PREVALENCE OF OBSTRUCTIVE SLEEP APNEA RISK STATUS AMONG AMBULATORY TYPE 2 DIABETES MELLITUS PATIENTS AT MOI TEACHING AND REFERRAL HOSPITAL

BY

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DECLARATION

Student's Declaration:

I hereby submit this thesis to the School of Medicine as part of the fulfillment of the requirements of the Master of Medicine degree in Internal Medicine Moi University.

I, hereby, declare that this dissertation is entirely my work and has never been published or submitted to any institution of higher education for the purpose of conferring a degree or any other academic qualifications unless otherwise stated, in which case citation or acknowledgment has been made.

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We submit this thesis for review with our approval as university supervisors.

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DEDICATION

First and foremost, I dedicate this work to my beloved parents, my father Adan Maalim and my dear mother, Deka Farah. Through their unwavering sacrifices, guidance and commitment to my education and upbringing, they laid the foundation that made this achievement possible.

I also dedicate this work to my dear wife, Tahani Ali, whose steadfast love, patience and encouragement were my pillar throughout this journey. Your constant support during my period of study kept me grounded and focused.

To my daughters, Amalia and Aaliya, you were my source of strength and motivation, inspiring me to persevere through every challenge.

ABBREVIATION OF TERMS

AASM	American Academy of Sleep Medicine
BMI	Body Mass Index
BQ	Berlin Questionnaire
CSA	Central Sleep Apnea
ESS	Epworth Sleepiness Scale
HBA1C	Glycated Hemoglobin A1C
IDF	International Diabetes Federation
MTRH	Moi Teaching and Referral Hospital
OSA	Obstructive Sleep Apnea
PSG	Polysomnography
REM	Random Eye Movement
T2DM	Type 2 Diabetes

OPERATIONAL DEFINITIONS

1. **High Risk for OSA:** participants will be categorized to have high risk of OSA if they'll meet at least two clinical criteria specified by the Berlin Questionnaire (appendix). On the converse, those that will meet only 1 or no criteria, will be categorized as Low Risk of OSA.
2. **Obstructive Sleep Apnoea**: A clinical syndrome characterized by recurrent episodes of partial or complete upper airway obstructions during sleep resulting in snoring, nocturnal oxygen desaturation, multiple and sudden arousals from sleep and excessive daytime sleepiness (Di Maria, 2015)
3. **The modified Mallampati classification**: Is a simple scoring system that provides an estimate of space available for endotracheal intubation and risk for developing obstructive sleep apnea.
4. **Type 2 Diabetes Mellitus**: Is a metabolic condition defined by abnormally high blood glucose levels that results from increased resistance and impaired insulin secretion. For the purpose of this study, a participant was considered to have T2DM if they had a documented diagnosis of type 2 diabetes mellitus in their medical records and were actively attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital (MTRH) for at least three months.

ABSTRACT

Background: Obstructive Sleep Apnoea (OSA) is a prevalent yet under-diagnosed sleep-related breathing disorder associated with considerable metabolic and cardiovascular morbidity. In patients with Type 2 Diabetes Mellitus (T2DM), the coexistence of OSA exacerbates glycemic dysregulation and amplifies the risk of diabetic complications. Despite the global evidence highlighting this bidirectional relationship, data from Kenyan diabetic populations remain limited.

Objectives: To determine the prevalence of OSA risk status and associated factors among ambulatory T2DM patients attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital (MTRH), Eldoret, Kenya, using the Berlin Questionnaire.

Methods: A hospital-based cross-sectional study was conducted in 334 adult T2DM patients. Participants were systematically sampled and screened for risk of OSA using the Berlin Questionnaire. Data on sociodemographic variables, metabolic markers such as glycated hemoglobin A1C (HbA1c), anthropometric indices such as Body Mass Index (BMI), neck circumference and anatomical factors such as nasal obstruction, and Mallampati score were collected. Bivariate analyses and multivariate logistic regression were performed to identify independent predictors of high OSA risk, with significance set at $p < 0.05$.

Results: The prevalence of OSA risk status was 41.3%, highlighting a substantial burden among patients with type 2 diabetes mellitus. Poor glycemic conrol (HBA1C > 8%) was independently associated with more than a two-fold increase in the odds of high OSA risk, while overweight status conferred a 1.5-fold higher likelihood of OSA risk. Anatomical abnormalities such as deviated nasal septum and nasal polyps, as well as advancing age above 50 years and higher Mallampati scores, were significantly associated with OSA risk. In contrast, hypertension demonstrated an inverse association with OSA risk, suggesting a potential protective effect, although this finding may reflect residual confounding or limitations within the regression

Conclusion: Nearly half of patients with type 2 diabetes mellitus at MTRH were at high risk for obstructive sleep apnoea, with significant associations observed between poor glycemic control, overweight status, nasal obstruction and elevated Mallampati scores. These findings are consistent with international evidence of demonstrating a multifactorial interraction between metabolic and anatomical determinants of OSA risk among populations with type 2 diabetes..

Recommendations: Incorporating routine screening for obstructive sleep apnea (OSA) into standard diabetes care. In addition, further research is required to develop and assess practical, cost-effective and evidence-based strategies for the screening and management of OSA.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Background Information

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by repetitive episodes of complete or partial obstruction of the upper airway during sleep, resulting in intermittent hypoxia, arousals from sleep, and fragmented sleep architecture (American Academy of Sleep Medicine AASM, 2017). These episodes often lead to reduced or absent airflow despite continued respiratory effort, contributing to disrupted sleep and subsequent daytime impairment. The most common symptoms include loud snoring, gasping or choking during sleep, and excessive daytime sleepiness (Di Maria, 2015)

Obstructive Sleep Apnea (OSA) is typically diagnosed using overnight polysomnography, which assesses the apnea-hypopnea index (AHI), an important indicator that quantifies the frequency of apneic and hypopneic events per hour of sleep. An AHI of $\geq$5 events per hour with accompanying symptoms, or an AHI of $\geq$15 events per hour irrespective of symptoms, confirms the diagnosis of obstructive sleep apnea (AASM, 2017).

Sleep-disordered breathing encompasses a spectrum of conditions, including central sleep apnea (CSA), obstructive sleep apnea (OSA), and mixed apneas, although OSA is the most prevalent form in the general population (Franklin & Lindberg, 2015).

When OSA is associated with excessive daytime sleepiness and other functional impairments, it is termed obstructive sleep apnea-hypopnea syndrome (OSAHS) (Patel et al., 2019). Obstructive sleep apnea often leads to intermittent hypoxemia, increased sympathetic activity, and oxyhemoglobin desaturation all of which have implications for metabolic and cardiovascular health (Punjabi, 2008).

The prevalence of OSA increases with age and is particularly higher in men. A landmark study by Young et al. (1993) found that OSA was significantly more common in older males. Following menopause, the prevalence in women also rises, likely due to hormonal changes, resulting in comparable rates between genders in older age groups (Bixler et al., 2001). OSA is typically characterized by upper airway collapse during sleep, especially in the pharyngeal region, leading to airflow limitation (Patrick & Trallo, 2013). Most patients with OSA present with symptoms like excessive daytime sleepiness, and their bed partners often report loud snoring, gasping, choking, snorting, or episodes of breathing cessation (Kapur et al., 2017).

Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition characterized by insulin resistance and impaired glucose metabolism, leading to hyperglycemia (Srinivasan et al., 2018) The global disease burden of both obstructive sleep apnea (OSA) and type 2 diabetes mellitus (T2DM) is escalating, particularly in developing countries such as Kenya, due to urbanization, sedentary lifestyles, and increasing obesity rates (World Health Organization, WHO, 2023).

The International Diabetes Federation reported that 4.8% of people in Africa have diabetes. By 2030, it is predicted that this percentage will increase to 5.3%. This is according to an article by Ayah et al titled "A population-based survey of the prevalence of diabetes in an urban slum community in Nairobi". According to the results, the prevalence ranges from 4.2% to 5.3% (Ayah et al., 2013).

According to the International Diabetes Federation, 90% of the global cases of diabetes mellitus are type 2 diabetes.

OSA affects approximately 9–25% of adults worldwide, and there is strong evidence of a bidirectional relationship between Obstructive sleep apnea and type 2 diabetes mellitus (Peppard et al., 2013; Reutrakul & Mokhlesi, 2017). Patients with Type 2 diabetes mellitus often have an increased likelihood of developing sleep disturbances, including obstructive sleep apnea, which may further worsen glycemic control and contribute to cardiovascular risk (Tasali et al., 2008).

Conversely, disrupted sleep patterns and intermittent hypoxia from OSA are known to impair glucose metabolism, promote insulin resistance, and elevate systemic inflammation (Punjabi, 2004). The common risk factors between OSA and T2DM, specifically obesity, age, and physical inactivity, facilitate their simultaneous occurrence (Reutrakul & Van Cauter, 2014).

However, emerging evidence indicate a more intricate relationship, wherein the pathophysiological mechanisms of one condition may exacerbate the onset or severity of the other. Intermittent hypoxia resulting from obstructive sleep apnea can stimulate sympathetic nervous system pathways and the hypothalamic-pituitary-adrenal axis, leading to the release of diabetogenic hormones including cortisol and catecholamines (Yi-Wen Tsai et al., 2012). These hormonal alterations diminish insulin sensitivity and lead to hyperglycemia.

Sleep deprivation and obstructive sleep apnea can also exacerbate insulin resistance through increased oxidative stress and inflammation. Several studies have confirmed the prevalence of OSA among individuals with type 2 diabetes mellitus and highlighted the metabolic consequences of untreated OSA (Einhorn et al., 2007; Kent et al., 2011). Notably, individuals with OSA but without diabetes often exhibit insulin resistance and impaired glucose tolerance, indicating that OSA may precede or contribute to the development of type 2 diabetes mellitus. (Aronsohn et al., 2010).

In a study conducted by Aronsohn et al. (2010), the severity of OSA was independently associated with poorer glycemic control, as reflected by elevated HbA1c levels. Importantly, this relationship persisted even after adjusting for confounding variables such as BMI, age, race, and gender. These findings underscore the clinical importance of screening for OSA among individuals with T2DM and suggest that management of sleep-disordered breathing may improve metabolic outcomes in this population**.**

Given the increased morbidity and mortality associated with both OSA and T2DM, particularly in relation to cardiovascular complications, their coexistence may result in synergistic health consequences that warrant early identification and integrated management.

The International Diabetes Federation recommends the routine screening of obstructive sleep apnea (OSA) in patients with type 2 diabetes as a means to augment the existing data on the condition (Shaw, Punjabi et al., July 2008)

In addition, a study by Sokwalla et al. (2017) that was conducted in Kenyatta National Hospital, a tertiary referral facility in Kenya, looked at the connection between OSA risk and sleep quality in ambulatory patients with T2DM. The study concluded that patients who had diabetes mellitus were considerably more likely to have sleep disturbances, emphasizing the importance of clinicians screening for OSA in their evaluation of these patients.

Polysomnography (PSG) is considered the gold standard in the diagnosis of OSA.

The method involves simultaneously assessing a number of physiological markers associated with alertness and sleep. The number of respiratory episodes and the resulting hypoxemia that these events result in can be immediately observed and counted.

Nevertheless, the integration of PSG within our existing framework is impeded by various factors, including the need for extensive labor, significant financial costs, and limited accessibility.

Given the aforementioned challenges, it is suggested that those who are at risk for obstructive sleep apnea (OSA) be identified using inexpensive and straightforward methods. An example is the Berlin Questionnaire (BQ).

1.2 Problem Statement

Obstructive Sleep Apnea (OSA) is increasingly recognized as a common but underdiagnosed condition among patients with type 2 diabetes mellitus (T2DM), with growing evidence highlighting its significant impact on metabolic and cardiovascular health outcomes (Yaggi et al.2005)

Undiagnosed and untreated OSA can lead to repeated episodes of nocturnal hypoxia and sleep fragmentation, triggering a cascade of adverse physiological responses including sympathetic nervous system over activity, systemic inflammation, and hormonal dysregulation. These pathophysiological changes are known to contribute to poor glycemic control in individuals with T2DM, thus exacerbating disease progression and increasing the risk of complications (Punjabi & Beamer, 2009)

The coexistence of T2DM and OSA has been linked to a significantly higher risk of macrovascular and microvascular events, such as myocardial infarction, stroke, retinopathy, and nephropathy (Resnick et al., 2003)

The presence of OSA in individuals with diabetes is associated not only with elevated glycated hemoglobin (HbA1c) levels but also with an increased incidence of insulin resistance, hypertension, and dyslipidemia—all of which contribute to the development of diabetic complications and greater morbidity (Aronsohn et al., 2010) Despite the well-documented consequences of OSA in T2DM patients, awareness and screening remain extremely limited, particularly in resource-constrained settings such as Kenya. In many developing countries, including Kenya, sleep disorders remain under-recognized in clinical practice, and OSA is rarely considered in routine diabetes management due to low public awareness, limited diagnostic tools, and a lack of sleep medicine expertise (Sokwalla et al., 2017) This results in a significant number of patients with undiagnosed OSA, further complicating the management of diabetes and increasing the burden on healthcare systems.

Although some studies from high-income countries have demonstrated a high prevalence of OSA risk among individuals with T2DM, there is a noticeable lack of context-specific data from sub-Saharan Africa and Kenya in particular. Most of the existing evidence has been generated from Western populations, and may not adequately reflect the unique sociocultural, genetic, and environmental factors that influence disease presentation and progression in African populations (Omondi et al., 2022; East African Medical Journal, 99)

Furthermore, there is limited empirical data regarding the use of validated screening tools such as the Berlin Questionnaire or STOP-Bang in local healthcare settings. This knowledge gap hinders early identification and timely intervention for patients at risk of OSA, particularly those living with diabetes, and impedes the development of evidence-based screening protocols and health policy reforms tailored to Kenyan contexts.

Considering the significant and escalating prevalence of T2DM in Kenya, along with rising obesity rates and sedentary lifestyles, it is imperative to assess the magnitude of OSA risk in this at-risk population. This study aims to fill the existing information gap by evaluating the prevalence of obstructive sleep apnea risk in patients with type 2 diabetes mellitus at Moi Teaching and Referral Hospital in Eldoret, Kenya. The results will yield significant insights for enhancing patient outcomes by focused screening, clinical awareness, and complete diabetes management regimens.

1.3 Justification

There is a significant lack of evidence regarding the prevalence of obstructive sleep apnea (OSA) risk in Kenya, especially among individuals with type 2 diabetes mellitus (T2DM). Despite global studies emphasizing the significant prevalence of obstructive sleep apnea (OSA) among people with diabetes, sub-Saharan Africa remains significantly inadequate in both research and clinical awareness. The absence of context-specific data restricts healthcare practitioners and policymakers in Kenya from fully understanding the impact of OSA and its effects on diabetes outcomes. The lack of sufficient evidence hinders the development of focused screening guidelines, diagnostic methods, treatment paths, and preventative initiatives for obstructive sleep apnea in patients with diabetes.

OSA is a condition that is frequently underdiagnosed, yet it poses significant risks to individuals with T2DM due to its negative influence on glycemic control and its association with an increased risk of cardiovascular events, hypertension, and poor quality of life. In Kenya, the situation is further compounded by limited access to sleep medicine services, lack of diagnostic equipment such as polysomnography, and insufficient training of healthcare workers on sleep disorders. As such, many patients with T2DM who may also have undiagnosed OSA continue to receive suboptimal care. Determining the magnitude of OSA risk within this population is therefore a critical first step toward improving early detection and timely intervention.

The current study seeks to fill this important knowledge gap by evaluating the prevalence of OSA risk among patients with T2DM attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital (MTRH) in Eldoret, Kenya. As a tertiary referral center, MTRH serves a large and diverse diabetic population from across western Kenya and neighboring regions, making it an ideal site for generating representative data that can inform national health policy and clinical practice. By providing empirical evidence on OSA risk, the study will enhance understanding of the burden of this condition in diabetic patients and support advocacy for the integration of routine OSA screening in diabetes care.

The anticipated findings of this study will contribute valuable insights into the prevalence of OSA risk among individuals with T2DM. This evidence will be essential in raising awareness among healthcare providers about the importance of evaluating sleep disorders in diabetic populations. Ultimately, improved recognition of OSA risk may lead to earlier diagnoses, better clinical outcomes, and reduced rates of diabetes-related complications through more comprehensive and individualized treatment strategies.

Furthermore, by elucidating the factors associated with increased OSA risk in T2DM patients, the study will help to identify subgroups that may benefit most from targeted interventions. Such data can be used to inform the development of context-appropriate screening tools, patient education programs, and multidisciplinary care models that combine sleep medicine with diabetes management.

Notably, this study builds upon the foundational work conducted by Sokwalla et al. (2017), who investigated sleep quality in ambulatory patients with type 2 diabetes in Nairobi, Kenya. Their study concluded with a recommendation for further research into sleep disorders among people with diabetes particularly the assessment of conditions such as OSA. By focusing specifically on the prevalence of OSA risk among patients with diabetes at MTRH, this study extends their work by addressing a critical and understudied aspect of diabetes care in the Kenyan context.

In sum, this study aims not only to generate local data on the prevalence of OSA risk in T2DM patients but also to drive future improvements in diabetes management, inform national health strategies, and ultimately improve health outcomes for a population at high risk of both metabolic and sleep-related complications.

1.4 Research Question

What is the prevalence of OSA risk status, and what factors are associated with OSA risk status among patients with T2DM patients attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital?

1.5 Objectives

1.5.1Broad Objective

To determine the prevalence of OSA risk status among T2DM patients attending the diabetes outpatient clinic in MTRH.

1.5.2 Specific Objectives

1. To determine the prevalence of OSA risk status among patients with T2DM attending the Diabetes outpatient clinic at MTRH.
2. To determine the various factors that are associated with OSA risk among patients with T2DM enrolled in the diabetes clinic at MTRH.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Background

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by repeated episodes of complete or partial obstruction of the upper airway during sleep, leading to reduced or absent airflow despite continued respiratory effort (American Academy of Sleep Medicine (AASM, 2017)

These apneic episodes generally result in repeated oxygen desaturations, disrupted sleep, and variations in intrathoracic pressure, ultimately causing considerable physiological repercussions, including cardiovascular, metabolic, and neurocognitive deficits (Jordan et al., 2014). OSA is frequently characterized clinically by symptoms such as loud snoring, gasping, nocturnal choking, witnessed apneas, and excessive daytime sleepiness (Epstein et al., 2009)

The diagnosis of obstructive sleep apnea (OSA) generally demands polysomnographic confirmation of an apnoea-hypopnea index (AHI) of ≥5 events per hour in symptomatic patients or ≥15 events per hour in asymptomatic patients (AASM, 2017). Sleep-disordered breathing, which includes OSA, central sleep apnea (CSA), and mixed apneas, affects a large proportion of the adult population worldwide and represents a significant public health concern (Franklin & Lindberg, 2015)

When OSA is accompanied by excessive daytime sleepiness and functional impairments, the condition is termed obstructive sleep apnea-hypopnea syndrome (OSAHS), a more clinically severe manifestation of sleep-disordered breathing (Patel et al. 2019). Oxyhemoglobin-desaturation during sleep is a characteristic feature of obstructive sleep apnea (OSA) and leads to sympathetic activation, oxidative stress, and systemic inflammation, all of which may have subsequent metabolic consequences. (Punjabi, 2008)

The prevalence of obstructive sleep apnea (OSA) increases with advancing age and is consistently higher in males than females, with population-based studies estimating prevalence rates ranging from 9% to 38% in the general adult population, with higher rates observed in older individuals and men (Senaratna et al., 2017)

Post-menopause, women exhibit an increased prevalence of obstructive sleep apnea, presumably attributable to hormonal alterations, hence diminishing the gender discrepancy in older demographics (Bixler et al., 2001)

The fundamental anatomical cause of obstructive sleep apnea (OSA) is upper airway collapsibility during sleep, particularly in the pharyngeal segment, which may occur partially or totally based on neuromuscular tone and anatomical predisposition (Patrick & Trallo, 2013). Common symptoms reported by patients include excessive daytime sleepiness, while bed partners often observe loud snoring, snorting, gasping, and apneic pauses (Kapur et al. 2017)

Type 2 diabetes mellitus (T2DM) is a metabolic disorder characterized by insulin resistance and inadequate insulin secretion, leading to hyperglycemia (Srinivasan et al., 2018). Both OSA and T2DM are increasing in prevalence worldwide, especially in low- and middle-income countries (LMICs), due to rapid urbanization, obesity, and sedentary lifestyles (World Health Organization (WHO 2023)

OSA affects approximately 9–38% of the general adult population, and this prevalence increases substantially among individuals with comorbid conditions like obesity and T2DM (Senaratna et al., 2017)

Multiple studies suggest that patients with T2DM have an increased risk of OSA due to shared risk factors and pathophysiological mechanisms, including visceral adiposity and chronic inflammation (Reutrakul & Mokhlesi, 2017)

Sleep fragmentation and intermittent hypoxia in OSA are believed to promote insulin resistance and impair glucose metabolism, contributing to poor glycemic control in T2DM patients (Punjabi, 2004)

One key reason for this overlap is the presence of shared modifiable risk factors such as obesity, which contributes to both insulin resistance and upper airway collapsibility (Kent et al., 2011)

Additionally, there is increasing evidence to suggest a bidirectional relationship between OSA and T2DM, where not only does OSA worsen glycemic control, but poor glycemic control may also predispose to OSA through mechanisms like autonomic neuropathy (Pamidi & Tasali, 2012)

Both conditions independently increase cardiovascular morbidity and mortality, and their coexistence may have synergistic negative effects on patient outcomes (Aronsohn et al., 2010)

Several physiological theories have been proposed to explain how poor sleep quality can affect glucose metabolism, including activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system (Spiegel et al., 1999)

This neuroendocrine activation leads to increased release of cortisol and catecholamines, which are known to impair insulin sensitivity and raise blood glucose levels (Yi-Wen Tsai et al., 2012)

Sleep deprivation or disruption, a hallmark of OSA, increases cortisol concentrations and sympathetic tone, further exacerbating insulin resistance (Tasali & Van Cauter, 2006)

Numerous cross-sectional and cohort studies have identified a strong association between OSA and T2DM, with prevalence estimates suggesting that between 40% and 86% of T2DM patients are at high risk of having undiagnosed OSA (Einhorn et al., 2007)

Even in patients without diabetes, OSA is associated with impaired glucose tolerance and elevated fasting glucose levels, suggesting it may independently contribute to metabolic dysfunction (Kent et al., 2011)

A landmark study by Aronsohn et al. (2010) demonstrated that the severity of OSA, measured by AHI, correlated positively with HbA1c levels in patients with T2DM, even after adjusting for confounders such as BMI, age, and gender (Aronsohn et al., 2010)

This suggests that undiagnosed or untreated OSA may be a critical contributor to suboptimal glycemic control in patients with T2DM and should be actively screened for in clinical practice

In Kenya and much of sub-Saharan Africa, there is an increasing acknowledgment of the burden imposed by non-communicable diseases (NCDs), particularly type 2 diabetes mellitus (T2DM), which shares numerous risk factors with obstructive sleep apnea (OSA). Ministry of Health, Kenya, 2015

Urbanization, dietary changes, escalating sedentary lifestyles, and increasing obesity rates are contributing to a dual burden of disease encompassing metabolic disorders and sleep-related breathing problems such as obstructive sleep apnea (WHO, 2021).

Despite the growing epidemiological association, there is a gap in clinical knowledge, systematic screening, and diagnostic capability for obstructive sleep apnea risk among diabetes people in numerous African healthcare settings (Adewole et al., 2009).

This figure aligns with results from similar African populations. For example, in Nigeria, a hospital-based study using the STOP-Bang questionnaire among T2DM patients found that 47.5% were at high risk of OSA, particularly those with obesity and hypertension (Obaseki et al., 2014)

In South Africa, research among urban black adults identified a high frequency of OSA symptoms such as loud snoring and excessive daytime sleepiness, underscoring the underdiagnosed nature of OSA in African settings (Pienaar et al., 2016)

Yet, due to the limited availability of sleep labs and trained personnel, most hospitals in Kenya including Moi Teaching and Referral Hospital (MTRH) do not routinely perform polysomnography or formal sleep assessments, leading to under recognition of OSA risk)

Given these barriers, screening tools such as the Berlin Questionnaire, STOP-Bang, and the Epworth Sleepiness Scale provide useful and low-cost ways to identify patients at high risk for OSA in resource-limited settings (Netzer et al., 1999)

The rising prevalence of obesity in Kenya has further heightened the urgency to screen for OSA among high-risk groups like T2DM patients. According to the Kenya Stepwise survey, about 28% of adults are overweight or obese risk factors that contribute to both insulin resistance and airway obstruction during sleep (KNBS et al., 2015)

In Eldoret, where Moi Teaching and Referral Hospital is located, rapid urban growth and changes in diet have led to a surge in diabetes-related complications, but data on OSA risk among diabetics remain sparse, highlighting a critical research gap that your thesis addresses (Muchiri et al., 2018)

Physiology of Normal Sleep

Sleep is a complex and vital physiological activity controlled by sophisticated brain mechanisms. It fulfills a restorative role for both the body and brain, essential for energy conservation, memory consolidation, hormonal equilibrium, immunological modulation, and cardiovascular stability (Krause et al., 2017). The structure of sleep is regulated by two fundamental processes: the homeostatic sleep drive (Process S) and the circadian rhythm (process C). The two systems collaborate to control the timing, depth, and duration of sleep.

Sleep Architecture

Human sleep is a non uniform condition that progresses cyclically through several stages, each marked by distinctive physiological, neurological, and electrophysiological alterations. This framework, known as sleep architecture, adheres to a systematic pattern that generally recurs every 90 to 120 minutes during an average 7–8-hour nocturnal period. Each sleep cycle comprises two primary types:

non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep, which alternate in a systematic rhythm throughout the night (Iber et al., 2007).

Non-Rapid Eye Movement (NREM) Sleep

NREM sleep is categorized into three stages: N1, N2, and N3, each indicating a progressively deeper level of sleep. NREM constitutes roughly 75–80% of total sleep duration in healthy persons.

N1 Stage

Also, known as light sleep. It comprises 5-10% of the total sleep. The electroencephalogram characteristics include low-voltage, mixed-frequency oscillations with diminished alpha activity. It is usually the transitional phase between waking and slumber. The heart rate and respiration rate commence a decline.

Muscle activity diminishes, and the eyes rotate gradually.

Clinical significance: Easily interrupted; frequently not recognized as genuine sleep by individuals. The neurological foundation involves diminished activity in the reticular activating system, leading to cortical deactivation (Carskadon & Dement, 2011).

N2 Stage

Intermediate stage sleep of the NREM sleep cycle. Comprises 45-55% of total sleep, which is the largest proportion. EEG features include sleep spindles (12-14 Hz bursts) and K-complexes. The Physiological Changes comprise a further drop in body temperature, heart rate, and respiration rate. There is also reduced sensitivity to external stimuli. Muscle relaxation deepens.

Clinical relevance: Spindles are thought to have a function in memory consolidation and sensory gating (SM Fogel 2011).

Stage N3

This is the Slow-Wave Sleep or Deep Sleep.

Proportion of total sleep is 15–25%. The Electroencephalogram characteristics include pronounced, low-frequency delta waves (0.5–2 Hz).

Physiological alterations include Significant reduction in sympathetic tone, predominated by the parasympathetic activity which includes minimal values of heart rate, blood pressure, respiration rate, and body temperature. The release of growth hormone is at its peak. Sleepwalking and night terrors typically manifest during this stage.

Clinical relevance: The restorative sleep stage is crucial for physical recovery and immunological function. Difficult to wake up from; confusion or sleep inertia may ensue if aroused.

Neurological Basis: Increased thalamocortical synchronization with decreased cortical response to stimuli (Dang-Vu et al., 2008).

Rapid Eye Movement (REM) Sleep

REM sleep comprises roughly 20–25% of total sleep duration and is distributed irregularly throughout the night, being shorter in initial cycles and lengthier in subsequent ones. It is also known as "paradoxical sleep" due to its combination of physiological characteristics of both deep sleep and wakefulness.

EEG characteristics include low-voltage, mixed-frequency waves indicative of wakefulness and desynchronized activity. It is also associated with rapid, conjugate, horizontal ocular movements.

Muscle activity: Significant skeletal muscle atonia (resulting from the inhibition of spinal motor neurons by glycine and GABA).

Autonomic changes include irregularities in heart rate, respiratory rate, and fluctuating blood pressure, elevated cerebral blood flow and oxygen utilization.
In this stage, vivid and emotionally intense dreams are predominant.
REM sleep is crucial for the consolidation of memory, particularly procedural and emotional memory (Walker & Stickgold, 2004). Synaptic pruning and neuroplasticity are also present in this stage

Neurological regulation: Coordinated by cholinergic neurons in the pons, namely the sublaterodorsal nucleus, and influenced by monoaminergic systems (Brown et al., 2012).

Relevance of Sleep Physiology in the Pathogenesis of Obstructive Sleep Apnea

Understanding the physiology of normal sleep provides a crucial foundation for appreciating the pathogenesis of obstructive sleep apnea (OSA). While sleep is intended to promote rest and systemic restoration, certain inherent physiological changes during sleep particularly those affecting respiratory control and upper airway patency can paradoxically predispose susceptible individuals to obstructive events. OSA occurs when these normal physiological processes are exaggerated or when structural vulnerabilities compromise airway stability during sleep.

REM sleep is notably linked to the highest incidence and severity of apneic occurrences. The significant atonia present during this period affects most upper airway dilator muscles, rendering the pharyngeal airway particularly susceptible to collapse (Malhotra & White, 2002). The inconsistency in respiratory drive and autonomic instability during REM sleep exacerbates this risk. Consequently, people with OSA frequently have a concentration of apneas during REM phases, resulting in disrupted sleep and increased cardiovascular burden (Jordan et al. 2014).

Reduced Chemoreflex Sensitivity and Ventilatory Drive

Normal sleep is characterized by diminished response to hypercapnia and hypoxia, resulting from reduced activity in central and peripheral chemoreceptors (T Douglas et al., 1982). In healthy persons, this drop has little effects. However, in patients with obstructive sleep apnea, decreased ventilatory drive during sleep restricts the capacity to restore airflow if obstruction arises. This may extend the length of apneas and exacerbate desaturation episodes. Moreover, persistent hypoxia due to recurrent apneic episodes can recalibrate chemoreceptor sensitivity over time, resulting in erratic breathing patterns and elevated loop gain, a phenomenon where minor alterations in ventilation induce significant fluctuations in respiratory output, thereby sustaining apnea–hyperpnea cycles (Wellman et al., 2013).

Arousal Threshold and Sleep Fragmentation

Arousal from sleep serves as a compensatory mechanism that terminates apneic episodes, facilitating the reopening of the airway. The arousal threshold differs among individuals and sleep stages, with deeper stages, particularly N3, necessitating more intense respiratory stimulation to induce wakefulness (Berry et al. 2019). In patients with obstructive sleep apnea, recurrent arousals reestablish airflow but lead to sleep fragmentation, hindering advancement to restorative sleep stages.

This cycle leads to significant daytime drowsiness, cognitive impairment, and disrupted neuroendocrine regulation. In certain individuals, a diminished arousal threshold may lead to premature awakenings prior to complete airway compensation, rendering treatment modalities such as CPAP less tolerable and underscoring the necessity for personalized therapy (DJ Eckert et al.2013).

Sleep-Induced Hormonal and Metabolic Effects

The interaction between sleep physiology and metabolic regulation is crucial to the pathophysiology of OSA. Sleep loss or disturbance, as seen in OSA, results in elevated evening cortisol levels, increased sympathetic activation, insulin resistance, and dysregulation of appetite hormones such as leptin and ghrelin (K Spiegel et al. 2004). These hormonal abnormalities facilitate weight gain, especially visceral obesity, a significant risk factor for obstructive sleep apnea, so creating a detrimental cycle between disrupted sleep and metabolic dysfunction.

In conclusion, the features that describe normal sleep, including muscle atonia, diminished respiratory drive, stage-specific physiological alterations, and modified neurohormonal patterns, can predispose individuals to obstructive sleep apnea when combined with anatomical or neuromuscular abnormalities. The pathophysiology of OSA is fundamentally linked to sleep physiology, necessitating a comprehensive understanding of these mechanisms for the formulation of effective screening, risk stratification, and personalized treatment approaches.

PATHOPHYSIOLOGY OF OBSTRUCTIVE SLEEP APNEA SYNDROME

The pathogenesis of obstructive sleep apnea (OSA) is complex and multifaceted, fundamentally characterized by the upper airway's inability to maintain patency during sleep.

The fundamental abnormality involves the inability of the pharyngeal dilator muscles particularly the genioglossus and other muscles of the upper airway—to resist the negative intraluminal pressure generated during inspiration.

Under typical physiological settings, these dilatory muscles are recruited in a precisely coordinated manner with each respiratory cycle. Their contraction stabilizes the airway, therefore preventing collapse as negative pressure is created in the upper airway during the inspiratory phase of breathing (Deegan & McNicholas, 1995).

Any disruption in this delicate neuromuscular equilibrium, whether due to decreased muscle responsiveness, weakened neuronal drive during sleep, or heightened negative inspiratory pressure can result in partial or total upper airway collapse. Such episodes lead to temporary hypopneas or apneas, which are defining characteristics of obstructive sleep apnea (OSA).

The vulnerability of the airway is particularly increased during rapid eye movement (REM) sleep, a phase characterized by a natural reduction in muscular tone, which further intensifies the likelihood of obstruction.

The narrowing of the upper airway, a central contributor to this pathophysiology, can arise from several anatomical and physiological factors. These include fixed structural elements, such as craniofacial skeletal anomalies, like a retrognathic mandible or a narrow maxillary arch, which inherently reduce the airway caliber. In addition, soft tissue enlargement or encroachment can also reduce airway patency. In obese individuals, for instance, fat deposition around the pharyngeal walls, tongue, and soft palate significantly decreases the cross-sectional area of the airway, increasing its collapsibility during inspiration.

Other anatomical contributors include adenotonsillar hypertrophy, particularly relevant in the pediatric population but still observed in adults. Moreover, transient factors such as fluid shifts during sleep may exacerbate upper airway narrowing. When a person lies in a recumbent position, fluid accumulated in the lower extremities during the day can redistribute to the neck and upper body at night. This rostral fluid shift increases tissue pressure around the upper airway and may exacerbate its tendency to collapse, especially in individuals with predisposing anatomical traits or comorbidities such as heart failure or nephrotic syndrome.

In the Kenyan context, where obesity, hypertension, and other metabolic risk factors are rising among individuals with type 2 diabetes mellitus the likelihood of encountering such airway-narrowing mechanisms is high. Additionally, diagnostic limitations such as the unavailability of sleep studies and lack of clinical suspicion may delay appropriate interventions in such patients. Understanding this pathophysiology is therefore essential in evaluating and managing OSA risk in diabetic populations, especially within resource-limited healthcare settings such as Moi Teaching and Referral Hospital (MTRH), Eldoret.

Upper Airway Anatomy and Vulnerability

The upper airway extends from the nasal cavity and oral cavity down to the larynx and comprises several important anatomical structures. These include the soft palate, tongue, uvula, tonsils, lateral pharyngeal walls, and various muscles responsible for maintaining airway tone. In healthy individuals, these structures remain open during sleep due to continuous neuromuscular activity. However, in people predisposed to OSA, these tissues may be enlarged, relaxed, or poorly supported by surrounding muscles and bones, leading to narrowing or collapse during inspiration (Malhotra & White, 2002).

Key anatomical risk factors for airway narrowing include hypertrophied tonsils, a low-positioned soft palate, retrognathia (posteriorly set jaw), and increased fat deposition around the neck and tongue. These features are common in individuals who are obese, a significant portion of whom also have T2DM. In the Kenyan context, rising obesity rates have contributed to an increased burden of non-communicable diseases such as diabetes and potentially OSA, especially in urban and semi-urban areas such as Eldoret.

Airway Obstruction During Sleep

During sleep, there is a natural reduction in the tone of skeletal muscles, including those that maintain the patency of the upper airway. In individuals with OSA, this normal relaxation is exaggerated due to anatomical and neuromuscular factors. As the pharyngeal muscles relax, the already narrowed airway becomes further compromised, leading to partial obstruction (hypopnea) or complete blockage (apnea). These episodes are more likely to occur during rapid eye movement (REM) sleep, where muscle tone is lowest, and the respiratory control system is less responsive to changes in oxygen and carbon dioxide levels (Jordan et al. 2014)

CONSEQUENCES OF OBSTRUCTION

Hypoxia and Hypercapnia

When the airway collapses, airflow into the lungs is significantly reduced or completely stopped, resulting in a drop in oxygen saturation (hypoxemia) and a buildup of carbon dioxide (hypercapnia). This imbalance triggers several compensatory mechanisms, including increased sympathetic nervous system activity, which attempts to restore normal breathing. In T2DM patients, these repeated episodes of oxygen desaturation may exacerbate insulin resistance and promote a pro-inflammatory state, worsening metabolic control (Reutrakul & Van Cauter, 2014)

In individuals with diabetes, these hypoxic episodes may further impair pancreatic beta-cell function and reduce insulin sensitivity. Studies have shown that intermittent hypoxia, even in the absence of obesity, can lead to glucose intolerance by disrupting glucose uptake and impairing insulin signaling pathways (Punjabi & Polotsky, 2005)

Arousal Response and Sleep Fragmentation

In response to low oxygen and high carbon dioxide levels, chemoreceptors in the brainstem activate an arousal mechanism. This process causes the individual to briefly awaken or transition into lighter stages of sleep, which reactivates the upper airway muscles and reopens the airway. These arousals are often accompanied by noticeable symptoms such as loud snoring, choking, gasping, or snorting. However, most patients do not remember these events, which may happen dozens or even hundreds of times per night (Malhotra & White, 2002).

Repeated arousals severely disrupt sleep continuity and architecture. Individuals with OSA are unable to maintain prolonged periods of deep, restorative sleep stages, including slow-wave sleep and REM sleep. The resulting sleep fragmentation leads to daytime symptoms such as excessive sleepiness, poor concentration, memory lapses, and mood disturbances. These impairments can reduce the ability of T2DM patients to adhere to medication regimens, maintain lifestyle changes, or manage glucose levels effectively (Tasali et al., 2008)

Systemic Effects of Chronic Sleep Disruption and Hypoxia

The effects of untreated OSA go beyond poor sleep and fatigue. The condition exerts far-reaching consequences on multiple body systems through a combination of chronic intermittent hypoxia, oxidative stress, inflammation, and sympathetic over activity. These changes contribute to endothelial dysfunction, hypertension, cardiac arrhythmias, and an increased risk of coronary artery disease and stroke. All these complications are already prevalent among patients with T2DM, and OSA may accelerate their progression (Somers et al., 2008)

From a metabolic perspective, OSA promotes insulin resistance through activation of the hypothalamic-pituitary-adrenal axis, which increases cortisol levels. Elevated cortisol worsens glucose metabolism and raises fasting glucose levels. Studies have shown that patients with untreated OSA have higher HbA1c levels compared to patients with diabetes without sleep-disordered breathing, even when BMI is similar (Aronsohn et al. 2010)

Moreover, patients with OSA have increased levels of inflammatory markers such as interleukin-6 and tumor necrosis factor-alpha, which are also elevated in poorly controlled diabetes. The synergistic effect of chronic inflammation from both conditions may accelerate microvascular and macrovascular complications such as nephropathy, retinopathy, neuropathy, and cardiovascular disease (Punjabi & Polotsky, 2005)

At Moi Teaching and Referral Hospital (MTRH) in Eldoret, Kenya, the lack of routine screening for OSA in diabetes clinics contributes to under-diagnosis. The symptoms of OSA, such as daytime fatigue or poor concentration, are often attributed to poorly controlled diabetes itself rather than underlying sleep-disordered breathing. Additionally, sleep medicine is still an emerging field in most low- and middle-income countries, with limited access to diagnostic tools such as polysomnography. Therefore, understanding the pathophysiology of OSA and its overlap with diabetes is crucial in helping clinicians suspect and address it even in resource-limited settings.

Using validated screening tools such as the Berlin Questionnaire or STOP-BANG can aid in identifying high-risk individuals. These tools do not require advanced equipment and can be easily integrated into routine outpatient care. Early identification and intervention through lifestyle modification, weight management, glucose control, and where possible, continuous positive airway pressure (CPAP), can greatly reduce complications and improve patient outcomes.

Systemic Effects

OSA exerts extensive impacts on multiple body systems as a result of chronic hypoxia, fragmentation of sleep by activating the sympathetic component of the autonomic nervous system.

The aforementioned effects have the potential to augment the likelihood of developing conditions such as hypertension, coronary syndromes and cerebrovascular accidents, metabolic disturbances (such as insulin resistance and diabetes), cognitive impairments, daytime somnolence, mood disorders, and an overall decline in quality of life.

It is noteworthy to acknowledge that in addition to obstructive factors, there exists a distinct form of sleep apnea known as central sleep apnea that is characterized by a deficiency in respiratory effort resulting from a malfunction in the brain's respiratory control center. Nevertheless, OSA is the more commonly observed variant, with its pathophysiology primarily centered on airway obstruction.

Central Sleep Apnea vs. Obstructive Sleep Apnea

Central sleep apnea (CSA) and obstructive sleep apnea (OSA) are the two primary types of sleep apnea. While they share comparable symptoms such as disturbed sleep, daily weariness, and snoring, their underlying causes, pathophysiology, and therapeutic modalities differ greatly.

Central Sleep Apnea (CSA)

When a person has central sleep apnea, their breathing repeatedly stops and starts while they are asleep. This condition is caused by the brain's inability to send the right signals to the respiratory muscles rather than a physical obstruction in the airway. In CSA, the issue lies in the central nervous system's respiratory control centers, primarily in the brainstem, which intermittently fail to initiate respiratory effort during sleep (Dempsey et al., 2010)

Compared to OSA, this type of sleep apnea is less frequent and typically linked to certain underlying medical disorders. For instance, CSA frequently occurs in patients with congestive heart failure, stroke, renal failure, or certain neurological diseases. It can also be triggered by the use of opioid medications or may be seen in high-altitude environments, where oxygen levels are reduced (Badr et al., 2022). In some cases, CSA is idiopathic, meaning no apparent etiology is established.

During a central apnea episode, there is no respiratory effort, and both the airflow and the movement of the chest and abdominal muscles are absent. This is in contrast to OSA, where respiratory effort is present, but airflow is blocked due to physical airway collapse. Because of the different mechanisms involved, patients with CSA often do not exhibit the loud snoring, choking, or gasping typically associated with OSA. Instead, they may experience silent pauses in breathing, which may only be observed by a bed partner or identified during sleep studies (Dempsey et al., 2010)

OSA is diagnosed using polysomnography (PSG), where central apneas are identified by the lack of both airflow and effort. Management depends on the underlying cause. For example, optimizing treatment for heart failure or adjusting medications can alleviate CSA symptoms. Continuous positive airway pressure (CPAP) or adaptive servo-ventilation (ASV) devices may be used in certain situations, though their efficacy varies based on the clinical setting (Badr et al., 2022).

Comparison with Obstructive Sleep Apnea (OSA)

While CSA is caused by neurological dysregulation, Obstructive Sleep Apnea (OSA) arises from physical obstructionof the upper airway during sleep. In OSA, the brain continues to send signals to breathe, but the airway becomes blocked, typically due to muscle relaxation and anatomical narrowing of the pharyngeal structures. Unlike CSA, individuals with OSA show persistent respiratory effort during apneic episodes, but airflow is reduced or absent due to obstruction (Malhotra & White, 2002

Another key difference lies in symptom presentation. OSA is often accompanied by loud snoring, choking, gasping, and noisy breathing, while CSA tends to be quieter. Despite these distinctions, both diseases can induce disturbed sleep, daytime drowsiness, exhaustion, and increased cardiovascular risk. However, OSA is far more common and is particularly relevant in the context of type 2 diabetes, obesity, and metabolic syndrome, all of which are risk factors for upper airway collapse (Punjabi

& Polotsky, 2005)

Due to restricted access to sleep studies and diagnostic tools, it can be difficult to differentiate between CSA and OSA in resource-constrained countries like Kenya. Nevertheless, OSA remains the primary focus of concern given its strong association with modifiable risk factors and higher prevalence in patients with type 2 diabetes. Additionally, the STOP-BANG and Berlin Questionnaires, commonly used to screen for sleep apnea risk in outpatient settings, are more sensitive to detecting OSA than CSA (Chung et al., 2008)

In conclusion, whereas both OSA and CSA are types of sleep-disordered breathing, their pathogenesis and therapeutic approaches are very different. CSA involves a failure of the brain's respiratory signaling and is generally associated with cardiac or neurological disorders, while OSA is related to mechanical obstruction of the upper airway, most commonly associated with obesity and diabetes. In the context of this study at MTRH in Eldoret, the emphasis is placed on OSA risk, given its greater prevalence and more direct link to type 2 diabetes in the Kenyan population.

2.2 The Prevalence of Obstructive Sleep Apnoea

Obstructive sleep apnoea (OSA) is globally regarded as the most prevalent form of sleep-disordered breathing. It is characterized by repeated episodes of partial or complete blockage of the upper airway during sleep, leading to disrupted sleep, intermittent low oxygen levels, and a broad spectrum of health consequences. In 2019, Benjafield et al. estimated that approximately 936 million adults aged 30–69 worldwide experience at least mild OSA (AHI ≥ 5), with around 425 million suffering from moderate-to-severe forms (AHI ≥ 15) (Benjafield et al., 2019) This underlines OSA’s significance as a global health priority.

A robust body of epidemiological work in developed countries including Australia, China, the United States, India, South Korea, and Spain, offers insights into OSA prevalence determined through polysomnography. Sharma et al. (2006) reported prevalence rates ranging from 3% to 19.7% for adult males and 2% to 7.4% for adult females. These disparities across genders are attributed to differences in upper-airway anatomy, hormonal influences, and fat distribution.

Global and Regional Prevalence of OSA Risk in T2DM

The risk of obstructive sleep apnea (OSA) in patients with type 2 diabetes mellitus (T2DM) has garnered heightened global attention due to an increasing prevalence of both illnesses and their bidirectional link. The global prevalence of Type 2 Diabetes Mellitus (T2DM) is escalating rapidly, with around 537 million persons impacted as of 2021, and predictions indicate this figure may rise to 783 million by 2045 (International Diabetes Federation, IDF, 2021).

Obstructive Sleep Apnea (OSA) is currently acknowledged as one of the most common chronic sleep disorders globally, impacting between 9% to 38% of the adult population, with considerable variation influenced by age, sex, obesity, and diagnostic techniques (Senaratna et al., 2017).

When T2DM and OSA coexist, the public health implications are profound, as both conditions independently contribute to increased cardiovascular risk, and their co-occurrence may have synergistic adverse outcomes (Reutrakul & Mokhlesi, 2017)

Globally, multiple studies using validated screening tools have documented a high prevalence of OSA risk among individuals with T2DM. In the United States, studies using the Berlin Questionnaire and STOP-Bang have shown that approximately 58% to 86% of patients with T2DM are at high risk for OSA (Einhorn et al., 2007)

A cross-sectional study conducted by Foster et al. (2009) utilizing polysomnography in a large U.S. cohort revealed that 86% of obese individuals with type 2 diabetes mellitus were undiagnosed with obstructive sleep apnea syndrome, and 33% exhibited severe obstructive sleep apnea (Foster et al., 2009).

A study conducted in Canada involving 300 patients with T2DM, utilizing the Berlin Questionnaire, indicated that 47.7% were at elevated risk for OSA, highlighting the necessity for regular OSA screening in diabetic clinics (Mokhlesi et al., 2011).

Comparable prevalence rates have been recorded in Europe. In Germany, Ficker et al. (1998) discovered that 69% of patients with T2DM were diagnosed with OSA via polysomnography, and the severity of OSA exhibited a positive correlation with inadequate glycemic management (Ficker et al., 1998).

In the United Kingdom, West et al. (2006) conducted a sleep study on patients with T2DM and discovered that 48% exhibited moderate to severe OSA, hence underscoring the necessity of OSA screening in diabetic populations (West et al., 2006).

The prevalence of obstructive sleep apnea risk among individuals with diabetes in Asia is notably elevated. A comprehensive study conducted in China by Zhang et al. (2013) utilizing the Berlin Questionnaire revealed that 57.4% of patients with T2DM were at an increased risk for OSA (Zhang et al., 2013).

In a comparable manner, a study that was carried out in India by Surani et al. (2015) discovered that 66 percent of diabetes patients were at a high risk for obstructive sleep apnea (OSA) based on the results of the STOP-Bang and Berlin Questionnaire (Surani et al., 2015).

These high rates of OSA risk in Asia can be partially attributed to the high burden of central obesity and the increasing prevalence of metabolic syndrome in the region, both of which are strong risk factors for OSA (Lam et al., 2010)

In Latin America, studies also show a substantial burden of OSA risk in T2DM populations. For instance, in Brazil, Lorenzi-Filho et al. (2002) reported that approximately 60% of obese individuals with T2DM had moderate to severe OSA (Lorenzi-Filho et al., 2002)

There is a common pathophysiological link between type 2 diabetes and obstructive sleep apnea, as indicated by these data from a variety of countries, which highlight the consistency of the association between the two conditions, independent of regional or ethnic variances.

In Africa, however, the data on the prevalence of OSA risk among individuals with T2DM is still emerging and relatively sparse. A study by Worku et al in Ethiopia, found that patients in Africa who have type 2 diabetes mellitus have a pooled prevalence of obstructive sleep apnea risk of 41.13 percent. The research underscores the necessity of incorporating obstructive sleep apnea screening and assessment protocols into standard follow-up therapy for individuals with type 2 diabetes mellitus (Worku et al, 2024)

Abdissa et al. identified a prevalence of obstructive sleep apnoea risk of 45.4% among type 2 diabetes mellitus patients at Jimma Medical Centre in southwestern Ethiopia and recommended early detection and appropriate treatments for this high-risk group.

A comparative cross-sectional study conducted in southern Ethiopia in 2021 identified a high prevalence of obstructive sleep apnea (OSA) in patients with type 2 diabetes mellitus (T2DM) and recommended early screening for OSA during follow-up to facilitate preventive measures and prompt treatment. (Alemayehu et al, 2022)

A study conducted by Umoh et al. in Nigeria investigated the prevalence of obstructive sleep apnea (OSA) risk among patients with type 2 diabetes mellitus (T2DM) using the Berlin Questionnaire, revealing a high OSA risk of 49.5%. The study recommended that T2DM patients undergo routine evaluation for OSA risk. (Umoh et al.,2020)

Embarak et al. (2019) examined the correlation between obstructive sleep apnea (OSA) and diabetic retinopathy at Zagazig University in Egypt, revealing that 60% of patients with type 2 diabetes mellitus (T2DM) had OSA. They further established that OSA was independently linked to diabetic retinopathy and maculopathy. (Embarak at el., 2019)

Sweed et al. examined obstructive sleep apnea in patients with type 2 diabetes mellitus among the Egyptian population and determined the prevalence to be 78%. Of these, 86.8% exhibited inadequate glycemic control. They determined that patients with type 2 diabetes mellitus exhibited a greater prevalence of obstructive sleep apnea, particularly among those with poorly managed blood glucose levels.

In Nigeria, Obaseki et al. (2014) assessed 200 patients with T2DM using the STOP-Bang questionnaire and found that 47.5% were at high risk for OSA (Obaseki et al., 2014)

A study conducted in Benin examining the prevalence of obstructive sleep apnea (OSA) and sleep quality among adult patients with type 2 diabetes mellitus (T2DM) revealed a significantly elevated risk for OSA and poor sleep quality. The recommendation emphasizes need for improved systematic screening of obstructive sleep apnea in individuals with type 2 diabetes mellitus.

In Kenya, Sokwalla et al. (2017) looked at the prevalence of poor sleep quality and risk of OSA utilizing the Pittsburgh Sleep Quality Index and the Berlin Questionnaire, respectively. The prevalence of OSA risk was 44%. They indicated that there is a significant prevalence of sleep disturbances in patients with type 2 diabetes mellitus and urged practitioners to screen for obstructive sleep apnea in this cohort.

Due to the limited availability of formal sleep studies, such as polysomnography, in numerous African healthcare settings, validated questionnaires like the Berlin Questionnaire, STOP-Bang, and Epworth Sleepiness Scale are essential instruments for assessing the prevalence of obstructive sleep apnea risk in vulnerable populations, including individuals with type 2 diabetes mellitus (Netzer et al., 1999).

Despite the expanding international literature, the African continent, including Kenya, remains short in comprehensive population-level data regarding the prevalence of obstructive sleep apnea risk among patients with type 2 diabetes mellitus, highlighting the necessity of localized research such as the present study at Moi Teaching and Referral Hospital (MTRH).

This work addresses a significant gap by supplying foundational data applicable for public health planning, clinical awareness, and comprehensive screening techniques for obstructive sleep apnea risk in diabetic populations in Kenya.

To build upon this evidence, the current study investigates obstructive sleep apnea risk prevalence among type 2 diabetes mellitus patients attending the diabetic outpatient clinic at Moi Teaching and Referral Hospital (MTRH), Eldoret, Kenya. Conducting this research in Kenya’s second-largest referral hospital allows comparison with earlier findings from KNH, enhancing understanding of regional variations.

Obesity is a well-established risk factor, with studies across different populations consistently showing that increased body mass index significantly elevates OSA risk. However, racial and ethnic variations exist independent of weight. In the U.S., Redline et al. (1997) reported that African Americans younger than age 35 exhibited higher OSA prevalence compared to White Americans, even after adjusting for BMI, suggesting genetic or structural predisposition.

Asian cohorts present another important case. Despite having lower obesity rates than Western populations, numerous studies including both community-based work in India (Sharma et al., 2006; Reddy et al., 2009) and research in South Korea ((Sunwoo JS, Hwangbo Y, et al.) have found similarly high OSA prevalence. This supports theories that smaller craniofacial dimensions, rather than obesity per se, may predispose Asians to OSA.

In summary, the global prevalence of OSA is alarmingly high, affecting nearly one in four adults worldwide, with even greater risks among individuals with T2DM. Regional and racial variations further complicate the epidemiological landscape. By focusing on patients at MTRH, this study aims to contribute invaluable data to the evidence base from sub-Saharan Africa, and to inform improved clinical screening and management practices across diverse Kenyan contexts.

2.3 Factors Associated with Obstructive Sleep Apnoea

To gain a thorough and nuanced understanding of the risk factors that contribute to the development and progression of obstructive sleep apnea (OSA), it is essential for both researchers and clinicians to focus their diagnostic evaluations and screening efforts on individuals who are most vulnerable to the condition. This approach allows for more targeted intervention, improves early detection, and ensures that limited resources especially in resource-constrained settings are optimally utilized to identify high-risk groups before complications arise.

In a pivotal study conducted by Muxfeldt et al. (2014), several demographic, metabolic, and physiological parameters were independently found to be significantly associated with moderate to severe forms of OSA. The study population included 422 adult patients who were being followed up for resistant hypertension a condition that itself is frequently comorbid with OSA.

Through comprehensive overnight monitoring and multivariate regression analysis, the researchers identified a range of predictive factors for moderate to severe OSA, defined by an apnea-hypopnea index (AHI) of ≥15 events per hour. These included male sex, advancing age, the presence of diabetes mellitus, general obesity as measured by body mass index (BMI), central adiposity indicated by increased waist circumference, and neck circumference all of which are indicators of upper airway compromise. In addition, higher nocturnal systolic blood pressure values were also observed to be closely linked with increased OSA severity.

Among the participants in the study, a significant 234 individuals representing approximately 55.5% of the total sample, were diagnosed with moderate to severe OSA based on objective sleep study findings. This high prevalence highlights the intricate interplay between metabolic disorders, cardiovascular dysfunction, and sleep-disordered breathing. The inclusion of resistant hypertensive patients further underlines the bidirectional relationship between poorly controlled blood pressure and the presence of untreated OSA. This study not only reinforces the importance of integrated care models that link cardiology, endocrinology, and sleep medicine but also serves as a reminder that high-risk groups, such as patients with T2DM and hypertension, warrant routine OSA screening.

Furthermore, the findings by Muxfeldt et al. support the growing consensus that clinicians should incorporate anthropometric markers like neck circumference and waist circumference into routine clinical assessments, especially in populations at risk of cardiometabolic diseases. These simple bedside measurements have shown strong predictive value in identifying individuals who are more likely to suffer from upper airway obstruction during sleep, and thus, should be considered essential tools in both primary care and specialty settings.

Given the overlap in risk profiles between patients with type 2 diabetes mellitus and those with resistant hypertension, the relevance of these findings extends directly to diabetic clinics and general medical practice.

Identifying such risk factors in outpatient settings such as MTRH can greatly improve early diagnosis, leading to timely intervention and potentially reducing the burden of OSA-related complications including cardiovascular morbidity, poor glycemic control, and diminished quality of life.

2.3.1 Sex

A number of research have repeatedly produced persuasive evidence demonstrating that males have a much greater sensitivity to obstructive sleep apnea (OSA) compared to females. Clinical and epidemiological investigations in a variety of populations have provided ample evidence of this gender-based discrepancy in prevalence. The reported male-to-female ratio in OSA diagnoses ranges from 5:1 to as high as 8:1, according to Punjabi (2008), indicating a considerable predominance of the illness among men.

thus validating this gender difference, Sharma (2006) observed in a population-based study that men were roughly ten times more likely than women to acquire OSA, thus underlining the disproportionate burden of the condition among males. These changes are not only coincidental but are believed to arise from a complex interplay of anatomical, physiological, hormonal, and behavioral factors (Sharma, 2006)

One explanation for this differential is that OSA may be systemically under-recognized and underdiagnosed in women due to differences in symptom presentation. Lin, Davidson, and Ancoli-Israel (2008) argued that the clinical presentation of OSA in females may differ considerably from that in males, which could result in delayed diagnosis or misattribution of symptoms to other illnesses. Women are more likely to report nonspecific problems including exhaustion, morning headaches, mood swings, insomnia, and general malaise than men, who often describe hallmark symptoms such as loud snoring, gasping, or observed apneas (Lin et al., 2008). These discrepancies in symptomatology may lead healthcare providers to underappreciate the risk of OSA in females, particularly in the absence of typical symptoms.

Young, Skatrud, and Peppard (2004) indicated that this delay in diagnosis may partially account for the potentially lower survival rates reported in women with OSA.

Because women often present later in the disease course, or with atypical symptoms, they may not receive timely interventions such as continuous positive airway pressure (CPAP) therapy, resulting in more advanced disease and worse outcomes (Young et al., 2004)

Behavioral and cultural patterns in symptom reporting may be another factor contributing to the observed gender disparity in diagnosis. Studies have found that men are more likely to report symptoms such snorting, gasping, and choking during sleep, which are commonly recognized indications of OSA, while women may be less forthcoming with such information or may not see similar symptoms as problematic. As a result, there exists a large gap in symptom recording between the sexes, which can prevent correct and prompt diagnosis of OSA in females (Punjabi, 2008)

According to Xie et al. (2011), women with OSA are more likely than men to self-report symptoms such generalized body weakness, extreme weariness, and loss of energy. Despite being clinically significant, these symptoms are frequently non-specific and, if not taken into account in the context of sleep-disordered breathing, may result in an underdiagnosis or incorrect diagnose (Xie et al., 2011).

Moreover, the perception and response of bed partners to sleep disturbances may play a subtle but important role in the detection of OSA. According to Young et al. (1993), bed mates who typically function as crucial observers of nocturnal symptoms may reveal gender-based disparities in the observation and reporting of disturbed breathing. In particular, female bed companions may be more sensitive to snoring and episodes of apnea in their male partners, necessitating medical assessment. Conversely, a lower percentage of clinical referrals for suspected OSA in females may result from male partners' lack of awareness of or concern for identical symptoms in women (Young et al., 1993).

Lastly, there is a persistent assumption among both patients and professionals that OSA is largely a male disorder. This common assumption may unwittingly lower the index of suspicion for OSA in females, resulting to diagnostic overlook. Consequently, healthcare providers may more quickly consider and investigate sleep-disordered breathing in men, while attributing identical symptoms in women to alternative diagnoses such as melancholy, anxiety, or chronic exhaustion (Punjabi, 2008) This gender bias in diagnosis methods further adds to the underrepresentation of women in OSA statistics and may have substantial ramifications for clinical care and outcomes.

In summary, the male gender remains a well-established risk factor for obstructive sleep apnea, supported by a number of studies and clinical findings. However, diagnostic biases, variations in symptom manifestation, and socio-behavioral factors that influence the disorder's detection and reporting may also contribute to the apparent lower prevalence among women rather than a genuine biological difference.

2.3.2 Age

The occurrence of sleep-related diseases, particularly obstructive sleep apnea (OSA), seems to increase steadily with increasing age. Advancing age has been significantly connected to alterations in sleep architecture, which may appear as problems in beginning sleep, increased frequency and duration of nocturnal awakenings, fragmented sleep, and a reduced proportion of restorative deep sleep (Reddy et al., 2009). These age-related abnormalities in sleep quality are compounded by physiological changes in upper airway patency and respiratory control mechanisms, making older persons particularly vulnerable to sleep-disordered breathing such as OSA.

Epidemiological studies reveal that more than 50% of adults aged 65 years and older exhibit chronic sleep-related issues, underlining the heightened sensitivity of this population to illnesses such as OSA (Punjabi et al., 2008) Multiple large-scale cohort studies have consistently showed that individuals aged 65 and beyond have a two- to three-fold greater risk of developing OSA compared to those in younger age ranges, notably those aged 30 to 64 years (Young et al., 2004)

This association between aging and increased OSA prevalence is believed to be complex, impacted by anatomical, neuromuscular, and metabolic changes that normally occur with age.

One of the primary studies addressing this relationship, conducted across many counties in Pennsylvania, indicated that the prevalence of OSA in men grew gradually with age. According to Bixler et al. (2001), the prevalence of OSA in males was 3.2% in the 20–44 age group, 11.3% in the 45–64 age group, and 18.1% in the 61–100 age group. Though at a slightly lower degree, female cohorts also showed same pattern of rising incidence with age.

Several anatomical and physiological processes have been postulated to explain why OSA becomes increasingly frequent with advancing age. One commonly recognized concept refers to age-related anatomical changes in the upper airway, such as elongation of the soft palate and greater flexibility in pharyngeal tissues. These alterations may lead to heightened airway collapsibility during sleep, hence favoring the initiation and progression of obstructive episodes (Malhotra & White, 2002) Furthermore, increased adipose tissue deposition in the parapharyngeal area is frequently associated with aging, which further narrows the upper airway and increases resistance to airflow during sleep (White, 2005).

Neuromuscular control of the upper airway also appears to diminish with age, lowering the ability of pharyngeal dilator muscles to maintain airway patency during sleep, especially during the rapid eye movement (REM) phase (Mezzanotte et al., 1992) Furthermore, aging is associated with a slowed ventilatory response to hypoxia and hypercapnia, which could impede arousal responses necessary to terminate apneic episodes (Skatrud et al., 2000). The increased frequency and intensity of apneas and hypopneas in older people are caused by these physiological declines taken together.

Interestingly, despite the rising body of evidence linking age to increased OSA risk, there remains considerable disagreement regarding the clinical presentation of the illness in older persons compared to younger or middle-aged individuals. While some research indicates that older individuals may be less sleepy during the day, other studies show that older adults have a comparable or even higher burden of symptoms such mood swings, morning headaches, and cognitive deterioration (Young et al. 2004).

However, this area remains underexplored, and greater study is necessary to identify whether the clinical aspects of OSA genuinely differ with age, or whether age-related cognitive changes complicate symptom reporting and interpretation.

Another problem is that older persons may present with various comorbid illnesses such as cardiovascular disease, diabetes mellitus, and neurodegenerative disorders that can conceal or mimic the symptoms of OSA. This overlap complicates the diagnosis and management of OSA in the elderly and may contribute to under-recognition in clinical settings. Moreover, age-related alterations in sleep patterns, such as increased napping or changed circadian rhythms, may also disguise the characteristic presentation of excessive daytime drowsiness that characterizes OSA in younger populations (Ancoli-Israel et al., 1991)

In conclusion, there is substantial and consistent evidence that age is an essential risk factor for the development and progression of obstructive sleep apnea. Both anatomical and functional alterations in the upper airway and respiratory control systems play significant roles in mediating this connection. Although the particular symptom profile of OSA in older persons remains a topic of ongoing inquiry, there is universal acceptance that advanced age considerably increases the probability of developing sleep-disordered breathing. As the world population continues to age, it becomes increasingly vital to recognize and manage the burden of OSA in older persons through increased screening, diagnosis, and age-appropriate interventions

2.3.3 Obesity

Obesity is one of the most well-established and modifiable risk factors for obstructive sleep apnea (OSA), with significant data supporting its role in the development and progression of the disorder. The prevalence of obesity has been rising globally at an alarming rate, affecting individuals across various racial and ethnic groupings, including both Caucasian and African populations.

Numerous studies have established that individuals with high body mass index (BMI) are much more likely to develop OSA, regardless of geographic location or socioeconomic background. According to Punjabi (2008), over 60% of persons referred for clinical examination and diagnostic testing for OSA were found to be obese, demonstrating a strong link between excess body weight and sleep-disordered breathing (Punjabi, 2008)

The association between obesity and OSA is complicated and bidirectional. Obesity leads to upper airway narrowing due to increased fat deposition in the parapharyngeal and submental regions, which reduces airway diameter and raises the risk of airway collapse during sleep. In addition, visceral and thoracic fat buildup worsens pulmonary mechanics by diminishing functional residual capacity, decreasing lung volume, and increasing the work of breathing variables that further exacerbate obstructive episodes during sleep (Schwartz et al., 2008)

The impact of body weight on OSA risk is further supported by evidence from international research. For example, a cross-sectional study carried out in New Delhi, India, revealed that those with a BMI $\geq$ 25 kg/m^2 were 4.98 times more likely to have OSA than people with a BMI below that cutoff (Sharma et al., 2006). This statistically significant correlation implies that even modest BMI increase significantly increases the risk of OSA, especially in South Asian populations where body composition changes may result in lower BMI cutoffs for overweight and obesity.

Numerous population-based studies from high and low-to-middle-income countries have proven a direct association between rising BMI and the prevalence of OSA, which is consistent with these findings. For instance, the Wisconsin Sleep Cohort Study found a linear correlation between weight gain and elevated apnea-hypopnea index (AHI), suggesting that even slight increase in body weight over time may exacerbate OSA or cause it to develop in people who were not previously affected (Peppard et al., 2000).

Moreover, studies have indicated that for every 10% rise in body weight, the chance of developing moderate to severe OSA increases by up to six -fold, underlining the major involvement of adiposity in OSA pathophysiology (Peppard et al., 2000)

The pathophysiological pathways linking obesity to OSA are complicated and involve both anatomical and functional abnormalities. Adipose tissue covering the upper airway structures, including the soft palate, tongue, and lateral pharyngeal walls, promotes pharyngeal collapsibility during sleep. In addition to these mechanical impacts, obesity is associated with systemic inflammation, oxidative stress, and hormonal abnormalities, including leptin resistance and diminished ghrelin regulation that may change ventilatory control and respiratory drive (Ryan & Bradley, 2005)

These variables collectively weaken neuromuscular compensatory processes that typically function to keep the airway open during sleep, leading to more frequent and protracted apneas.

Importantly, the severity of OSA has been demonstrated to rise with increasing BMI, and obese persons tend to present with more frequent respiratory episodes, lower oxygen saturation levels, and more episodes of daytime somnolence. This has clinical significance for screening and care, as weight reduction through lifestyle modification, pharmacological therapy, or bariatric surgery has been associated with considerable improvements in OSA severity and even complete remission in some individuals (Tuomilehto et al., 2009)

While obesity considerably increases the incidence of OSA, it is equally crucial to highlight that not all individuals with OSA are obese and not all obese individuals develop OSA. This shows that other factors such as craniofacial architecture, fat distribution, genetic predisposition, and sex hormones also modify the individual risk. Nevertheless, the presence of obesity, especially central obesity, remains one of the most powerful predictors of OSA, and its incidence continues to rise in both industrialized and developing nations.

In conclusion, obesity constitutes a key and modifiable risk factor for obstructive sleep apnea, with a substantial evidence from data confirming its causative and contributing involvement. Increased BMI not only predisposes persons to OSA but also worsens its severity and complicates its treatment. Reducing the rising prevalence of sleep-disordered breathing requires tackling this risk factor through preventative measures and public health initiatives, given the growing worldwide burden of obesity.

2.3.4 Cigarette Smoking

Tobacco, the principal constituent of cigarettes, has long been recognized as a major risk factor for a wide array of respiratory and cardiovascular diseases. In the context of sleep medicine, there exists a growing body of interest in the potential association between cigarette smoking and obstructive sleep apnea (OSA). One of the primary hypotheses suggests that smoking may contribute to the development or exacerbation of OSA by inducing upper airway inflammation, promoting oxidative stress, altering neuromuscular control, and negatively impacting the structure and function of airway tissues (Taveira et al., 2018)

Cigarette smoke is composed of thousands of harmful chemicals that can irritate and inflame the upper airway mucosa, leading to increased airway resistance and collapsibility, two central features in the pathophysiology of OSA. Chronic exposure to tobacco smoke has also been linked to structural changes in the pharyngeal tissues and increased mucosal edema, which could further narrow the airway lumen during sleep (Kales et al., 1984)

Additionally, nicotine, a major active component of tobacco, has complex effects on sleep architecture. Initially, nicotine acts as a central nervous system stimulant, delaying sleep onset and reducing total sleep time. However, as blood nicotine levels decline overnight, individuals may experience withdrawal-like effects, leading to fragmented sleep and instability of ventilatory control mechanisms (Taveira et al., 2018).

Despite the biologically plausible mechanisms linking smoking to OSA, the current body of literature presents mixed and sometimes contradictory findings, making it a contentious and unsettled topic in sleep medicine. For example, in their cross-sectional analysis, Taveira et al. (2018) concluded that there was no statistically significant association between cigarette smoking and the diagnosis of OSA, suggesting that smoking alone may not serve as a reliable independent risk factor

when considered in isolation (Taveira et al., 2018)

Similarly, findings from a population-based study conducted in Taiwan by Hsu, Chiu, Chang, Chang, and Lane (2019) did not report a significant relationship between tobacco use and OSA. The researchers accounted for potential confounders such as age, sex, and body mass index (BMI), and concluded that smoking was not associated with an increased risk of OSA in their cohort (Hsu et al., 2019) 3q
These findings reinforce the notion that while smoking may affect sleep quality and respiratory health, it may not be a direct etiological factor in the development of obstructive sleep apnea.

Contrasting these results, however, are studies that do report a significant association between smoking and the severity of OSA. Yosunkaya, Kutlu, and Vatansev (2021) found that the frequency and duration of cigarette smoking were positively correlated with the severity of apneic episodes and sleep-disordered breathing. Their findings suggest that individuals who were heavy or long-term smokers had more severe manifestations of OSA, including higher apnea-hypopnea index (AHI) scores and greater oxygen desaturation during sleep (Yosunkaya et al., 2021)
The authors proposed that smoking may exacerbate underlying vulnerabilities in susceptible individuals, thus amplifying the severity of sleep-related breathing disturbances.

In contrast, a study by Esen and Akpinar (2021) did not observe any significant association between cigarette smoking and OSA severity, leading to further ambiguity in literature. They reported that smoking habits, including the number of cigarettes smoked per day and duration of smoking, were not predictive of OSA diagnosis or severity based on polysomnographic findings (Esen & Akpinar, 2021) As such, the role of cigarette smoking in the pathogenesis and progression of OSA remains inconclusive, with studies yielding differing results depending on sample size, population demographics, and methodological design.

Some scholars have argued that the conflicting evidence in the literature may be due to the complex interactions between smoking and other coexisting risk factors such as obesity, alcohol use, nasal congestion, or chronic bronchitis, which can independently influence sleep and breathing patterns. It is also possible that differences in genetic susceptibility, fat distribution, or inflammatory responses may mediate how smoking affects individuals' risk for OSA. Moreover, smoking may serve as a surrogate marker for other unmeasured behavioral or environmental exposures that contribute to the risk of disordered breathing during sleep.

In conclusion, while there are biologically plausible mechanisms that support a potential link between cigarette smoking and obstructive sleep apnea, the empirical evidence remains mixed. Some studies report a positive association with OSA severity, while others find no significant correlation after adjusting for confounders. As such, the relationship between cigarette smoking and OSA cannot be definitively characterized at this time. Further well-designed longitudinal and interventional studies are needed to clarify whether cigarette smoking contributes independently to OSA risk or whether it acts synergistically with other factors to worsen disease severity.

2.4 Complications of Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is not only a disorder of disrupted sleep but also a condition with a wide range of systemic complications that affect various physiological systems. The chronic and repetitive nature of sleep disturbances caused by OSA leads to hypoxia, sleep fragmentation, and heightened sympathetic nervous system activity. These effects, when left unmanaged, result in significant impairment in daytime functioning, mental and physical health, and long-term morbidity. The complications of OSA can be broadly classified into neurocognitive, cardiovascular, metabolic, and psychosocial domains.

2.4.1 Daytime Sleepiness

One of the most well-documented consequences of OSA is persistent and excessive daytime sleepiness**.** This symptom arises primarily due to the repeated interruption of normal sleep architecture by apneic and hypopneic events throughout the night. Each episode of airway obstruction causes a brief arousal from sleep, even if the patient is not consciously aware of waking up. Over time, these repeated arousals result in cumulative sleep deprivation and reduced sleep efficiency, both of which significantly impair daytime alertness and function (Johns, 1991)

Daytime sleepiness caused by OSA has been shown to reduce concentration, slow reaction times, and impair memory, making routine tasks such as reading, driving, and working more challenging. In fact, studies have found that patients with untreated OSA have an increased risk of involvement in road traffic accidents due to impaired vigilance and microsleeps while driving (Tregear et al., 2009)

The Epworth Sleepiness Scale (ESS) remains a widely used self-reporting tool for measuring daytime somnolence, helping clinicians gauge the severity of functional impairment (Esen & Akpinar, 2021).

2.4.2 Reduced Quality of Life

Obstructive sleep apnoea has profound and often underestimated effects on the quality of life of affected individuals. People living with OSA frequently experience chronic fatigue, irritability, difficulty concentrating, low mood, and impaired interpersonal relationships. These symptoms cumulatively reduce productivity, interfere with occupational functioning, and increase dependence on others for daily activities (Al Lawati et al., 2009)

Several studies have demonstrated that the impact of OSA on QoL is comparable to, and sometimes exceeds, that observed in other chronic medical conditions, such as diabetes, hypertension, or congestive heart failure (Flemons & Tsai, 1997)

More severe forms of OSA are associated with significantly lower scores on quality of life scales. According to Al Lawati et al. (2009), individuals with more advanced disease had an approximately 1.5-fold higher likelihood of reporting poor quality of life compared to those with milder forms. Left untreated, these effects can lead to social isolation, occupational underperformance, and psychological distress.

2.4.3 Cardiovascular Diseases

Perhaps the most serious and well-established complications of OSA are its strong associations with cardiovascular diseases (CVD)**.** A growing body of evidence links OSA with hypertension, coronary artery disease, heart failure, atrial fibrillation, stroke, and sudden cardiac death (Ayas, Taylor, & Laher, 2016). Recurrent episodes of oxygen desaturation and arousal during sleep result in sympathetic nervous system activation, elevated blood pressure, and endothelial dysfunction, each of which contributes to the progression of cardiovascular pathology.

Chronic intermittent hypoxia, a hallmark of OSA, triggers oxidative stress and promotes systemic inflammation. These processes play a central role in atherogenesis and endothelial damage. Moreover, OSA is associated with dysregulation of metabolic hormones such as leptin, ghrelin, and adiponectin, which can influence appetite control, lipid metabolism, and glucose homeostasis, indirectly contributing to cardiovascular risk (Al Lawati et al., 2009)

In patients with resistant or poorly controlled hypertension, the prevalence of undiagnosed OSA is notably high, and treating OSA with continuous positive airway pressure (CPAP) has been shown to significantly reduce nighttime blood pressure and improve overall cardiovascular outcomes (Peppard et al., 2000)

Similarly, the presence of OSA is now recognized as an independent risk factor for atrial fibrillation and has been associated with higher recurrence rates of arrhythmias following cardioversion or catheter ablation (AS Gami et al., 2007)

2.4.4 Neurocognitive and Psychological Impairment

Beyond cardiovascular consequences, OSA has significant effects on cognitive function and mental health. Individuals with OSA frequently report memory difficulties, poor concentration, impaired executive function, and decreased processing speed. These effects are primarily due to the cumulative burden of fragmented sleep and intermittent nocturnal hypoxia on brain structures involved in cognition, particularly the prefrontal cortex and hippocampus (Beebe & Gozal, 2002)

Moreover, OSA has been independently associated with higher rates of depression and anxiety, which may both precede and follow the development of the disorder. Emotional dysregulation and mood instability in these individuals can worsen interpersonal relationships and reduce motivation for self-care and medical adherence. The bidirectional relationship between OSA and mental health disorders further highlights the need for early identification and comprehensive management.

2.4.5 Metabolic and Endocrine Dysregulation

OSA is also closely linked to metabolic disturbances, including insulin resistance, glucose intolerance, and dyslipidemia. The intermittent hypoxia and sleep fragmentation characteristic of OSA lead to increased cortisol secretion, insulin resistance, and alterations in glucose metabolism. These mechanisms predispose individuals to type 2 diabetes mellitus, even independent of obesity (Tasali & Ip, 2008)

Patients with untreated OSA are more likely to develop components of metabolic syndrome, such as central obesity, elevated triglycerides, reduced HDL cholesterol, and increased fasting glucose. This association underscores the critical role of OSA not only as a respiratory disorder but also as a systemic condition with wide-ranging metabolic consequences.

In summary, obstructive sleep apnoea is a complex and multisystemic disorder that extends far beyond disrupted sleep. Its complications affect nearly every domain of health, including daytime sleepiness, psychological well-being, cardiovascular function, and metabolic homeostasis. Failure to identify and treat OSA in a timely manner can result in significant morbidity and impaired quality of life. Early diagnosis and appropriate intervention, particularly through CPAP therapy and lifestyle modification, are essential to reducing the burden of complications and improving long-term outcomes.

2.5 Diagnosis of OSA

Obstructive sleep apnea is defined on the basis of nocturnal and daytime symptoms as well as sleep study findings.

Diagnosis requires the patient to have either:

1.Symptoms of nocturnal breathing disturbances (snoring, snorting, gasping or breathing pauses during sleep) or daytime sleepiness or fatigue that occurs despite sufficient opportunity to sleep and not explained by other medical problems.

2.Five or more episodes of obstructive apnea or hypopnea per hour of sleep (the apnea-hypopnea index-AHI, calculated as the number of episodes divided by the number of hours of sleep) documented during a sleep study.

The Apnea-Hypopnea Index (AHI) quantifies the total number of apneas and hypopneas per hour of sleep; apnea is characterized by a cessation of airflow lasting at least 10 seconds, while hypopnea is characterized by a decrease in respiratory effort accompanied by a minimum of 4% oxygen desaturation.

Polysomnography also quantifies another sleep parameter, referred to as respiratory disturbance index (RDI). The Respiratory Disturbance Index is defined as the total number of apneas, hypopneas, and atypical respiratory episodes per hour of sleep. The two terms, apnea hypopnea index and respiratory disturbance index are frequently utilized interchangeably

OSA may also be diagnosed in the absence of symptoms if the Apnea hypopnea index is more than 15 episodes/hour. Each episode of apnea or hypopnea represents a reduction in breathing for at least 10 seconds and commonly results in more than 3% drop in oxygen saturation or a brain cortical arousal. OSA severity can be characterized by the frequency of breathing disturbances (AHI) the amount of oxyhemoglobin desaturation with respiratory events, the duration of apnea and hypopneas, the degree of sleep fragmentation and the level of reported daytime sleepiness or functional impairment.

There is no definitive gold standard for the diagnosing obstructive sleep apnea, complicating the calibration of any diagnostic tool. Historically, PSG, conducted in a monitored environment (sleep laboratory) has served as the benchmark for diagnosing OSA. This is according to a report by the medical secretariat of the American Academy of Sleep Medicine.

POLYSOMNOGRAPHY

Polysomnography is a comprehensive test for diagnosing sleep disorders. It is widely regarded as the gold standard in the diagnosis of obstructive sleep apnea. However, a paper by the American Academy of Medicine argues that there is no definite gold standard diagnostic for obstructive sleep apnea (AASM, Medical secretariat) It encompasses the concurrent measurement of various physiological parameters associated with both sleep and wakefulness. The device has the capability to directly observe and measure the frequency of respiratory events, as well as the resulting decrease in oxygen levels associated with these events.

Polysomnography measures the following:

- Brain Waves: Electroencephalogram (EEG) monitors the electrical activity in the brain to assess sleep stages.
- Eye movements: Electrooculogram (EOG) tracks rapid eye movement (REM) during sleep.
- Heart rate: Electrocardiogram (ECG) monitors heart rate and rhythm. This reveals autonomic dysfunction e.g. elevated nighttime heart rate in type 2 diabetes and obstructive sleep apnea patients.
- Breathing: Measures airflow, apnea-hypopnea index, chest and abdominal movement, and blood oxygen levels to detect breathing problems.

- Muscle activity: Electromyography (EMG) monitors muscle tone, especially in the legs and chin. It can also detect restless leg syndrome.
- Body position: Sensors track changes in body position during sleep.

PROCEDURE

The patient will typically spend a night in a sleep laboratory with sensors attached to their body. The sensors are connected to a computer that records data from various physiological parameters. The data is then analyzed by a sleep specialist to identify sleep patterns and notes any abnormalities.

Other than Obstructive sleep apnea, PSG can also diagnose other sleep disorders such as insomnia, narcolepsy, restless leg syndrome, REM sleep behavior disorders and parasomnias.

PSG, however, is labor-intensive, expensive and not available in our setup. Furthermore, it requires the expertise of sleep specialists, who are not readily available in many local health facilities. As a result, many patients with OSA remain undiagnosed therefore not getting the deserved treatment modalities.

With these challenges, the use of simple, less expensive and easy to use tools to identify patients at risk is recommended. Many are lengthy and complicated which makes them inconvenient to use and vulnerable to variability among clinicians performing the upper airway assessment (Chung, Abdullah, & Liao, 2016) However, two tools for screening OSA stood out because of their low cost and are easy to use or administer.

These tools are STOP-BANG and Berlin questionnaires (Amra et al., 2018)

STOP-BANG QUESTIONNAIRE

The STOP-Bang Questionnaire is a validated screening instrument intended to identify persons at elevated risk for OSA, especially in preoperative contexts. Developed in 2008 by Dr. Frances Chung and associates at the University of Toronto, it fulfills a need for a swift, economical approach to identify undiagnosed obstructive sleep apnea (OSA), which impacts around 80% of surgery patients and heightens perioperative difficulties. The term is an acronym representing eight principal risk factors: Snoring, tiredness, observed apnea, high blood pressure, body mass index (BMI), age, neck circumference, and gender (Edwin Seet et al, Singapore Medical Journal)

STRUCTURE AND ADMINISTRATION

Eight yes/no questions grouped into two domains:

STOP (Symptom-based):

Snoring: "Do you snore loudly?"

Tiredness: "Do you often feel fatigued?"

Observed apnea: "Has anyone seen you stop breathing during sleep?"

Pressure: "Do you have hypertension?"

Bang (Demographic-based):

5. BMI >35 kg/m² (or >30 kg/m² in Asian populations)
6. Age >50 years.
7. Neck circumference >40 cm (16 inches).
8. Male gender

Scoring: Each "yes" = 1 point; total score ranges from 0–8.

RISK STRATIFICATION

Score	OSA Risk Level	Clinical Implication
0–2	Low risk	Unlikely to have moderate/severe OSA
3–4	Intermediate risk	Requires further assessment (e.g., sleep study)
5–8	High risk	High probability of moderate/severe OSA

CLINICAL APPLICATIONS

Perioperative Risk Stratification

STOP-Bang scores of 3 or above are associated with a 5.5-fold elevated risk of intraoperative and postoperative adverse events, such as cardiac and pulmonary problems.

Scores of 5 or higher indicate a 34–44% likelihood of perioperative problems, compared to a 5.5% likelihood for scores below 3.

Sleep clinics: Triage instrument for polysomnography referrals

Type 2 diabetes: Screening for concomitant obstructive sleep apnea (prevalence up to 56%)

Cardiology: Forecasts recurrence of cardiovascular events

The STOP-BANG and Berlin questionnaires have been evaluated for their diagnostic properties in several research. Amra et al, (2018) compared the sensitivity and specificity of these two tools and found out that the Berlin questionnaire was more sensitive than the STOP-BANG questionnaire and that the STOP-BANG questionnaire was more specific compared to the Berlin questionnaire (Amra et al., 2018). However as noted earlier on in this chapter that OSA worsens DM control and may contribute to DM-related complications (Aronsohn et al., 2010), we intend to use Berlin questionnaire with higher sensitivity compared to STOP-BANG and include all type 2 DM patients who are most likely to have OSA

Berlin Questionnaire

The Berlin Questionnaire is a widely used diagnostic tool for obstructive sleep apnea (OSA), a common sleep disorder characterized by repeated episodes of complete or partial upper airway obstruction during sleep. The questionnaire is designed to identify individuals who are at high risk of OSA, and its findings can be used to guide further evaluation and treatment.

Development of the Berlin Questionnaire

The Berlin Questionnaire was developed in the late 1990s by a team of researchers at the University of Berlin. The questionnaire was designed to be a simple, self-administered tool that could be used to identify individuals who were at high risk of OSA. The development of the questionnaire was based on a comprehensive review of the literature on OSA, as well as expert opinion and input from clinicians and researchers in the field.

The questionnaire consists of 11 questions that assess a range of symptoms and risk factors associated with OSA, including:

SECTION 1: DEMOGRAPHIC INFORMATION

Height:	________ cm/in	Weight:	________ kg/lbs
Age:	________ years	Gender:	☐ Male ☐ Female

SECTION 2: SNORING ASSESSMENT

2. Do you snore?

☐ Yes ☐ No ☐ Don't know

IF YES TO QUESTION 2, PLEASE COMPLETE:

3. Loudness of snoring:

☐ Slightly louder than breathing

☐ As loud as talking

☐ Louder than talking

☐ Very loud (heard in adjacent rooms)

4. Snoring frequency:

☐ Nearly every day

☐ 3-4 times/week

☐ 1-2 times/week

☐ 1-2 times/month

☐ Never/nearly never

5. Has your snoring bothered others?

☐ Yes ☐ No

SECTION 3: SLEEP SYMPTOMS

6. Breathing pauses noticed during sleep:

☐ Nearly every day

☐ 3-4 times/week

☐ 1-2 times/week

☐ 1-2 times/month

☐ Never/nearly never

7. Tiredness after waking:

☐ Nearly every day

☐ 3-4 times/week

☐ 1-2 times/week

☐ 1-2 times/month

☐ Never/nearly never

8. Daytime fatigue while awake:

☐ Nearly every day

☐ 3-4 times/week

☐ 1-2 times/week

☐ 1-2 times/month

☐ Never/nearly never

9. Nodded off while driving?

☐ Yes ☐ No

If YES, frequency:

☐ Nearly every day

☐ 3-4 times/week

☐ 1-2 times/week

☐ 1-2 times/month

☐ Never/nearly never

SECTION 4: MEDICAL HISTORY

10. High blood pressure?

☐ Yes ☐ No ☐ Don't know

11. BMI: ________ kg/m^2

(Formula: weight(kg) / $[height(m)]^2$)

<u>Scoring the Berlin Questionnaire</u>

The questionnaire consists of 3 categories related to the risk of having sleep apnea. Patients can be classified into High Risk or Low Risk based on their responses to the individual items and their overall scores in the symptom categories.

Categories and Scoring:

Category 1: items 2, 3, 4, 5, and 6;

Item 2: if ‘Yes’, assign 1 point

Item 3: if either of the last two options is the response, assign 1 point

Item 4: if either of the first two options is the response, assign 1 point

Item 5: if ‘Yes’ is the response, assign 1 point

Item 6: if either of the first two options is the response, assign 2 points

Add points. Category 1 is positive if the total score is 2 or more points.

Category 2: items 7, 8, and 9.

Item 7: if either of the first two options is the response, assign 1 point

Item 8: if either of the first two options is the response, assign 1 point

Item 9: if ‘Yes’ is the response, assign 1 point

Add points. Category 2 is positive if the total score is 2 or more points.

Category 3 is positive if the answer to item 10 is ‘Yes’ or if the BMI of the patient is greater

than 30kg/m2. (BMI is defined as weight (kg) divided by height (m) squared, i.e ..., kg/m2).

High Risk: if there are 2 or more categories where the score is positive.

Low Risk: if there is only 1 or no categories where the score is positive.

Additional Question: item 9 should be noted separately.

A score of 2 or more on the Berlin Questionnaire indicates a high risk of OSA. Individuals with a high-risk score are more likely to have OSA and should undergo further evaluation and testing, such as polysomnography (PSG) or home sleep apnea testing (HSAT).

Interpretation of the Berlin Questionnaire

The Berlin Questionnaire is not a definitive diagnostic tool for OSA, but rather a screening tool to identify individuals who may be at high risk of the disorder. A high-risk score on the questionnaire should be followed up with further evaluation and testing to confirm the diagnosis.

The questionnaire has been shown to be effective in identifying individuals with OSA, with a sensitivity of 86% and a specificity of 77%. However, the questionnaire is not perfect, and false negatives and false positives can occur.

Limitations of the Berlin Questionnaire

While the Berlin Questionnaire is a useful diagnostic tool for OSA, it has several limitations. One of the main limitations is that it relies on self-reported symptoms and risk factors, which may not be entirely accurate. Additionally, the questionnaire does not take into account other sleep disorders that may be present, such as insomnia or restless leg syndrome.

Another limitation of the Berlin Questionnaire is that it may not be effective in identifying individuals with mild OSA or those who do not exhibit typical symptoms of the disorder. Furthermore, the questionnaire has not been validated in certain populations, such as children or pregnant women.

Advantages of the Berlin Questionnaire

Despite its limitations, the Berlin Questionnaire has several advantages that make it a useful screening tool for OSA. One of the main advantages is that it is simple and easy to administer, making it a useful screening tool in primary care settings. Additionally, the questionnaire is self-administered, which reduces the burden on healthcare providers and makes it more accessible to individuals who may not have access to specialized sleep clinics.

Another advantage of the Berlin Questionnaire is that it is cost-effective and does not require any specialized equipment or training. This makes it a useful tool for healthcare providers in resource-limited settings or for individuals who are unable to access specialized sleep clinics.

Clinical Applications of the Berlin Questionnaire

The Berlin Questionnaire has a range of clinical applications, including:

Primary care settings: The questionnaire can be used as a screening tool in primary care settings to identify individuals who may be at high risk of OSA.

Sleep clinics: The questionnaire can be used to evaluate individuals who are referred to sleep clinics for suspected OSA.

Pre-operative assessment: The questionnaire can be used to identify individuals who may be at high risk of OSA before undergoing surgery.

Public health campaigns: The questionnaire can be used to raise awareness about OSA and to identify individuals who may be at high risk of the disorder.

Future Directions

While the Berlin Questionnaire is a useful diagnostic tool for OSA, there are several areas for future research and development. One area is the development of more accurate and reliable diagnostic tools that can be used in a range of clinical settings. Another area is the use of technology, such as mobile apps and wearable devices, to detect OSA symptoms and risk factors.

Additionally, there is a need for more research on the epidemiology and pathophysiology of OSA, as well as the development of more effective treatments for the disorder. The Berlin Questionnaire is an important tool in the diagnosis and management of OSA, but it is only one part of a comprehensive approach to addressing this complex and multifaceted disorder.

In conclusion, the Berlin Questionnaire is a valuable diagnostic tool for OSA, and its use can help to identify individuals who are at high risk of the disorder. By raising awareness about OSA and its symptoms, and by providing a simple and effective screening tool, the Berlin Questionnaire can help to improve the diagnosis and management of OSA, and reduce the burden of this complex and multifaceted disorder.

Treatment of Obstructive Sleep Apnea (OSA)

The treatment of obstructive sleep apnea (OSA) involves a comprehensive strategy that is both patient-focused and evidence-based. Due to the numerous causes of obstruction of the airway and the differing severity of the condition, treatment options must be customized to each patient's characteristics, including symptom severity, comorbidities, anatomical features, and therapy tolerance. The principal objectives of OSA treatment are to eradicate apneas and hypopneas, enhance sleep quality, alleviate daytime symptoms, and mitigate the long-term cardiovascular and metabolic risks linked to untreated conditions.

The treatment can be categorized as follows into non-pharmacological, pharmacological and surgical.

Non-Pharmacolgical Therapy

Lifestyle Modification

Lifestyle modification is regarded as a first line treatment strategy especially for individuals with mild obstructive sleep apnea or those with modifiable risk factors like obesity. Weight reduction has repeatedly demonstrated a substantial enhancement in the severity of obstructive sleep apnea (OSA), as even small weight loss can decrease the apnea-hypopnea index (AHI) and increase oxygen saturation levels during sleep.

A Very Low Calorie Diet, coupled with active lifestyle counseling, resulting in significant weight loss is a feasible and effective intervention for most patients with mild-moderate OSA, with positive results sustained at one-year follow-up. (Tuomilehto et al., 2009)

Lifestyle interventions also encompass positional therapy, which involves avoiding the supine posture during sleep and has been shown to be advantageous for individuals whose apneas are mostly dependent on the supine position (Oksenberg et al., 2006)

Other lifestyle modifications include reducing alcohol intake and cigarette smoking cessation.

Pharamacological Intervention/Mechanical Therapy.

Continuous Positive Airway Pressure (CPAP) Therapy

Continuous Positive Airway Pressure (CPAP) therapy is the definitive treatment for moderate to severe obstructive sleep apnea (OSA). CPAP functions by providing a continuous flow of pressured air via a nasal or oronasal mask, which maintains the upper airway's patency and prevents its collapse during sleep.

Multiple studies have established the effectiveness of CPAP in decreasing AHI, enhancing sleep architecture, and mitigating excessive daytime sleepiness (SP Patil et al., 2007)

Furthermore, continuous positive airway pressure (CPAP) therapy has been associated with decreases in cardiovascular morbidity, as well as improvements in blood pressure control and glycemic regulation in diabetic patients (Marin et al., 2005).

Nonetheless, adherence to CPAP presents a considerable therapeutic obstacle, since certain patients experience discomfort or intolerance stemming from mask leaks, nasal congestion, or feelings of claustrophobia. To mitigate this, auto-titrating CPAP (APAP) and bilevel PAP (BiPAP) systems may be employed in specific instances to enhance comfort and tolerance.

Oral Appliance Therapy

For patients with mild to moderate obstructive sleep apnea (OSA) or those who cannot tolerate continuous positive airway pressure (CPAP), oral appliances, especially mandibular advancement devices (MADs), present a feasible alternative. These devices advance the mandible anteriorly, thereby expanding the upper airway and mitigating pharyngeal collapse. MADs are particularly efficacious in patients exhibiting retrognathia, small jaw features, or positional obstructive sleep apnea (Sutherland et al 2014)

TIRZEPATIDE

Tirzepatide is currently the only FDA-approved pharmacological intervention for OSA. It is a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide (GLP)1 receptor agonist (Twincretin)

Tirzepatide in the management of OSA

Evidence from the SURMOUNT-OSA trial demonstrated that tirzepatide significantly reduced AHI in patients with moderate-severe OSA. The therapeutic benefit was observed irrespective of baseline glycemic status including those patients without T2DM. Tirzepatide is also used to treat obesity thus offering an extra benefit as most patients with OSA have a high BMI. This drug therefore represents an emerging pharmacological therapy for moderate-severe OSA expanding treatment options beyond traditional device-based interventions.

Other Pharmacologic and Adjunct Therapies

. Modafinil and armodafinil are wakefulness-promoting medications occasionally utilized to address persistent daytime somnolence in patients compliant with CPAP therapy but nonetheless suffer from excessive drowsiness (Schwartz et al., 2003).

Furthermore, novel medicines aimed at modulating upper airway muscle tone or respiratory control systems are now being explored. Carbonic anhydrase inhibitors, such as acetazolamide, have demonstrated potential in diminishing central apneas or elevated loop gain in mixed obstructive sleep apnea phenotypes (Edwards et al., 2012).

Surgical Interventions

Surgery may be indicated in specific instances where anatomical anomalies substantially cause airway obstruction or when non-invasive treatments like CPAP and oral devices are unsuccessful or poorly tolerated. The predominant surgical intervention for obstructive sleep apnea (OSA) is uvulopalatopharyngoplasty (UPPP), which entails the excision of superfluous tissue from the soft palate and pharynx to widen the airway. While UPPP may diminish snoring and slightly enhance AHI, its efficacy rates are markedly inconsistent and reliant on meticulous patient selection. (E Sher et al 1996)

Alternative surgical interventions include nasal operations (e.g., septoplasty, turbinate reduction) for patients experiencing considerable nasal obstruction, tongue base reduction, and maxillomandibular advancement (MMA), which has demonstrated substantial effectiveness in severe obstructive sleep apnea (OSA), particularly in individuals with craniofacial anomalies (Li et al., 2000). Advances in minimally invasive techniques, such as hypoglossal nerve stimulation (HNS), are also gaining attention. HNS entails the implantation of a device that activates the hypoglossal nerve to constrict airway muscles during sleep, therefore mitigating airway collapse. Research indicates that this therapy markedly decreases AHI and enhances quality of life in specific patients (Strollo et al 2014)

CHAPTER THREE

3.0 METHODOLOGY

3.1 Study Setting

The study was conducted at Moi Teaching and Referral Hospital's (MTRH) diabetes outpatient clinic. With a bed capacity of 1020 specialized beds, an average of 1300 inpatient patients at a given time and 1500 outpatients each day, MTRH is a National/International Referral Hospital that provides outpatient, inpatient, and specialist medical services. The hospital, which serves roughly 24 million people in portions of 23 Kenyan counties, Eastern Uganda, South Sudan, Tanzania, and the Democratic Republic of the Congo, is situated along Nandi Road in Eldoret Uasin Gishu County.

MTRH also serves as the teaching hospital for the Moi University College of Health Sciences, which trains over 240 postgraduate students (Registrars) in a variety of fields, including many Masters in Medicine programs.

The diabetes outpatient clinic is located on the ground floor of the Chandaria cancer and chronic disease center. The clinic operates a total of three days per week, specifically on Mondays, Thursdays, and Fridays, with operational hours from 9:00 AM to 3:00 PM. On average, approximately 50 patients seek medical attention at the clinic each day, resulting in a cumulative weekly patient count of 150 individuals.

3.2 Study Design

A cross-sectional study was conducted to identify the prevalence and factors associated with OSA risk in Type 2 Diabetes.

3.3 Population

The study included adults 18 years or older attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital during the specified study period as long as they meet the eligibility criteria for the study.

3.4 Eligibility Criteria

3.4.1 Inclusion Criteria

a) Adults aged 18 years and older.

b) On care at MTRH for Type 2 diabetes mellitus for at least three months

c) Ability to provide informed consent

3.4.2 Exclusion Criteria

a) Pregnancy

b) Those undertaking night shift jobs-Individuals working night shifts were excluded due to the high prevalence of circadian rhythm disturbances, sleep deprivation, and shift work sleep disorder in this population. These sleep pattern alterations can mimic or exacerbate symptoms such as excessive daytime sleepiness, fatigue, and poor sleep quality, which may be misclassified as OSA risk on screening tools

c) Patients with prior diagnosis of sleep apnoea and on treatment. - Participants with a known diagnosis of OSA and already receiving treatment (e.g., CPAP therapy) were excluded because their OSA status had already been established and managed, thus not reflecting undiagnosed or untreated OSA risk within the diabetic cohort. Including them could have led to inaccurate prevalence estimates and potentially confounded the analysis of risk factors, since their symptoms and outcomes may be modified by treatment

3.5 Sample Size

3.5.1 Objective 1

To determine the prevalence of OSA risk status among ambulatory type 2 diabetes patients attending the diabetes outpatient clinic at MTRH.

The estimated prevalence from a study by S.Ade et al showed a prevalence of risk of OSA of 18%.

Sample size for the prevalence= We used the formula by Fishers et al

$$n = \frac{Z^2 P (1-P)}{e^2}$$

$$N = \frac{(1.96*1.96)\ (0.44)\ (1-0.44)}{(0.05)^2}$$

$N = (3.8416*0.18*0.82) / 0.0025$

Therefore, we needed to recruit a minimum of 227 patients who met the inclusion criteria.

3.5.2 Objective 2

To determine the factors associated with Obstructive sleep apnoea (OSA) risk status.

Sample size for factors associated= We used the formula by Peduzzi et al, 1998

$n=10\ Kp$

$n=10 \times 60/0.18=333.3$

We considered only six factors, that is, age, sex, obesity, neck circumference, smoking, alcohol and recruited a minimum of 334 patients who met the inclusion criteria. This is the sample size that was used for this study.

3.6 Sampling Method

A Systematic sampling technique was used to select participants after identifying eligible participants using inclusion and exclusion criteria.

3.7 Study Variables

3.7.1 Independent

The predictor variables in this study included:

1. Social demographic characteristics including age, sex, alcohol consumption, marital status, occupation, education level. Among those that drink alcohol, details of alcohol consumption was collected including how often one drinks alcohol, hours spent before sleep after alcohol intake.
2. Clinical characteristics including BMI, abdominal circumference, neck circumference, recent HbA1C levels, sleep position, and Mallampati score.
3. Exposure to air pollutants including whether one smokes tobacco or has ever smoked tobacco, whether fossil fuel is used as a source of energy for cooking

3.7.2 Dependent

The outcome variable in this study was the presence or the absence of OSA risk as estimated using the Berlin Questionnaire

3.8 Study Procedure

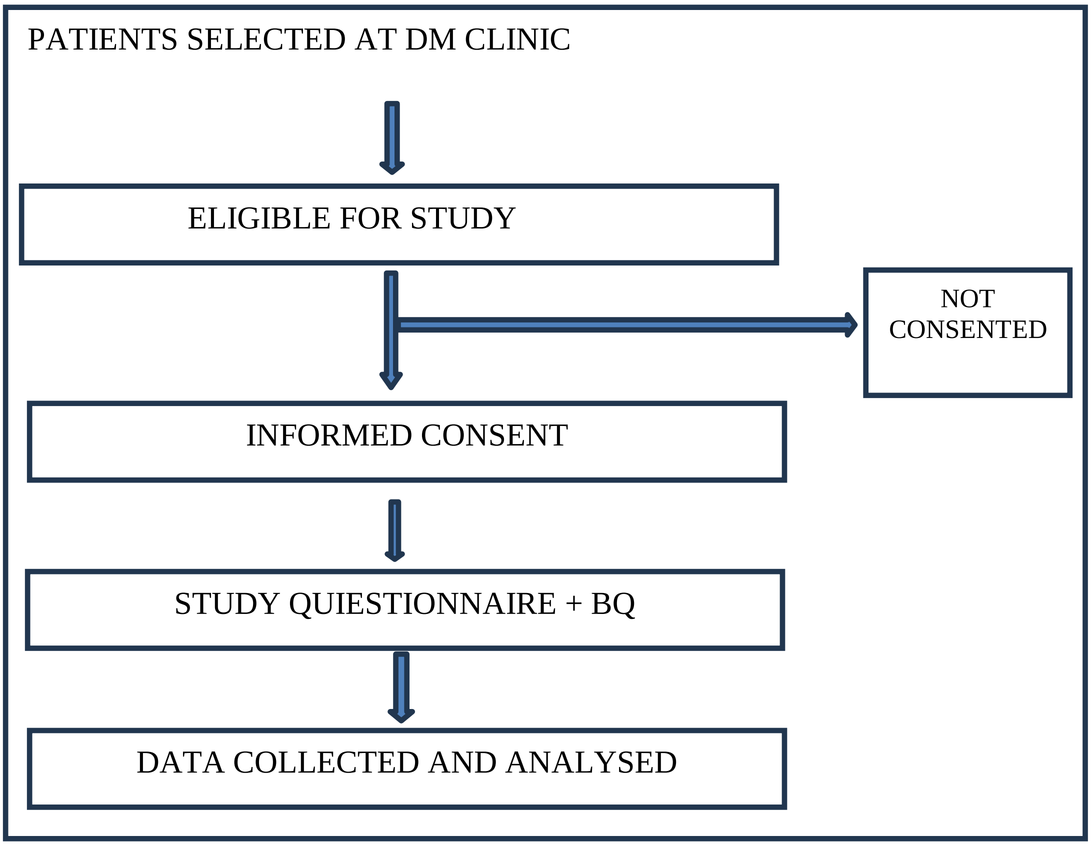
The research was conducted at the diabetes outpatient clinic of MTRH, located on the ground floor of the Chandaria Cancer and Chronic Diseases Center. A dedicated room for the study was labeled and made known to the clinic staff for ease of referral. Patients attending the clinic were screened for eligibility to participate in the study. Those who met the prespecified inclusion and exclusion criteria were consented by the investigator or a trained research assistant. Thereafter, relevant data were obtained and captured in the questionnaire and later transcribed into a digital version for electronic storage. Vital signs were measured, followed by physical assessment for features of OSA as per the modified Mallampati method. Findings were categorized according to the modified Mallampati criteria. All questionnaires and other source materials containing patient data were stored in a locked cabinet accessible only to the research personnel to ensure confidentiality, while the electronic version was secured with a password.

The modified Mallampati score was documented. During the physical examination, participants were instructed to open their mouths, and the examiner assigned a classification (I–IV) based on visualization. Class I denoted visibility of the soft palate, hard palate, and uvula; Class II indicated visibility of the soft palate and uvula; Class III denoted visibility of only the soft palate and base of the uvula; and Class IV indicated visibility of only the hard palate. The physical examination and subsequent classification were performed by the principal investigator, a second-year internal medicine registrar with six years of prior experience as a medical officer in the accident and emergency department of a busy level 5 hospital.

History of nasal congestion was also obtained. Participants were asked whether they experienced unilateral or bilateral congestion, nasal obstruction without other symptoms, post-nasal drip, facial pain, or anosmia. Physical examination was performed to identify visible features contributing to nasal obstruction, such as turbinate hypertrophy or deviated septum.

Neck circumference was measured using standard anatomical landmarks for consistency, with the thyroid cartilage as the reference point. The measurement was taken completely around the neck with a tape measure. Waist and hip circumferences were also measured. Waist circumference was assessed at the level of the umbilicus, with the participant standing upright and feet together, while hip circumference was measured at the widest part of the hips. The waist–hip ratio was subsequently computed and recorded.

The most recent HbA1c value (3 months) of each participant was documented. In the absence of a recent HbA1c measurement, a venous blood sample was obtained for determination. Eligible participants then completed the Berlin Questionnaire (BQ). Responses were scored as “High Risk” or “Low Risk” for OSA based on the established criteria. Study findings were presented using figures, tables, and graphs, and statistical analyses were performed. A p-value <0.05 was considered statistically significant.



The study questionnaire, which incorporated the BQ, also captured data on comorbidities, complications, occupation, education level, marital status, alcohol consumption (including frequency, timing before sleep, and associated effects), smoking history, fossil fuel use for cooking, neck circumference, nasal congestion, sleeping position, and Mallampati score. The BQ contained five items on snoring, three on daytime somnolence, and one on BMI. A category was considered positive if responses indicated persistent symptoms (≥3–4 times per week). A positive score in the third category required a history of BMI ≥25 kg/m². Participants were classified as being at high risk for OSA if they had a positive score in two or more categories; otherwise, they were classified as low risk (Netzer et al., 1999).

The Berlin Questionnaire, with a sensitivity of 86% and specificity of 77% for identifying OSA, had been validated against polysomnography. Furthermore, this tool had been validated in the Kenyan setting by Sokwalla et al. (2017). The questionnaire was administered during the patients' visits to the diabetes outpatient clinic at MTRH. Whenever possible, responses regarding snoring and other OSA-related symptoms were verified by a family member or bed partner. However, the absence of a companion did not constitute an exclusion criterion; unaccompanied patients were also interviewed and included in the study.

3.8.1 Data management and analysis plan

Preliminary analysis involved summarizing the demographic and clinical characteristics of study participants. Categorical variables such as sex, alcohol consumption, occupation, educational level, abdominal circumference, and sleep position were summarized as frequencies with their corresponding percentages. Numerical variables such as age were summarized using means or medians with their respective standard deviations or interquartile ranges. Body Mass Index (BMI) and HbA1c, although initially collected in numerical form, were subsequently categorized into clinically meaningful groups and summarized as categorical variables.

Further analysis was done as per each objective as summarized in the table below

Objective	Outcome	Independent	Statistical Test
One: To determine the prevalence of OSA risk in patients with T2DM enrolled in the Diabetes clinic at MTRH using the Berlin Questionnaire	OSA risk (Yes/ No) – binary categorical variable		Frequency & proportion
Two: To determine the various factors that are associated with OSA among patients with T2DM enrolled in the diabetes clinic at MTRH	OSA risk (Yes/ No) – binary categorical variable	Age, neck circumference, Sex, obesity, smoking, alcohol	*t*-test, Mann Whitney U Test Chi Square, Fishers exact test Binary logistic regression

3.9 Ethical Consideration

In adherence to ethical standards, approval for the study was obtained from the Institutional Research and Ethics Committee (IREC) and NACOSTI prior to its commencement. Permission was also granted by the administration of Moi Teaching and Referral Hospital. Informed consent was obtained from each participant before enrollment into the study. Furthermore, the privacy and confidentiality of all participants' data were strictly maintained throughout the study

CHAPTER FOUR

4.0 RESULTS

Table 1: Sociodemographic Characteristics of Study Participants

CHARACTERISTIC	**FREQUENCY (N)**	**PERCENTAGE (%)**
AGE (YEARS)		
<30	1	0.3
31–40	43	12.9
41–50	46	13.8
51–60	74	22.2
>60	170	50.9
GENDER		
FEMALE	215	64.4
MALE	119	35.6
MARITAL STATUS		
MARRIED	268	80.2
SINGLE	27	8.1
WIDOWED	39	11.7
EDUCATIONAL LEVEL		
NONE	70	21.0
PRIMARY	121	36.2
SECONDARY	90	26.9
TERTIARY	53	15.9
OCCUPATION		
PEASANT FARMER	121	36.2
TRADER	63	18.9
HOUSEWIFE	61	18.3
CIVIL SERVANT	25	7.5
NONE	24	7.2
CASUAL LABOURER	20	6.0
RETIRED	17	5.1
SECURITY PERSONNEL	2	0.6
ACCOUNTANT	1	0.3
ALCOHOL USE		
NO	315	94.3
YES	19	5.7
SMOKING TOBACCO		
NO	332	99.4
YES	2	0.6
FOSSIL FUEL USE		
YES	316	94.6
NO	18	5.4

Table 2: Clinical and Biophysical Characteristics of Study Participants

Characteristic	Frequency (n)	Percentage (%)
Nasal Congestion While Sleeping		
Yes	114	34.1
No	220	65.9
Sleeping Position		
Side	290	86.8
Supine	37	11.1
Abdomen/Stomach	7	2.1
Anthropometric Measurements		
Neck circumference (cm)	Mean = 36.75 ± 2.96	—
Waist circumference (cm)	Mean = 100.26 ± 14.31	—
Hip circumference (cm)	Mean = 108.79 ± 11.26	—
Waist-to-hip ratio (WHR)	Mean = 0.93 ± 0.08	—
Mallampati Score		
Class I	236	70.7
Class II	84	25.1
Class III	14	4.2
Glycemic Control (HbA1c %)		
Mean HbA1c	9.45 ± 2.59	—
Optimal (<7)	65	19.5
Good (7.1–8.0)	78	23.4
Poor (8.1–9.9)	68	20.4
Very Poor (>10)	123	36.8
Nasal Obstruction		
Normal	227	68.0
Deviated Nasal Septum	70	21.0
Nasal Polyps	37	11.1
History of Hypertension		
Yes	198	59.3
No	136	40.7
Snoring		
Yes	143	42.8
No	187	56.0
Don’t know	4	1.2

OBJECTIVE 1: PREVALENCE OF OSA RISK

Table 3: Obstructive Sleep Apnea (OSA) Risk

OSA Risk	Frequency (n)	Percentage (%)
Low Risk	196	58.7
High Risk	138	41.3

According to the Berlin Questionnaire, 58.7% of the participants were categorized as low risk for obstructive sleep apnea (OSA), while 41.3% were at high risk.

OBJECTIVE 2: FACTORS ASSOCIATED WITH OSA

Table 4: OSA Risk and Gender

Gender	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Female	127	88	215	0.847
Male	69	50	119	

There was no statistically significant association between gender and risk of obstructive sleep apnea (OSA) among type 2 diabetes patients ($\chi^2 = 0.037$, $p = 0.847$). Both males and females had similar distributions across low and high OSA risk categories.

Table 5: OSA Risk and Marital Status

Marital Status	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Married	162	106	268	0.015
Single	19	8	27	
Widowed	15	24	39	

A statistically significant association was observed between marital status and OSA risk ($\chi^2 = 8.443$, $p = 0.015$). Married individuals constituted the majority in both risk groups, but widowed participants had a higher proportion in the high-risk group compared to other marital statuses.

Table 6: OSA Risk and Educational Level

Education Level	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
None	34	36	70	0.074
Primary	73	48	121	
Secondary	51	39	90	
Tertiary	38	15	53	

Educational level did not show a statistically significant association with OSA risk ($\chi^2 = 6.941$, $p = 0.074$). However, a trend was observed where participants with tertiary education were less represented in the high-risk category.

Table 7: OSA Risk and Sleeping Position

Sleeping Position	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Abdomen	2	3	5	0.100
Side	176	114	290	
Stomach/Abdomen	2	0	2	
Supine	16	21	37	

Although side sleeping was the most common position among both risk groups, sleeping position was not significantly associated with OSA risk ($\chi^2 = 6.247$, $p = 0.100$).

Table 8: OSA Risk and Mallampati Score

Mallampati Score	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
1	151	85	236	0.009
2	38	46	84	
3	7	7	14	

There was a statistically significant association between Mallampati score and OSA risk ($\chi^2 = 9.432$, $p = 0.009$). Participants with higher Mallampati scores were more likely to be in the high OSA risk category.

Table 9: OSA Risk and Nasal Obstruction

Nasal Obstruction	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
DNS	20	50	70	<0.001
Nasal Polyps	19	18	37	
Normal	157	70	227	

A highly significant association was found between nasal obstruction and OSA risk ($\chi^2 = 37.280$, $p < 0.001$). Participants with a deviated nasal septum (DNS) had a notably higher prevalence of high OSA risk compared to those with normal nasal anatomy.

Table 10: OSA Risk and HbA1c Category

HbA1c Category	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Optimal (<7)	36	29	65	0.005
Good (7.1–8)	52	26	78	
Poor (8.1–9.9)	28	40	68	
Very Poor (>10)	80	43	123	

Glycemic control as measured by HbA1c categories, was significantly associated with OSA risk ($\chi^2 = 12.988$, $p = 0.005$). A higher proportion of participants with poor and very poor glycemic control fell into the high OSA risk category.

Table 11: OSA Risk and BMI Category

BMI Category	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Underweight (<18)	6	0	6	<0.001
Normal (18.1–25)	77	25	102	
Overweight (25.1–30)	75	67	142	
Obese (>30)	36	44	80	

BMI category showed a strong and statistically significant association with OSA risk ($\chi^2 = 24.318$, $p < 0.001$). Overweight and obese individuals had a higher likelihood of being at high risk for OSA.

Table 12 OSA Risk and Age Group

Age Group	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
<30 years	0	1	1	0.015
31–40 years	33	10	43	
41–50 years	32	14	46	
51–60 years	42	32	74	
>60 years	89	81	170	

Age was significantly associated with OSA risk ($\chi^2 = 12.375$, $p = 0.015$). The prevalence of high OSA risk increased with age, particularly among participants older than 50 years.

Table 13: OSA Risk and Occupation

Occupation	Low OSA Risk (n)	High OSA Risk (n)	Total (n)	p-value
Accountant	0	1	1	0.343
Casual Labourer	11	9	20	
Civil Servant	20	5	25	
Housewife	38	23	61	
None	13	11	24	
Peasant Farmer	67	54	121	
Retired	9	8	17	
Security Personnel	2	0	2	
Trader	36	27	63	

No statistically significant association was observed between occupation and OSA risk ($\chi^2 = 8.996$, $p = 0.343$). While peasant farmers and traders formed the largest occupational groups, their OSA risk distribution was proportional.

ASSOCIATION TABLES

Pearson Correlation Between HbA1c Control (Binary) and OSA Risk

Variables	Pearson Correlation (r)	p-value	N
HbA1c vs OSA Risk	0.050	0.361	334

A Pearson correlation was conducted to assess the relationship between glycemic contro; using HbA1c control and risk of obstructive sleep apnea (OSA).

The analysis showed a **very weak positive correlation** between poor glycemic control and high OSA risk ($r = 0.050$), which was **not statistically significant** ($p = 0.361$). This suggests that there is no meaningful linear relationship between HbA1c control and OSA risk in this population.

Spearman's Rank Correlation Between HbA1c Control (Binary) and OSA Risk

Variables	Spearman's rho (ρ)	p-value	N
HbA1c Binary vs OSA Risk	0.050	0.361	334

A Spearman's rank-order correlation was performed to assess the monotonic relationship between glycemic control (HBA1C) and OSA risk.

The result showed a **very weak positive correlation** ($\rho = 0.050$), which was **not statistically significant** ($p = 0.361$). This indicates that there is no meaningful monotonic relationship between glycemic control and OSA risk among the study participants.

BINARY LOGISTIC

Variable	B	S.E.	Wald	df	p-value	Adjusted Odds Ratio (AOR)	95% CI for AOR
HbA1c	0.207	0.226	0.840	1	0.359	1.230	0.790 – 1.913

A binary logistic regression was performed to assess the relationship between glycemic control (HbA1c) and the risk of obstructive sleep apnea (OSA). The model was not statistically significant, $\chi^2(1) = 0.843$, $p = 0.359$, indicating that HbA1c control was not a significant predictor of OSA risk.

Participants with poor glycemic control had **1.23 times higher odds** of being at high risk of OSA compared to those with good or optimal control. However, the association was **not statistically significant** ($p = 0.359$). Thus, no meaningful relationship could be confirmed between HbA1c control and OSA risk in this model.

MULTIVARIATE ANALYSIS

Predictor Variable	AOR (Exp(B))	95% CI for AOR	p-value
HbA1c (poor control)	2.28	1.25 – 4.13	0.007
BMI: Overweight (25.1–30)	1.48	1.37 – 3.21	<0.001
Hypertension (Yes)	0.20	0.10 – 0.39	<0.001
Nasal Obstruction: DNS	5.98	2.69 – 13.26	<0.001
Nasal Obstruction: Polyps	3.63	1.45 – 9.07	0.006

A multivariate logistic regression model was used to assess the independent predictors of high risk for obstructive sleep apnea (OSA) among type 2 diabetes patients. The overall model was statistically significant ($\chi^2 = 125.893$, $df = 30$, $p < 0.001$), indicating good model fit. The model explained approximately **43% of the variance** in OSA risk (Nagelkerke $R^2 = 0.427$) and correctly classified **77.3%** of cases.

Key findings include:

- **Poor glycemic control (HbA1c ≥8%)** was significantly associated with a **2.3-fold increased risk** of high OSA risk (AOR = 2.28, 95% CI: 1.25–4.13, *p* = 0.007).

- Being **overweight (BMI 25.1–30)** was associated with a **significantly lower adjusted odds** of high OSA risk compared to the reference group, although this result may reflect complex model dynamics or interactions (*AOR* = 0.175, $p < 0.001$).

- Having a history of **hypertension** was associated with a **reduced odds** of high OSA risk (*AOR* = 0.20, 95% CI: 0.10–0.39, $p < 0.001$), which is counterintuitive and may suggest confounding or model misclassification.

- Participants with **deviated nasal septum (DNS)** had **nearly six times** higher odds of being at high risk for OSA (*AOR* = 5.98, $p < 0.001$), while those with **nasal polyps** also had significantly increased odds (*AOR* = 3.63, *p* = 0.006).

Other variables such as gender, age group, marital status, education level, occupation, sleeping position, and Mallampati score were not independently significant in the adjusted model.

CHAPTER FIVE

5.0 DISCUSSIONS, STUDY STRENGHTS AND LIMITATIONS.

This chapter discusses the findings of the study on the prevalence of obstructive sleep apnea risk and associated factors in patients with type 2 diabetes mellitus (T2DM) attending the diabetes outpatient clinic at Moi Teaching and Referral Hospital (MTRH), Eldoret, Kenya. The discussion compares the findings with current literature, interprets the important findings and examines potential rationales for noted correlations or their absence. The ramifications for clinical practice and public health are also addressed.

5.2 Prevalence of OSA Risk

Our study revealed that 41.3% of T2DM patients were classified as high risk for OSA according to the Berlin Questionnaire. This prevalence corresponds with earlier studies from diverse settings. Research conducted in India and Egypt among patients with diabetes indicated that the prevalence rates of obstructive sleep apnea (OSA) risk ranged from 35% to 48% (Khalyfa et al., 2011; Sharma et al., 2006). Another study by Pamidi and Tasali (2012) also showed that almost one-third of people with type 2 diabetes were at increased risk for undiagnosed OSA.

These findings confirm that OSA is a common yet unrecognized comorbidity in diabetic populations, necessitating regular screening in diabetes management.

The high risk of obstructive sleep apnea in this population can be linked to a number of overlapping risk factors, including age, obesity, poor glycemic management, and upper airway structural abnormalities. Furthermore, the use of the Berlin Questionnaire, which includes major risk factors such as snoring, daytime drowsiness, and hypertension, likely increased the sensitivity of recognizing those at risk, even in the absence of rigorous polysomnographic testing (Netzer et al. 1999).

5.3 Factors Associated with OSA Risk

5.3.1 Age

The study found a statistically significant link between age and risk of OSA ($p = 0.015$). Participants over the age of 50 were more likely to fall into the high-risk category. This finding is consistent with previous research indicating that OSA prevalence increases with age, particularly beyond midlife (Bixler et al., 2001; Heinzer et al., 2015). Airway collapsibility during sleep is caused by age-related structural changes, including decreased upper airway muscle tone and increased pharyngeal fat deposition (Jordan et al., 2014).

5.3.2 Marital Status

A significant correlation was discovered between marital status and OSA risk ($p = 0.015$), with bereaved participants falling disproportionately into the high-risk group. While this finding has not been widely documented in the literature, it is possible that the absence of a bed companion reduces the likelihood of snoring or apneic episodes being observed or reported. Furthermore, social isolation among widowed adults may correlate to decreased healthcare-seeking behavior and delayed OSA discovery (Yaffe et al., 2014).

5.3.3 Glycemic Control

In our study, glycemic control, as measured by HbA1c levels, was found to be significantly associated with the risk of obstructive sleep apnea (OSA). Multivariate logistic regression demonstrated that participants with poor glycemic control (HbA1c $\geq 8\%$) had a 2.3-fold increased odds of being categorized as high risk for OSA (AOR = 2.28, 95% CI: 1.25–4.13, $p = 0.007$). Although correlation analysis did not reveal a statistically significant linear association between HbA1c and OSA risk, the multivariate model distinctly demonstrated that glycemic control was an independent predictor when accounting for other variables.

This finding corresponds with an expanding collection of literature indicating a bidirectional link between obstructive sleep apnea and inadequate glycemic control. Untreated obstructive sleep apnea (OSA) adversely affects glucose metabolism through mechanisms including intermittent hypoxia, oxidative stress, systemic inflammation, and stimulation of the autonomic nervous system (Bonsignore et al., 2019) These alterations diminish insulin sensitivity and pancreatic β-cell functionality, exacerbating the onset and progression of insulin resistance, a defin ing characteristic of type 2 diabetes mellitus (T2DM) (Reutrakul & Mokhlesi, 2017). Conversely, persons with inadequately managed diabetes may exhibit heightened vulnerability to obstructive sleep apnea due to neuropathy impacting the upper airway musculature, modifications in central respiratory regulation, and augmented adipose accumulation in the cervical and trunk areas, particularly in the context of obesity. Research indicates that diabetic autonomic neuropathy may hinder the neuronal regulation of pharyngeal muscles, consequently worsening airway collapsibility during sleep (Sharma 2010).

Furthermore, increased HbA1c levels have been associated with the severity of obstructive sleep apnea in multiple studies. One such study by Aronsohn et al. (2010), showed that patients with moderate to severe obstructive sleep apnea (OSA) exhibited significantly elevated HbA1c levels as compared to individuals with mild or no OSA, irrespective of body mass index (BMI) and age.

In our study, a significant trend was identified wherein people exhibiting "very poor" glycemic control (HbA1c > 10%) represented the largest proportion (36.8%) of the sample, with many classified within the high OSA risk category. The findings are clinically significant as ongoing hyperglycemia associated with undetected obstructive sleep apnea may lead to the onset of diabetes-related comorbidities, including nephropathy, retinopathy, and cardiovascular disease. The confluence of obstructive sleep apnea and inadequate glycemic control constitutes a high-risk metabolic phenotype necessitating immediate therapeutic intervention (Kent et al., 2014)

Furthermore, the treatment of OSA has been demonstrated to enhance glycemic control. Continuous Positive Airway Pressure (CPAP) therapy, the gold standard in the treatment of obstructive sleep apnea (OSA), has shown moderate yet consistent decreases in HbA1c levels in individuals with type 2 diabetes mellitus (T2DM), especially with strong adherence to the therapy (Sharma et al., 2011). The enhancements in glycemic measures after OSA treatment reinforce the concept that sleep-disordered breathing significantly contributes to the pathophysiology of hyperglycemia.

Despite these findings, it is important to acknowledge that certain randomized controlled trials have yielded inconclusive results about the effect of CPAP on HbA1c levels, particularly in patients with mild to severe OSA or inadequate CPAP adherence (Pamidi et al., 2015). Consequently, although existing evidence indicates a correlation between inadequate glycemic control and obstructive sleep apnea (OSA), long-term, rigorously controlled intervention studies are necessary to establish the causative relationship and determine if OSA management should be routinely incorporated into diabetes care.

The results of this investigation underscore the necessity for OSA screening in T2DM patients exhibiting inadequate glycemic control, particularly in resource-constrained environments where the prevalence of undiagnosed sleep apnea is presumably elevated. Incorporating OSA screening instruments like the Berlin Questionnaire or

STOP-BANG into standard diabetes therapy may facilitate the prompt identification of at-risk individuals, enhance metabolic control, and reduce complication rates.

5.3.4 Body Mass Index (BMI)

A significant correlation was discovered between BMI and the risk of OSA ($p < 0.001$). The incidence of elevated OSA risk was highest among overweight and obese adults. These findings reinforce the well-established role played by obesity in the pathogenesis of obstructive sleep apnea (OSA). Excess adipose tissue in the neck region and upper airway enhances airway collapsibility, whereas visceral fat induces systemic inflammation and diminishes lung capacities (Peppard et al., 2000）

5.3.5 Nasal Obstruction

Individuals with deviated nasal septum (DNS) and nasal polyps exhibited markedly elevated odds of belonging to the high-risk category for obstructive sleep apnea (OSA). The DNS significantly influenced the probability of OSA, exhibiting an approximate sixfold increase (AOR = 5.98, $p < 0.001$). Anatomical anomalies can elevate upper airway resistance during sleep, leading to snoring and hypopneas (Koutsourelakis et al., 2008). The robust statistical correlation identified herein advocates for the regular evaluation of nasal patency during obstructive sleep apnea screening.

5.3.6 Mallampati Score

The Mallampati classification demonstrated a strong correlation with the risk of obstructive sleep apnea (OSA) ($p = 0.009$), indicating that participants with elevated Mallampati scores (Class II or III) were more likely to be at high risk. This aligns with the current literature, which identifies the Mallampati score as a dependable indicator of upper airway obstruction and the likelihood of obstructive sleep apnea (Kirkness, Punjabi et al. 2006).

5.4 Non-significant Associations

Certain characteristics, including gender, occupation, level of education, and sleeping posture, exhibited no significant correlation with OSA risk in this cohort. The absence of gender correlation contradicts other research indicating a higher frequency of OSA in males (Franklin & Lindberg, 2015) potentially reflecting disparities in symptom perception or underreporting among females. The lack of correlation between sleeping position and OSA risk may be attributed to the significant prevalence of side-sleeping in the sample, which serves as a protective posture against airway collapse.

5.5 Interpretation of Multivariate Model

The multivariate regression model accounted for 43% of the variance in OSA risk (Nagelkerke $R^2 = 0.427$) and accurately classified 77.3% of patients. This indicates that although key predictors including inadequate glycemic management, nasal obstruction, and BMI are essential, additional unmeasured factors may also affect the likelihood of OSA. These factors may encompass craniofacial anatomy, genetic susceptibility, levels of physical activity, and nutritional practices.

Hypertension was found to have an inverse association with the probability of obstructive sleep apnea (AOR = 0.20, $p < 0.001$), contradicting the established bidirectional link between obstructive sleep apnea and hypertension (Drager et al., 2013). This paradoxical conclusion may result from misclassification, treatment-related confounding, or survivor bias, underscoring the complex nature of evaluating observational data.

5.6 Strengths and Limitations

This study possesses numerous significant strengths. This is the inaugural study on sleep medicine, particularly obstructive sleep apnea, conducted at Moi Teaching and Referral Hospital (MTRH), an area that has not been extensively studied in our institution and by extension our country.

A key strength of this study is the sample size of 334 participants which offered adequate statistical power to identify significant correlations between risk factors and obstructive sleep apnea (OSA) risk in patients with type 2 diabetic mellitus (T2DM).

The sample's diversity derived from both urban and rural environments within the Moi Teaching and Referral Hospital (MTRH) catchment area enhances the generalizability of the findings to comparable diabetic populations in low- and middle-income nations.

A further strength lies in the utilization of validated tools, including the Berlin Questionnaire for evaluating OSA risk and standardized metrics such as the Mallampati score, neck and waist circumference, and glycated hemoglobin (HbA1c) levels. Such tools have been extensively utilized in both research and clinical settings, ensuring reliability and comparability of findings with other global studies.

The study's systematic data gathering method, compliance with uniform measuring methodologies, and stringent statistical analysis enhance its internal validity.

The thorough evaluation of various potential risk factors including sociodemographic data, lifestyle choices (e.g., alcohol, tobacco), anatomical characteristics (e.g., nasal obstruction), and metabolic markers (e.g., HbA1c, BMI) offers a complete understanding of OSA risk in this diabetic population. This multifactorial research identifies independent determinants of OSA risk, providing valuable insights for tailored screening programs.

5.7 Study Limitations

This study provides significant insights into the association between obstructive sleep apnea and type 2 diabetes mellitus; nevertheless, some limitations must be acknowledged when interpreting the findings:

The cross-sectional design of the study limits any causal inferences regarding the association between OSA risk and its associated factors. While associations were identified, the directionality of these relationships remains ambiguous. Poor glycemic control might increase the risk of obstructive sleep apnea (OSA), but undetected OSA may also adversely affect glycemic results, exemplifying a classic case of bidirectionality.

Secondly, employing a screening questionnaire, such as the Berlin questionnaire instead of polysomnography (PSG), which is the gold standard for detecting obstructive sleep apnea (OSA), may have resulted in classification bias. The Berlin Questionnaire, although validated for use in resource poor setups, may inaccurately assess risk due to the reliability of self-reported data and the patient's awareness of symptoms like snoring and fatigue. Objective confirmation by polysomnography or home sleep testing would have enhanced diagnosis accuracy.

CHAPTER SIX

6.0 CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

This study aimed to ascertain the prevalence OSA risk status and associated risk factors in patients with type 2 diabetes mellitus (T2DM) at Moi Teaching and Referral Hospital (MTRH), Eldoret. 41.3% of participants were identified as being at high risk for obstructive sleep apnea (OSA), suggesting that nearly half of the patients with diabetes may be unknowingly affected by this potentially catastrophic and underdiagnosed disorder.

The results indicate that OSA is prevalent among patients with T2DM and is significantly associated with several metabolic, anatomical, and demographic variables. Inadequate glycemic management, as shown by increased HbA1c levels, was identified as a major and independent predictor of OSA risk. This finding reinforces the increasing evidence indicating a bidirectional link between sleep-disordered breathing and glucose metabolism.

Other factors substantially correlated with elevated OSA risk included advanced age, elevated body mass index (BMI), anatomical nasal abnormalities (particularly deviated nasal septum), and higher Mallampati scores, indicative of increased upper airway obstruction. Conversely, variables like gender, occupation, education level, and sleeping position exhibited no statistically significant relationships in the adjusted analysis.

6.2 Recommendations

- Incorporate routine OSA screening into standard management of diabetes. Healthcare practitioners overseeing T2DM patients should consistently evaluate OSA risk utilizing simple, validated tools, such as, the Berlin Questionnaire. This is especially crucial for patients exhibiting inadequate glycemic control, or those with obesity, nasal obstruction, or having symptoms such as fatigue and snoring.
- Implement interdisciplinary care strategies: Collaboration among diabetologists, sleep medicine specialists, ENT surgeons, and nutritionists can improve the holistic management of individuals at elevated risk for OSA. Addressing anatomical variables like nasal blockage in conjunction with metabolic regulation can produce improved results.
- Educate patients about sleep health: Numerous individuals lack awareness regarding the health hazards linked to inadequate sleep quality and obstructive sleep apnea (OSA). Patient education regarding symptom recognition, the significance of sleep hygiene, and current treatment modalities (e.g., CPAP, weight reduction) should be integrated into diabetes counseling.
- It is essential to establish specific guidelines in hospitals and diabetes clinics that specify when and how to to screen patients for obstructive sleep apnea.
- Invest in accessible diagnostic resources and set up a sleep laboratory in Moi Teaching and referral hospital and other regional health facilities in the country. Given that comprehensive polysomnography may be challenging for all patients, particularly in resource-constrained environments, the utilization of portable sleep monitors or home-based sleep evaluations should be considered to enhance diagnostic accessibility.

- Educate physicians and allied health professionals in sleep medicine to provide specialized and proficient treatments for individuals with obstructive sleep apnea.
- Future research should utilize prospective designs to elucidate the temporal link between obstructive sleep apnea (OSA) and glycemic control, along with the effects of OSA treatment on diabetes outcomes.

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APPENDICES

Appendix 1: Questionnaire

A. SOCIAL DEMOGRAPHIC FACTORS. ***Please tick where applicable***

1. Age [ ]

2. Gender

a. Male [ ]

b. Female [ ]

3. Are you married?

a. Yes [ ]

b. No [ ]

4. Education level of respondent

a. None [ ]

b. primary [ ]

c. secondary [ ]

d. Tertiary [ ]

5. Occupation

a. peasant farmer [ ]

b. casual laborer [ ]

c. housewife [ ]

d. student [ ]

e. civil servant [ ]

f. trader [ ]

other (specify)……………………………………………….

6. Do you take alcohol

a. Yes [ ]

b. No [ ]

7. If yes to question 4 above, how often do you take alcohol?

a. Daily [ ]

b. Once a week [ ]

c. once a month [ ]

d. Rarely [ ]

8. If yes to question 4 above, after how many hours do you go to sleep after taking

alcohol?

a. less than 1 hour [ ]

b. 1 hour [ ] **c.** 2 hours [ ]

d. 3 hours [ ]38

e. 4 hours [ ]

f. more than 4 hours [ ]

9. Do you smoke tobacco

a. Yes [ ]

b. No [ ]

10. If yes to question 7 above, how much do you smoke?

a. a packet / day [ ]

b. 10 sticks / day [ ]

c. less than 10 sticks / day [ ]

d. 10-20 sticks / day [ ] **e**. other (specify)………………………………………..

11. Do you use any fossil fuel e.g firewood or charcoal as a source of energy for cooking?

a. yes [ ]

b. no [ ]

12. Do you get nasal congestion while sleeping?

a. Yes [ ]

b. No [ ]

13. **If yes to question 12 above, do you suffer from any condition such as vasomotor rhinitis that could cause nasal blockage?**

a. Yes [ ]

b. No [ ]

13. Common sleeping position

a. supine (on the back) [ ]

b. side (on left or right) [ ]

c. stomach/ abdomen [ ]

B. CLINICAL EXAMINATIONS

14. neck circumference in cm ……….

15. waist circumference in cm………………

16. hip circumference in cm …………

15. Waist-hip ratio………………**Mallampati score**

16. Throat abnormalities: enlarged tonsils, abnormal pharyngeal anatomy, crowded mouth with

low-extending soft palate, large uvula, large tongue (macroglossia).

The Mallampti score: Class I…………Class II……….Class III………..ClassIV……………

17. Nasal obstruction

a. deviated nasal septum…………….

b. nasal polyps………………………

c. normal…………………………….

18. Blood pressure measurements

a. systolic………………

b. diastolic…………….

Appendix 2: Berlin Questionnaire

1. Complete the following:

Height: _____________ Weight: ____________

Age: ____________ Gender: ____M ____F

2. Do you snore?

_____Yes

_____No

_____Don't know

If you snore:

3. Your snoring is…

_____ Slightly louder than breathing

_____ As loud as talking

_____ Louder than talking

_____ Very loud, can be heard in adjacent rooms

4. How often do you snore?

_____ Nearly every day

_____ 3-4 times a week

_____ 1-2 times a week

_____ 1-2 times a month

_____ never or nearly never

5. Has your snoring ever bothered other people?

_____ Yes

_____ No

6. Has anyone noticed that you quit breathing during your sleep?

______ Nearly every day.

______ 3-4 times a week

______ 1-2 times a week

______ 1-2 times a month

______ never or nearly never

7. How often do you feel tired or fatigued after your sleep?

______ Nearly every day

______ 3-4 times a week

______ 1-2 times a week

______ 1-2 times a month

______ never or nearly never

8. During your wake time, do you feel tired, fatigued, or not up to par?

______ Nearly every day

______ 3-4 times a week

______ 1-2 times a week

______ 1-2 times a month

______ never or nearly never

9. Have you ever nodded off or fallen asleep while driving a vehicle?

______ Yes

______ No

_____ If yes, how often does it occur?

_____ Nearly every day.

_____ 3-4 times a week

_____ 1-2 times a week

_____ 1-2 times a month

_____ never or nearly never

10. Do you have high blood pressure?

_____ Yes

_____ No

_____ Don't know

11. BMI (Body mass index) = ______________

Scoring the Berlin Questionnaire

The questionnaire consists of 3 categories related to the risk of having sleep apnea. Patients can be classified into High Risk or Low Risk based on their responses to the individual items and their overall scores in the symptom categories.

Categories and Scoring:

Category 1: items 2, 3, 4, 5, and 6;

Item 2: if ‘Yes’, assign 1 point

Item 3: if either of the last two options is the response, assign 1 point

Item 4: if either of the first two options is the response, assign 1 point

Item 5: if ‘Yes’ is the response, assign 1 point

Item 6: if either of the first two options is the response, assign 2 points

Add points. Category 1 is positive if the total score is 2 or more points.

Category 2: items 7, 8, and 9.

Item 7: if either of the first two options is the response, assign 1 point

Item 8: if either of the first two options is the response, assign 1 point

Item 9: if ‘Yes’ is the response, assign 1 point

Add points. Category 2 is positive if the total score is 2 or more points.

Category 3 is positive if the answer to item 10 is ‘Yes’ or if the BMI of the patient is greater

than 30kg/m2. (BMI is defined as weight (kg) divided by height (m) squared, i.e ..., kg/m2).

High Risk: if there are 2 or more categories where the score is positive.

Low Risk: if there is only 1 or no categories where the score is positive.

Additional Question: item 9 should be noted separately.

Berlin Questionnaire - Tafsiri ya Kiswahili

1.Jaza yafuatayo:

Urefu: _____________ Uzito:

____________ Umri: ____________ Jinsia: M____F ____

2. Je, wewe hukoroma?

_____ Ndiyo

_____ Hapana

_____ Sijui

Ikiwa unakoroma:

3. Kukoroma kwako ni…

_____ zaidi ya kupumua

_____ Kama kusema

_____ zaidi ya kusema

_____ zaidi kiasi Cha kuweza kusikika vyumba jirani

4. Wewe hukoroma:

_____ Karibu kila siku

_____ Mara 3-4 kwa wiki

_____ Mara 1-2 kwa wiki

_____ Mara 1-2 kwa mwezi

_____ Kamwe au karibu kamwe

5. Je, kukoroma kwako kumewahi kuwakera watu wengine?

_____ Ndiyo

_____ Hapana

6. Je, mtu yeyote amegundua kuwa unaacha kupumua wakati wa usingizi wako?

_____ Karibu kila siku

_____ Mara 3-4 kwa wiki

_____ Mara 1-2 kwa wiki

_____ Mara 1-2 kwa mwezi

_____ Kamwe au karibu kamwe

7. Mara ngapi unahisi uchovu au uchovu baada ya usingizi wako?

_____ Karibu kila siku

_____ Mara 3-4 kwa wiki

_____ Mara 1-2 kwa wiki

_____ Mara 1-2 kwa mwezi

_____ abadani au karibu abadani

8. Wakati wa kuamka kwako, je,unahisi uchovu, uchovu, au hauko sawa?

_____ Karibu kila siku

_____ Mara 3-4 kwa wiki

_____ Mara 1-2 kwa wiki

_____ Mara 1-2 kwa mwezi

_____ abadani au karibu abadani

9. Je, umewahi kukunjuka au kulala wakati wa kuendesha gari?

_____ Ndiyo

_____ Hapana

_____ Ikiwa ndivyo, mara ngapi hutokea?

_____ Karibu kila siku

_____ Mara 3-4 kwa wiki

_____ Mara 1-2 kwa wiki

_____ Mara 1-2 kwa mwezi

_____ abadani au karibu abadani

10. Je, una shinikizo la damu kubwa?

_____ Ndiyo

_____ Hapana

_____ Sijui

11. BMI (Index ya Mwili wa Misa) = ______________

Appendix 3: Informed Consent

Study Title: **PREVALENCE OF OBSTRUCTIVE SLEEP APNOEA RISK STATUS AMONG AMBULATORY TYPE 2 DIABETES MELLITUS PATIENTS AT MOI TEACHING AND REFERRAL HOSPITAL.**

Name of Principal Investigator(s): Dr. Maalim Abdirahman Adan

Co-investigator(s): Prof. L. Diero and Dr. D. Lagat

Name of Organization: Moi University School of Medicine

Address: P.O Box 4606 030100 Telephone Number: +254 53 2033235

Name of Sponsor/Funding Agency: None

Informed Consent Form for: Obstructive sleep apnea Study Participants

This Informed Consent Form has two parts:

• **Part I**: Information Sheet [to share information about the study with you]

• **Part II**: Certificate of Consent [for signatures if you choose to participate]

PART I: INFORMATION SHEET

Introduction:

You are being asked to take part in a research study. This information is provided to tell you about the study. Please read this form carefully. You will be given a chance to ask questions.

Taking part in this research study is voluntary. Saying no will not affect your rights to health care or any other services. Your treatment/payment or enrollment in any health plans or eligibility for benefits will not be affected if you decide not to take part. You are also free to withdraw from this study at any time. If after data collection you choose to quit, you can request that information provided by you be destroyed under supervision. This would be before data is de-identified and aggregated. You will be notified if new information becomes available about the risks or benefits of this research. You will receive a copy of this form after it is signed

Purpose of the study:

This study aims to find out the prevalence of obstructive sleep apnea risk status among people with diabetes. We also would like to study the factors that are associated with it. The information that will be gathered from this study will help us understand how common obstructive sleep apnea is among those with diabetes thereby diagnosing and managing it appropriately.

Study site: This study is being conducted at the MTRH Diabetes Outpatient Clinic.

Study population:

You are eligible to take part in this research study because you are older than 18 years old and are enrolled in the MTRH diabetes outpatient clinic for management of diabetes.

Study procedures:

This will be a cross-sectional study which means we will complete all study procedures on your day of enrolment in the shortest time possible. We aim to recruit up to 334 participants.

If you agree to participate in the study, the following will be done:

Your social demographic characteristics will be taken and recorded safely.

You will be asked questions using the Berlin questionnaire and you will be scored as either high risk or low risk.

Your blood pressure, Waist, hip and neck circumferences will be measured**.** The most recent measurement of your HBA1C will also be documented. In the event that a recent HBA1C measurement is unavailable, a blood sample will be drawn from your arm to ascertain your HBA1C level. This procedure will aid in understanding your glucose control over the preceding three-month period. There will be no obligation for you to provide payment for this examination. The financial burden will be assumed by us.

Benefits:

There may be no direct benefits to you. However, the study findings will help clinicians managing your diabetes find out how common obstructive sleep apnea in diabetics is, diagnose and manage it accordingly. It will also help us come up with policies for screening of obstructive sleep apnea among diabetes patients so that we have a holistic approach to diabetes care and offer our patients the highest standards of care at MTRH.

Risks/Discomforts:

We will ensure that high standards are applied during our information collection and avoid exposure to any risks to the study participants during the time of study.

Payments and Reimbursements:

No payments or reimbursements will be provided to you for your participation in this study.

Confidentiality:

All reasonable efforts will be made to keep your protected information private and confidential. Using or sharing ("disclosure") of such information will follow National privacy guidelines. By signing the consent document for this study, you are giving permission ("authorization") for the use and disclosure of your study information. We may need to share your protected information with the community advisory board, MTRH//MU-IREC, NACOSTI or the healthcare team. We will retain our research records for at least seven years after the study is completed. At that time, the research information is destroyed by shredding any paper documents, and deleting any computer records. If you decide to withdraw your permission for use of your personal data, contact Dr. Maalim Abdirahman in writing and let them know your decision. At that time, we will stop further collection of any information about you. However, the health information collected before this withdrawal may continue to be used for the purposes of reporting and research quality. You have the right to see and copy your personal information related to the research study for as long as the study team holds this information

Compensation for injury:

We do not anticipate that any injury may occur to you resulting from participating in this study.

PART II: CONSENT OF PARTICIPANT

I have read or have had someone read to me the description of the research study. The investigator or his/her representative has explained the study to me and has answered all the questions I have at this time. I have been told of the potential risks, discomfort, and possible benefits (if any) of the study. I freely volunteer to take part in this study.

[If the participant is illiterate, or for some reason is unable to write, they should provide a thumbprint and a competent witness must be engaged during the consent process]

____________________________ ___________________________ ___________

Name of Participant Signature of participant/Thumbprint Date & Time

____________________________ ___________________________ ___________

Name of Witness [Optional] Signature of Witness Date & Time

____________________________ ___________________________ ___________

Name of the person obtaining consent Signature of person obtaining consent Date & Time

Dr. Maalim Abdirahman ________________________ __________

Printed name of the Principal Investigator Signature of Investigator Date

Contacts for questions about the study

Questions about the study: Dr. Maalim Abdirahman |Phone: 0716038285 |

Email: adoabdirahman@gmail.com

Questions about your rights as a participant: You may contact the Institutional Ethics and Research Committee (MTRH//MU-IREC) 0787723677 or email irec@mtrh.go.ke or irecoffice@gmail.com. The MTRH//MU-IREC is a group of people that review studies for safety and to protect the rights of participants.

FOMU YA KIBALI ILIYOARIFIWA

Anwani Ya Utafiti: KUENEA KWA NA MAMBO YANAYOHUSIANA NA UGONJWA YA APNEA YA USINGIZI MIONGONI MWA WAGONJWA WA KISUKARI AINA YA 2 KATIKA HOSPITALI YA MAFUNZO NA RUFAA YA MOI, ELDORET, KENYA

(Ma)Jina la Mchunguzi Mkuu: Dkt. Maalim Abdirahman

Wachunguzi wenza: Prof L. Diero na Dkt. D. Lagat

Jina la Shirika: Chuo Kikuu cha Moi-Shule ya Matibabu

Anwani: Sanduku la Posta 4606 030100 Nambari ya Simu: +254 53 2033235

Jina la Mfadhili/Wakala wa Ufadhili: Hakuna

Fomu ya Kibali ya: Kuenea kwa na mambo yanayohusiana na ugonjwa wa apnea ya usingizi miongoni mwa wagonjwa ya kisukari aina ya 2 katika hospitali ya MTRH

Hii Fomu ya Kibali ina sehemu mbili:

• **Sehemu ya I**: Karatasi ya Habari [kushiriki habari na wewe kuhusu utafiti]

• **Sehemu ya II**: Cheti cha Kibali [ya saini ikiwa utachagua kushiriki]

SEHEMU YA I: KARATASI YA HABARI

Utangulizi:

Unaulizwa kushiriki katika somo la utafiti. Taarifa hii inapeanwa kukuambia kuhusu utafiti. Tafadhali soma fomu hii kwa makini. Utapewa nafasi kuuliza maswali.

Kushiriki katika huu utafiti ni kwa hiari. Kusema hapana haitaathiri haki zako kwa huduma ya afya au huduma zingines. Matibabu yako/malipo au kujiandikisha kwa mpango yoyote ya afya au kustahiki kwa manufaa haitaathiriwa ikiwa utaamua kutoshiriki. Pia uko huru kujiondoa kutoka kwa utafiti huu wakati wowote. Ikiwa baada ya ukusanyaji wa data utaamua kujiondoa, unaweza omba kwamba taarifa uliyopeana yaharibiwe chini ya usimamizi. Hii itafanyika kabla ya data kutotambuliwa na kujumlishwa. Utajulishwa ikiwa habari mpya inapatikana kuhusu hatari au manufaa ya utafitu huu. Utapokea nakala ya fomu hii baada ya kutiwa saini

Madhumuni ya utafiti: Tafiti hii inalenga kupata kama ugonjwa wa apnea ya usingizi ni changamoto kubwa au la miongoni mwa wagonjwa wa kisukari. Taarifa ambazo zitapatikana kutoka tafiti hii zitasaidia kutambua uhusiano kati ya mambo hayo mawili na kuchangia katika kutengeneza sera za kusimamia kisukari.

Eneo la Utafiti: Utafiti huu unafanyika katika kliniki ya ugonjwa wa kisukari ya MTRH.

Idadi ya Watu kwa Utafiti:

Unastahili kushiriki katika somo hili la utafiti kwa sababu wewe ni mkubwa zaidi ya miaka 18, na umejiandikisha katika kliniki ya ugonjwa ya kisukari ya MTRH.

Utaratibu wa Utafiti:

Hii itakuwa utafiti wa sehemu mbali ambayo inamaanisha tutakamilisha taratibu zote za masomo katika siku yako ya usajili kwa muda mfupi iwezekanavyo. Tunalenga kuandikisha hadi washiriki 334.

Ikiwa utakubali kushiriki katika utafiti huu, utafanya yafuatayo:

Wafanyakazi wa utafiti

Manufaa:

Hautaweza kupata manufaa yoyote ya moja kwa moja kutoka kwa utafiti..

Hatari/Usumbufu:

Tutakuwa tunakusanya habari ya afya na demografia ya jamii ambayo ni sehemu ya mambo ya kila siku ya huduma.

Malipo na Marejesho:

Hakuna malipo au marejesho yatakayotolewa kwako kwa ushiriki wako katika utafiti huu.

Usiri:

Jitihada zinazowezekana zitafanywa ili kuweka habari yako iliyolindwa ya kibinafsi na ya siri. Kutumia au kushiriki ("ufichuzi") ya habari hiyo itafuata miongozo ya kibinafsi ya Kitaifa. Kwa kusaini hati ya idhini ya utafiti huu, unatoa ruhusa ("idhini") kwa matumizi na ufichuzi wa maelezo yako ya utafiti. Tunaweza kuhitaji kushiriki maelezo yako yaliyolindwa na bodi ya ushauri wa jamii, MTRH / / MU-IREC, NACOSTI au timu ya huduma ya afya. Tutahifadhi rekodi zako za utafiti kwa angalau miaka sita baada ya utafiti kukamilika. Kwa wakati huo, habari za utafiti huharibiwa kwa kukatakata karatasi yoyote, na kufuta rekodi zozote za kompyuta. Ukiamua kuondoa ruhusa yako ya matumizi ya data yako ya kibinafsi, wasiliana na Dkt. Maalim Abdirahman kwa maandishi na uwajulishe uamuzi wako. Wakati huo, tutaacha ukusanyaji zaidi wa habari yoyote kukuhusu. Hata hivyo, taarifa za afya zilizokusanywa kabla ya kujiondoa huu zinaweza kuendelea kutumika kwa madhumuni ya kutoa taarifa na ubora wa utafiti. Una haki ya kuona na kunakili maelezo yako ya kibinafsi kuhusiana na somo la utafiti kwa muda mrefu kama timu ya utafiti inashikilia habari hii

Fidia kwa majeraha:

Hatutarajii jeraha lolote litatokea kwako kutokana na kushiriki katika utafiti huu.

SEHEMU YA II: IDHINI YA MSHIRIKI:

Nimesoma au mtu amenisomea maelezo ya somo la utafiti. Mchunguzi au mwakilishi wake amenielezea utafiti kwangu na amejibu maswali yangu yote niliyonayo kwa wakati huu. Nimeambiwa hatari zinazowezekana, usumbufu na faida zinazowezekana (ikiwa kunayo) ya utafiti. Najitolea kwa hiari kushiriki katika utafiti huu.

[Ikiwa mshiriki hajasoma, au kwa sababu fulani hawezi kuandika, wanafaa kupeana kidole gumba na shahidi mwenye uwezo lazima wahusishwe kwa mchakato wa kibali]

__________________________ _________________________ __________

Jina la Mshiriki Saini ya mshiriki/Kidole Gumba Tarehe na Saa

__________________________ __________________________ __________

Jina la Shahidi [Hiari] Saini ya Shahidi Tarehe na Saa

_________________________ ________________________ __________

Jina la mtu anayetafuta idhini Saini ya mtu anayetafuta idhini Tarehe na Saa

Daktari Maalim Abdirahman ________________________ __________

Jina lililochapishwa la mchunguzi Saini ya Mchunguzi Tarehe

Maelezo ya mawasiliano kwa maswali kuhusu utafiti

Maswali kuhusu utafiti: Daktari Maalim Abdirahman |Simu: 0716038285 |

Barua pepe: adoabdirahman@gmail.com

Maswali kuhusu haki zako kama mshiriki: Unaweza kuwasiliana na Kamati ya Kitaasi ya Maadili na Utafiti (MTRH//MU-IREC) 0787723677 au barua pepe irec@mtrh.go.ke au irecoffice@gmail.com. The MTRH//MU-IREC ni kikundi cha watu wanaohakiki utafiti kwa usalama na kulinda haki ya wanaoshiriki

Appendix 4: IREC Approval

MTRH/MU-INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471/2/3

MOI UNIVERSITY
COLLEGE OF HEALTH SCIENCES
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

Reference: IREC/555/2023
Approval Number: 0004643

14th December, 2023

Dr. Maalim Abdirahman Adan,
Moi University,
School of Medicine,
P.O. Box 4606-30100,
ELDORET-KENYA.

Dear Dr. Maalim,

PREVALENCE OF OBSTRUCTIVE SLEEP APNEA RISK AMONG AMBULATORY TYPE 2 DIABETES MELLITUS PATIENTS AT MOI TEACHING AND REFERRAL HOSPITAL

This is to inform you that ***MTRH/MU-IREC*** has reviewed and approved the above referenced research proposal. Your application approval number is ***FAN: 0004643.*** The approval period is **14th December, 2023- 13th December, 2024.** This approval is subject to compliance with the following requirements;

i. Only approved documents including (informed consents, study instruments, Material Transfer Agreements (MTA) will be used.
ii. All changes including (amendments, deviations, and violations) are submitted for review and approval by ***MTRH/MU-IREC.***
iii. Death and life threatening problems and serious adverse events or unexpected adverse events whether related or unrelated to the study must be reported to ***MTRH/MU-IREC*** within 72 hours of notification.
iv. Any changes, anticipated or otherwise that may increase the risks or affected safety or welfare of study participants and others or affect the integrity of the research must be reported to ***MTRH/MU-IREC*** within 72 hours.
v. Clearance for export of biological specimens must be obtained from ***MOH at the recommendation of NACOSTI*** for each batch of shipment.
vi. Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. Attach a comprehensive progress report to support the renewal.
vii. Submission of an executive summary report within 90 days upon completion of the study to ***MTRH/ MU-IREC.***

Prior to commencing your study; you will be required to obtain a research license from the National Commission for Science, Technology and Innovation (NACOSTI) https://oris.nacosti.go.ke and other relevant clearances from study sites including a written approval from the CEO-MTRH which is mandatory for studies to be undertaken within the jurisdiction of Moi Teaching & Referral Hospital (MTRH) and its satellites sites.

Sincerely,

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
14 DEC 2023
APPROVED

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc CEO - MTRH Dean - SOP Dean - SOM
Principal - CHS Dean - SON Dean - SOD

Appendix 5: Hospital Approval (MTRH)

An ISO 9001:2015 Certified Hospital

MOI TEACHING AND REFERRAL HOSPITAL

Telephone: (+254)-0532033471/2/3/4
Fax: 0532061749
Email: ceo@mtrh.go.ke/ceooffice@mtrh.go.ke

NANDI ROAD
P.O. BOX 3-30100
ELDORET,KENYA

Ref: ELD/MTRH/R&F/10/2/V.2/2010

14th December, 2023

Dr. Maalim Abdirahman Adan,
Moi University,
School of Medicine,
P.O. Box 4606-30100,
ELDORET-KENYA.

PREVALENCE OF OBSTRUCTIVE SLEEP APNEA RISK AMONG AMBULATORY TYPE 2 DIABETES MELLITUS PATIENTS AT MOI TEACHING AND REFERRAL HOSPITAL

You have been authorised to conduct research within the jurisdiction of Moi Teaching and Referral Hospital (MTRH) and its satellites sites. You are required to strictly adhere to the regulations stated below in order to safeguard the safety and well-being of staff, patients and study participants seen at MTRH.

1. The study shall be under Moi Teaching and Referral Hospital regulation.
2. A copy of MTRH/MU-IREC approval shall be a prerequisite to conducting the study.
3. Studies intending to export human bio-specimens must provide a permit from MOH at the recommendation of NACOSTI for each shipment.
4. No data collection will be allowed without an approved consent form(s) to participants unless waiver of written consent has been granted by MTRH/MU-IREC.
5. Take note that data collected must be treated with due confidentiality and anonymity.

The continued permission to conduct research shall only be sustained subject to fulfilling all the requirements stated above.

The approval period is 14th December, 2023 – 13th December, 2024.

For DR. WILSON K. ARUASA, *MBS, EBS*
CHIEF EXECUTIVE OFFICER

14 DEC 2023

c.c. - Senior Director, Clinical Services
- Director, Nursing Services
- HOD, HRISM

All correspondences should be addressed to the Chief Executive Officer
Visit our Website: www.mtrh.go.ke
TO BE A GLOBAL LEADER IN THE PROVISION OF EXCEPTIONAL MULTI-SPECIALTY HEALTH CARE, TRAINING AND RESEARCH

Appendix 6: Nacosti Approval

REPUBLIC OF KENYA

NATIONAL COMMISSION FOR SCIENCE,TECHNOLOGY & INNOVATION

Ref No: 972014

Date of Issue: 06/March/2025

RESEARCH LICENSE

This is to Certify that Dr.. MAALIM ABDIRAHMAN ADAN of Moi University, has been licensed to conduct research as per the provision of the Science, Technology and Innovation Act, 2013 (Rev.2014) in Uasin-Gishu on the topic: PREVALENCE OF OBSTRUCTIVE SLEEP APNEA RISK AMONG AMBULATORY TYPE 2 DIABETES MELLITUS PATIENTS AT MOI TEACHING AND REFERRAL HOSPITAL for the period ending : 06/March/2026.

License No: NACOSTI/P/25/416362

972014

Applicant Identification Number

Director General
NATIONAL COMMISSION FOR SCIENCE,TECHNOLOGY & INNOVATION

Verification QR Code

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See overleaf for conditions