is similar to JNC-8, except that drug therapy for JNC-7 is from five classes.
while for JNC-8 is from four classes.\textsuperscript{3, 5} A population based policy and systems change approach which involves lifestyle changes in population, that decreases the BP level in the general population by even modest amounts has the potential to substantially reduce morbidity and mortality or at least delay the onset of hypertension.\textsuperscript{2, 14} For instance, it has been estimated that a 5mmHg reduction of SBP in the population would result in a 14 percent overall reduction in mortality due to stroke, a nine percent reduction in mortality due to coronary heart disease (CHD), and a seven percent decrease in all-cause mortality.\textsuperscript{2}

When high blood pressures are not controlled, complications such as coronary heart diseases, stroke, heart failure, peripheral vascular disease, renal impairment, retinal haemorrhage and visual impairment can occur.\textsuperscript{3}

One of the risk factors for poor BP control in hypertension is insufficient sleep and poor sleep quality.\textsuperscript{15, 16} Sleep is one of the most important elements of human lives and improves the physical and mental abilities.\textsuperscript{17} Sleep quality is one of the dimensions of sleep, other dimensions include sleep duration, which is the total amount of sleep obtained per 24 hours; sleep efficiency or continuity which is the ease of falling asleep and returning to sleep.\textsuperscript{18} Others include timing of sleep, ability to maintain attentive wakefulness (alertness/sleepiness) and satisfaction.\textsuperscript{18} Sleep quality is the assessment of sleep as either good or poor.\textsuperscript{18} It measures both the quantitative and the qualitative component of sleep.\textsuperscript{19} Using the Pittsburg Sleep Quality Index (PSQI), good sleep is defined as PSQI score of five or less, while poor sleep is defined as score of greater than five.\textsuperscript{19} Sleep duration is also used to define sleep quality. Normal sleep is defined as sleeping between six to eight hours, while sleeping less than six or more than eight hours is referred to as short and long sleep respectively and represent poor sleep quality.\textsuperscript{19, 20, 21} Complaints about sleep quality are common in epidemiological surveys and indicate that 15-35% of adult population complain of frequent sleep quality disturbance.\textsuperscript{19}
Study by Bruno and colleagues, showed that resistant hypertension is associated with poor sleep quality, increased sleep latency and reduced sleep efficiency. Short sleep duration is associated with elevated blood pressure and the risk of other cardiovascular diseases. Insufficient sleep may increase the risk of hypertension and cardiovascular disease through effects on sympathetic nervous system activation by alteration of the hypothalamus-pituitary-adrenal axis leading to secretion of cortisol and renin-angiotensin system and augmented systemic levels of inflammation such as elevation of C-reactive protein levels. However, there are evidences that a good night sleep is associated with better control of blood pressure. Blood pressure declines to its lowest levels during nighttime sleep when the parasympathetic activity is highest. Consequently, the arteries of people with short sleep duration benefit the least from this positive effect. Indeed, part of the association was explained by adjusting for BMI, total HDL cholesterol ratio, systolic blood pressure, and prevalence of type 2 diabetes. Therefore, there is the need to establish the relationship between sleep quality and blood pressure control in hypertensive patients.

Sleep is increasingly recognized as important to public health, with sleep insufficiency linked to motor vehicle crashes, industrial disasters, and medical and other occupational errors. It can even contribute to cardiovascular disorders and mortality. Activation of the sympathetic nervous system is an important mechanism that links sleep disorders with combined systolic and diastolic hypertension, which is common in the middle-aged hypertensive patients. Individuals with both shorter and longer sleep had an increased risk of hypertension. Short sleep and long sleep duration are also associated with weight gain, obesity, type 2 diabetes, cardiovascular disease, and premature mortality.

Technological advancements have afforded modern society with 24-hour work operations, trans-meridian travel and exposure to a myriad of electronic devices such as televisions, computers and cellular phones that keep people awake most of the times leading to shorter
sleep duration. There is a growing evidence which suggests that these advancements take their toll on human functioning and health through their damaging effects such as increase in sedentary lifestyle and its effect on sleep quality, quantity and timing.

The factors that affect sleep duration are socio-economic, occupation and race/ethnicity. Studies showed that usual sleep duration above or below the median of seven to less than eight hours per night is associated with an increased prevalence of hypertension, particularly at the extreme of less than six hours per night indicating the effect of sleep duration on blood pressure. Short sleepers according to Knutson et al were defined as those reporting < 6 hours of sleep in their time diary. They also showed that percentages of short sleepers ranged from 7.6% in 1975 to 9.3% in 2006. The study showed that the highest proportion of short sleepers was observed in the 1998-99 data, which had the smallest sample size. A Study by Egan and the report by the National Sleep Foundation in US showed that sleep duration has decreased in the general population over the last 30 years. It also reported an increase from 12% to 16% of subjects sleeping less than 6 hours on workdays confirming the reduction in the sleep duration among the general population. Other studies also showed that poor sleep quality and sleep disorders can affect blood pressure and vice versa. This is because decrease sleep duration and sleep quality leads to prolonged exposure to activated sympathetic nervous system which could accentuate structured remodeling and augment renal sodium retention. Sleep restriction and sleep disorders are both associated with increased prevalence and incidence of hypertension. Sleep deprivation studies in normotensive subjects have demonstrated that BP was increased after nights of sleep restriction.

All the major physiological changes are influenced by sleep. There are two stages of sleep: the non rapid eye movement(NREM) and the rapid eye movement(REM), each marked by characteristic physiologic changes. The Non rapid eye movement(NREM) sleep constitutes about 75 to 80% of total sleep time in adults. It consists of 4 stages in increasing
depth of sleep. Stage one is the state between wakefulness and sleep, the muscle are active and the eye roll around slowly. It is the period of transition from a relatively unsynchronized beta and gamma brain waves to more synchronized but slower alpha and then to theta waves. Stage two is characterized by further decrease in muscle activity and conscious awareness of the outside world, the theta waves predominate and is characterised by sleep spindles and k-complexes. In stage three, the individual is less responsive to the outside environment and unaware of any sound or stimuli and blood pressure, neuronal activity, temperature, heart and respiratory rates are all at their lowest level. This stage is characterised by delta brain wave or slow wave sleep with some sleep spindles fewer than in stage two. Stage four is characterized by delta wave that exceed more than 50% of the total wave. Stages three and four are referred to as deep or delta sleep because arousal threshold is high; people may perceive these stages as high-quality sleep.

Cardiovascular changes, which include the decrease in blood pressure and heart rate, occur during the NREM particularly during the slow-wave sleep. The REM sleep follows each cycle of NREM sleep. It is characterized by low-voltage fast activity on the EEG and postural muscle atonia. Respiratory rate and depth fluctuate dramatically. Most dreams occur during REM sleep. Most adults sleep 7–8 hours per night, although the timing, duration, and internal structure of sleep vary among healthy individuals and as a function of age. Report by European Society of Hypertension said that Blood pressure (BP) and heart rate (HR) decrease throughout NREM sleep, particularly during slow-wave sleep (dipping pattern). However, good sleep hygiene has been found to reduce blood pressure. The move toward the use of non-drug approach to improved sleep is due to the unwanted side effects of drugs.

1.2 STATEMENT OF THE PROBLEM

It is worthy of note that not all hypertensive patients who are on drugs have achieved blood pressure control. In a study on prevalence of hypertension in Lagos, it was found that control
of hypertension was poor in 78.79% of the subjects possibly because of low earning capacity of these subjects. A study in Zaria, North Western Nigeria showed that only 12.4% of patients attending Tertiary Health Centre had their blood pressure controlled while only 67.3% reported compliance with their medication. In Ilorin, North Central Nigeria Blood Pressure control is still unacceptably poor among hypertensive Nigerians. This may not be unconnected with the poor knowledge of high blood pressure and adverse practices by the patients. Because of these, many studies have been carried out to find ways of improving blood pressure control. These include lifestyle modifications and improving quality of sleep. Sleep plays an important role in the life of human being, and that is why the National Sleep Foundation suggested that most adults need 7–9 hours of sleep per night, although individual variations exist. It also reported that 35.3% of Americans are having less than 7 hours of sleep on average during a 24-hour period. Studies have shown a relationship between sleep duration and hypertension. In Japan both long and short habitual sleep duration were significantly associated with high blood pressure values and hypertension occurrence in Japanese male subjects. Other studies showed that sleeping 5 hours or less per night was associated with a 60% increased risk of incident hypertension and subjects sleeping more than 9 hours per night had 30% higher prevalence of hypertension. Further studies carried out establish the relationship between sleep and hypertension. These studies showed that usual sleep duration above or below the median of 7 to less than 8 hours per night is associated with an increased prevalence of hypertension, particularly at the extreme of less than 6 hours per night. Short term experimental sleep restriction for as little as one night has been reported to increase blood pressure in both healthy and hypertensive subjects. In a natural experiment of sleep deprivation occurring in Japanese workers intermittently exposed to long shifts, in which sleep duration was curtailed to 3.6
hours compared to nights when sleep was reported to be 8 hours was associated with an average increase in systolic and diastolic blood pressure of 6 and 3mmHg respectively.\textsuperscript{20} Redline reported that after adjusting for numerous confounders, the odds of hypertension increased by approximately 70\% and 20\% for those reporting sleep duration of less than 6 hour and between 6 and 7 hour per night respectively.\textsuperscript{20} Those reporting more than 9 hours of sleep per night had an approximately 30\% increased odds of hypertension.\textsuperscript{20} A decreased odds of hypertension was also observed in association with improved sleep efficiency.\textsuperscript{20} Intervention studies are therefore needed to assess whether improving the quality of sleep will help to reduce blood pressure since in Nigeria there is no study on the effect of sleep on blood pressure control. Increasing evidence have suggested that adequate duration and quality of sleep are important for overall health.\textsuperscript{42} A structured sleep education is aimed at improving the quality of sleep by educating the patients to practice good sleep hygiene. Sleep hygiene has been defined as those behaviors that are believed to promote and improve quantity and quality of sleep.\textsuperscript{43} This can be achieved by keeping a regular sleep schedule; avoiding stimulating activities (e.g., vigorous exercise) within 2 hours of bedtime; avoiding caffeine, nicotine, and alcohol in the evening; avoiding going to bed on a full or empty stomach; and sleeping in a dark, quiet, well-ventilated space with a comfortable temperature.\textsuperscript{34, 44} Because chronic sleep loss has a cumulative effect on mental and physical well-being, potentially exacerbating depression, obesity, diabetes, and other chronic conditions, treatment of patients with chronic diseases might benefit from counseling about the importance of sufficient sleep.\textsuperscript{41} Several studies have been carried out on the relationship between sleep duration, sleep disorders and hypertension in other countries but only little is known about the effect of
sleep on blood pressure control in my institution; the Jos University Teaching Hospital. 14, 15, 23, 24, 27
1.3 OBJECTIVES OF THE STUDY

General Objectives
To assess the effect of structured sleep education on blood pressure control with the view of incorporating it in the management of adult hypertensive patients attending the general outpatient clinic (GOPC) of the Jos University Teaching Hospital (JUTH)

Specific objectives

1. To determine the socio-demographic characteristics of the adult hypertensives presenting at GOPC of JUTH.

2. To determine the sleep quality in the study population using the Pittsburg sleep quality index.

3. To compare the blood pressure changes from baseline in the group of hypertensive patients who had structured sleep education and those without structured sleep education.

1.4 SIGNIFICANCE OF THE STUDY

This study is worth carrying out because studies in Zaria, Nsukka, Ilorin and Ife showed that most hypertensive patients have not achieved good blood pressure control.\textsuperscript{36, 45, 46, 47} Hypertension has been shown to affect sleep quality and vice versa.\textsuperscript{16,22,32} Several studies have linked insufficient sleep and poor sleep quality to development of hypertension and their effect on quality of life.\textsuperscript{14,16,24,27} Therefore, this research if found significant will help in the reduction of blood pressure among hypertensive patients by improving the quality of sleep among them and thereby improving their quality of life. Information from this study would be utilized in providing care and management of hypertensive patients. It will be incorporated into the lifestyle modifications approach in the management of high blood pressure in hypertensive patients to reduce the burden and cost of treating hypertension.
CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

The purpose of the various committees on hypertension is to provide an evidence-based approach to the prevention and management of hypertension.\(^2\,\,5\) Hypertension is the most common condition seen in primary care and leads to myocardial infarction, stroke, renal failure, and death if not detected early and treated appropriately.\(^5\) Abundant evidence from randomized controlled trials (RCTs) have shown the benefit of antihypertensive drug treatment in reducing important health outcomes in persons with hypertension.\(^5\) The panel members appointed to the eighth Joint National Committee (JNC 8) used rigorous evidence-based methods to develop evidence statements and recommendations for blood pressure (BP) treatment based on a systematic review of the literature to meet user needs, especially the needs of the primary care clinician.\(^5\) It is important to note that the key messages in both JNC-7 and JNC-8 is that age is an important factor in the development of hypertension.\(^2\,\,5\) Two-thirds of individuals aged older than 65 years have hypertension.\(^2\) This was similar to the finding of Ostchega and colleagues where they showed that individuals older than 60 years had a higher prevalence of hypertension when compared with those in younger age group.\(^7\) Other risk factors include family history, sedentary life style, poor diet, cigarette smoking, sex and race.\(^48,\,49\) One unconventional and often overlooked risk factor is sleep.\(^48\) Several studies have shown insufficient sleep as a risk factor for elevated BP and hypertension.\(^16,\,23,\,24\) The risk associated with increasing blood pressure is continuous, with each 2mmHg rise in systolic blood pressure associated with a 7% increased risk of mortality from ischaemic heart disease and a 10% increased risk of mortality from stroke.\(^50\)
2.2 Epidemiology

The World Health Organization (WHO) report on non-communicable diseases in 2008 showed that globally, approximately 40% of adults aged 25 and above had been diagnosed with hypertension; the number of people with the condition rose from 600 million in 1980 to one billion in 2008. By 2025, the number of people with hypertension (HTN) will increase by about 60% to a total of 1.56 billion as the proportion of elderly people will increase significantly. This may be due to an increase in detection rather than a temporal increase as the observation is limited by lack of serially conducted studies in any of the population. Approximately 30% of adult Americans and an estimated 1 in 3 adults in the United States have hypertension and 7% of adults with HTN have not been told by their physician that they have high blood pressure (HBP).

The lowest prevalence of raised blood pressure was in the Americas, with 35% for both sexes, this is because of high income earning in the population. In the West African sub-region, hypertension prevalence rates between 1970 and 2009 in Ghana, revealed a range of 19% to 48% and in 2007, hypertension was said to be the second leading cause of outpatient morbidity in the Greater Accra region. Study in South-South semi-urban Nigeria showed that 47.0% of the study population in a community based study, had a raised BP >140/90mmHg. In 2010, WHO reported that prevalence of hypertension in Nigeria was 42.8%, while in a meta analysis in South-West Nigeria the prevalence of hypertension ranged from a minimum of 12.4% to a maximum of 34.8%. The combined prevalence was as high as developed countries and as reported by Ojumu in Jos, North-Central Nigeria but lower than in African region and other part of the North-Central zone. He reported a prevalence of 36.6% which was higher than 22.7% reported by Adediran and colleagues in other part of the North-Central zone. The major reason for this trend included age, increase prevalence of obesity, physical inactivity, unhealthy diet, stress, increased longevity, short and poor sleep.
reported a prevalence of 21.3%. This was due to the inclusion of younger individuals in the study.

Age has been shown to be the most likely determinant of blood pressure and the development of hypertension. Blood pressure increase steadily with age from the youngest to the oldest age brackets, irrespective of gender. The prevalence of hypertension increased with age among all the participants. This could be due to progressive arterial stiffness as a result of structural and functional changes within the vessel wall. Ji-Rong and colleagues in China also showed that with advancing age, there is increasing prevalence of systolic and isolated systolic hypertension because of the progressing arterial stiffness and there is decline in diastolic blood pressure. This suggest that both systolic and diastolic blood pressure have different clinical significance in the elderly. Ekanem and colleagues in 2013, showed that in South-South Nigeria, the age range for hypertension was 16 to 65 years with a mean age of 31.7 ± 7.6 years, among the residents in military barracks which consist mainly of young individual. The mean age was lower than 59.05 ± 9.06 years seen in the study by Iyalomhe et al which also showed a higher range of 35 – 80 years with a mean years and the modal age group of 56 – 60 years. On the other hand, Adediran et al in 2013 showed that in the Middle-belt Nigeria, SBP increased with age with a peak in the 60-69 age groups, while the DBP peaked in the 40 – 49 age groups. They also revealed that the prevalence of hypertension increased with age from 11% in the 20- 29 age group to 40.7% in the 60-69 age group. Ugwuja and colleagues found just like in other studies that the prevalence of hypertension was significantly higher among the older age groups of 46–55 (P = 0.02) and >55 (P = 0.02) years. This show that age also contributes to the prevalence of hypertension. However, least prevalence of hypertension was observed in the age group 26–35 years which was 9.7%. In those that were between 70 -79 years of age, they noticed a drop in the
prevalence of hypertension. This may be accounted for by the few number of participants in this age group and the short life span in the environment. Ekanem and colleagues showed that both systolic and diastolic blood pressure increased with increase in age in younger age group, except for those aged >60 years. The study also showed that for every year increase in age, there was an increased risk of high BP to the tune of 8%, p<0.001. Ekwunife reported that prevalence of hypertension increases with advancing age to the point where more than half of people aged 60 to 69 years old and approximately three-quarters of those aged 70 years and older are affected.

Several studies on the relationship between gender and the prevalence of hypertension showed that hypertension is more common among men than in women. Ugwuja and colleagues in Southeast Nigeria, showed that males had a higher prevalence of hypertension in comparison to their female counterparts; 28.2% versus 20.9%. This was similar to the findings of Ekanem et al where they reported a prevalence of 30.2% in males and 16.8% in females (p<0.001). This was similar to other studies but lower than 40.3% and 24.7% for males and females respectively as reported by Ekwunife et al in Nsukka. However, both the systolic and diastolic BP increased with age in both men and women.

Conversely, Ogah et al in Abia showed a higher prevalence in women than men (3.5%-68.8% vs. 7.9%-50.2%, respectively). This was also similar to the findings of Wokoma and colleagues in Barako, a rural community in Rivers where there was predominance of females (almost twice as men). This may be a reflection of rural-urban drift of able bodied men in search of job opportunities in the cities and local government headquarters, leaving behind women and children in the rural area and also due to higher healthcare seeking behavior among women than men. Similarly, Maas and colleague reported that in the middle-aged and elderly women who have reached menopause or experiencing perimenopausal syndrome, have high risk factor for hypertension and coronary heart disease. This was due to decline in
the cardio protective function of oestrogen which was believed to inhibit the progression of atherosclerosis.\textsuperscript{60} Oestrogen and oestrogen-androgen ratio in women start to decrease after the age of 40 years.\textsuperscript{60} In aging women, systolic blood pressure rises more steeply compared with men and is due to hormonal changes during menopause.\textsuperscript{60} Another reason may be due to increasing obesity. Abiodun et al in Ibadan showed that female subjects were more obese (P<0.0001) and systolic-diastolic hypertension (SDH) was prevalent among the obese group. They also found that among the study population, the prevalent of systolic-diastolic hypertension and isolated systolic hypertension were 77.6\% and 73.5\% respectively. This study also showed that IDH was more prevalent in females (4.9\%) than males (4.7\%) whereas, ISH was more common in males (10.1\%) than females (6.2\%) with (P= 0.048).\textsuperscript{61} In 2010, WHO reported that the prevalence of high blood pressure in Nigeria was 42.8\% in both male and female.\textsuperscript{3} While Adeniran et al also showed that there was no significant difference in the mean SBP or DBP in both sexes, even though hypertension prevalence differed little in men and women with rates of 21.2\% versus 22.4\% respectively ($\chi^2$=0.274, p = 0.60).\textsuperscript{52} However more females had hypertension than males.\textsuperscript{52}

Ekanem and colleagues in Uyo, Akwa Ibom showed that majority of the participants (44.6\%) earned between ₦30, 000 and ₦50,000 per month (equivalent of $200 and $333).\textsuperscript{10} Awobusiyi and colleagues in 2012, showed that the monthly income among hypertensive in Lagos was generally low with 44\% percent of all subjects studied earning less than ₦10,000.00 per month which is lower than the country minimum wage of ₦18,000.\textsuperscript{33} Most of the hypertensive patients (67.5\%) had at least a secondary education.\textsuperscript{10} Other study showed that Literacy level was high 72.56\% and level of education appears not to affect control of hypertension.\textsuperscript{33} The study by Awobusiyi and colleagues also showed that 21.34\% hypertensive had no formal education, more than forty-eight percent (48.17\%) hypertensives had either primary or secondary education, while 30.49\% had tertiary education.\textsuperscript{36} Report by
United Nations Educational, Scientific and Cultural Organization (UNESCO) in 2012 on literacy level showed that in Nigeria, adult literacy rate was 56.9%. It also reported that literacy level is higher in males than in females due to low girl child education. Egbi and colleagues showed that participants with primary level of education had higher proportions of hypertensives compared with those with a minimum of a secondary level of education. It is more likely that education, as a marker of socio-economic status, may be associated with lifestyle habits that help account for differences in both high blood pressure and lower hypertension prevalence rates. The low level of hypertension among the high socio-economic class is due to improved capacity to seek and pay for medical services. On the other hand it can worsen the condition due to adoption of unhealthy lifestyle. In the low socio-economic class, the high prevalence of hypertension is due to inability to procure drug because of the cause, limited access to health facility etc. While in some there is low prevalence of hypertension due to adoption of healthy lifestyle practices.

Egbi and colleagues reported that marital status had a significant relationship with hypertension. They also reported a lower prevalence of HTN among those who were unmarried compared with their married counterparts, this is due to increasing age. This is similar to a report by Onwuchekwa et al. Lipowicz and colleagues on the other hand observed an increased risk of higher blood pressure among unmarried individuals compared with married people, this was due to prolonged stress, low social support and economic aspect of living alone. Although being single may have a relative adverse effect on health, including the likelihood of having high blood pressure, marital transition from unmarried to married life has also been associated with changes in lifestyle, some of which may negatively impact on blood pressure. It may be due to loss of spousal support and associated stress which may be experienced by separated or widowed individual.
The increased in body mass index (BMI) is also a risk factor for hypertension. Several studies have shown that increasing body mass index was associated with hypertension. In a study by Ekanem and colleagues in a cross sectional study in semi-urban Nigerian community in 2013, showed that 46.6% of hypertensive patient had normal BMIs, while the prevalence of overweight and obesity were 35%, and 17.2% respectively. The higher prevalence of overweight and obesity in the urban population may indicate differences in lifestyle. Other reasons include increase in salt and fat intake from consumption of processed foods and participating in jobs with minimal physical activities. The urban populations are more likely to eat processed foods which are high in salt and fat content. Obesity which is a risk factor for hypertension is also higher in urban areas than in rural areas. Adeniran et al showed that hypertension was more prevalent in the urban than rural dwellers with rates of 32.7% and 12.9% respectively ($\chi^2=12.80, p<0.001$). The mean BMI and waist circumference were significantly higher in the urban than rural dwellers while the BMI was more prevalent with rates of 40.7% versus 4.3% ($\chi^2 = 43.81, p<0.001$) respectively.

Several studies have linked the increase in obesity to a high prevalence of hypertension in urban than rural population. This was contrary to the study of Ogah and colleagues in which they reported that hypertension was high in rural population as in urban population, this was likely because most of the rural population are older as more people move to rural areas after retirement. Bosu in his systemic review reported that hypertension was a significant problem not only in urban populations but also in poorest and leanest rural populations. Addo and colleagues in Ghana reported a prevalence ranging from 19.3% in rural to 54.6% in urban areas, this was due to increase number of the elderly, excess alcohol intake and increasing weight gain.
Majority of the hypertensive patients attending University of Ilorin Teaching Hospital were either traders or business men/women (44.5%). Black people clearly have a higher prevalence than either group of white people. This is due to lower plasma rennin activity when compared to the white people. The rennin activity also declines with age.

Lifestyle practices play an important role in the development of hypertension. It was surprising to note that cigarette smoking was a statistically significant predictor of high BP at the univariate level only. There could be some degree of under-reporting of smoking among women which may have to do with the fact that the culture in most parts of Nigeria frowns at smoking particularly among the females. On the other hand, Awobusuyi showed that 7.19% of hypertensives were active smokers and 43.6% had history of intake of alcohol.

The other risk factor for hypertension includes family history of hypertension. Egbi and colleagues in Yenogoa, showed that alcohol consumption, history of cigarette smoking, family history of HTN and family history of diabetes did not show statistically valid higher rates of HTN even though they are commonly considered as risk factors. It is possible that doses of exposure in these individuals may not have been enough to cause significant association with hypertension.

Hypertension often occurs with co-morbidities such as obesity, dyslipidaemia and diabetes.

Study on profile of hypertensive patients in Lagos, showed that control of hypertension was poor in 78.79% of the subjects possibly because of low earning capacity of these subjects. Ekwunife also reported that 22.2% were taking antihypertensive medication, but only 6.2% had optimal blood pressure control. Similarly, in Zaria, Albert et al showed that only 12.4% of patients attending Tertiary Health Centre had their blood pressure controlled while only 67.3% reported compliance with their medication. While in the middle belt of Nigeria Blood Pressure control is still unacceptably poor among hypertensive Nigerians. This may not be
unconnected with the poor knowledge of high blood pressure and adverse practices by the patients. Because of these many studies have been carried out to find ways of improving blood pressure control. These include lifestyle modifications and improving the quality of sleep.

The JNC-7 panel reported that pre-hypertensive individuals’ (systolic BP 120–139 mm Hg and or diastolic BP 80–89 mm Hg) require health-promoting lifestyle modifications to prevent the progressive rise in blood pressure and CVD. For uncomplicated hypertension, thiazide diuretic should be used in drug treatment for most, either alone or combined with drugs from other classes. While in JNC-8 there is strong evidence to support treating hypertensive persons aged 60 years or older to a BP goal of less than 150/90mmHg and hypertensive persons between the ages of 30 to 59 years of age to a diastolic goal of less than 90mmHg. There is insufficient evidence in the goal to be achieved in hypertensive persons younger than 60 years for a systolic blood pressure and those younger than 30 years for a diastolic blood pressure. So the panel recommends a BP of less than 140/90mmHg as the goal BP for those groups based on expert opinion. In individuals with diabetes mellitus or non-diabetic chronic kidney disease (CKD), JNC-7 recommended a target blood pressure of less than 130/80mmHg, while JNC-8 recommended a target blood pressure of less than 140/90mmHg.

2.3 Pathophysiology of hypertension

Despite major advances in understanding the pathophysiology of hypertension, availability of effective and safe antihypertensive drugs, suboptimal blood pressure (BP) control is still the most important risk factor for cardiovascular mortality and is globally responsible for more than 7 million deaths annually. In humans primary (essential) hypertension, the precise causes are not completely understood, although excessive weight gain and dietary factors appear to play a major role since hypertension is rare in non-obese hunter-gatherers living in
non-industrialized societies.\textsuperscript{64} Recent advances in genetics offer opportunities to discover gene-environment interactions that may also contribute to hypertension, although success thus far has been limited mainly to identification of rare monogenic forms of hypertension.\textsuperscript{64} The pathogenic mechanisms of BP involve increased cardiac output (CO), total peripheral vascular resistance (TPR), or both because, BP equal to CO X TPR.\textsuperscript{65} In primary hypertension, CO is normal or slightly increased and TPR is increased. There is abnormal Na\textsuperscript{+} transport across the cell wall because of defect in Na\textsuperscript{+}-K pump leading to increase intracellular sodium, accumulation of intracellular calcium with resultant increase in sympathetic stimulation. This will enhance the effect of norepinephrine and increase BP.\textsuperscript{65} Sympathetic stimulation increases BP more in patients with pre-hypertension or hypertension than in normal patients. The role of renin-angiotensive-aldosterone system in primary hypertension is not established. However, in black and elderly patients with hypertension, renin level tend to be low.\textsuperscript{65} Study by Egan and colleague showed that insufficient sleep may increase the risk of hypertension and cardiovascular disease through effects on sympathetic nervous system activation by alteration of the hypothalamus -pituitary- adrenal axis leading to secretion of cortisol and renin-angiotensin system and augmented systemic levels of inflammation such as elevation of C-reactive protein levels, tumour necrosis factor and interleukin as shown below.\textsuperscript{3,26,66}

2.3.1 Pathophysiology of sleep alteration and hypertension

The pathophysiological mechanisms underlying the association between short sleep duration, sleep alteration and hypertension as shown in appendix c is best understood in the context of psychoneuroimmunologic theory, which is a bidirectional communication between the brain and the immune system. It is related to inappropriate physiological arousal due to alteration in the stress system functions.\textsuperscript{66} Good sleep has important homeostatic functions, including
suppressive effects on the stress system, while poor sleep is related to the activation of the sympathetic nervous system and proinflammatory pathways and increase in oxidative stress.\textsuperscript{14, 66} This would impair the adaptation to stress through allostasis and contribute to allostatic load, thus compromising stress-resiliency and increasing blood pressure.\textsuperscript{66, 69} The mechanisms underlying the association of short duration of sleep with CVD may include sleep related disturbances in endocrine and metabolic functions.\textsuperscript{67, 68}

Prolonged poor sleep quality and other sleep disorders act as chronic stressors, activating the sympathetic nervous system and systemic inflammation, the resultant response to the stress involves bidirectional signaling between the brain and autonomic, cardiovascular and immune systems via the neural and endocrine mechanism, the so-called allodynamic processes which can be adaptive in short term but become maladaptive in the long term consequently leading to long term deregulation of allostasis and promote maladaptive stress mechanism.\textsuperscript{66}

The bidirectional communication between the brain and the immune system is carried out through a complex network of cytokines, autonomic and endocrine systems through progression of the atherosclerotic process and induction of endothelial dysfunction to initiate plague activation.\textsuperscript{66}

Sleep lost has been experimentally found to induce sustained increased in blood pressure in either normotensive, prehypertensive or hypertensive subjects in both elderly, young adults, male and female subjects.\textsuperscript{66}

Yue and colleague reported that insomnia is associated with an overall hypersecretion of ACTH and cortisol, suggesting an activation of the hypothalamic-pituitary-adrenal (HPA) axis in these patients.\textsuperscript{29} Given the well-established association of hypercortisolemia with significant medical morbidity (eg, hypertension, metabolic syndrome, osteoporosis), insomnia can induce hypertension through an activation of the hypothalamic-pituitary-
adrenal (HPA) axis. These could lead to elevated sympathetic nervous system activity, waking physical and psychosocial stressors, increased salt retention and changed exposure to raised 24-hour blood pressure and heart rate.

2.4 Cardiovascular and hypertension risk factors

Arterial hypertension is an important risk factor for occurrence of cardiovascular disease (CVD). The impact of hypertension in cardiovascular morbidity and mortality is higher than any cardiovascular risk factor including traditional factors such as obesity, dyslipidaemia, age, family history, lifestyle, cigarette smoking and nontraditional risk factors such as increased inflammation and hypercoagulable state. Therefore, blood pressure (BP) control is an important therapeutic goal for the slowing of progression as well as for the prevention of Cardiovascular disease. Maraj and colleagues in the United States, showed that among individuals aged 40-90 years, each 20/10mmHg rise in blood pressure doubles the risk of fatal coronary events.

Overweight and obesity are established risk factors for hypertension and other non-communicable diseases such as diabetes mellitus. This is because weight gain and dietary factors appear to play a major role since hypertension is rare in non-obese hunter-gatherers living in non-industrialized societies. Study in Portharcourt on prevalence of hypertension and its modifiable risk factors by Ordinioha showed that 60% were overweight, while 22.67% were obese and only 17.33% of the lecturers were of normal weight. The high prevalence of overweight and obesity was due to high socioeconomic status of the subjects and the fact that they ate on daily basis, an average of one meal daily that was not prepared at home. This showed that there is a relationship between hypertension and weight gain. In Jos, North-Central Nigeria, the prevalence of overweight and obesity was 21.4% (19.4% in males and 21.4% in females), this is because most of the subjects had post secondary education and were more likely to have adopted western lifestyle and diet.
Study by Ugwuja et al in South-West Nigeria showed that there seems to be increase in hypertension prevalence with advancement in educational status such that participants with tertiary education had significantly higher prevalence of the disease in comparison with those with primary education; 22.7% versus 14.7%, (P = 0.03). This may be due to increasing sedentary lifestyles due to increase in socio-economic status.56

High BP is also more prevalent in individual with diabetes mellitus than in those without it. In a follow up study of 2,629 America Indians for 12 years who were free from hypertension at baseline examination, the prevalence of hypertension was significantly higher in those who subsequently developed diabetes mellitus than in those who did not.73

Sleep is increasingly recognized as important to public health, with sleep insufficiency linked to motor vehicle crashes, industrial disasters, and medical and other occupational errors.23 Short sleep duration is associated with weight gain, obesity, type 2 diabetes, elevated blood pressure, cardiovascular disease, and premature mortality, this is by influencing the level of leptin, ghrelin, appetite and insulin sensitivity.15,20 In a study linking sleep and hypertension, Pandey et al in America, showed that both white and black short sleepers had a greater likelihood of reporting hypertension than those who reported sleeping 6 to 8 hours. Unadjusted logistic regression analysis exploring the race/ethnicity interactions between insufficient sleep and hypertension indicated that black short sleepers (<6 hours) and long sleepers (>8 hours) were more likely to report hypertension than their white counterparts (OR = 1.34 and 1.37, resp.; P < 0.01).74 This may be due to differences in social and environmental elements in the epidemiological study of hypertension.74 Significant interactions of insufficient sleep with race/ethnicity were also observed even after adjusting to effects of age, sex, income, education, body mass index, alcohol use, smoking, emotional distress, diabetes, coronary heart disease and stroke.74 Similarly, results from the National interview survey in United State on sleep duration and cardiovascular disease showed a
positive association between both short and longer sleep durations and CVD in representative sample of the population.\textsuperscript{75} The survey in addition found that alteration in sleep duration is independently associated with CVD.\textsuperscript{75} This was independent of age, sex, race-ethnicity, smoking, alcohol intake, body mass index, physical activities, diabetes mellitus, hypertension, and depression. Comparing with sleep duration of 7 hours, the multivariate odds ratio (95% confidence interval) of CVD was 2.20 (1.78, 2.71), 1.33 (1.13, 1.57), 1.23 (1.06, 1.41), and 1.57 (1.31, 1.89) for sleep duration ≤ 5 h, 6 h, 8 h, and ≥ 9 h respectively.\textsuperscript{75} The non-traditional cardiovascular risk factors, such as markers of inflammation have been linked to development of hypertension, NHAHES III found that individuals with elevated BP had a higher prevalence of C-reactive protein than normotensive subjects even after adjusting for age, gender, race, smoking, BMI, exercise, diabetes, and medication usage.\textsuperscript{76} In addition to C-reactive protein, Zhang and colleagues also reported an increase tumour necrosis factor, amyloid and homocystein as well as higher white blood cell counts.\textsuperscript{77} These studies demonstrated that hypertension is often associated with multiple cardiovascular risk factors.

2.5 Subclinical disease and cardiovascular markers

Hypertension is associated with subclinical and cardiovascular disease and a study by Gerber et al showed that before and after controlling for sex, race/ethnicity, age, body mass index at baseline, and change in body mass index, urinary albumin-to-creatinine ratio was found to be a significant independent predictor of change in awake and sleep systolic and diastolic BPs (all P < .05). It also independently predicted hypertension status at follow-up.\textsuperscript{78} Albuminuria is a well-known predictor of poor renal outcomes in patients with type 2 diabetes and in essential hypertension.\textsuperscript{79} It has also been shown more recently to be a predictor of cardiovascular outcomes in diabetic and hypertensive population. There is also emerging data that reduction of albuminuria leads to reduced risk of adverse renal and cardiovascular
Data from the Framingham study showed that in 1,568 non-hypertensive participants without diabetes, urinary albumin excretion less than the microalbuminuria threshold predicted the development of cardiovascular disease.\textsuperscript{81} Therefore, the presence of microalbuminuria may need to be viewed in the same light as other risk factors such as blood pressure, cholesterol, and blood glucose.\textsuperscript{79} In the Rotterdam Study, a prospective population-based study with 1,900 participants (≥ 55 years of age including 739 with normal blood pressure and 1,161 with elevated blood pressure), showed that individuals with hypertension had significantly smaller arteriolar and venular diameter and arteriolar-venular ratios than normotensive individuals, indicating presence of microvascular damage.\textsuperscript{82} While Cecelja reported that arterial stiffness increases with age by approximately 0.1 metre per second per year in East Asian populations with low prevalences of atherosclerosis.\textsuperscript{83} While some authors report a linear relationship between arterial stiffness and age,\textsuperscript{83} others have found accelerated stiffening between 50 and 60 years of age.\textsuperscript{84} Classic cardiovascular risk factors, which include hyperlipidaemia, diabetes mellitus, elevated body mass index and smoking, have been implicated in accelerated arterial stiffening.\textsuperscript{83} However, findings with respect to risk factors other than age and blood pressure have been inconsistent and negative findings not highlighted in many studies.\textsuperscript{83} In the presence of atherosclerosis, stiffness of large arteries is increased and it has been suggested that arterial stiffness may be a useful marker of the extent of atherosclerosis in the aorta.\textsuperscript{83}

\subsection*{2.6 Cardiovascular morbidity and mortality}

For some patient groups, detection of hypertension was an issue, whereas for other groups it was the management that was described as the challenge. Hypertension has been associated with variety of cardiovascular diseases and with cardiovascular-associated all cause mortality.\textsuperscript{85} Study by Cooper and colleagues on the other hand showed that decreasing
systolic BP to lower than 130mmHg in patients with diabetes and CAD was not associated with further reduction in morbidity beyond that associated with systolic BP lower than 140mmHg.\textsuperscript{85} Hublin et al in Finland observed increased risk of mortality both for short sleep in men (26\%) and in women (21\%), and for long sleep (24\% and 17\%) among twins population.\textsuperscript{86} However, reduction in systolic and diastolic blood pressure resulted in significant reduction in morbidity and mortality associated with hypertension.\textsuperscript{85} In the Monitoring Trends and Determinants on Cardiovascular Diseases/Cooperative Research in the Region of Augsburg (MONICA/KORA ) study on variation on changes in the body composition and changes in blood pressure levels, after 10 years of follow-up, hypertension developed in 50.1\% of individuals with pre-hypertension and only 6.76\% went from hypertensive to pre-hypertensive blood pressure levels. An increase in body weight and fat mass in the study was a risk factor for the development of sustained hypertension, whereas a decrease was predictive of a decrease in blood pressure.\textsuperscript{87} The Pre-hypertension group was characterized by a marked increase in body weight (5.71\% [95\% confidence interval (CI): 4.60\% to 6.83\%]) that was largely the result of an increase in fat mass (17.8\% [95\% CI: 14.5\% to 21.0\%]). Insufficient sleep is a direct contributor to injury and death from motor vehicle and workplace accidents.\textsuperscript{88} These sleep-related problems incur financial costs relating to health and other expenditures and non-financial costs relating to loss of quality of life. Economic estimates demonstrate that sleep disorders are associated with large financial and non-financial costs. Given that the greatest financial costs appear to be non-medical costs related to loss of productivity and accident risk, it is likely that inclusion of the effects of sleep restriction from poor sleep habits or choice could add considerably to this already substantial amounts.\textsuperscript{88}
The pre-hypertensive and normotensive group showed no significant change in body weight (−1.55% [95% CI: −3.70% to 0.61%]) and fat mass (0.20% [95% CI: −6.13% to 6.52%]) In the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) study, in which the risks of white-coat or masked hypertension for the development of sustained hypertension were evaluated; 60% of patients, developed hypertension from normotension and just 8% of the subjects reverted from true hypertension to normotension.

2.7 Sleep quality

Sleep is an important component of a healthy lifestyle. Sleep quality is an important factor in the physiologic recovery of the body during sleep, and good sleep quality may prevent CVD. There is growing evidence mostly from populations in Western countries pointing to downward trends in the average duration of sleep, and increasingly higher prevalence of insomnia and other sleep disturbances. On the prevalence of poor sleep quality Shittu and colleague showed the prevalence to be 44.0% and overall sleep quality was very good, fairly good, fairly bad and very bad in 30.8%, 33.2%, 19.5%, and 16.5% respectively. Sleep duration of 7-8 hours is generally accepted as total sleep requirement for adults. Habitual sleep duration was coded as either short sleep duration (<6 hours per night) or long sleep (>8 hours per night) with a reference of 6–8 hours per night representing normal sleep duration. Stranges et al observed that a large number of adults greater than 50 years in low-income settings are currently experiencing sleep problems. They observed a higher prevalence of sleep problems in women and older age groups than men. This was as a result of greater burden of psychological morbidity (anxiety, depression) in women, gender-specific role such as mothering and related stresses and disparities in socioeconomic status. While a study on the effect of menopause on sleep quality by Zahra and colleagues reported hormonal changes as the reason for poor sleep quality and increased sleep problems in women. Survey by
Cohen et al showed that duration of nighttime sleep of more than 9 hours was significantly related to increased mortality in comparison with sleeping 7-9 hours (hazard ratio [HR] = 1.31, P < 0.01) after adjusting for demographic, health, and function variables, whereas for short nighttime sleep of fewer than 7 hours mortality did not differ from that of 7-9 hours of sleep. For those who sleep more than 9 hours per night, there is a significant increase in mortality risk (HR = 1.385, P < 0.05) as a result of fatigue, depression and decrease in physical activity, while in the older population it may represent end of life process such as frailty, boredom, inactivity or dementia. Cohen and colleagues also observed that short sleep may have protective or no effect on mortality (for those who nap). On the contrary, both short and long duration of sleep have been associated with increased mortality. Pooled analyses conducted in a recent review of 16 studies of persons whose mean age ranged from 41 to 77 years indicated that sleeping fewer than 7 hours per night increased mortality risk by 12%, and sleeping more than 8-9 hr per night increased mortality risk by 30% compared with those sleeping 7-8 hr per night. Epidemiological studies on sleep duration showed that short sleep duration of less than 7 hours was associated with higher incidence of overweight, obesity, and hypertension, total cholesterol, hemoglobin A (1c), and triglycerides. Many previous studies showed that there was no association of sleep quality with arterial blood pressure in the elderly aged 60 years or older. In a study of elderly population over 90 years in China, it showed that the association between sleep quality and arterial blood pressure changed with age; that in a population aged less than 60 years it was significant but that in a population aged more than 60 years (including general elderly aged 60 years or older and very elderly aged 90 years or older) it was non-significant. Cohort studies reported conflicting results for the association of short sleep duration with the occurrence of cardiovascular diseases (CVD). The relation between short sleep duration and CVD incidence
could be due to an effect of short sleep on intermediate biological CVD risk factors such as BMI, blood lipids, high blood pressure, and prevalence of diabetes.\textsuperscript{24, 94, 95} Subjects with chronic insomnia have a higher risk for hypertension and U-shaped associations have also been demonstrated between sleep quality and hypertension.\textsuperscript{23, 96, 97} This means that within normal sleep duration and quality, blood pressure is normal or reduced but is elevated when sleep quality is poor. On the other hand, it has also been confirmed that subjects with hypertension are more likely to report emotional disorders such as anxiety, depression, and sleep disorders, which are usually viewed as one of their clinical features. This indicate that hypertension is a risk factor for sleep disorders.\textsuperscript{98} Shankar and colleague also showed both short and long sleep duration to be independently associated with poor sleep rated health, and is independent of age, sex, race-ethnicity, smoking, alcohol intake, body mass index, physical activity, depression, diabetes mellitus, hypertension, and CVD. When they compared with sleep duration of 7 h, there was a positive association between both shorter and longer sleep duration and poor self-rated health in a representative sample of US adults.\textsuperscript{99}

A number of factors affect sleep quality and sleep health, these include obesity, poor sleep hygiene, depression, anxiety, certain medical conditions, and the use of certain medications and substances such as alcohol and nicotine, and just to name a few.\textsuperscript{100} Sleep related health problems are an important public health concern. Impaired sleep quality, duration and sleep disorders are associated with diabetes, depression, social discord, and even inflammation.\textsuperscript{101} On the other hand, sleep loss from these problems is associated with disturbances in cognitive and psychomotor function including mood, thinking, concentration, memory, learning, vigilance and reaction times. These disturbances have adverse effects on wellbeing, productivity and safety.\textsuperscript{88} Gu et al reported that oldest-old adults aged 80 or older tended to sleep either shorter or longer as compared to young elders.\textsuperscript{101} This is because when the old
adults get older, their health condition gets worse, on average. The quality of sleep of the oldest-old did not differ much from that of young elders; and the former even had better sleep quality than the latter when demographics, socioeconomic status (SES), family and or social support and connection, health practice, and health conditions were controlled for. They also showed that resource-rich elders slept longer than resource-poor elders, and the former had better sleep quality than the latter. More economic and personal resources allow those individuals with higher SES to respond more favorably to adverse events and avoid risky behaviors. Men’s self-rated quality of sleep was consistently better than that of women. Sleep hygiene principles have been shown to improve sleep quality.

2.7.1 Sleep and cardiovascular risk

The Sleep Heart Health study, which is a community-based prospective study showed that both short and long habitual sleep durations are associated with prevalent hypertension in community-dwelling middle-aged and older adults. The association of sleep duration with hypertension persisted after adjustment for factors believed a priori to be potential confounders of the association between sleep duration and hypertension, including age, sex, race and apnoea-hypopnoea index (AHI). It also showed that the association of sleep duration with hypertension was not confounded by caffeine or alcohol consumption or cigarette smoking, which might influence sleep habits. The study suggested that levels of habitual sleep restriction that are common in the adult population may contribute to the high population prevalence of hypertension. Shabanayagam showed that in US adults, there was a positive association between both shorter and longer sleep durations and CVD. These results suggest that sleep duration may be an important marker of CVD. Shittu et al in Nigeria showed that obese class 3 were poor sleepers which were similar to the findings of Alebiosu et al and Pearson et al where they respectively showed that patients who
are poor sleepers have elevated BMI and that obese patients had difficulty with sleep.\textsuperscript{19, 102, 103} These will affect the cardiovascular system.

Shittu and colleagues also showed that there was strong statistical association between poor sleep quality, depression (p-value 0.000), and body mass index (p-value 0.05).\textsuperscript{19} While Alebiosu et al showed that 42.4\% of hypertensive subjects were poor sleepers (global PSQI > 5), with a global mean PSQI of 5.03 ± 3.28. This was significantly more than 17.3\% of control subjects with a mean global PSQI of 3.10 ± 0.83 in the study.\textsuperscript{102}

The Monitoring Project on Risk Factors and Chronic Diseases in the Netherlands (MORGEN) study revealed that during 10-15 years of follow-up, 1,486 CVD and 1,148 coronary heart disease (CHD) events occurred. Short sleepers (≤ 6 h) had a 15\% higher risk of total CVD (HR: 1.15; 95\%CI: 1.00-1.32) and a 23\% higher risk of CHD (HR: 1.23 [1.04-1.45]) when compared to normal sleepers (7 h) after adjustment for all confounders. Additional adjustment for intermediate biological risk factors attenuated these relative risks to 1.11 (0.97-1.27) for total CVD and to 1.19 (1.00-1.40) for CHD. Short sleepers with poor sleep quality had a 63\% higher risk of CVD (HR: 1.63 [1.21-2.19]) and a 79\% higher risk of CHD incidence (HR: 1.79 [1.24-2.58]) compared to normal sleepers with good sleep quality, after adjustments for all confounders. The study observed no associations between long sleep duration (≥ 9 h) and CVD or CHD incidence.\textsuperscript{95}

In a recent meta-analysis incorporating 15 studies by Cappuccio et al they revealed that short sleep compared to normal sleep duration was not related to total CVD (RR: 1.03 [95\% CI 0.93-1.15]), though it was related to coronary heart disease (CHD) (RR:1.48 [1.22-1.80]) and stroke (RR 1.15 [1.00-1.31]). The residual confounding and lack of specificity of the outcome measures may explain the above findings.\textsuperscript{104} For long sleep compared to normal sleep duration, they reported an RR of 1.41 (1.19-1.68) for total CVD, 1.38 (1.15-1.66) for CHD, and 1.65 (1.45-1.87) for stroke.\textsuperscript{104}
Shittu and colleague in their study, demonstrated a strong positive statistical association between increasing body mass index and PSQI Score for sleep quality.\textsuperscript{19} This was similar to the finding of Pearson et al where they found that obese patient had difficulty with sleep.\textsuperscript{103} This result was consistent with the finding of Anne et al.\textsuperscript{105} Abdulsalam on the other hand found no significant correlation between age, weight, height and BMI.\textsuperscript{26} The Quebec family study showed that changes in adiposity indices when compared between short- (5-6 hours), average- (7-8 hours), and long- (9-10 hours) duration sleeper groups and after adjustment for age, sex, and baseline body mass index, short-duration sleepers gained 1.98 kg (95% confidence interval: 1.16-2.82) more and long-duration sleepers gained 1.58 kg (95% CI: 1.02-2.56) more than did average-duration sleepers over 6 years. Short and long duration sleepers were 35% and 25% more likely to experience a 5kg weight gain respectively, as compared with average-duration sleepers over 6 years. The risk of developing obesity was elevated for short- and long-duration sleepers as compared with average-duration sleepers, with 27% and 21% increases in risk, respectively.\textsuperscript{107} Short sleep duration was associated with weight gain and the development of obesity over one year in men, but not in women.\textsuperscript{102} The odds ratios for the development of obesity were 1.91 (95%CI 1.36, 2.67) and 1.50 (95%CI 1.24, 1.80) in men who slept < 5 and 5-6 h, respectively. There was no significant association between sleep duration and weight gain or obesity found for women.\textsuperscript{108} This may be due to lower prevalence of obesity in these women.\textsuperscript{108} However, Gangwisch et al and Stranges et al reported a negative and no association respectively in their studies on the relationship between sleep duration and obesity or weight gain.\textsuperscript{19, 92} Several studies have reported a significant inverse relation-ship between self-reported sleep duration and weight.\textsuperscript{109, 110} Short sleep duration could have an impact on obesity by influencing lifestyle and habits. Lack of sleep may cause daytime sleepiness and fatigue, which can lead to restriction of physical activity and, in turn, start a vicious circle of short
sleep duration and physical inactivity.\textsuperscript{111,112} Theorell et al reported an inverse relationship between short sleep duration and central obesity in women after adjusting for confounders. These associations were stronger in the younger (< 50 years) than in the older (≥ 50 years) women.\textsuperscript{111} Hairston on the other hand showed that eight hours or more of sleep was also significantly related to a greater accumulation of BMI (0.8 kg/m\textsuperscript{2}, P < 0.001), subcutaneous adipose tissue (20 cm\textsuperscript{2}, P < 0.01) and derived visceral adipose tissue (6 cm\textsuperscript{2}, P < 0.05) compared to sleep duration between 6 and 7 hours.\textsuperscript{113}

This association between both short and long durations of sleep and CVD was consistently present in both men and women. This is consistent with previous reports from other countries examining the association between sleep duration and CVD mortality.\textsuperscript{25,114}

2.7.2 Sleep quality and hypertension

Habitually, sleep problems could lead to the development and maintenance of hypertension. Ji-Rong and Julia reported that the extended exposure to these forces could make the cardiovascular system to operate at elevated pressure equilibrium through structural adaptations such as arterial and left ventricle hypertrophic remodeling.\textsuperscript{29, 97} This could also contribute to hypertension by disrupting circadian rhythmicity and autonomic balance.\textsuperscript{29}

Several studies showed that sleep quality correlated with risk for hypertension. Kai et al showed that good and poor sleep quality were associated with hypertension in males, with odds ratios of 1.20 (95\% CI, 1.01–1.42) and 1.67 (95\% CI, 1.32–2.11) respectively.\textsuperscript{115} This was similar to other studies where they reported that a strongly significant association of sleep quality with arterial blood pressure or with the risk of hypertension among adolescents and adults (aged 60 years or younger).\textsuperscript{16,116} Poor sleep quality, defined by a Pittsburgh Sleep Quality Index score as a score of five or less, was more frequent among those with hypertension.\textsuperscript{117} Study by Knutson found that lower sleep quality, as indicated by shorter or longer sleep duration and poor sleep maintenance, was associated with higher systolic and
diastolic blood pressure. These were similar to the findings in Nigerian hypertensive patients; Alebiosu et al found that 42.4% of hypertensive patients have poor sleep. This study showed that about two-thirds of patients with elevated systolic blood pressure were poor sleepers. In Nigeria, Shittu et al showed that there was a strong statistical association between poor sleep quality, blood pressure (p-value 0.002), depression (p-value 0.000) and body mass index (p-value 0.05).

On the other hand, Ji-Rong et al showed that there was no association between sleep quality and arterial blood pressure in nonagenarian and centenarians. This is because the association between sleep quality and arterial blood pressure changed with age. In addition the study was cross-sectional study in which the patients could change their sleep habits. Similarly, other studies showed that there was no association between sleep quality and arterial blood pressure in the elderly aged 60 years or older. This shows that the association between sleep quality and arterial blood pressure changed with age. This showed that in a population aged less than 60 years the relationship between sleep quality and hypertension was significant, but that in a population aged more than 60 years (including general elderly aged 60 years or older and very elderly aged 90 years or older) it was non-significant. This is because aging is associated with changes in the length and quality of sleep as well as an increase in sleep complaints, and sleep problems increase with age.

This study also showed that no category of sleep quality was found to be related to hypertension prevalence among those with normal sleep duration. This therefore demonstrated that the association of sleep duration and sleep quality with hypertension was an additive relationship. Alebiosu et al showed that 27.8% of hypertensive subjects that had good sleep had abnormal systolic blood pressure compared with 66.7% of hypertensive subjects with poor sleep.
2.7.3 Sleep duration and hypertension

Two major community-based cohort studies, the Sleep Heart Health Study (SHHS) and the National Health and Nutrition Examination Survey (NHNES) have reported that there is a relationship between self-reported short sleep duration and prevalence and incidence of hypertension.\textsuperscript{16,23} Gottlieb et al compared subjects sleeping 7 to less than 8 hours per night, they showed that those sleeping less than 6 and between 6 and 7 hours per night had adjusted odds ratios for hypertension of 1.66 (95% confidence interval 1.35-2.04) and 1.19 (1.02-1.39), respectively, whereas those sleeping between 8 and 9 and 9 or more hours per night had adjusted odds ratios for hypertension of 1.19 (1.04-1.37) and 1.30 (1.04-1.62), respectively (p < .0001 for association of sleep duration with hypertension).\textsuperscript{23} Gottlieb et al.\textsuperscript{23} have demonstrated from SHHS that short and long habitual sleep durations are associated with prevalent hypertension in community-dwelling middle-aged and older Adults, when compared with subjects sleeping between 7 and 8 hours per night.\textsuperscript{23} This is because those who reported sleeping 9 or more hours per night reported 15% less physical activity per week than those sleeping 7 to 8 hours per night, and inactivity may place these long sleepers at increased risk of hypertension.\textsuperscript{23} after adjustment for possible confounders such as age, sex, race, obesity, apnea, hypopnea index, or lifestyle habits.\textsuperscript{23} Short sleep duration was associated with higher prevalence of hypertension in the Korean National Health and Nutrition survey 2001.\textsuperscript{119} The biologic mechanisms underlying an association of short sleep duration and hypertension are uncertain. Pandey and colleagues reported that long sleep may actually be more detrimental to health than short sleep; this could be due to sleep fragmentation and photoperiodic abnormalities, which could all lead to increased blood pressure levels.\textsuperscript{74} This study also found higher prevalence of systemic hypertension among those with sleep fragmentation as a result of frequently waking up which may result in shorter sleep duration and due to frequent arousal and transient increase in BP and
sympathetic activity due to these arousals. Subjects participating in NHNES who had self-reported less than 5 hours of sleep by night demonstrated a higher incidence of hypertension after 8 to 10 years followup. Other studies showed that participants who slept < 5 or < 7 hours per night had a greater frequency of hypertension than individuals sleeping 7 to 8 hours per night. These studies also showed that participants who slept >8 h or >9 hours were more likely to have elevated blood pressure than those sleeping 7 to 8 hours per night. These studies underscore the potential consequences of obtaining too little or too much sleep. The relationship between sleep duration and hypertension is also age and gender dependent. Adolescents with shorter sleep duration assessed by actigraphy demonstrated higher prevalence of pre-hypertension. Conversely, an association between sleep restriction and incident hypertension was not found in subjects between 60 and 86 years of age in the NHNES study. Hypertension was not associated with sleep duration assessed by either self-report or actigraphy in a cross-sectional study of 5058 participants, aged 58 to 98 years of age in the Rotterdam Study. William et al also reported that both short and long sleep duration in early pregnancy were associated with increased mean third trimester SBP and DBP. They showed that the mean third trimester SBP was 3.72, and 2.43 mm Hg higher for women reporting ≤ 6 hours and 7-8 hours sleep, respectively. When compared with women reporting 9 hours of sleep, the mean third trimester SBP was 4.21 mm Hg higher for women reporting long sleep (≥ 10 hours) vs. the control group. Short and long sleep durations were associated with increased risks of pregnancy induced hypertension (PIH) and preeclampsia (PE). The ORs for very short (< 5 hours) and long (≥ 10 hours) sleepers were 9.52 (95% CI 1.83 to 49.40) and 2.45 (95% CI 0.74 to 8.15) for PE . As mentioned earlier, several studies have implicated insufficient sleep as a risk factor for elevated blood pressure and hypertension in men and non-pregnant women. Shabayagam et al found that both short and long durations of sleep (≤ 5 and ≥ 9 hours) were
positively associated with CVD. This association was consistently present in analysis stratified by age, sex, race-ethnicity, and BMI, as well as in the subgroup of apparently healthy study subjects, defined as those without diabetes, hypertension, or depression. Knutson et al showed that several socio-demographic factors were associated with short sleep. They also reported that women of 65 years of age and older, Asians, Hispanics, and married people are likely to be short sleepers. The odds of short sleep were higher among full-time workers, those with some college education, African Americans, and among single or divorced/separated individuals compared to married individuals. Income level was not associated with short sleep. Finally, considering short sleep duration, hypertension was both more prevalent and more incident in women only, in the Whitehall II Study.

2.7.4 Effects of sleep on blood pressure control

BP decreases during sleep, and reduced dipping of BP during sleep increases cardiovascular risk. Calhoun et al reported that during normal sleep, there is a decrease in BP relative to wakefulness and partly is attributable to decrease in sympathetic output. While Ogawa et al showed that diastolic blood pressure was significantly higher after total sleep deprivation than after control sleep (66.5 ±1.7 vs 57.4 ± 3.3 mm Hg) and this was due to increase in sympathetic activity. This showed that maintaining and improving sleep will reduce blood pressure and improve blood pressure control in hypertensive patients. The Sleep Heart study showed that a night of sleep restriction to a mean of 3.6 hours due to working overtime was associated with a 6mmHg increase in mean SBP and a 3-mm Hg increase in mean DBP, compared with a night of 8 hours sleep. Lusardi and colleagues found that a single night of experimental sleep restriction of 4 hours of sleep in the home setting resulted in a 4mmHg and 7mmHg increase in mean morning SBP in normotensive and hypertensive subjects respectively. Smaller increases in mean morning DBP was also observed. The same group reported increases of 22mmHg in mean SBP and 17mmHg in mean DBP across 10 days of
partial sleep deprivation to 4 hours per night in four experimental subjects, but this was not significantly different from the 10 mmHg increase in mean SBP and 13 mm Hg increase in mean DBP observed in the 5 control subjects. On the other hand, study by Haack and colleagues demonstrated that increasing average nightly sleep duration by approximately 30 min over a 6-week period led to a significant reduction of beat-to-beat SBP and DBP in hypertensive and pre-hypertensive subjects, while the control group who maintained their habitual sleep duration did not show a significant reduction in BP. The findings suggested that behavioral interventions designed to increase sleep duration may serve as an effective strategy in the treatment of high BP. The study also showed that when compared with standard behavioral intervention strategies, such as exercise and dietary interventions, the BP reduction in response to increased sleep duration appears to be of comparable magnitude. Systolic and diastolic beat-to-beat blood pressure averaged across the 24-hour recording period significantly decreased from pre- to post-intervention visit in the sleep extension group that had improved by 14 ± 3 and 8 ± 3 mmHg, respectively (P < 0.05). Though the reduction of 7 ± 5 and 3 ± 4 mmHg in the sleep maintenance group was not significant, it did not differ from the blood pressure reduction in the sleep extension group (P = 0.15 for interaction effect). Improving sleep by increasing sleep extension in the study reduced systolic blood pressure by 14 mmHg and the arm without sleep extension had a reduced SBP by 7 mmHg. This reduction of BP in the sleep maintenance group may reflect habituation to the Clinical Research Centre environment, but may also reflect the fact that the sleep maintenance group was an “active” Control (i.e. subjects in this group, as well as in the sleep extension group, were asked to follow a set of sleep hygiene instructions, such as maintaining regularity of bedtimes). While total sleep deprivation over 3 days in healthy volunteers showed an average increase of 7 and 4 mmHg for SBP and DBP, respectively. Haack et al showed that BP-
elevating effect of short sleep duration is reversible by increasing habitual sleep duration. This BP-lowering effect of sleep extension was evident after an intervention period of 6 weeks.\textsuperscript{45} The time course of this effect is unknown; that is whether the change in BP was a function of successive days of sleep extension, because the study was designed with only a single visit.\textsuperscript{45} On the other hand, Kubo et al failed to find a BP-lowering effect of sleep extension of 2 hours per weekend day over a period of one week in workers with habitual sleep durations of less than 6 hours.\textsuperscript{128} Comprehensive lifestyle interventions over a 9-week period, including dietary and exercise interventions, reduced 24-hour ambulatory SBP and DBP by 10 and 5mmHg, respectively.\textsuperscript{129} Compared with these standard behavioral interventions, it appears that the sleep extension approach to improve sleep may serve as an effective strategy in the treatment of high BP, either alone or in combination with standard lifestyle modification.\textsuperscript{45}

Vgontzas and colleague showed that chronic insomniacs (individual with sleep difficulty) who slept five or less hours or 5-6 hours, respectively, for more than one year had a high risk of hypertension than subjects who slept > 6 hours and had no sleep complaint.\textsuperscript{124} In contrast, insomniacs who slept ≥ 6 hours did not show an increased risk for hypertension compared to the control group. Subjects with the milder form of insomnia (difficulty falling or staying asleep) had a significantly higher risk for hypertension when they slept ≤ 5 hours and a non-significantly increased risk when they slept 5-6 hours compared to the control group.\textsuperscript{124} Gangwisch et al found that progressively, older age groups had increasingly lower percentages of subjects who reported 7-hour sleep durations and increasingly higher percentages who reported long and short sleep durations. The most likely explanations for this finding are a cohort effect and changes in sleep durations over time, whereby, as individuals age, they become less likely to sleep 7 hours and more likely to sleep 5 hours or less or 9 hours or more.\textsuperscript{130}
2.8 Treatment of hypertension

Many guidelines exist for the management of hypertension. Two of the most widely used recommendations are those from the American Diabetes Association (ADA) and the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7). The Eighth Report of the JNC (JNC 8) was released in December 2013.\(^5\) The risk associated with hypertension can be reduced by controlling the BP, therefore, the goal of managing hypertension is to maintain BP at normal level. Management of most hypertensive patients consists of non-pharmacological interventions and in addition the use of BP lowering medications.

2.8.1 Non-Pharmacology treatment of hypertension

2.8.1.1 Lifestyles measures

Both JNC-7 and JNC- 8 recommend lifestyle approach as first line for the management of hypertension. Major lifestyle modifications shown to lower BP include weight reduction in those individuals who are overweight or obese, adoption of the Dietary Approaches to Stop Hypertension (DASH) eating plan, which is diet rich in potassium and calcium, dietary sodium reduction. Physical activity, and moderation of alcohol consumption.\(^2\) The table appendix D showed the various lifestyle modifications and the blood pressure reduction.\(^2\)

2.8.1.2 Sleep education

Sleep education programs are still in their infancy and vary widely in their scope, delivery and outcomes.\(^{131}\) There are two specific types of sleep education programs that help to improve sleep health, those that sought to change sleep behavior and those that sought simply to disseminate information.\(^{131}\) Improving sleep quality has been suggested as a therapeutic target for the modification and prevention of cardiovascular risk factors, such as hypertension.\(^{24}\)
The provision of high-quality education and training that is responsive, relevant, accessible and evidence based is critical if the vision for quality sleep education programs is to be fulfilled.\textsuperscript{131} The role of the structured sleep education is to ensure that there was improvement in both sleep knowledge, quality and duration.\textsuperscript{24,132} Improving sleep duration to between 7-8 hour per night has been shown to improve blood pressure\textsuperscript{13,23,24} Similarly, extension of sleep duration has been found to lower blood pressure in those with short sleep duration.\textsuperscript{45} This is a behavioural approach to control blood pressure. While in a study by Haas on improving blood pressure through enhanced sleep showed that hypertensive patients taking estazolam to improved their sleep showed that they had a reduction of 10.5mmHg in systolic blood pressure and 8.1mmHg in diastolic blood pressure and also improvement in sleep quality.\textsuperscript{133} The control group did not achieve significant reduction in either the systolic or diastolic blood pressure during the intervention.\textsuperscript{133} Promotion of sleep hygiene through structured sleep education will positively influence sleep behaviour.\textsuperscript{132}

2.8.2 Pharmacologic treatment

Pharmacological treatment is inevitable in high-risk populations such as those with CHD. The recommended target blood pressure for individuals with CHD or CHD equivalents is <130/80mmHg. JNC-8 recommended that in the general population aged 60 years or older, pharmacologic treatment to lower BP should start at systolic blood pressure (SBP) of 150mmHg or higher or diastolic blood pressure (DBP) of 90mmHg or higher and treat to a goal SBP lower than 150mmHg and goal DBP lower than 90mmHg.\textsuperscript{5} This is because there is moderate- to high-quality evidence from RCTs that in the general population aged 60 years or older, treating high BP to a goal of lower than 150/90 mm Hg reduces stroke, heart failure, and coronary heart disease (CHD).\textsuperscript{5} The trials on which these evidence statements and these recommendations were based include Hypertension in the Very Elderly Trial (HYVET),
Systolic Hypertension in Europe (Syst-Eur) trial, The Systolic Hypertension in the Elderly Program (SHEP), Japanese Trial to Assess Optimal Systolic blood Pressure in Elderly hypertensive patients (JATOS), Valsartan in elderly Isolated Systolic Hypertension (VALISH) and Italian Study on the Cardiovascular Effects of Systolic Blood Pressure Control (CARDIO-SIS).134-136 Also in the general population younger than 60 years, JNC-8 recommended initiation of pharmacologic treatment to lower BP at DBP of 90 mm Hg or higher and treat to a goal DBP of lower than 90mmHg.5 This was based on high-quality evidence from 5 DBP trials: Hypertension Detection and Follow-up Program (HDFP), Hypertension-Stroke Cooperative, Medical Research Council (MRC), Australian National Blood Pressure Study (ANBP) and Veterans Administration (VA) Cooperative that demonstrate improvements in health outcomes among adults aged 30 through 69 years with elevated BP.5 There is moderate evidence to support initiating drug treatment with an angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, calcium channel blocker, or thiazide-type diuretic in the nonblack hypertensive population, including those with diabetes. In the black hypertensive population, including those with diabetes, a calcium channel blocker or thiazide-type diuretic is recommended as initial therapy.5 JNC- 7 Recommended 5 classes to be considered as initial therapy but recommended thiazide-type diuretics as initial therapy for most patients without compelling indication for another class it specified particular antihypertensive medication classes for patients with compelling indications, such as diabetes, CKD, heart failure, myocardial infarction, stroke, and high CVD risk while JNC-8 recommended selection among 4 specific medication classes (ACEI or ARB, CCB or diuretics) and doses based on RCT evidence recommended specific medication classes based on evidence review for racial, CKD, and diabetic subgroups.2,5
2.9 Prevention

Hypertension is typically diagnosed and managed in the outpatient setting and is one of the most common reasons that patients visit a family physician. With almost a quarter of the adult population and almost half of people aged 50 years and older having hypertension, the burden of this disease is undeniably high. As treatment of hypertension is associated with a 20% to 25% reduction in cardiovascular events, getting control of this generally asymptomatic disease might be one of the most important preventive measures that family physicians can take. Family physicians improved the quality of care for patients with hypertension through continuous medical education. Since the first-line approach to the management of hypertension is lifestyles modifications. Family physicians can improve the quality of life of hypertensive patients through healthy lifestyle, patient education, family counselling, social support networks and health promotion programmes in order to enhance adherence of hypertensive patients with the therapeutic regimen and to improve their quality of life.

The prevention and management of hypertension are major public health challenges. Public health strategy, which complements the hypertension treatment strategy, is warranted. To prevent BP levels from rising, primary prevention measures should be introduced to reduce or minimize these causal factors in the population, particularly in individuals with pre-hypertension.

A population approach that decreases the BP level in the general population by even modest amounts has the potential to substantially reduce morbidity and mortality or at least delay the onset of hypertension.

Barriers to prevention include cultural norms, insufficient attention to health education by health care practitioners, lack of reimbursement for health education services, lack of physical activity, large amounts of sodium added to foods by the food industry and
restaurants, and the higher cost of food products that are lower in sodium and calories.\textsuperscript{2} Overcoming these barriers will require a multipronged approach directed not only to high-risk populations, but also to communities, schools, worksites, and the food industry.\textsuperscript{2,72}
CHAPTER THREE

3.0 METHODOLOGY

3.1 Study setting

The study was carried out in the general outpatient clinic (GOPC) of the Jos University Teaching Hospital (JUTH).

The hospital is located in Jos, the capital city of Plateau State, North-central Nigeria. It is located at latitude 9° 55' N and longitude 8° 53' E with an altitude of about 1250 m (about 4100 ft) above sea level. According to the 2006 National census, the State had a population of 3,206,531 while Jos North had a population of 437,217 with area of 291km².\textsuperscript{139}

The Jos University Teaching Hospital is a 600-bed facility that provides primary, secondary and tertiary care to the population of Plateau and the neighbouring States of Nasarawa, Benue, Kaduna, Bauchi, Gombe, Adamawa and Taraba. The GOPC provides continuous, holistic, and comprehensive healthcare for both acute and chronic medical conditions, while referring patients to specialist departments or clinics in a two-way referral system as necessary.

3.2 Study population

This comprised of all adults from 18 years and above who are known to have essential hypertension for at least six months and above and have been on antihypertensive medications for at least six months presenting at the general outpatient clinic of the Jos University Teaching Hospital, Jos

3.3 Study period

The study was carried out between the month of May and November, 2014
3.4 Eligibility

3.4.1 Inclusion criteria

All consenting adults 18 years and above known to have hypertension that are already on medications who presented to GOPC, JUTH within the study period were included in the study.

3.4.1 Exclusion criteria

1. Those that were ill enough to require hospital admission and Specialist care
2. Those taking any drugs causing sedation e.g. antihistamine, antitussives
3. Those taking more than two antihypertensive medications or recently (within the last 12 weeks) changed the type or dose of their antihypertensive medication or are planning to do so in the next 12 weeks because of resistance and difficulty in controlling the blood pressure
4. Those with psychiatric illnesses e.g. depression, dementia
5. Security men that work in the night and those on night shift
6. Those with co-morbidities like diabetes mellitus, renal disease
7. Those currently undergoing sleep hygiene education or related sleep cognitive behavioural therapy
8. Those with history of sleep disorders such as narcolepsy, hypersomnias, night terrors
9. Breast feeding mothers

3.5 Sample size determination

The sample size for the study was obtained using the formula for comparing the means of two groups:
\[ N = \left[ 2\sigma^2 \left( Z_{\alpha/2} + Z_\beta \right)^2 \right]^{1/2} \]

Where

- \( N \) = the sample size required in each group (double this for total sample).
- \( \sigma \) = standard deviation of the mean systolic blood pressure = 24.8 mmHg\(^5\)
- \( \delta \) = difference to be detected or larger difference between the mean blood pressure values of the intervention and control group = 10 mmHg
- \( Z_{\alpha/2} \) = The desired level of statistical significance (Typically 1.96 for 95% CI)
- \( Z_\beta \) = The desired power (Typically 0.84 for 80% power)

\[
N = 2 \times 24.8^2 \left( 0.84 + 1.96 \right)^2\]
\[
10^2
\]
\[
= 96
\]

The sample size in each group was 106 using a 10% attrition rate which is approximately 10.

### 3.6 Ethical consideration

Approval was obtained from the ethical committee of Jos University Teaching Hospital, Jos.

The nature, aim and objectives of the study were explained to the participants. A written informed consent was also obtained from each participant. The information obtained from the study was treated as confidential. At any time in the course of the study, the participants had the option to opt out of the study without affecting his or her care.

### 3.7 Instrument of data collection

1. Study Questionnaire (Appendix E)
2. Pittsburg Sleep Quality Index (Appendix F)
3. Structured sleep hygiene instruction (Appendix G)
4. Sphygmomanometer (Made in England by Dekamet MK.3)

5. Littmann stethoscope (3M™ Littmann™ Classic II S.E IIG 37027 Made in U.S.A)

6. Wall-mounted Stadiometer: for height

7. Digital Bathroom Weighing Scale (Health scale RGZ-120)

3.8 Study design

The study was a randomized controlled trial.

The study set out to test the hypothesis that structured sleep education administered to patient with hypertension will lead to a significant reduction in blood pressure.

3.9 Study protocol

Consecutive patients aged 18 years and above who were attending the GOPC and who met the inclusion criteria were enrolled into the study after obtaining an informed written consent.

The resident doctors in the department assisted in the assessment of the hypertensive patients and all hypertensive patients were referred to the researcher for further assessment. The participants were randomly assigned into either the control or the intervention groups using random numbers generated by using the Open Epi version 2.2.1. One hundred and six random numbers were generated between one and two hundred and twelve. The generated numbers were allocated to control while the remaining were allocated to the intervention. The random allocation of subjects into each group was done by means of previously sealed opaque envelopes that were only opened at the time of allocation of the subjects.

All subjects were interviewed after an informed written consent (Appendix D) was obtained using the study questionnaire (Appendix E). The component of the questionnaire included socio-demographic data, medical history, family and social history.

Focused physical examination involved the height measurement to the nearest 0.1 meter (m) using the wall-mounted stadiometer (SALTON RED-307, made in China) and weight to the
nearest 0.1 kilogram (kg) using a digital portable bathroom weighing scale (Health scale RGZ-120 by Easy way medical England). The weighing balance was calibrated daily using known weights. The height and the weight were measured in the patient with minimal wears and without footwear and in erect position. Patients were asked to remove their head wears. The height and the weight were used to calculate the body mass index (BMI) in kilogram per meter square using the formula:

\[ \text{BMI} = \frac{\text{Weight}}{\text{Height}^2} \]

The researcher using a mercury type sphygmomanometer (Made in England by Dekamet MK.3) and Littmann stethoscope (3MTM LittmannTM Classic II S.E IIG 37027 Made in U.S.A) after making the participant feel relaxed and rested for at least five minutes measured blood pressure in millimeters of mercury (mmHg). It was checked using appropriate sized cuff for the participants. The systolic blood pressure was recorded when korotkoff sound was heard while the diastolic blood pressure was the value when the sound disappears. The mercury level in the sphygmomanometer was maintained by locking the column at the end of the day to prevent spillage.

Sleep quality in both groups was assessed using the Pittsburg sleep quality index (Appendix F) The Pittsburg Sleep Quality Index tool was designed by University of Pittsburgh, Sleep Medicine Institute. It is a nine-item questionnaire that consist of 19 questions that are grouped into seven components or dimensions scores, each weighted equally on a 0- to 3-point scale. The seven dimensions were then added to yield a global PSQI score (range, 0-21points). Higher scores indicate poor quality of sleep. The seven dimension of PSQI are subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Responses are based on the majority of days (and nights) of the previous month. Patients with a PSQI score of greater than five are defined as poor sleepers whereas those with a score of five or less are
considered good sleepers.\textsuperscript{141} The tool has been used in Nigeria among hypertensive patients and the general population,\textsuperscript{19,102} It has a sensitivity of 89.6\% and specificity of 86.5\%.\textsuperscript{141}

After the assessment using the PSQI, structured sleep education in the form of sleep hygiene instruction was administered to the subjects in the intervention group with the aid of a clinical psychologist in the hospital. This was done to each individual.

The structured sleep education was re-enforced at four, eight and 12 weeks for the subjects in the intervention groups to ensure adherence. Both the control and the intervention groups were followed up at four, eight and 12 weeks.

All the questionnaires used in the study were interviewer-administered and were done by the researcher only in order to prevent inter-rater bias.

The subjects in both groups continued with their antihypertensive medications. They were reminded of their follow up visit a day before the clinic by either phone call or text message.

The follow up visits were on different days for the intervention and control groups to prevent contaminations.

\textbf{3.10 Method of data analysis}

The results obtained were analyzed using the computer software Epi Info version 3.5.3 (Centre for Disease Control, 2011, Georgia USA).\textsuperscript{142} Background descriptive analysis was done to compare the two groups. The primary outcome variables of interest were blood pressure and sleep quality. The means at 95\% confidence interval was determined using the student $t$-test and the proportion of categorical variable were compared using the $X^2$ test. Paired $t$-test analysis was used to compare the differences in mean of systolic and diastolic blood pressure in both groups before and after intervention. Other outcome variables investigated were sleep quality, sleep duration, weight, height, BMI, frequencies and percentages of various characteristics were determined.
The data was analyzed on the intention to treat (ITT) basis. A p-value of <0.05 was considered significant in all analysis.

ITT is described as “once randomized, always analyzed.” It is an analysis that includes every subject who is randomized according to randomized treatment assignment. It ignores noncompliance, protocol deviations, withdrawal, and anything that happens after randomization.

It avoids over-optimistic estimates of the efficacy of an intervention resulting from the removal of non-compliers by accepting that non-compliance and protocol deviations are likely to occur in actual clinical practice. The Consolidated Standards of Reporting Trials (CONSORT) statement for improving the quality of reports of RCTs states that number of participants in each group should be analyzed by intention-to-treat principle. This is because it maintains prognostic balance generated from the original random treatment allocation. It gives an unbiased estimate of treatment effect and preserves the sample size, because if non-compliant subjects and dropouts are excluded from the final analysis, it might significantly reduce the sample size, leading to reduced statistical power. One advantage of ITT is that it minimized type I error due to cautious approach and allows for the greatest generalizability. However, its estimate of treatment effect is generally conservative because of dilution due to non-compliance. Also, heterogeneity might be introduced if noncompliants, dropouts and compliant subjects are mixed together in the final analysis. Moreover, end-point data will differ markedly among non-compliant, dropouts and compliant subjects, and interpretation might become difficult if a large proportion of participants cross over to opposite treatment arms. ITT analysis has been criticized for being too cautious and thus being more susceptible to type II error. In ITT, the missing data are dealt with by using the last observation carried forward (LOCF) method, whereby the last available
measurement for each individual at the time point prior to withdrawal from the study is retained in the analysis.\textsuperscript{146}

ITT differs from the per protocol (PP) analysis which refers to inclusion in the analysis of only those patients who strictly adhered to the protocol. The PP analysis provides an estimate of the true efficacy of an intervention, i.e., among those who completed the treatment as planned. However, its results do not represent the real life situation and it is likely to show exaggerated treatment effect.\textsuperscript{147} The per protocol analysis may be biased. This is because the participants were excluded if they did not complete the protocol of the allocated treatment and the original comparability of the treatment groups in their baseline characteristics achieved after randomisation may not have been maintained.\textsuperscript{146-147}
CHAPTER FOUR

4.0 RESULTS

4.1 Introduction

Two hundred and twelve subjects fulfilled the inclusion criteria and participated in the study, out of which there were One hundred and six each in the control and the intervention group. Two hundred and six (97.2%) completed the study while six (2.8%) did not complete the study. Out of the six that did not complete the study, three each were from the control and the intervention group. In the control group, one participant dropped out at fourth week and the remaining two at eighth week. In the intervention group, one dropped out at fourth week, one at eighth week and one did not present at twelfth week. Analysis was carried out based on the intention to treat basis.\textsuperscript{146-147} The chart in figure 1 below shows the flow of the participants.
Recruited and randomized
N=212

Control
N=106
Lost to follow up 1 at 4wks, 2 at 8wks
Completed study
N=103
Analyzed
N=106

Intervention
N=106
Lost to follow up 1 each at 4wks, 8wks and 12wks
Completed study
N=103
Analyzed
N=106

Figure 4.1 Trial Profile of participants through the study period.
### 4.2 Baseline characteristics of the study groups

Table 4.1: Socio-demographic characteristics

<table>
<thead>
<tr>
<th>Socio-demographic characteristics</th>
<th>Study group</th>
<th>χ²</th>
<th>Df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total No. (%)</td>
<td>N=106(100%)</td>
<td>N=106(100%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age group (yrs)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-44</td>
<td>57(26.9)</td>
<td>29(27.4)</td>
<td>28(26.4)</td>
<td>0.430</td>
</tr>
<tr>
<td>45-64</td>
<td>132(62.3)</td>
<td>67(63.2)</td>
<td>65(61.3)</td>
<td></td>
</tr>
<tr>
<td>&gt;64</td>
<td>23(10.8)</td>
<td>10(9.4)</td>
<td>13(12.3)</td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>27(12.7)</td>
<td>14(13.2)</td>
<td>13(12.3)</td>
<td>0.042</td>
</tr>
<tr>
<td>Female</td>
<td>185(87.3)</td>
<td>92(86.8)</td>
<td>93(87.7)</td>
<td></td>
</tr>
<tr>
<td><strong>Religion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Christianity</td>
<td>88(41.5)</td>
<td>47(44.3)</td>
<td>41(38.7)</td>
<td>0.699</td>
</tr>
<tr>
<td>Islam</td>
<td>124(58.5)</td>
<td>59(55.7)</td>
<td>65(61.3)</td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hausa/Fulani</td>
<td>93(43.9)</td>
<td>41(38.7)</td>
<td>52(49.1)</td>
<td>4.795</td>
</tr>
<tr>
<td>Plateau indigenous tribes</td>
<td>73(34.4)</td>
<td>39(36.8)</td>
<td>34(32.1)</td>
<td></td>
</tr>
<tr>
<td>Yoruba</td>
<td>11(5.2)</td>
<td>4(3.4)</td>
<td>7(6.6)</td>
<td></td>
</tr>
<tr>
<td>Igbo</td>
<td>3(1.4)</td>
<td>2(1.9)</td>
<td>1(0.9)</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>32(15.1)</td>
<td>20(18.9)</td>
<td>12(11.3)</td>
<td></td>
</tr>
<tr>
<td>Marital status</td>
<td>Married</td>
<td>Divorced</td>
<td>Unmarried</td>
<td>Widowed/widower</td>
</tr>
<tr>
<td>------------------</td>
<td>---------</td>
<td>----------</td>
<td>-----------</td>
<td>-----------------</td>
</tr>
<tr>
<td></td>
<td>152(71.7)</td>
<td>2(0.9)</td>
<td>5(2.4)</td>
<td>53(25.0)</td>
</tr>
<tr>
<td></td>
<td>76(71.7)</td>
<td>1(0.9)</td>
<td>2(1.9)</td>
<td>27(25.5)</td>
</tr>
<tr>
<td></td>
<td>76(71.7)</td>
<td></td>
<td></td>
<td>26(24.5)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Govt employed</th>
<th>Self employed</th>
<th>NGO</th>
<th>Student</th>
<th>Unemployed</th>
<th>p-value</th>
<th>X²-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>42(19.8)</td>
<td>133(62.7)</td>
<td>8(3.8)</td>
<td>2(0.9)</td>
<td>27(12.7)</td>
<td>2.200</td>
<td>0.699</td>
</tr>
<tr>
<td></td>
<td>22(20.8)</td>
<td>68(64.2)</td>
<td>2(1.9)</td>
<td>1(0.9)</td>
<td>13(12.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20(18.9)</td>
<td>65(61.3)</td>
<td>6(5.7)</td>
<td>1(0.9)</td>
<td>14(13.2)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Educational level</th>
<th>None formal</th>
<th>Primary</th>
<th>Secondary</th>
<th>Tertiary</th>
<th>p-value</th>
<th>X²-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>107(50.5)</td>
<td>47(22.2)</td>
<td>19(9.0)</td>
<td>39(18.4)</td>
<td>0.709</td>
<td>0.871</td>
</tr>
<tr>
<td></td>
<td>51(48.1)</td>
<td>25(23.6)</td>
<td>9(8.5)</td>
<td>21(19.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>56(52.8)</td>
<td>22(20.8)</td>
<td>10(9.4)</td>
<td>18(17.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Income per month</th>
<th>≤₦18,000.00</th>
<th>&gt;₦18,000.00</th>
<th>p-value</th>
<th>X²-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>144(67.9)</td>
<td>68(32.1)</td>
<td>0.779</td>
<td>0.377</td>
</tr>
<tr>
<td></td>
<td>69(65.1)</td>
<td>37(34.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>75(70.8)</td>
<td>31(29.2)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The socio-demographic characteristics of the study population are as shown in table 4.1. A total number of 212 patients were analysed. The mean age with standard deviation of the population (N=212) was 50.76±10.7years. The age ranged from 18 to greater than 64 yrs. There was no significant difference in the mean age distribution between the control (50.19±10.87years) and intervention group (51.33±10.54years) (t=0.817, p=0.415).
Among the 212 subjects that participated in the study, 57(26.9%) were between 18-24 years, 132(62.3%) were 45-64 years while the remaining were more than 64 years. One seventh of them were males 27(12.7%) while most of them were females 185(87.3%). (M:F ratio=1:6.9). Majority were married 152(71.7%), 53(25%) were widowed, 2(0.9%) were divorced and 5(2.4%) were unmarried.

Most of the subjects were Muslims 124(58.5%) while the rest were Christians.

Ninety-three (43.9%) of the patients were Hausa/Fulani, 73(34.5%) were Plateau indigenous tribes. Yoruba were 11(5.2%), Igbo 3(1.4%) while others were 32(15.1%).

Majority of the participants 107 (50.5%) had no formal education, while only 39(18.4%) had tertiary education.

The occupation status of the participants indicated that 133(62.7%) were self-employed, 42(19.8%) were employed by government, while 27(12.7%) were unemployed.

Most of the employed participants earned less than ₦18, 000 per month 144(67.9%) while the remaining earned more than ₦18, 000 per month 68(32.1%).

Overall table 4.1 showed that randomization was effective as there was no significant difference in the baseline socio-demographic characteristic across the groups.
### 4.3 Personal characteristics of the study groups

Table 4.2: Personal characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total (%)</th>
<th>Study group</th>
<th>χ²</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Control n=106(%)</td>
<td>Intervention n=106(%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Smoking cigarette</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6(2.8)</td>
<td>3(2.8)</td>
<td>3(2.8)</td>
<td>0.000</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>206(97.2)</td>
<td>103(97.2)</td>
<td>103(97.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Alcohol intake</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>28(13.2)</td>
<td>12(11.3)</td>
<td>16(15.1)</td>
<td>0.658</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>184(86.8)</td>
<td>94(88.7)</td>
<td>90(84.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Duration of past hypertension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-5</td>
<td>98(46.2)</td>
<td>46(43.4)</td>
<td>52(49.1)</td>
<td>0.873</td>
<td>2</td>
</tr>
<tr>
<td>6-10</td>
<td>63(29.7)</td>
<td>33(32.0)</td>
<td>31(29.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10</td>
<td>51(24.1)</td>
<td>28(26.4)</td>
<td>23(21.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>History of stroke</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2(0.9)</td>
<td>2(1.9)</td>
<td>0(0.0)</td>
<td>2.020</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>210(99.1)</td>
<td>104(98.1)</td>
<td>106(100.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Medication for hypertension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>212(100.0)</td>
<td>106(100.0)</td>
<td>106(100.0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

The medical characteristics of the group are as shown in table 4.2. Majority 98(46.2%) were hypertensive between 0-5 years, 63(29.7%) between 6-10 while 51(24.1%) were hypertensive for more than 10 years. All participants 212(100.0%) were taking medications for hypertension. Six (2.8%) smoked cigarette, 28(13.2%) of the participants drink alcohol and only two (0.9%) had history of stroke.
4.4 Family characteristics

Table 4.3: Family characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total No (%</th>
<th>Study group</th>
<th>(\chi^2)</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Control n=106(%)</td>
<td>Intervention n=106(%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family member having Hypertension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>111(52.5)</td>
<td>58(54.7)</td>
<td>53(50.0)</td>
<td>0.473</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>101(47.6)</td>
<td>48(45.3)</td>
<td>53(50.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family member having diabetes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>28(13.2)</td>
<td>17(16.0)</td>
<td>11(10.4)</td>
<td>1.481</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>184(86.8)</td>
<td>89(84.0)</td>
<td>95(89.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family member having stroke</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>19(9.0)</td>
<td>13(12.3)</td>
<td>6(5.7)</td>
<td>2.833</td>
<td>1</td>
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<tr>
<td>No</td>
<td>193(91.0)</td>
<td>93(87.7)</td>
<td>100(94.3)</td>
<td></td>
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<tr>
<td><strong>Family member having heart disease</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3(1.4)</td>
<td>2(1.9)</td>
<td>1(0.9)</td>
<td>0.338</td>
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</tr>
<tr>
<td>No</td>
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<td>104(98.1)</td>
<td>105(99.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sleep disorder</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>212(100.0)</td>
<td>106(100.0)</td>
<td>106(100.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family member having Psychiatric/mental illness</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>No</td>
<td>212(100.0)</td>
<td>106(100.0)</td>
<td>106(100.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Majority of the family relations of the participants had hypertension 111(52.5%), 28(13.2%) had diabetes mellitus, 19(9.0%) had stroke, while three (1.4%) had heart disease.
### 4.5 Sleep quality before and after intervention among the study group

Table 4.4A: Sleep quality before and after intervention

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group</th>
<th>t-test</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Control n=106</td>
<td>Intervention n=106</td>
<td></td>
</tr>
<tr>
<td>Mean PSQI</td>
<td>Initial</td>
<td>6.86±3.52</td>
<td>6.51±3.41</td>
<td>7.21±3.56</td>
</tr>
<tr>
<td></td>
<td>Final</td>
<td>4.82±3.31</td>
<td>6.55±3.21</td>
<td>3.43±2.70</td>
</tr>
<tr>
<td>Mean sleep duration (hrs)</td>
<td>Initial</td>
<td>5.60±2.6</td>
<td>5.73±1.18</td>
<td>5.47±1.33</td>
</tr>
<tr>
<td></td>
<td>Final</td>
<td>6.25±0.96</td>
<td>5.98±1.01</td>
<td>6.51±0.83</td>
</tr>
</tbody>
</table>
### 4.6 Assessment of sleep quality in study groups

Table 4.4B Sleep quality before and after intervention

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group</th>
<th>( \chi^2 )</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total No. (%)</td>
<td>Control n=106(%)</td>
<td>Intervention n=106(%)</td>
<td></td>
</tr>
<tr>
<td>PSQI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>Good sleep</td>
<td>75(100.0) 45(60.0) 30(40.0)</td>
<td>3.000 1 0.083</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>137(100.0) 61(44.5) 76(55.5)</td>
<td>1.642 1 0.200</td>
<td></td>
</tr>
<tr>
<td>Final</td>
<td>Good sleep</td>
<td>143(100.0) 48(33.6) 95(66.4)</td>
<td>14.362 1 0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>69(100.0) 58(84.1) 11(15.9)</td>
<td>31.154 1 0.001</td>
<td></td>
</tr>
<tr>
<td>Sleep duration</td>
<td>Short</td>
<td>161(100.0) 80(49.4) 81(50.6)</td>
<td>0.025 1 0.875</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>39(100.0) 19(46.3) 20(53.6)</td>
<td>0.220 1 0.639</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Long</td>
<td>12(100.0) 7(58.3) 5(41.7)</td>
<td>0.657 1 0.414</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Short</td>
<td>123(100.0) 81(65.9) 42(34.1)</td>
<td>12.366 1 0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>84(100.0) 21(25.0) 63(75.0)</td>
<td>21.000 1 0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Long</td>
<td>5(100.0) 4(80.0) 1(20.0)</td>
<td>1.800 1 0.180</td>
<td></td>
</tr>
</tbody>
</table>

The sleep quality among the study participants is presented in table 4.4A and 4.4B. Results of the PSQI assessment revealed a mean score of 6.86±3.52 at the baseline and 4.82±3.31 after
the intervention with a p=0.001. This showed that there was statistical significant difference between sleep quality at baseline and after intervention.

Seventy-five (35.4%) of the hypertensive patients had good sleep quality at baseline, 45(60%) in the control group compared to 30(40%) in the intervention group. Majority of the study participants had poor sleep quality 137 (64.6%). Sixty-one (44.5%) were in the control group while 76(55.5%) were in the intervention group. There was no statistically significant difference at baseline(x^2=3.000, df=1, p=0.083)

The participants with short sleep duration at baseline, in both control and intervention groups were 49.7% and 50.3% respectively.

After intervention the number of participant with good sleep quality increased to 143(67.5%) while those with poor sleep quality decreased to 69 (32.5%) among the participants. Good sleep quality at the end of the study in the intervention group was 95(66.4%) and 48(33.6%) in the control group. This was statistically significant (p=0.001). Fifty-five (79.7%) participants had poor sleep quality in the control while only 14(20.3%) had poor sleep in the intervention group at the end of the study. This was also statistically significant. (p=0.001).

There were more participants with normal sleep 63(75%) in the intervention than 21(25%) in the control group after the intervention. (p=0.001).
Figure 4.2: Changes in the number of participants relating to quality of sleep of the study groups over the study period.
Table 4.5: Quality of sleep of study groups over 12 weeks

<table>
<thead>
<tr>
<th>Period of study</th>
<th>Control</th>
<th>Intervention</th>
<th>$\chi^2$</th>
<th>Df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Good sleep</td>
<td>45(60.0)</td>
<td>30(40.0)</td>
<td>3.000</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>61(44.5)</td>
<td>76(55.5)</td>
<td>1.642</td>
<td>1</td>
</tr>
<tr>
<td>4 weeks</td>
<td>Good sleep</td>
<td>46(56.1)</td>
<td>36(43.9)</td>
<td>3.126</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>60(46.2)</td>
<td>70(53.8)</td>
<td>2.669</td>
<td>1</td>
</tr>
<tr>
<td>8 weeks</td>
<td>Good sleep</td>
<td>57(40.1)</td>
<td>85(59.9)</td>
<td>5.521</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>49(70.0)</td>
<td>21(30.0)</td>
<td>11.200</td>
<td>1</td>
</tr>
<tr>
<td>12 weeks</td>
<td>Good sleep</td>
<td>48(33.6)</td>
<td>95(66.4)</td>
<td>14.362</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Poor sleep</td>
<td>58(84.1)</td>
<td>11(15.9)</td>
<td>31.154</td>
<td>1</td>
</tr>
</tbody>
</table>

Figure 4.2, Tables 4.5 shows the changes in the quality of sleep of the study participants over the period of 12 weeks duration. At the end of the intervention, the number of participants with good sleep in the control group reduced from 60% to 33.6% while in the intervention arm it increased from 40% to 66.4%. On the other hand, the participants with poor sleep quality increased in the control group from 44.5% to 79.9% while in the intervention group, it reduced from 55.5% to 20.3%. This was statistically significant. (p=0.001).
## 4.7 Participants mean blood pressure changes before and after intervention

Table 4.6: Comparing the initial and final blood pressure changes in the study group

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Study group</th>
<th>Study Phase</th>
<th>Total mean</th>
<th>Initial</th>
<th>Final</th>
<th>Change</th>
<th>t-test</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td></td>
<td>141.92±19.30</td>
<td>141.92±19.30</td>
<td>137.92±15.10</td>
<td>4.000</td>
<td>1.681</td>
<td>104</td>
<td>0.094</td>
</tr>
<tr>
<td></td>
<td>Intervention</td>
<td></td>
<td>131.00±15.28</td>
<td>145.19±21.88</td>
<td>127.26±16.19</td>
<td>17.93</td>
<td>6.782</td>
<td>104</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td></td>
<td>90.47±11.21</td>
<td>89.15±10.79</td>
<td>86.70±8.92</td>
<td>2.45</td>
<td>1.804</td>
<td>104</td>
<td>0.073</td>
</tr>
<tr>
<td></td>
<td>Intervention</td>
<td></td>
<td>82.48±8.33</td>
<td>91.66±11.26</td>
<td>80.00±6.76</td>
<td>11.66</td>
<td>9.140</td>
<td>104</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table 4.6 showed that there was reduction in the mean systolic blood pressure in the control group by 4mmHg (p=0.094), and in the intervention group by 17.93mmHg (p=0.001). In the diastolic blood pressure the reduction in control group was 2.45mmHg (p=0.073), while in the intervention the reduction was by 11.66mmHg (p=0.001).
### 4.8 Report of the mean blood pressure changes over the study period

Table 4.7: Changes in the mean blood pressure of study groups over the study period

<table>
<thead>
<tr>
<th>Study period</th>
<th>Systolic Blood pressure</th>
<th>Diastolic Blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Intervention</td>
</tr>
<tr>
<td>Baseline</td>
<td>141.92±19.30</td>
<td>145.19±21.88</td>
</tr>
<tr>
<td>4weeks</td>
<td>136.50±15.13</td>
<td>133.30±19.77</td>
</tr>
<tr>
<td>8weeks</td>
<td>137.77±14.95</td>
<td>126.99±16.26</td>
</tr>
<tr>
<td>12weeks</td>
<td>137.92±15.10</td>
<td>127.26±16.19</td>
</tr>
</tbody>
</table>
4.9: Reduction in blood pressure

Figure 4.3: Baseline and post intervention systolic blood pressure between the control and intervention groups

Figure 4.4: Baseline and post intervention diastolic blood pressure between the control and intervention groups
The changes in the mean blood pressure of the study groups over the 12 weeks is as shown in Table 4.7 and figures 4.3 and 4.4 which revealed that reduction in blood pressures in both arms of the intervention was more at 12 weeks, but more in the intervention groups than in the control groups.
### 4.10 Logistic regression analysis of the association between PSQI, Systolic BP and Diastolic BP in relation to study group

Table 4.8: Logistic regression analysis of the association between PSQI, Systolic BP and Diastolic BP in relation to study group

<table>
<thead>
<tr>
<th>Study group</th>
<th>OR</th>
<th>95% C.I.</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PSQI at 12 weeks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>10.656</td>
<td>4.991-22.752</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>PSQI at 4 weeks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>3.858</td>
<td>2.053-7.250</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Duration of sleep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>0.650</td>
<td>0.088-0.310</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Family member having stroke</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>0.306</td>
<td>0.095-0.984</td>
<td>0.047</td>
</tr>
<tr>
<td><strong>Systolic BP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>5.066</td>
<td>2.696-9.518</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Diastolic BP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention group</td>
<td>11.524</td>
<td>5.394-24.621</td>
<td>0.001</td>
</tr>
</tbody>
</table>
The logistic regression showed that the odd ratios for reduced systolic blood pressure was 5.066 (95% CI, 2.696-9.518), 11.524 (95% CI, 5.394-24.621) for diastolic blood pressure and 10.656(95% CI, 4.991-22.752) for good sleep quality. (p=0.0001)

Table 4.6 showed that the mean systolic blood pressure reduction in the intervention after 12weeks was 17.93mmHg, while the mean diastolic blood pressure after the same period was 11.66mmHg. The paired t-test analysis revealed a statistical significant difference in the mean systolic blood pressure at the end of the study t [(104) 6.782, p=0.001. There was also significant difference in the mean diastolic blood pressure t [(104) 9.140, p=0.001].
CHAPTER FIVE

Discussion

Several studies have been carried out to look at the quality of sleep among hypertensive patients and also on the relationship between sleep duration, sleep disorders and hypertension in other countries but only little is known about the effect of sleep on blood pressure control in our environment. In a search through the literature, limited studies that investigated the effect of improved sleep quality in controlling blood pressure were found. This study sought to look at the effect of structured sleep education on blood pressure control among adult hypertensive patients in this environment. At baseline there was no significant difference between the control and the intervention groups confirming that randomization was effective with respect to the distribution of the participants in the two arms of the study.

This study has demonstrated that improvement in sleep quality through structured sleep education led to reduction in blood pressure among hypertensive patients.

5.1 Socio-demographic characteristics of the participant

The mean age of the participants was 50.76±10.72, comparable to previous findings in Nigeria by Wokoma et al. (48.9 + 14.8 years), and Ordinioha (46.06 ± 9.62 years). However, other studies reported a lower mean age; Ekanem et al. (31.7 ± 7.6 years) in South-south Nigeria and Adediran (41.91±15.08) in rural Nigeria. This could be accounted for since the study by Ekanem et al. was carried out among the residents in military barracks which consisted mainly of young adults. The higher age in this study supports the fact that the prevalence of hypertension increases with age as reported by Iyalomhe et al. (59.05± 9.06), Alebiosu (58.15±9.65 years) in semi-urban Nigeria and Ostchega (>60 years) in the United States. The WHO as well as Ekwunife et al. showed that age is an important risk factor for hypertension. Ostchega and colleagues also showed that individuals older than
60 years had a higher prevalence of hypertension when compared with those in younger age groups. In this study, individuals older than 64 years accounted for only 10.8%. This may be explained for by the small number of participants in this age group and the shorter life span in our environment resulting in small number of the elderly in the study. The finding was lower than that of Ekwunife et al where they reported that prevalence of hypertension increased with advancing age to the point where approximately three-quarters of those aged 70 years and older are affected.

The study participants were made up of 27 (12.7%) male and 185 (87.3%) females (M:F = 1:6.9). This finding is similar to an earlier studies from Osun, Barako and Abia in Nigeria and China. The higher proportion of females in this study could be as a result of the characteristics of the study population which consisted of more females presenting with hypertension. It may also be due to higher health seeking behavior among women. This was similar to the finding by Qureshi et al in the United States (4.1 million males and 6.9 million females). Ekwunife et al (40.3% versus 24.7%) and Ugwuja et al (28.2% versus 20.9%) in Nigeria. This could be explained in part by life-style changes as well as environmental and social factors typical of urbanized places which are taking place in Nigeria and population growth. This is because the population were based in the urban areas. Other studies also showed a similar trend. However, WHO in 2010 Global report showed that the prevalence of hypertension was 46% for both sexes aged 25 and above, while the lowest prevalence of raised blood pressure was in the Americas, with 35% for both sexes due to improvement in socioeconomic status.

Majority of the study participants had no formal education. This may be because most of them were women and educational status of women in this community is low. Also the low literacy level in Northern Nigeria among women is due to low level of girl child education.
This is contrary to the study in Lagos, Nigeria where most of the hypertensive patients (67.5%) had at least a secondary education. While in the developed world majority of hypertensive patients had tertiary education. Other studies have shown that Literacy level was higher among hypertensive and level of education did not affect control of hypertension. Ugwuja and colleagues also reported an increase in hypertension prevalence with advancement in educational status such that participants with tertiary education had significantly higher prevalence of the disease in comparison with those with primary education. This is because education as a marker of socio-economic status may be associated with lifestyle habits that put the individual at risk of developing hypertension. On the contrary, Egbi and colleagues reported a better blood pressure control among hypertensives with high socioeconomic status. This is due to improved capacity to seek and pay for medical services. However, the low prevalence of hypertension in the study group with tertiary education may be due to high socioeconomic status resulting in improved health care as seen with low prevalence of hypertension in developed world.

The majority of the participants earned at least eighteen thousand naira or less probably because of the low level of education. Most of them are self employed or involved in petty trading or subsistence farming. This was similar to the findings of Awobusuyi and colleagues in Lagos where they showed that monthly income was found to be generally low with 31.8% earned between ₦10,000 to ₦20,000, while majority(44%) earned less than ₦10,000 per month. Other reasons being that majority of the hypertensive patients attending the outpatient clinic were either traders or small scale business men/women and are in the low socioeconomic class. This was contrary to the study by Ekanem and colleagues, in semi-urban community in South-south Nigeria, which showed that majority earned between ₦30,000 and ₦50,000 per month which is above the country’s minimum wage of ₦18,000.
More than sixty percent of the participants (62.7%) were self employed, this may be explained by the low socioeconomic status of the study population. This was similar to the findings of Katibi and colleagues in the Middle-belt, Nigeria where they reported that most of the hypertensive patients attending University of Ilorin Teaching Hospital were either traders or business men/women 44.5%.\textsuperscript{38}

One hundred and fifty-two (71.7%) of the patients were married, fifty-three (25%) were widowed, two (0.9%) were divorced and only 5(2.4%) were unmarried. This was similar to the findings of Egbi et al (63.6%) in Yenagoa, Nigeria.\textsuperscript{53} They reported that increasing age, marital transition from unmarried to married life and the associated changes in lifestyle, some of which may negatively impact on blood pressure, leading to increased in blood pressure. These include loss of spousal support and associated stress which may be experienced by separated or widowed individual.\textsuperscript{53, 58, 139} Egbi reported that marital status had a significant relationship with hypertension and that there was a lower prevalence of hypertension among those who were unmarried compared with their married counterparts due to increasing age among the married hypertensive patients.\textsuperscript{53} This was similar to the finding in this study that showed that majority of the hypertensive patients were married, but was in contrast to the finding of Lipowicz et al., they observed an increase in risk of hypertension among the unmarried.\textsuperscript{53, 58}

5.2: Sleep quality

More than half of the participants (64.6%) had poor sleep quality and only 35.4% had good sleep. This is comparable to the findings of Pearson et al (66.7%)\textsuperscript{103} but higher than the findings of Alebiosus et al in a prospective study on quality of sleep among hypertensive patients in Nigeria, where they reported a 42.4% poor sleep quality and 27.8% good sleep quality among hypertensive patients.\textsuperscript{102} Shittu et al reported 44\%\textsuperscript{19} prevalence of poor sleep
quality in Nigeria while Bruno et al reported 38.2% in Italy.\textsuperscript{13} The poor sleep quality was due to elevated blood pressure and high BMI.\textsuperscript{14,97,102} On the contrary, Ji-Rong et al reported a high prevalence (58.4%) of good sleep quality among hypertensive patients.\textsuperscript{29} This is because most of the participants were younger, they also showed that there was no association between sleep quality and arterial blood pressure.\textsuperscript{29} This is because the association between sleep quality and arterial blood pressure changed with age, with increased difficulties in sleep initiation and maintenance in the elderly.\textsuperscript{29}

Kai et al in China showed that good and poor sleep qualities were associated with hypertension with odds ratios of 1.20 (95% CI, 1.01–1.42) and 1.67 (95% CI, 1.32–2.11) respectively.\textsuperscript{115} This showed that individuals with poor sleep quality are more likely to develop hypertension than individuals with good sleep quality. This was similar to other studies in the US where they reported that there is a strong significant association between sleep quality with blood pressure or with the risk of hypertension among adolescents.\textsuperscript{16,116}

The mean PSQI score in the present study before intervention was 6.86±3.52, which was high and signifies poor sleep quality and is comparable to the findings of Alebiosus et al (5.03±3.28) in Nigeria, Ji-Rong et al (6.84±2.150) in China\textsuperscript{29}. In the present study, majority of the participants with poor sleep were women. This is similar to findings by Stranges et al in a multi-center cross sectional study on sleep problems, where they observed that a large number of women in low-income settings are currently experiencing sleep problems.\textsuperscript{92} This is because women have different physiology, greater burden of psychologic comorbidity (anxiety, depression), gender-specific roles such as mothering and related stresses, disparities in socioeconomic status and social role, and differential self-reporting of sleep habits between men and women.\textsuperscript{92} They also observed a higher prevalence of sleep problems in older age groups.\textsuperscript{92} Other studies have shown that there is no association between sleep quality with blood pressure in people aged 60 years or older.\textsuperscript{95, 97} This finding is in agreement with that of
the present study since there is a statistical significant relationship between sleep quality and hypertension in subjects less than 60 years of age (p<0.001).

After intervention, the present study showed a statistical significant improvement in sleep quality (p<0.001). This therefore, demonstrated the effect of the structured sleep education on improving sleep quality.

5.3 Sleep duration

There was a statistical significant association between sleep duration and hypertension in the present study. The mean duration of sleep for the control group was 5.60±1.26 hours while it was 6.25±0.96 hours for the intervention group. At baseline, 75.9% of the participants slept less than 7 hours, while 18.4% slept between 7-8 hours and 5.7% slept more than 8 hours per night in the control group. The higher percentage of those who sleep less than 7 hours was comparable to the findings of Vgontzas et al where they showed that individual who slept ≤ 5 h or 5-6 h, respectively, had a risk of hypertension than those who slept > 6 hours and had no sleep complaint. Similarly, Gottlieb et al in an epidemiologic study in the United States on association of usual sleep and hypertension reported that participants aged 32 and 59 years at baseline who reported sleeping ≤ 5 hours of sleep per night were at an increased risk for developing hypertension over the follow-up period. This was similar to the findings by Choi et al in 2008, in the Korean National Health and Nutrition survey 2001 where they observed that short sleep duration was associated with higher prevalence of hypertension. This was because those who have short sleep duration have an increase in sympathetic nervous system activity which may cause sustained hypertension and overactivity of the renin-angiotensin-aldosterone system, proinflammatory endothelial dysfunction, and renal impairment. While those who sleep 9 or more hours per night reported 15% less physical activity per week than those having a normal sleep 7 to 8 hours per night, and inactivity may place these long sleepers at increased risk of hypertension.
After intervention, majority (75%) of the participants slept between 7 to 8 hours per night. This was comparable to the findings of Gottlieb et al where they reported that individuals who slept between 7 and 8 hours had low incidence of developing hypertension. This may explain the reason why there was reduction in the blood pressure following intervention with correspondent increase of up to 75% of hypertensive patients that slept between seven and eight hours. The present study also shows that following intervention, there was no individual with sleep duration of greater than 8 hours. This may be due to improvement in sleep quality. Conversely, in 2013, Pandey and colleagues reported that long sleep may actually be more detrimental to health than short sleep. This could be due to sleep fragmentation and photoperiodic abnormalities, which could all lead to increased blood pressure levels. Pooled analyses conducted in a recent review of 16 studies of persons whose mean age ranged from 41 to 77 years indicated that sleeping fewer than 7 hours per night increased mortality risk by 12%, and sleeping more than 8-9 hours per night increased mortality risk by 30% compared with those sleeping 7-8 hours' per night.

On the other hand, Julia et al in a cross sectional study on sleep duration and hypertension in Rotterdam found no association between either self-reported or actigraphically measured sleep duration and hypertension or blood pressure, this was also reported by Gangwisch et al where they reported that sleep duration and hypertension are not related in those aged ≥60 years. This was because most of the participants were elderly and had short sleep duration due to insomnia or decrease need of sleep, another reason is that elderly people compensate for their nightly short sleep duration by daytime nappings.

5.4 Effect of structured sleep education on blood pressure

The results from this study shows that mean systolic blood pressure reduction in the intervention after 12 weeks was 17.93 mmHg, while the mean diastolic blood pressure after the same period was 11.66 mmHg. The paired t-test analysis revealed a statistical significant
difference in the mean systolic and diastolic blood pressure at the end of the study (p=0.001). This was similar to the findings of Haack et al in sleep extension interventional study, where they reported that improving sleep through sleep hygiene instructions, such as maintaining regularity of bedtimes etc after a period of 6 weeks reduce systolic blood pressure by 14mmHg. The result from the present study was higher than 1.38 mm Hg (95% CI, 3.6 to −0.88 mm Hg) reduction in systolic blood pressure and 1.52 mm Hg (95% CI, 3.1 to −0.07 mm Hg) in diastolic blood pressure reported by Calhoun et al. The results are not comparable because the studies though interventional were carried out on patients with obstructive sleep apnoea. Similarly, improving sleep by use of medication showed a reduction in systolic blood pressure by 10.5mmHg and by 8.1mmHg in diastolic blood pressure. On the other hand, total sleep deprivation over 3 days in healthy volunteers showed an average increase of 7 and 4mmHg for SBP and DBP, respectively. This was lower than the findings reported by Calhoun, where he showed an increase of 16.7±9.4mmHg in patients with periodic limb movement associated with microarousal during sleep. Similarly, Ogawa et al showed that diastolic blood pressure was significantly higher after total sleep deprivation than after control sleep (66.5 ±1.7 vs 57.4 ± 3.3 mm Hg) and this was due to increase in sympathetic activity. The findings suggested that behavioral interventions designed to increase sleep duration may serve as an effective strategy in the treatment of high BP. However, the limitation here was that the sample size was small.

There are limited data to demonstrate the effectiveness of increasing and reducing sleep quality on blood pressure. There were no studies found on the effect of structured sleep education or sleep hygiene on blood pressure in our setting.

In the present study, following the structured sleep education, it was observed that there was a statistically significant reduction in the mean blood pressure (p<0.001). The odd ratio for the effect of structured sleep education on blood pressure control among the hypertensive patients was 0.85 (95% CI, 0.75 to 0.96).
patients were 5.066 (95% CI 2.696-9.518; p=0.001) for the systolic blood pressure and 11.524 (95% CI 5.394-24.621; p=0.001) for the diastolic blood pressure. This may be because most of the participants are younger and diastolic blood pressure is more common in people younger than 60 years.\textsuperscript{5}.

This study evaluated the cumulative effect of structured sleep education by improving sleep quality through sleep hygiene education on blood pressure control. The mean reduction in the systolic and diastolic blood pressure was statistically significant. Despite other modalities of treatment, greater BP reduction was observed in the groups with improved sleep quality as compared to the group with poor sleep and in those that had structured sleep education. Therefore, it can be concluded that in addition to the other modalities of treating hypertension, improvement in sleep quality can also help in reducing blood pressure among hypertensive patients.

5.5 Strength and limitations of the study

The major strength of this study was that the participants were randomly allocated to either of the control or the intervention group without the knowledge of the investigator. This helped to reduce the effect of confounding variables. Another strength of this study is its design being an interventional study. The presence of a separate control group allowed for the comparison of the participants.

5.6 Limitation of the study

The major limitation of this study was that there are few interventional studies on the effect of improving sleep quality to control blood pressure among hypertensive patients.

However, the PSQI tool is sensitive and has been validated across many cultural settings in the world. Local validity and formal translation into Nigerian languages have not been done. This implies that the sensitivity and the validity of the tool in this study should be considered
with caution despite the fact that the tool had a positive result. To overcome this, only the researcher administered the tool.

Another major limitation of this study was the lack of local and insufficient international interventional journal studies on structured sleep education. This made comparison very difficult.

5.7 Relevance of the study to family medicine:

Hypertension is typically diagnosed and managed in the outpatient setting and is one of the most common reasons to visit a family physician. With almost a quarter of the adult population and almost half of people aged 50 years and older having hypertension, the burden of this disease is undeniably high. As treatment of hypertension is associated with a 20% to 25% reduction in cardiovascular events, getting control of this generally asymptomatic disease might be one of the most important preventive measures that family physicians can take.\textsuperscript{139}

This study is relevant to the specialty of family medicine, because this approach can improve the care of patient at little or no cost. Therefore improving blood pressure at a reduced cost is the aim of this study. Family physicians should therefore, always assess the quality of sleep among hypertensive patients and ensure that sleep hygiene is part of management of hypertensive patients.

Most patients would be interested in improving their sleep hygiene if advised by their doctor through education.

5. 8 Conclusion

Hypertension is the most common cardiovascular disease and one of the reasons patients visit Family Physicians.\textsuperscript{1} It is also worthy of note that not all hypertensive patients who are on drugs have achieved blood pressure control. One of the risk factors for poor BP control in hypertension is insufficient sleep and poor sleep quality.\textsuperscript{15,16} This study, therefore, was
designed to determine the effect of structured sleep education by improving sleep quality to control blood pressure among hypertensive patients in JUTH. This it did through its specific objectives. It also determined the quality of sleep among the patients.

The study was designed with a power of 80% to determine 10mmHg mean reduction in blood pressure with 95% level of significance. At baseline, there was no significant difference between the two groups

The study found a significant improvement in blood pressure in the intervention group. It also found a high prevalence of poor sleep quality which improved after intervention.

5.9 Recommendations

1. Based on the result of this study, there would be need for further interventional research to be conducted on the effect of improving sleep quality to control blood pressure among hypertensive patients.

2. There is need to always assess sleep quality among hypertensive patients since majority of them have poor sleep quality.

3. Structured sleep education should be included in the routine care of hypertensive patients. Since this study was hospital based and seem to imply that prevalence of hypertension was common in women than men. This question can be answered by community-based study to determine the prevalence of both sleep quality and hypertension.

4. Multicentre study in Nigeria validating the PQSI tool would be gratifying so as to enable easy comparison of the studies with other studies in other parts of the world.

5. In the light of the global increase in the prevalence and cost of treating hypertension and the findings from this study, there is need for physicians to educate patients on adopting healthy lifestyles in addition to improvement in their sleep hygiene and quality through a structured sleep education in order to reduce blood pressure and improve their quality of life.
The success of these recommendations will depend to a large extent on the degree to which they are applied and adopted for use by physicians and other care professionals and also the generalization of the research findings.

5.10 Suggestion for further studies

Currently no available research assesses the effect of structured sleep education on blood pressure control on hypertensive patients in Nigeria. Further studies are therefore required on this subject in order to draw evidence that is more conclusive from the study. Longer studies that will involve a community-based approach of population of hypertensive patients for the evaluation of structured sleep education on hypertensive patients and quality of sleep should be carried out.
REFERENCES


34. Centre for Disease Control, Morbidity and Mortality Weekly Report Weekly / Vol. 60/ No. 8 March 4, 2011.


75. Sabanayagam C, Shankar A. Sleep duration and cardiovascular disease: results from the National Health Interview Survey. SLEEP. 2010; 33(8):1037-42.


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## APPENDIX A

### CLASSIFICATION OF BLOOD PRESSURE

<table>
<thead>
<tr>
<th>BLOOD PRESSURE CLASSIFICATION</th>
<th>SBP mmHg</th>
<th>DBP mmHg</th>
</tr>
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<tbody>
<tr>
<td>NORMAL</td>
<td>&lt;120</td>
<td>and &lt;80</td>
</tr>
<tr>
<td>PREHYPERTENSION</td>
<td>120-139</td>
<td>or 80-89</td>
</tr>
<tr>
<td>STAGE 1 HYPERTENSION</td>
<td>140-159</td>
<td>or 90-99</td>
</tr>
<tr>
<td>STAGE 2 HYPERTENSION</td>
<td>≥160</td>
<td>≥100</td>
</tr>
<tr>
<td>Modification</td>
<td>Recommendation</td>
<td>Approximate SBP Reduction (Range)</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>Weight reduction</td>
<td>Maintain normal body weight (body mass index 18.5–24.9 kg/m²).</td>
<td>5–20 mmHg/10 kg weight loss</td>
</tr>
<tr>
<td>Adopt DASH eating plan</td>
<td>Consume a diet rich in fruits, vegetables, and low fat dairy products with a reduced content of saturated and total fat</td>
<td>8–14 mmHg</td>
</tr>
<tr>
<td>Dietary sodium reduction</td>
<td>Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride).</td>
<td>2–8 mmHg</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week).</td>
<td>4–9 mmHg</td>
</tr>
</tbody>
</table>
Moderation of alcohol consumption

Limit consumption to no more than consumption 2 drinks (1 oz or 30 mL ethanol; e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey) per day in most men and to no more than 1 drink per day in women and lighter weight persons.

Non-pharmacologic treatment to prevent or reduce elevated blood pressure

2–4 mmHg
APPENDIX C
APPENDIX D

CONSENT FORM

I am Dr Shurukuk Cletus Bako, of the department of Family Medicine, Jos University Teaching Hospital, Jos.

I want to carried out a study on the topic "EFFECT OF STRUCTURED SLEEP EDUCATION ON BLOOD PRESSURE CONTROL AMONG ADULT HYPERTENSIVES ATTENDING THE JOS UNIVERSITY TEACHING HOSPITAL GENERAL OUTPATIENT CLINIC" to incorporate it in the management of adult hypertensive patients attending the general outpatient clinic of Jos University Teaching Hospital

The study will involve asking you questions to fill in my questionnaire. You are free to participate in this study but if you decide not to, this study will not any in way affect the outcome of your treatment. All information filled in the questionnaire will be kept strictly confidential and your identity will not be disclosed.

If you accept to participate in the study, kindly sign the space provided below

I (Initials)_________________________ have asked questions regarding this research and have been satisfactorily answered. I therefore wish to participate in the research.

Thank you.

Signature of the patient /Date-----------------------------------------------------

Signature of Witness/Date----------------------------------------------------------

Signature of Investigator/Date-------------------------------------------------------
APPENDIX E

Questionnaire

Serial NO.------------------ Hospital NO.-------------------

Initials-----------------------------------------------

Address-----------------------------------------------------------------------

Phone Number---------------- Date------------------

Consent form signed Intervention Control

A. DERMGRAPHIC DATA

Age-------Years  Sex : Male/ Female  Ethnic group--------

Religion-------------  Occupation------------------------

Marital Status----------  Income per month------------

EDUCATIONAL STATUS

  No formal education

  Primary

  Secondary

  Tertiary

B. MEDICAL HISTORY

  Do you smoke cigarettes:  Yes  No  In the past

  How long have you been smoking    ------------------

  How many sticks do you smoke in day  ------------------

  Do you drink alcohol:  Yes  No  In the past

  How much quantity per week--------------
Which types of alcohol do you drink-----------------------

Do you have family history of

Hypertension  Yes  No
Diabetes   Yes  No
Stroke  Yes  No
Heart Disease  Yes  No
Sleep disorders   Yes   No
Psychiatric/Mental illness    Yes   No

How long have you had a high blood pressure?

Have you ever been treated for stroke:   Yes  No?

Are you taking any medications for?

Hypertension--------------------------

C. Source (s) of income -----------------------------

Any support from relatives   Yes   No

If yes, specify-----------------------------

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<th></th>
<th>Initial</th>
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<th>8weeks</th>
<th>12weeks</th>
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<tr>
<td>12WEEKS</td>
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</tbody>
</table>
APPENDIX F

The Pittsburgh sleep quality index (PSQI)

Instructions: the following questions relate to your usual sleep habits during the past month only. Your answer should indicate the most accurate reply for the majority of days and nights in past month. Please answer all questions. During the past month,
1. When have you usually gone to bed?....................... 
2. How long (in minutes) has it taken you to fall asleep each night:.........................
3. When have you usually gotten up in the morning?..............................................
4. How many hours of actual sleep do you get at night?(this may be different than the number of hours you spend in bed)
5. During the past month, how often have you had trouble sleeping because you... Not during the past month(0) Less than once a week(1) Once or twice a week (2) Three or more times a week (3)
   a. Cannot get to sleep within 30 minutes
   b. Wake up in the middle of the night or early morning
   c. Have to get up to use the bathroom
   d. Cannot breathe comfortably
   e. Cough or snore loudly
   f. Feel too cold
   g. Feel too hot
   h. Have bad dreams
   i. Have pain
   j. Other reason(s). Please describe, including how often you have had trouble sleeping because of this reason.
5. During the past month, how often have you taken medicine (prescribed or “over the counter) to help you sleep?
6. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity
7. During the past month, how much of a problem has it been or you to keep up enthusiasm to get things done?
Add the seven component score together: Global PSQI Score-----
Structured sleep hygiene instruction

Check those that apply

Avoid caffeine **6-8 hours before bedtime**. I will not have caffeine after ------ pm.

Avoid nicotine **before bedtime**. I will not have a cigarette after---------- pm.

Limit alcohol use. I will not have more than -------- drinks in the evening.

Avoid use of sleeping pills.

Do exercise regularly, but not within 2 hours of bedtime: ------------------

Ensure your bedroom is a comfortable temperature, quiet, and dark and your mattress and pillow are good.

Do take a hot bath 1-2 hours prior to bedtime.

Eat a light snack at bedtime but avoid large or problematic foods.

Avoid naps.

Limit time in bed.

Stay on a regular sleep schedule
Dr. Shurukuk Cletus Bako,  
Department of Family Medicine,  
Jos University Teaching Hospital,  
Jos-Nigeria.

RE: ETHICAL CLEARANCE/APPROVAL  
I am directed to refer to your application dated 29th October, 2013 on the research proposal titled:

“Effect of Structured Sleep Education on Blood Pressure Control among Adult Hypertensives Attending the Jos University Teaching Hospital General Out-Patient Clinic” and your appearance before the Ethical Committee on 1st November, 2013.

Following recommendation from the Institutional Health Research Ethical Committee, I am to inform you that Management has given approval for you to proceed on your research topic as indicated.

You are however required to obtain a separate approval for use of patients and facilities from the department(s) you intend to use for your research.

The Principal Investigator is required to send a progress report to the Ethical Committee at the expiration of three (3) months after ethical clearance to enable the Committee carry out its oversight function.

Submission of final research work should be made to the Institutional Health Research Ethical Committee through the Secretary in Room 2, Administration Department, please.

On behalf of the Management of this Hospital, I wish you a successful research outing.

Hajja R. Danfilo  
For: Chairman, MAC
Our Ref: AF/003/12/008/791

Date: 27th February, 2014

Dr. SHURKUK, Cletus B.
Dept. of Family Medicine
JUTH
Jos

Dear Sir/Madam,

RE: REGISTRATION OF TITLE OF DISSERTATION: ASSESSMENT OF PROPOSAL FOR THE PART II EXAMINATIONS IN THE FACULTY OF FAMILY MEDICINE

We wish to refer to your letter on the above subject matter and inform you that your proposal titled “EFFECTS OF STRUCTURED SLEEP EDUCATION ON BLOOD PRESSURE CONTROL AMONG ADULT HYPERTENSIVES ATTENDING JUTH GENERAL OUTPATIENT CLINIC, JOS” has been assessed.

The attached are the comments and recommendations of the assessors for guidance, in the conduct of your project.

You can now proceed with the study.

We wish you the best of luck.

Yours faithfully,

F Mrs. E. A. Akpabio
Examination Officer
E-mail- adanmaakpablo@yahoo.com
For: College Registrar