Faculty of Health Sciences

Department of Biomedical and Clinical Technology

The comparison between apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal

Yabanathi Kibi (20908471)

A research project submitted to the Department of Biomedical and Clinical Technology in the Faculty of Health Sciences at the Durban University of Technology in partial fulfilment of the requirement for the award of the Master of Health Sciences in Clinical Technology

August 2023

COPYRIGHT NOTICE

STUDENT DECLARATION OF ORIGINALITY OF THE WORK

I hereby declare that this research project is my original work, and to the best of my knowledge and belief, it contains no material previously published or written by any other person, nor material that has been accepted for the award of any other degree or diploma of the university or other institute of higher learning, except where due acknowledgment has been made in the text.

This dissertation was conducted in the Department of Biomedical and Clinical Technology, Faculty of Health Sciences, at the Durban University of Technology under the supervision of Dr Mogapi Jeremiah Mohapi (Senior Lecturer: Facilitation and Research Supervision in Department of Biomedical and Clinical Technology) and Dr Rosaley Prakaschandra (Senior Lecturer: Facilitation and Research Supervision in Department of Biomedical and Clinical Technology), including Mr Dalincebo Christopher Mdluli (Neurophysiology Lecturer in Department of Biomedical and Clinical Technology). The Department of Neurophysiology and Pulmonology at Inkosi Albert Luthuli Central Hospital, Durban, under the supervision of Prof. Kennedy Nyamande (Head of Discipline: Pulmonology and Critical Care).

SIGNED_____ DATE 17/08/2024

YABANATHI KIBI

(MASTER OF HEALTH SCIENCES IN CLINICAL TECHNOLOGY)

I hereby certify that the above statement is true and correct.

SIGNED_____ DATE 17/08/2024

DR ROSALEY PRAKASCHANDRA

(PhD: Cardiology)

SIGNED_____ DATE 17/08/2024

PROF. KENNEDY NYAMANDE

(MB ChB (UZ), FCP (SA), Cert Pulmonology Phys (SA), Ph.D. (UKZN)

SIGNED_____ DATE 17/08/2024

MR DALINCEBO CHRISTOPHER MDLULI

(Masters in Neurophysiology)

DEDICATION

I dedicate this research to my mother for her hard work in raising me and to my two children, my partner, my sister, and my brother for their continued support and encouragement.

I would like to thank Dr J. M. Mohapi for all his advice, encouragement, learning skills, and writing skills he taught me. I say thank you so much that I came across you in my tertiary journey, you taught me a lot and I continued with the work where we left off.

ACKNOWLEDGEMENTS

Thank you to Dr M. J. Mohapi, Dr R. Prakaschandra, and Mr D. C. Mdluli for all the help and time spent encouraging me to complete this research project. You reminded me of all the due dates and taught me strategies to employ so that I make my research meaningful.

Thank you to Prof. K. Nyamande and Prof. AI Bhigjee for all the writing skills and advice they have given me during this journey on strengthening my research while ensuring the safety of the patients taking part in the study.

Thank you to my unit Supervisor, Miss C van der Walt for your time, and for the help in facilitating some of the utensils to use for my research.

Thank you to Dr R. Steele for editing the dissertation and references for clarity and language (Annexure 24).

Lastly, I would like to thank my family. You have all been very supportive and encouraging. This study would not have been possible without the support of my mother, Nompumelelo Kibi, my partner Tawanda, my two siblings, Okuhle and Luthando, and my two amazing daughters, Tadiwa and Atandwa.

ABSTRACT

Background:

Sleep-disordered breathing (SDB) is a highly prevalent, though under-recognised, public health problem. The apnoea-hypopnoea index (AHI) is a standard measure used to assess the presence and severity of SDB. It is also a primary measure to assess the effectiveness of SDB treatment. This study aimed to compare the apnoea-hypopnoea index (AHI) between the overnight polysomnography (OPSG) and the afternoon-nap polysomnography (ANPSG) tests through the variables measured. This was accomplished by comparing the AHI values and other variables of the ANPSG and OPSG in diagnosing SDB.

Method:

The study investigated the correlation of the AHI, by measuring the dependent variables using the afternoon nap polysomnography (ANPSG) and overnight polysomnography (OPSG) on the same patients at Inkosi Albert Luthuli Central Hospital (IALCH).

A sample size of 25 was deemed adequate for analysis. Patients with the following symptoms and signs, highly suggestive of SDB, were recruited into the study: excessive sleepiness and tiredness during the day, snoring, nocturnal choking or gasping, and restlessness. A validated Epworth screening questionnaire was done at base hospital by the attending physician prior to booking the patient for sleep test. Afternoon nap PSG and overnight PSG data were compared using SPSS (Version 28®), where both descriptive and inferential statistics were used to analyse the data.

Results:

The majority of patients were females (56%) while males accounted for 44%. Most of the patients were older than 50 years of age (60%) with a mean age of 51.04 ± 10.65 years. The mean BMI was 37.6 ±10.9 (kg/m^2), the mean neck circumference was 41.4 ±4.6 cm, the mean weight 103.1 ± 28.6 kg, and the mean height 1.66 ± 0.1m. Furthermore, the correlation coefficient revealed a strong linear association between ANPSG and OPSG.

The paired sample test showed that there was a difference between afternoon and overnight polysomnography measured for RDI ($p < 0.001$), AHI ($p = 0.002$), NREM (RDI) ($p < 0.001$), NREM (AHI) ($p = 0.001$), REM (RDI) ($p = 0.005$), REM (AHI ($p = 0.027$), total sleeping time ($p < 0.001$), and initial REM latency ($p < 0.001$). The mean value measured for OPSG was higher when compared with the ANPSG for the following variables: RDI (72.85 ± 33.99), AHI (66.76 ± 33.73), NREM (RDI) (72.69 ± 36.48), NREM (AHI) (66.63 ± 36.18), REM (RDI) (76.72 ± 26.65), REM (AHI) (69.64 ± 28.97), total sleep time (421.96 ± 61.03), and initial REM latency (116.74 ± 71.46).

ANPSG underestimates the severity parameters of sleep-disordered breathing (SDB). ANPSG may confirm the presence of AHS, however may not grade the true nocturnal severity, and when SDB is at a mild stage, ANPSG may not rule out that SDB will be or will not be present in the OPSG. ANPSG underestimates the value of AHI, RDI and other variables when compared to OPSG, see Annexure 26.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF ANNEXURES

LIST OF ABBREVIATIONS/ACRONYMS

3 T MRI	3 Telsa Magnetic Resonance Imaging
ANPSG	Afternoon nap polysomnography
ALMA	Alternating leg muscle activation
AHI	Apnoea-hypopnoea index
BPAP-S	Bilevel positive airway pressure in a spontaneous mode
BPAP-ST	Bilevel positive airway pressure in spontaneous-timed mode
BPAP	Bilevel-positive airway pressure
CNS	Central nervous system
CSA	Central sleep apnoea
CPAP	Continuous positive airway pressure
CPS	Cycles per second
ENT	Ear-nose and throat
ECG	Electrocardiography
EEG	Electroencephalography
EMG	Electromyography
EOG	Electrooculography
EFM	Excessive fragmentary myoclonus
Hz	Hertz
HFT	Hypnagogic foot tremor
IALCH	Inkosi Albert Luthuli Central Hospital
ICSD-2	International classification of sleep disorders-2
IREC	Institutional research ethics committee
IRL	Initial REM Latency
IQR	Interquartile range
Msec	Milliseconds
MSA	Mixed sleep apnoea
MSLT	Multiple Sleep Latency Test
non-REM	Non-rapid eye movement sleep
OSA	Obstructive sleep apnoea
OPSG	Overnight Polysomnography
PLM	Periodic limb movements
Pcrit	Pharyngeal critical closing pressure

PSG	Polysomnography
REM	Rapid eye movement sleep
ROC	Reactive oxygen species
RMA	Rapid maxillary expansion
R & K criteria	Rechtschaffen and Kales criteria
RBD	REM sleep behaviour disorder
REB	Respiratory effort belts
RERAs	Respiratory effort-related arousals
RIP	Respiratory inductance plethysmography
RLS	Restless legs syndrome
RMD	Rhythmic movement disorder
SDB	Sleep-disordered breathing events
SEM	Slow eye movements
V waves	Vertex sharp waves

CHAPTER 1: INTRODUCTION AND BACKGROUND OF THE RESEARCH

1.1 Introduction to the study

The apnoea-hypopnoea index (AHI) is the standard measure used to assess sleep-disordered breathing (SDB) existence and extremity (Ho *et al.* 2015). It is also a primary measure to assess the effectiveness of SDB treatment (Boyd and Walters 2013). Afternoon-nap polysomnography (ANPSG) is a sleep study that records sleep and breathing in the sleep laboratory during the daytime, typically recording for 2-4 hours in the afternoon. Overnight polysomnography (OPSG) is a sleep study that records sleep and breathing in the sleep laboratory overnight, typically recording for 6-10 hours in the night (Gregório *et al.* 2011; Shrivastava *et al.* 2014).

The controversial narrative is that ANPSG cannot be fully used as an alternative to OPSG (Gregório *et al.* 2011). The ANPSG is a standard operating procedure used to diagnose (SDB) at Inkosi Albert Luthuli Central Hospital (IALCH). Shrivastava *et al*. (2014) state that an individual who spends only 3 to 4 hours of sleep in bed cannot solicitously have the normal amount of sleep and this may result in not going through all stages of sleep, including the appropriate sequence of sleep cycles.

Universally, the OPSG is the optimum standard for assessing SDB (Epstein *et al.* 2009). Screening methods have been developed to improve the current ways of diagnosing sleep disorders, but the diagnosis is still missed in a number of patients and this is worthy of attention. These existing methods include partial night polysomnogram (PSG), nasal-cannula flow signal, unattended portable monitors, and short induced-sleep PSG (Ramachandran and Josephs 2009).

A PSG is commonly known as a sleep study world-wide. It has been used for many years as a diagnostic tool for defining sleep and monitoring the major electrophysiologic changes that occur during sleep (Shrivastava *et al.* 2014). The term polysomnography is therefore used as the procedure includes monitoring of both

respiratory and cardiac parameters. In other words, PSG comprises overnight neuro-cardio-pulmonary monitoring (Glenn 2012). Researchers further emphasise that OPSG is indeed an ideal and highly recommended standard for evaluating many disorders of sleep and wakefulness, particularly sleep-disordered breathing (Glenn 2012).

Polysomnography is of importance in the inspection of patients with sleep-disordered breathing, disorders of commencing and maintaining sleep, excessive daytime somnolence, disorders of the sleep-wake cycle, and disorders associated with certain sleep stages, like parasomnias. PSG can quantify sleep time, differentiate sleep stages (see Chapter 3), and assess sleep fragmentation (Kryger, Roth and Dement 2017). In the centres where OPSG is used, only a small population of patients can be seen, whereas when simplified sleep study methods are used, a larger number of patients can be investigated and shorter patient follow-up appointment dates can be achieved. Sleep-disordered breathing is a chronic, devitalizing disease that results in serious cardiovascular and cerebrovascular illnesses and mortality.

To make a diagnosis of a sleep disorder, it is critical to understand normal sleep behaviour. Many sleep disorders involve a normal behaviour that extends beyond the range of normal. These may include but are not limited to bedwetting that continues into the teenage years or daytime sleepiness that exceeds a dip in alertness after lunch and progresses into inappropriate napping (Rosenberg 2010). The International Classification of Sleep Disorders Second Edition (ICSD-2) contains descriptions of over 80 sleep disorders (Medicine 2005). Some are relatively common, while others are rare. Each disorder has standard diagnostic set of rules that must be met before a patient is given a specific diagnosis. Some of these criteria require a sleep study, whereas others do not.

1.2 The importance of sleep

Sleep deprivation is a significant problem and is often hidden. It tends to affect, among other important tasks, cognitive and work-related demands (Loft and Cameron 2014). Poor sleep patterns can impede the functionality of immune system, appetite regulatory process, and other physiological processes. This influences health issues

as it has pernicious effects on health and well-being. Inability to sleep soundly poses a considerable risk by undermining physical activity, healthy diet choices to maintain wakefulness, and other behaviours requiring motivation and self-control (Taylor, Lichstein and Durrence 2003; Barber *et al.* 2010; Norra, Richter and Juckel 2011; Copinschi 2005 Brindle and Conklin 2012). Prioritising sleep is extremely important, as it supports critical thinking, allowing one to be more vigilant, and sustaining attention. Sleep also plays a vital role in emotional regulation. It is important to know that sleep is mostly useful in restoring alertness and waking functions, including health when it is amalgamated and not fragmented. This usually happens when sleep is permitted to go through the relevant physiological arrangements of non-REM and REM stages of sleep at night, and this takes place when the brain is temporarily programmed by the individuals' circadian clock (Worley 2018).

Individuals whose daily sleep duration is inadequate, or frequently interrupted for example by any type of sleep breathing disorder, sleep disorder like restless leg syndrome, pain or stress, shift-work or jet lag, often are not aware of the accumulating sleep deficits on the waking cognitive functions, performance, memory, and accuracy (Worley 2018).

1.3 Defining the factual duration of sleep

With investigations over many years, it appears that scientists in different regions of the world have gathered enough evidence and have come to the conclusion that the number of hours required for sleeping needs to be approximately seven to eight hours (Lamberg 2006). It has been noted that several different disorders begin to increase in prevalence when the duration of sleep drops below seven hours. Poor sleep management may contribute to accidents and catastrophes that happen daily (Medic, Wille and Hemels 2017).

Sleep-disordered breathing (SDB) is a highly prevalent, though under-recognised, public health problem. It includes a range of disorders characterised by repetitive episodes of abnormal respiratory patterns, in which partial or complete cessation of breathing occurs, with consequent hypoxemia during sleep. This leads to daytime excessive sleepiness and reduced quality of life with an increased probability of mood

disorders, neurocognitive impairment, and road traffic accidents (Sharma and Srijithesh 2013).

The basic set of assumptions that guide this inquiry is a positivist paradigm to compare AHI results obtained from the ANPSG and OPSG. The ontological philosophy is that SDB can manifest during the sleeping period irrespective of the duration of sleep (Gregório *et al.* 2011).

The study was conducted in a sleep laboratory. Both the ANPSG and OPSG were performed on the same patients, under the same conditions. The only difference was the duration: 2-4 hours and 6-10 hours, respectively. The measurement of the ANPSG and OPSG was performed according to the American Academy of Sleep Medicine (AASM) version 2.0 in conjunction with the minor changes published on the updated versions for standardisation. The recorded polysomnogram was stored in a computer database using Nicolet software. The statistician advised a sample size of 25 using a systematic probability sampling technique. Patients with clinical symptoms and questionnaire responses of excessive sleepiness and tiredness during the day, snoring, nocturnal choking or gasping, restlessness, and sleep disturbance were recruited for the study. The descriptive data include gender, age, race, neck circumference, waist circumference, weight, afternoon nap, and overnight PSG data. Statistical analysis was performed using SPSS 28.

1.4 A comprehensive summary of methods and methodology, as well as data analysis techniques

The basic set of assumptions that guided this inquiry was a positivist paradigm to compare AHI results obtained from the ANPSG and OPSG. The study was experimental, measurements were taken into consideration, and sought specificity and sensitivity of each sleep study in the diagnosis of SDB and other conditions were measured during the study. The ontological philosophy was that SDB could manifest during a sleeping period irrespective of the duration of sleep (Gregório *et al.* 2011). The independent variable in this study was sleep, and the dependent variable included all the indices measured and calculated depending on sleep duration and architecture.

All patients underwent the ANPSG and OPSG on consecutive days depending on which study was done first. Different recording times were expressed as x-h PSG, where x = 2-4hrs for an afternoon nap, and x = 6-10hrs for overnight PSG. Patients aged 18 years and older but younger than 70 years attending IALCH were recruited. Patients whose responses to the questionnaires were highly suggestive of sleep-disordered breathing were recruited to take part in the study. The questionnaires used were Berlin, Epworth (ESS), Functional Outcome (FOSQ), and STOP-Bang; this is explained in depth in the chapter 2 (Annexure 26).

Quantitative data from participants were analysed in terms of questionnaire results, gender, age, race, neck circumference, waist circumference, and weight. Afternoon nap PSG and Overnight PSG data were compared using SPSS® (Version 28), where both descriptive and inferential statistics were used to analyse the data. Univariate and bivariate analyses are most appropriate for descriptive statistics (Salkind and Frey 2019). Bar graphs and tables were used to present the data.

Non-parametric testing using Kolmogorov-Smirnov assisted in determining the normal distribution of the variables (Kinnear and Gray 2006). Concerning the inferential statistical analysis, the independent t-test was used to compare the mean difference between the AHI of overnight PSG and afternoon nap PSG. Barnes and Lewin (2011) indicated that the independent t-test identifies the mean difference most suitably, and is very useful in testing the significant difference of any kind between two variables (Barnes and Lewin 2011). In addition, the relationship between the demographic factors (age, race, neck and waist circumference, and weight), and the overnight versus afternoon nap PSG variables was determined by using the Pearson correlation coefficient.

1.5 Summary of salient findings

The paired sample test done for the afternoon (ANPSG) and overnight polysomnography (OPSG) showed that there were statistically notable differences between afternoon and overnight polysomnography measured for RDI ($p < 0.001$), AHI ($p = 0.002$), NREM (RDI) ($p < 0.001$), NREM (AHI) ($p = 0.001$), REM (RDI) ($p = 0.005$),

REM (AHI) ($p = 0.027$), total sleeping time ($p < 0.001$), and initial REM latency ($p < 0.001$).

The mean value measured for OPSG was higher when compared against the ANPSG for variables RDI (72.85 ± 33.99), AHI (66.76 ± 33.73), NREM RDI (72.69 ± 36.48), NREM (AHI) (66.63 ± 36.18), REM (RDI) (76.72 ± 26.65), REM (AHI) (69.64 ± 28.97), total sleep time (421.96 ± 61.03), and initial REM latency (116.74 ± 71.46). No differences could be detected between ANPSPG and OPSG for the variables; sleep efficiency ($p = 0.065$), breathing-related arousals ($p = 0.877$), and spontaneous-related arousal ($p = 0.368$). However, it was found that as the age of patients increased, the AHI measured for either ANPSG or OPSG decreased.

There was a statistically significant correlation measured for the neck circumference for ANPGS ($p = 0.017$) and OPSG ($p = 0.025$) against the sleep apnoea severity. Patients with a neck circumference of 42.6 ± 3.8 had severe ANPSG while those with a neck circumference of starting from 42.3 ± 4.0 had severe OPSG.

The differences and similarities found during the comparison of the dependent and independent variables stress the need and importance of OPSG for most patients. The latter is due to the values obtained during ANPSG being lower than the values obtained during OPSG. ANPSG gave lower severity scores for most parameters measured, thus underestimating the severity of SDB.

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

Sleep disordered breathing (SDB) is a condition that is characterised by repetitive incomplete or complete obstruction of the upper airway, or complete cessation of air thereby compromising gas exchange (Suzuki *et al.* 2005; Epstein *et al.* 2009). Escourrou and Rehel (2000: 69), Epstein *et al*. (2009: 263) and Chou *et al*. (2011: 1096) state that the overnight PSG study is the gold standard for diagnosing sleep-disordered breathing events (SDB). During the development of the polysomnogram (PSG) a sleep research board formed in the early 1960s put together standard operating procedures for recording and scoring of sleep stages in 1968, including the addition of simultaneous recording of respiratory and cardiac monitoring.

The deliberations of this committee remain the international ideal standard in the neurophysiological field and are known as the “Rechtschaffen and Kales criteria”, (R & K criteria) (Matheson, Singh and Packard 2007). Walker and Stickgold (2009:139), stated that in a case of suspected sleep disorder, ANPSG should be able to reveal the suspected condition if present, and not only during the use of a more extensive OPSG (Walker and Stickgold 2009). The authors further wrote that sleep apnoea diagnosis may be confirmed using ANPSG of 2-4 hours’ duration, however, this may only happen when all polysomnographic variables are recorded. Furthermore, all sleep stages (non-REM and REM) would need to be sampled, and lastly, some of the sleep time needs to be spent in a supine position. Kapur *et al*. 2017 suggested that a technically adequate OSA diagnostic test include a minimum of 4 hours with a technically competent oximetry and flow data during sleep period when using home sleep apnoea testing.

The use of drugs to induce sleep during a PSG or sleep deprivation prior to a sleep study may not be allowed, as some drugs are believed to negatively add an impact on the numbers of apnoeas. However, Ahmad *et al*. 2022 found contrary results with the use of zolpidem in their study. Zolpidem was found to reduce respiratory arousal index (39.7 ± 7.7 vs. 23.3 ± 4.4 events/h, P = 0.031). It also lowered the total AHI (55.6 ± 8.5 vs. 41.3 ± 7.5 events/hour, P = 0.033), and the other clinical and physiological parameters were not affected (Ahmad *et al.* 2022). These researchers concluded by

stating that as much as ANPSG can be used to confirm the presence of SDB, it is however still inadequate to definitively exclude a diagnosis of sleep apnoea.

It is estimated that in the United States, about 70 million people have at least a certain type of sleep disorder and this may be due to many different reasons (Worley 2018). The challenge faced by clinicians is identifying comorbidities, as the diagnosis of sleep apnoea often is complicated by the presence of another condition or disorder. Underlying conditions can complicate treatment and often require sleep specialists to work hand in hand with specialists in other disciplines. For example, a patient with insomnia may also experience mood disorder or depression. Therefore, a psychiatrist will be needed to collaborate with the primary care physician.

(Chang *et al.* 2020) Conducted a study of obstructive sleep apnoea awareness among primary care physicians in Africa. The researchers found that the physicians considered OSA to be important but had modest knowledge about OSA in adults and children, and had a low perceived confidence in adult and paediatric management. A suggestion of improving primary physicians' knowledge about OSA and its diagnosis and management through focused educational interventions during undergraduate training and continuing professional development programs was the utmost idea of dealing with the problem (Chang *et al.* 2020; Mbena 2022).

The complications associated with sleep apnoea are serious, hence the need to improve methods used in identifying and properly diagnosing sleep-related disorders. Undetected OSA in patients with other serious underlying illnesses can result in potentially dangerous complications. To give example, opioids are known to cause detrimental effects on respiration, this can lead to central sleep apnoea (CSA), shallow and irregular or interrupted breathing, and sustained hypoventilation, a condition capable of causing death. CSA is often defined as the presence of at least five central apnoeas per hour (Park and Helman 2015).

The standard recording analysis includes 11 parameters, namely, electroencephalography (EGG), electrooculography (EOG), electromyography (EMG) under the chin and tibialis anterior muscles, electrocardiography(ECG), nasal or oral airflow and pressure cannula, a thermistor for the nasal and oral airflow sensor, chest

and abdominal respiratory effort bands, oxygen saturation, and body position sensor. Glenn (2012) recommends that the period for recording a standard PSG should be at least 6 hours and a maximum of 8 hours (Glenn 2012). Shrivasta *et al*. (2014) elaborate on this by stating that 6-8 hours of PSG recording gives more than enough total sleep time value to be used for reporting. However, overnight PSG is costly, has limited availability, is time-consuming, and requires an overnight study with supervision. Due to these constraints with OPSG, some scholars suggest that short afternoon nap PSG constitutes a promising alternative method in the diagnosis of sleep apnoea as it is affordable and only takes a short duration of time to do the test (Chou *et al.* 2011; Gregório *et al.* 2011; Kahlke *et al.* 2013). Kushida *et al* (2008) further explains that although the OPSG is time-consuming and resource-intensive, it theoretically maximises the opportunities to comprehensively observe breathing response during the sleep states during the night (Kushida *et al.* 2008).

During uninterrupted sleep, non-rapid eye movement (NREM) and rapid eye movement (REM) stages of sleep alternate in 4 to 6 cycles of approximately 70 to 90 minutes. This makes it important for the patient to be given enough time to go through all of the stages of sleep during PSG recording, as some disorders are triggered and/or exacerbated by or occur only during certain sleep stages (Matheson, Singh and Packard 2007). The ANPSG is the only study used in IALCH to diagnose SDB events and is obtained during 2-4 hours of sleep. Shrivastava *et al.* (2014) state that an individual spending only 3-4 hours in bed is unable to practicably accumulate the normal amount of sleep and may not go through all required stages of sleep and the cycles. This may also result in the initiation of unnecessary treatment. Therefore, a reduced total time in bed during a sleep study may be of clinical significance and may support a diagnosis of insufficient sleep, with an inconclusive diagnosis.

The similarity of results was measured using AHI. The AHI is a calculated index, defined as the total number of hypopnoea, obstructive, or central sleep apnoea per hour of sleep. The severity of apnoeas was categorised according to AASM guidelines as no sleep apnoea if there were less than 5 apnoeas per hour, mild if greater than 5 and less than 15 per hour, moderate if greater than 15 and less than 30 apnoeas per hour, and severe if more than 30 apnoeas per hour (Epstein *et al.* 2009).

Young *et al*. (2004) noted in their writing that obese patients are more prone to develop SDB as fat deposits tend to be around the upper airway, obstructing breathing (Young, Skatrud and Peppard 2004). The thick neck circumference narrows the airway. A study dedicated to find the relationship between obesity and OSA in anaesthesiology setting, concluded that as obesity is a multi-systemic disease which carries significant morbidity and mortality. It is of utmost importance to identify the disease and grade its severity and assess the degree of dysfuction it has caused so that the patient can be optimally assessed by the anaesthetist (Naidoo 2015).
Smoking is another risk factor, particularly in older adults (Young, Skatrud and Peppard 2004). There is a lot that is believed to contribute to the variance occurring in prevalence between males and females. These variances may include but are not limited to differences in fat distribution, collapsibility and length of the upper airway, arousal response, and sex hormones (Lin, Davidson and Ancoli-Israel 2008). Shrivastava *et al.* (2014) stated that males are more affected by SDB compared to women. Their study showed that 10% of men compared to 3% of women between the ages of 30-49 years old, and 17% of men in comparison with 9% of women between the same ages of 50-70 years old were affected by SDB.

Sleep-disordered breathing includes but is not limited to disorders such as central sleep apnoea, obstructive sleep apnoea, mixed apnoea, and sleep-related hypoventilation syndrome. Therefore, PSG is a necessary and not just inconsequential test in the diagnosis and management of these sleep disorders (Matheson, Singh and Packard 2007). The guidelines of the American Sleep Disorders Association (ASDA) state that continuous positive airway pressure (CPAP) titration must be performed for one night under full PSG control using a manual and not an automated protocol. However, this recommendation is usually not followed in many institutes owing to cost and the long waiting list. This has led to the development of alternative methods of CPAP titration, including split-night studies where both the diagnostic PSG and titration are done on the same night, monitoring of respiratory variables only without the EEG part of the recording, and titrating with automatic CPAP systems in hospitals and at home (Lloberes *et al.* 2004).

The effectiveness of CPAP pressure on a patient with apnoeas is determined during a titration study conducted during a full PSG study at a sleep laboratory where a trained

and experienced neurophysiologist manually increases airway pressure progressively until apnoeas, hypopnoeas, and snoring eradicate, oxyhemoglobin saturation is normalised, and sleep fragmentation disappears. Lloberes *et al.* (2004) conducted a daytime CPAP titration study with full PSG performed in severe sleep apnoea-hypopnoea syndrome (SAHS) patients. At this hospital, the implementation of daytime studies achieved a reduction in the CPAP titration waiting list. Manual titration with full PSG was performed in the same manner as night-time titration. Titration began at 9 a.m. The neurophysiology clinical technologist would end daytime studies in agreement with a physician if patients achieved at least one REM period, which usually occurred during the first four hours.

Data from the daytime CPAP titration study were then calculated and presented as means and standard deviations. The one-way ANOVA and the t-test for independent variables were utilised to evaluate continuous variables from the different studies, with a 95 percent confidence interval considered significant. Qualitative variables were analysed using a w2 test. After three months of treatment with CPAP, Epworth's sleepiness score and objective CPAP use were recorded. Objective utilisation of CPAP and subjective sleepiness measured by the Epworth sleepiness scale did not vary substantially among the groups. This study demonstrated that the implementation of night-time sleep deprivation prior to daytime CPAP titration studies is useful in SAHS patients (Lloberes *et al.* 2004).

Sleep time, the total number of apnoeas and hypopnoeas, AHI, and oxygen saturation variations obtained with full night-time PSG and involuntary afternoon sleep PSG were analysed using the Mann-Whitney test (Gregório *et al.* 2011). The correlation between AHI obtained by both the PSGs was calculated by using the Pearson correlation coefficient. The accurate values for sensitivity and specificity were established using the receiver operating characteristic curve. To evaluate the correlation between both the PSGs in comparison, the Bland Alteman plot was utilised, with a 95 percent confidence interval considered significant. The PSGs were considered positive when the two studies recorded ≥ 30 events per hour or if the variance between the two PSGs was ± 10 or fewer events per hour. The study indicates that induced sleep while doing the afternoon nap PSG did not compromise the respiratory drive, although no patients presented stage 3 of sleep or REM sleep during the study (Gregório *et al.* 2011). In

other studies, the Pearson correlation coefficient was used to compare OPSG and WatchPAT in the diagnosis of obstructive sleep apnoea (Kenny, Christine and David 2007).

Khawaja *et al*. (2010)'s study on the diagnostic accuracy of split-night polysomnograms showed that the two and three-hour AHI strongly correlated with the full night-time AHI with a concordance correlation coefficient [CCC] of 0.93 and 0.97, respectively. Some studies found that ANPSG in the identification of OSA had a good positive predictive value (77-100%) while others found it had a poor negative predictive value (17-49%). An ANPSG may confirm sleep breathing disorder but may not grade its true nocturnal severity and does not rule out that sleep-disordered breathing will be found on an OPSG (Foldvary-Schaefer, Grigg-Damberger and Mehra 2019).

A split-night PSG is a night-time procedure performed with 2 hours of diagnostic PSG recording, followed by a CPAP titration study if the sleep apnoea value is clinically significant. Different laboratories use varying criteria, from 15 events per hour of sleep to 40 events per hour of sleep. CPAP titration is not recommended in the case of an inadequate number of disordered breathing events recorded over the first 2 hours of diagnostic recording. Since the degree of disordered breathing present over the entire night is not always reflected in the first 2 hours of sleep, neurophysiologists may have to revert to full night-time PSG. In some instances, SDB occurs mainly or solely during REM sleep, and these REM-related apnoeas may not be documented as REM sleep occurs mainly in the early hours of the morning (McNicholas 2008; Foldvary-Schaefer, Grigg-Damberger and Mehra 2019). The diagnostic PSG recording should provide adequate data to make a reliable sleep-related diagnosis. A positional apnoea can often only be fully assessed with an OPSG (McNicholas 2008).

Home sleep apnoea test (HSAT) is another alternative method to diagnose OSA. Although, it may be less costly compared to OPSG in the laboratory, it has its own limitations (Kapur *et al.* 2017). Physiological parameters collected may differ depending on whether the study is Type II, Type III or Type IV, and there is unavailability of a Neurophysiologist to adjust sensors when needed. Type I sleep studies are the full OPSGs conducted in the sleep laboratory. Type II sleep studies are unattended and can be performed outside of sleep laboratory as they use the same

parameters as Type I sleep studies. Devices measuring limited cardiopulmonary parameters; such as two respiratory variables (airflow, an effort to breathe), oxygen saturation, and a cardiac variable (heart rate or electrocardiogram) are used in Type III studies. The devices that measure only 1 or 2 parameters fall under Type IV sleep studies, the usually measured sensors are oxygen saturation and heart rate, sometimes, just air-flow (Kapur *et al.* 2017; Rosen *et al.* 2018).

HSAT devices in comparison with OPSG raise risk for technical failures due to lack of the actual times needed for the completion of the report, most devices have been reported to be unable to define sleep versus awake. Another potential disadvantage is that a split-night test cannot be done with HSAT, but can be initiated during an OPSG when needed (Kapur *et al.* 2017).

There may be a big measurement error with HSAT, compared to OPSG, when it comes to respiratory event index, as recording time rather than sleep time may be used to define the denominator during calculations (Kapur *et al.* 2017; Rosen *et al.* 2018). Other factors in the HSAT that could contribute to the underestimation of actual number of apnoeas is the failure of devices that use conventional sensors to detect hypopnoeas that are not associated with cortical arousal, with the additional contributors to the measuring error, including the sensor dislodgement and poor quality signal. This may result in the need of repeat studies due to inadequate data for diagnosis (Kapur *et al.* 2017).

A technically adequate HSAT device was suggested to comprise of a minimum of the following sensors: nasal pressure, chest and abdominal respiratory inducatance plethysmography, and oximetry, or peripheral arterial tonometry with oximetry and actigraphy (Kapur *et al.* 2017).

Guidelines do suggest that either OPSG or HSAT is recommended for the diagnosis of OSA in the context of considering two important statements. First, the choice of the study should be guided by clinical judgment together with patient preferences. Second, the choice of the study should not be judged only by patients that do not have too complicated conditions who are at increased risk for moderate to severe OSA, but in whom there is a concern for OSA based on a comprehensive sleep evaluation, OPSG

is recommended (Kapur *et al.* 2017; Rosen *et al.* 2018). The researcher continued by stating that the numbers of patients potentially misclassified by HSAT was high enough to be of clinical concern, especially when tests were inconclusive or negative, and misdiagnoses on patient outcomes can cause significant harm. These concerns support the use of diagnostic test with higher sensitivity, the OPSG. Furthermore, the potential cost, convenience, and comfort advantages of HSAT over OPSG are not as high as reflected by the cost difference of a single night of testing done during OPSG. However, the use of HSAT does not provide inferior clinical benefit, compared to OPSG when used in the appropriate context described in the recommendations (Kapur *et al.* 2017).

2.2 Basic anatomy and physiology of sleep

There has been an outstanding increase in the demand for knowledge and availability of affordable sleep-related evaluations and sleep studies in the fields of neurophysiology and pulmonology (Chokroverty 2010). A laboratory PSG requires hard work and somewhat multiplex and costly study, which results in its availability being limited. This has led to the building and using more simplified sleep study methods that are less labour-intensive, such as ANPSG. However, there have been considerable doubts and ongoing debate regarding their clinical use especially when it comes to the questionable incomplete sleep stage recording effects on the patients' outcomes (Chai-Coetzer and McEvoy 2017).

Sleep is a complex process that involves multiple structures, and the reciprocal interconnection is mandatory for the beginning and conservation of sleep (Kovacevic-Ristanovic and Kuzniar 2014). The area around the solitary tract in the medulla, and midline thalamus are the structures believed to contribute to sleep (Figures 1 and 2). The parts of the basal forebrain responsible for sleep include the preoptic area of the thalamus, especially the ventrolateral pre-optic nucleus (VLPO), these parts promote sleep using gamma-aminobutyric acid (GABA) or galanin activity (Kovacevic-Ristanovic and Kuzniar 2014) (Figure 1). The inhibitory action on the posterior hypothalamic awakening neurons nuclei results in the sleep-promoting role of the anterior hypothalamus, the mostly involved neurons are the tuberomammillary histaminergic neurons (TMN) projecting to the cortex (Kovacevic-Ristanovic and

Kuzniar 2014). The medulla relays information between the spinal cord and the higher regions of the brain. The link work by using white matter projection tracts including a network of grey and white matter known as reticular formation (Izac 2006). The other function of medulla oblongata involves the regulation of cardiac, respiratory rate and depth of breathing, and vasomotor control as it has both the sensory and motor nuclei (Izac 2006).

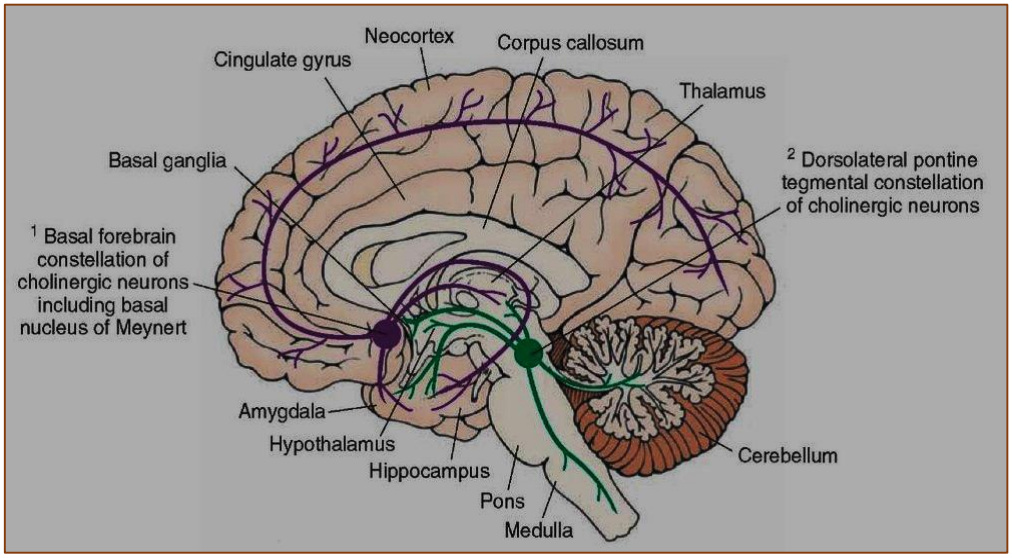


Figure 1: A more detailed form of basal forebrain
Source: Xu *et al.* (2015)

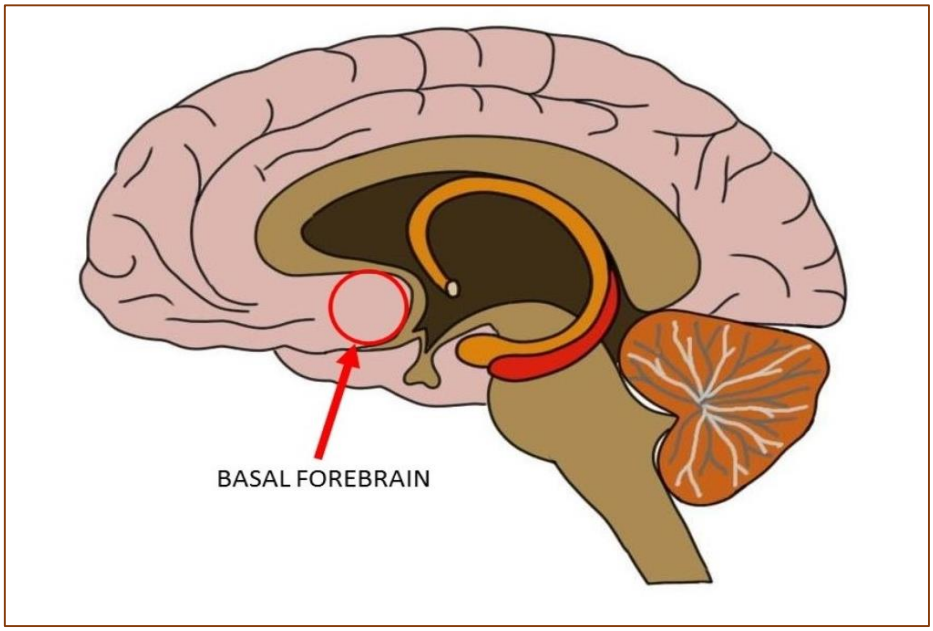


Figure 2: A less detailed form of basal forebrain
Source: Dingman (2017)

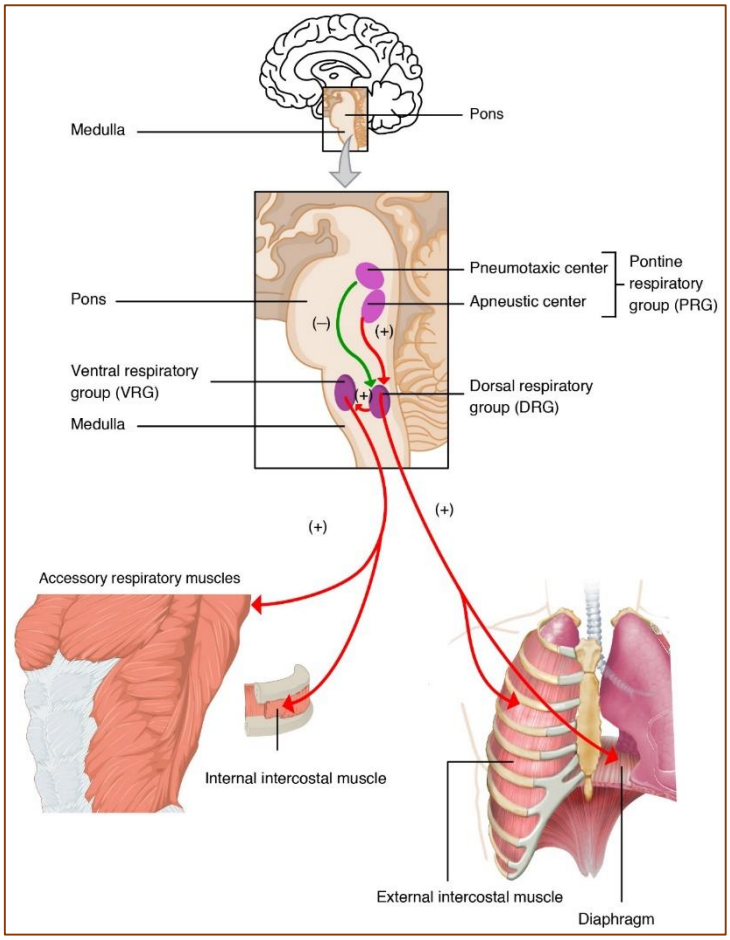


Figure 3: The respiratory centres of the brain
Source: Aung *et al.* (2019)

The wake-promoting hypocretin system, orexin, ascending reticular activating system of the pons, medulla, and posterior hypothalamus are the structures promoting the waking state (Kovacevic-Ristanovic and Kuzniar 2014). Therefore, elliptically, the VLPO (GABA/ galanin) system impedes the arousal system while the dorsolateral hypothalamic (hypocretin [orexin]) system initiates the arousal system. Any damage to the VLPO system results in insomnia, while any damage to the hypocretin system causes narcolepsy, sleep attacks or hypersomnolence, and cataplexy. Pons contains part of respiration: pneumotaxic, and apneustic centres (Figure 3). The pneumotaxic centre is a neural centre in the upper part of the pons, that regulates respiration by providing inhibitory impulses on the inspiration thereby preventing over-distension of the lungs and helping maintain alternatively recurrent inspiration and expiration. The apneustic centre stimulates the inspiratory neurons of the VLPO (GABA/galanin) system and the dorsolateral hypothalamic (hypocretin [orexin]) system (Izac 2006). The midbrain encompasses white matter tracts which allow it to continue reticular

formation (Figure 4). The two rope-like masses of white matter, known as the peduncles, relay impulses between the midbrain and the cerebrum. These fibres run down from the cortex to the ventral part of the midbrain, pontine grey matter, and the spinal cord. The reticular formation joins with parts of the hypothalamus and thalamus, as it has ascending and descending fibres from many of the structures within the brain (Izac 2006). The reticular formation works as the reticular activating system (RAS). The RAS maintains the cerebrum in a state of alertness and keeps track of the afferent impulses received by the cerebrum. Without the constant excitation of cortical neurons by the reticular activating impulses, a person remains comatose and cannot be awakened (Izac 2006).

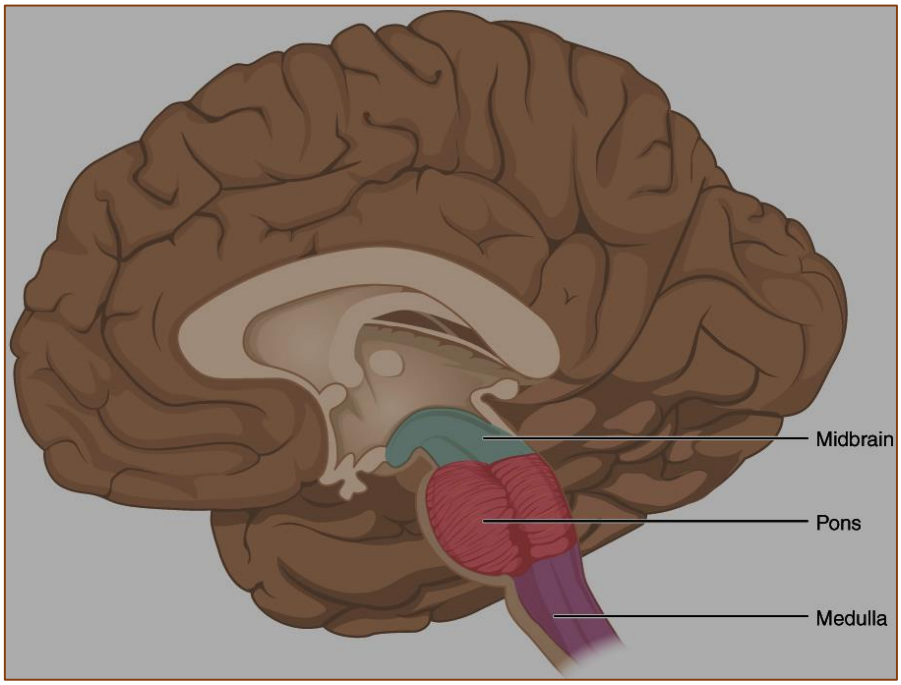


Figure 4: Midbrain
Source: https://teachmeanatomy.info/neuroanatomy/brainstem/midbrain/

The interface between antagonistic aminergic and cholinergic systems in the brainstem controls the alternating sleep stages, NREM and REM. The aminergic and cholinergic systems are part of the arousal process of the cortex, together with the dopaminergic system, which especially controls the alertness in the ventral tegmental area (VTA), area A10 (Kovacevic-Ristanovic and Kuzniar 2014; Carley and Farabi 2016). This involves the excitement of dopaminergic neurons of the VTA but not substantia nigra (SN) by hypocretin. Dopaminergic neurons in the frontal part of the periaqueductal grey

(PAG) are also activated during the awake state (Kovacevic-Ristanovic and Kuzniar 2014; Carley and Farabi 2016).

Circadian sleep rhythm is a 24-hour internal clock that cycles between sleepiness and alertness at consistent intervals within the brain (Victor, Ropper and Adams 2001). The sleep/wake cycle is controlled by the hypothalamic suprachiasmatic nucleus (SCN), as it sets the body clock period to nearly 25 hours. The SCN is situated above the optic chiasm as a pair of small clusters of nerve cells within the anterior ventral hypothalamus. The retina gives output to the SCNs by the way of the retinohypothalamic tract, which transports light stimuli to the SCN (Izac 2006). The SCN also receives input from the lateral geniculate nucleus in form of projections, while the thalamic relays the primary visual system (Izac 2006). During night time sleep the core body temperature is believed to be at its minimum point and melatonin levels at their maximum. Cortisol level is reduced during sleep onset but elevated in the morning during wake time (Kovacevic-Ristanovic and Kuzniar 2014).

Understanding the neurochemistry of sleep, which involves the neurotransmitters and neuromodulators that control both arousal and sleep is of great importance. This information makes things easy when it comes to prescribing a certain sleep disorder in the clinical setting. For example, knowing that hypocretin agonists and GABA/galanin antagonists are the treatment options for excessive sleepiness. GABA agonist treat insomnia and spasticity from multiple sclerosis among other things, as they decrease brain activity and relaxing muscles. Hepocretin/Orexin-1 (agonist) may induces wakefulness and reduce REM. Orexin antagonist may promote sleep (e.g Suvorexant which works by blocking the action of a certain natural substances in the brain that causes wekefulness) GABA antagonist inhibit the action of GABA. (Kovacevic-Ristanovic and Kuzniar 2014).

Sleep architecture is an umbrella word for the cyclical pattern of sleep as it shifts between awake, NREM, and REM (Rosenberg 2010). The researcher further highlighted that too much or too little sleep might be harmful. Research has shown that people who usually get less than 6 hours a night or more than 10 hours a night are at higher risk of death in comparison to people who usually sleep 7.5 hours per night. This was investigated over a 6-year follow-up duration (Rosenberg 2010).

There are three parameters from the sleep recording of PSG required to interpret the sleep stages, namely the electroencephalogram (EEG), electrooculogram (EOG), and an electromyogram (EMG) (Figures 13 and 14) (Kovacevic-Ristanovic and Kuzniar 2014). There are 2 types of sleep stages, the NREM and REM sleepIt has been documented by population studies that the percentage of time spent in each stage is dependent on individuals' age and sex (Altevogt and Colten 2006; Kovacevic-Ristanovic and Kuzniar 2014). Deeper sleep was found to occur more in women of the American Indians and blacks in comparison with lighter sleep noted in men relative to other ethnic groups. On the other hand, a relationship was noted between increasing age and impaired sleep in men, with less consistent associations in women (Redline *et al.* 2004).

2.3 Sleep fragmentation

Sleep fragmentation is described as numerous short periods of arousal during sleep. These are not the usual micro-arousals that almost everyone experiences at night during sleep (Morrell *et al.* 2000). They involve awakenings that one recalls later.. These arousals result in decreased total time spent asleep compared to the normal total sleep time percentage leading to daytime tiredness and sleepiness (Bonnet and Arand 2003). Some individuals who go through sleep fragmentation may be suffering from sleep maintenance insomnia, among other conditions. This is a type of insomnia characterised by the ability to fall asleep at bedtime, but an inability to maintain sleep through the night. Fragmented sleep matters because unrefreshing, and unfulfilling sleep is more than exasperation. It may have serious health repercussions. These may include apart from excessive daytime sleepiness, weight gain, and mood problems (Alomri *et al.* 2021). Sleep fragmentation is regarded as a symptom and a representation of underlying sleep disorder such as obstructive sleep apnoea or narcolepsy (Morrell *et al.* 2000).

It is known that sleep disorder measurements from different instruments may vary in terms of accuracy, misdiagnosis, and misclassification (Chou *et al.* 2011; Kapur *et al.* 2017). When sleep quantity is decreased and sleep efficiency is poor, AHI tends to be overestimated (de los Reyes 2014). When scoring OPSG and ANPSG, sleep is

grouped into NREM and REM sleep. The NREM and REM sleep take turns in recurring 4-6 cycles of almost 90 minutes. The first REM period is short lasting approximately 10 minutes, then later in the middle of the night longer periods of REM sleep are noted (Matheson, Singh and Packard 2007). Absent or only one REM sleep underestimates AHI since apnoeas and hypopnoeas tend to be worse in REM sleep as respiratory muscles are more hypotonic during this stage of sleep (de los Reyes 2014).

2.4 The respiratory system and sleep-disordered breathing

The respiratory system filters air into and out of the lungs to enable the exchange of oxygen and carbon dioxide between the air and the blood. Izac (2006) states that the way the skull and face are structured creates a unique breathing pattern. For example, prognathic features involve a chin that is behind the plane of the lips and teeth, while orthognathic face individuals have a flat plane structure of the face. The upper respiratory tract is represented by the parts above the chest cavity, namely, the passages of the nose, nasal cavities, pharynx, larynx, and upper trachea (Kleinstreuer and Zhang 2010). The respiratory muscles (diaphragm and intercostal muscles), pleural membrane, lungs, and lower trachea, all form the lower respiratory tract (Figure 5) (Krimsky and Leiter 2005).

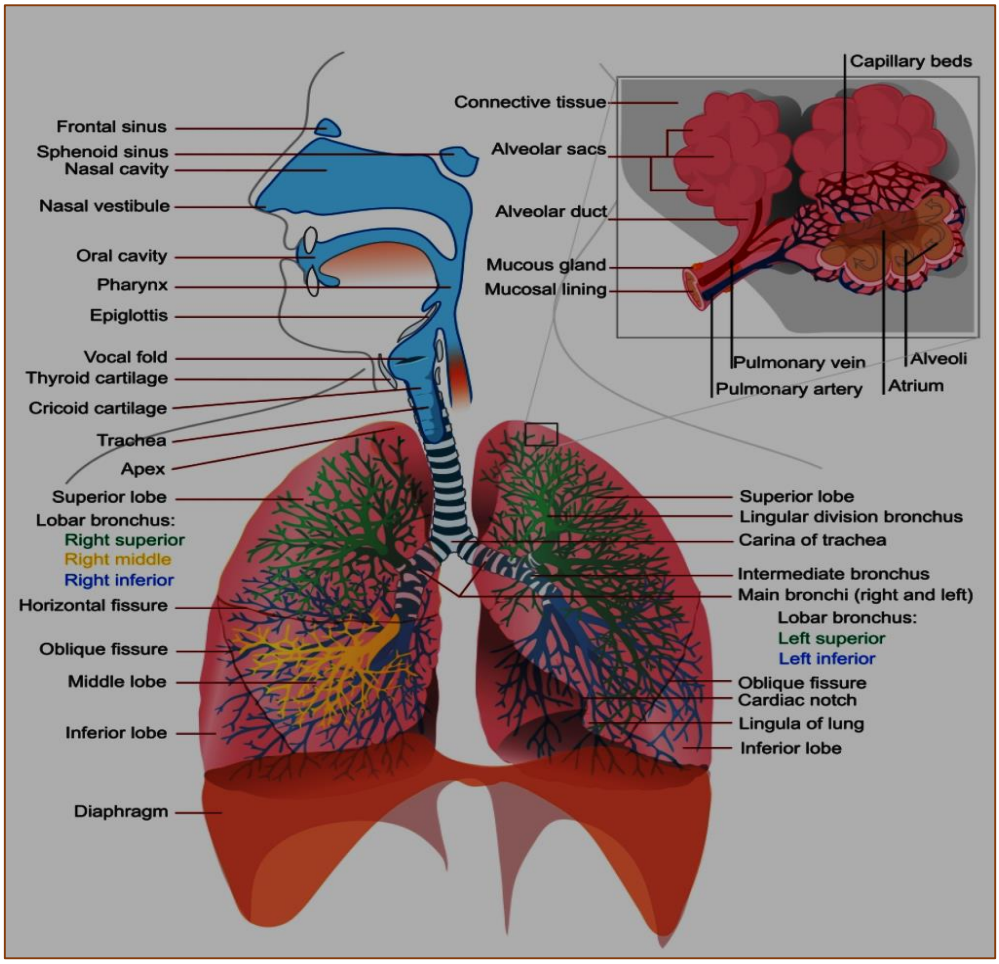


Figure 5: Respiratory system
Source: Quizlet.com: Chapter 12, respiratory system

Ventilation is induced by impulses from the medulla oblongata, through phrenic nerves to the diaphragm, then the message is passed from the intercostal nerves to the external intercostal muscles. The stretch in the lung tissue is then detected by baroreceptors, as the lungs inflate these receptors then create and pass sensory impulses to the medulla to begin the reduction in the inspiratory centre. This adjustment of the inspiration centre of the medulla, known medically as the Hering-Breuer inflation reflex, assists in inhibiting the over-filling of the lungs. All of this activity reduces the impulses to the respiratory muscles in order to activate expiration (Izac 2006). The two respiratory centres in the pons (apneustic and pneumotaxic centre) work hand in hand with the medulla's inspiratory centre to create a normal breathing rhythm. Apneustic centre impulses prolong inhalation, while the pneumotaxic centre adjourns the process to cause exhalation. Chemoreceptors located in the medulla, carotid, and aortic bodies detect changes in the blood gases and pH (Izac 2006). Changes which are then referred to as sensory impulses run along the glossopharyngeal and vagus nerves to

the medulla resulting in an elevated respiratory rate (Izac 2006). This brings extra air into the lungs to rectify hypoxia. In the case of reduced blood pH by carbon dioxide (CO_2), the medulla recovers this by increasing respiration, the idea is to cause more exhalation of the CO_2 to increase pH to the optimum level (Izac 2006).

Regular breathing patterns and more pronounced hypoventilation are noted during NREM sleep in comparison with drowsiness (Izac 2006). Low muscle tone in the upper airway during sleep may result in apnoeas, this mechanism is at its highest during REM sleep (Krimsky and Leiter 2005). Normally, the characteristics of REM sleep include intermittent irregular shallow breathing pattern that is accompanied by apnoeas of 10 to 30 seconds. These temporally normal cessations of breath are time-locked with REM bursts and do not occur at random (Krimsky and Leiter 2005).

2.5 Sleep architecture

Stages of sleep are defined using three parameters, the electroencephalogram (EEG), electromyogram, and electrooculogram (EOG) (Kumar 2013). Knowledge of sleep stages background in a PSG recording is important. 8-13 Hz alpha frequency, known as alpha activity appears in the posterior regions during eye closure in a normal, alert, and resting individual (Kumar 2013). NREM and REM are the two major stages of sleep. NREM is electrographically further divided into 3 stages of sleep. Stage N1 directly follows the state of wakefulness (Kovacevic-Ristanovic and Kuzniar 2014). During this stage, the EEG has low-voltage, mixed frequencies background dominated by theta activity, on the other hand, alpha activity is less than 50% when looking at the 30 seconds epoch, vertex sharp waves, and slow eye movements (Kovacevic-Ristanovic and Kuzniar 2014). The N2 sleep stage has a relatively low voltage mixed frequency background, however, differentiated from other sleep stages due to the presence of sleep spindles at a frequency of 12-14 Hz, K-complexes, as well as up to 20% of high-voltage delta waves may still be present (Kovacevic-Ristanovic and Kuzniar 2014). Stage N3 of NREM also known as slow wave sleep is characterised by EEG waveforms less than 2 Hz constituting at least 20% of the 30-seconds epoch. Slow wave sleep takes place mostly in the first third of the sleep period (Kovacevic-Ristanovic and Kuzniar 2014). REM and NREM sleep alternate at different intervals between adults and infants, 90 minutes and 60 minutes, respectively. The EEG pattern

of REM sleep looks like that of stage 1 sleep; however, the difference is the presence of rapid eye movements. Furthermore, EMG activity is of low amplitude during this period, and the autonomic nervous system is generally activated at higher than average levels, this includes respiratory rate, heart rate, and blood pressure. About 80% of people recall vivid dreams from REM sleep awakenings, compared to only 5% of NREM sleep awakenings. However, approximately 60% to 80% of some individuals may recall fragmented thoughts from NREM sleep awakenings (Kovacevic-Ristanovic and Kuzniar 2014). It has been written in population studies that the percentage of duration spent in each stage of sleep varies with age and gender. Following are the widely measured parameters when performing a PSG (LOC, left outer canthus; ROC, right outer canthus; A1, the left ear; T3/T4, the temporal regions; C3/Cz/C3, the central areas; O1, the occipital; EMG, the electromyogram under the chin and on the tibialis anterior surface; ECG, the electrocardiogram; LL, for left leg; RL, for the right leg; NA, for nasal airflow; OA, oral airflow which may be separate or connected with the NA; TM bands, for thoracic movement during breathing; AM bands, for abdomen breathing movement; IC, intercostal EMG; SAO2, oxygen saturation) (Yamada and Meng 2012).

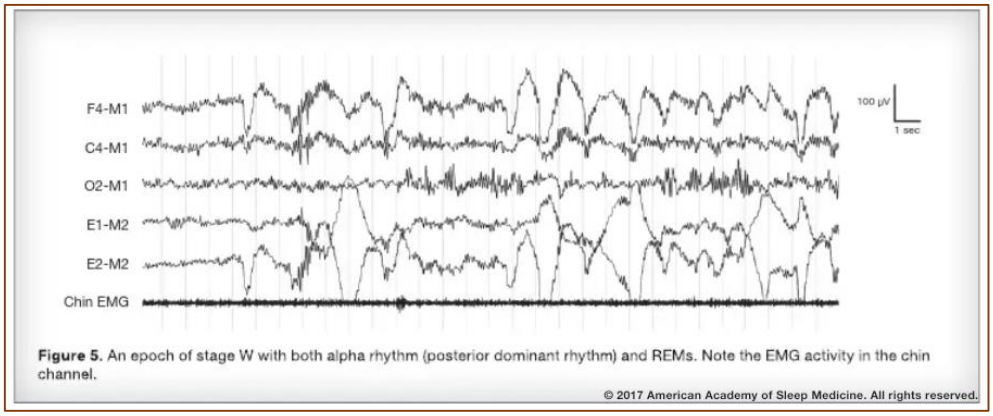


Figure 6: Sleep stage – Wake
Source: Kapur *et al.* (2017)

The alertness state known as Wake (Figure 6), ranges from full alertness to early drowsiness. Alpha rhythm is demonstrated during eye closure in the majority of individuals, and during eye-opening a low amplitude intermixed background activity of beta and alpha frequencies is noted without the posterior alpha rhythm (Whitten *et al.* 2011). During an awake state, EOG usually demonstrates rapid eye blinks at a frequency of about 0.5-2 Hz. The chin EMG during this period is of variable amplitude usually higher than during the sleeping stage (Berry *et al.* 2012a).

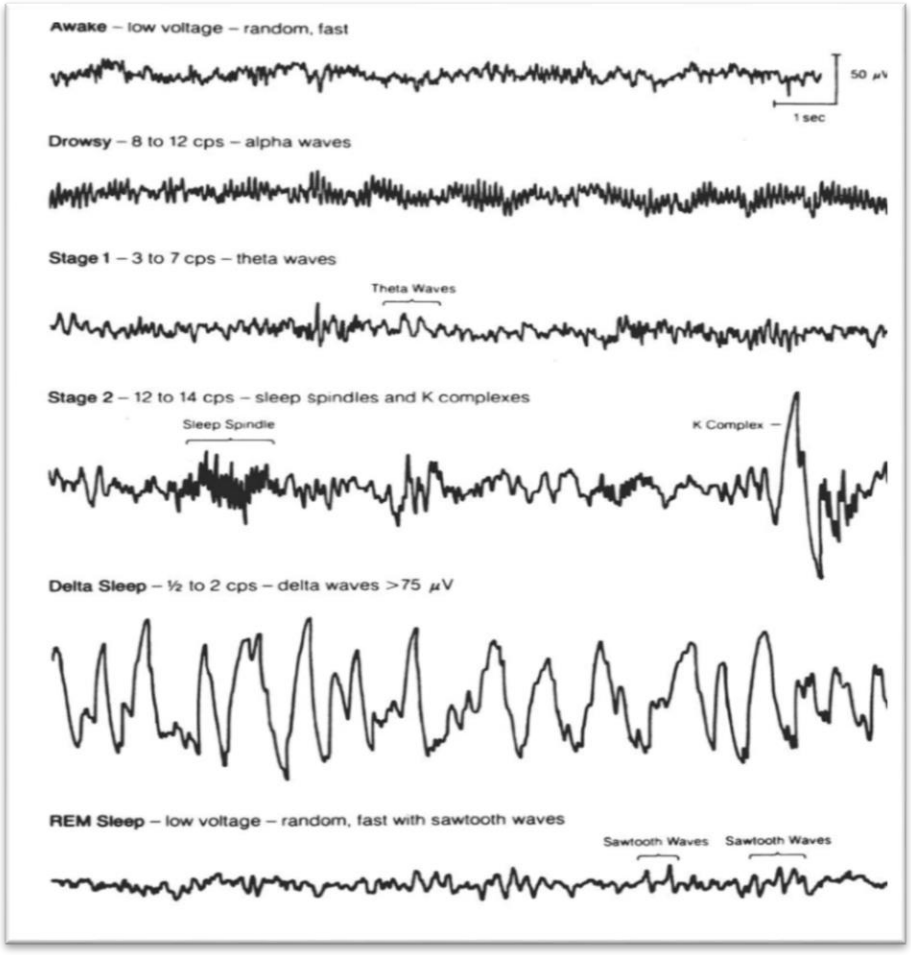


Figure 3: All sleep stages
Source: Radtke (1990), Duckrow and Zaveri 2005)

Scoring stage N1 involves looking for slow eye movements (SEM), that are conjugate, regular, and sinusoidal with an initial deflection that usually lasts > 500 msec (Berry *et al.* 2012a). The background during this period consists of low amplitude, mixed frequency activity (LAMF), predominantly 4-7 Hz or cycles per second (cps) delta to theta activity attenuating alpha rhythm (Berry *et al.* 2012a). Vertex sharp waves (V waves) are sharply contoured with a duration of < 0.5 seconds, usually maximal over the central regions (Figure 7).

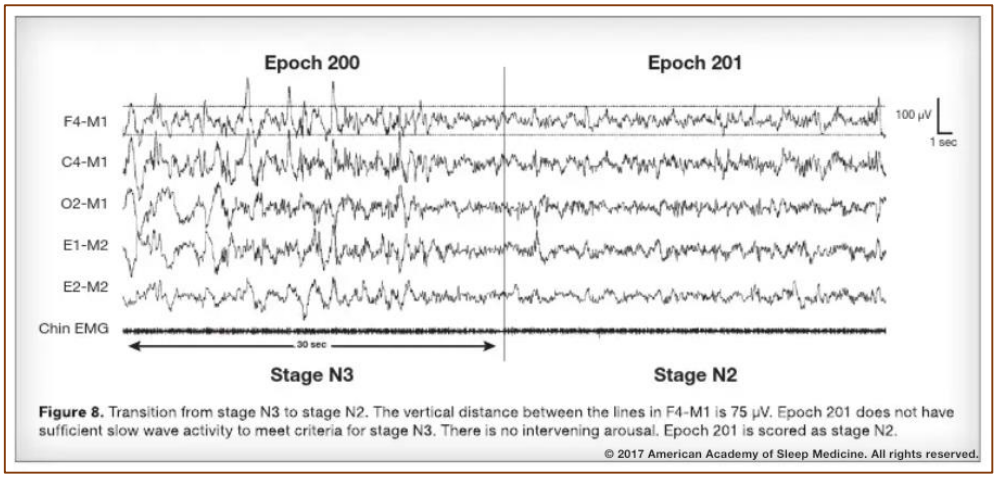


Figure 4: Sleep stage 2 (N2)
Source: Kapur *et al.* (2017)

Figure 8 shows an epoch scored as stage N2 because of the transition from stage N3 to stage N2.

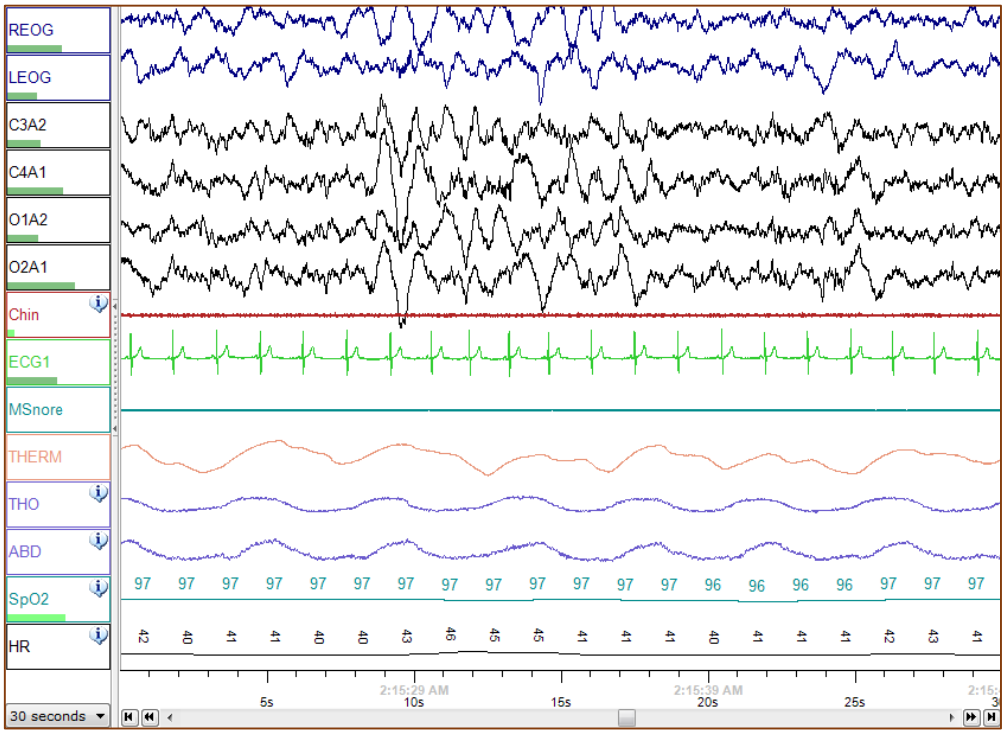


Figure 5: Sleep stage N3
Source: Sleep stage N3. Png (commons.m.wikimedia.org)

Stage N3 of sleep (Figure 9) is scored in the presence of slow activity. This activity consists of 0.5 Hz to 2 Hz frequency and amplitude of greater than 75 micro-volts measured from peak-to-peak over the most anterior regions of the placements in the recording. A 30-sec epoch filled with greater than or equal to 20% of slow wave activity

is scored as Stage N3 of sleep, irrespective of age. Sleep spindles may persist in the N3 sleep stage. The chin EMG amplitude varies in N3, often lower than that of stage N2, at times it is as low as in the REM sleep stage (Berry *et al.* 2012a).

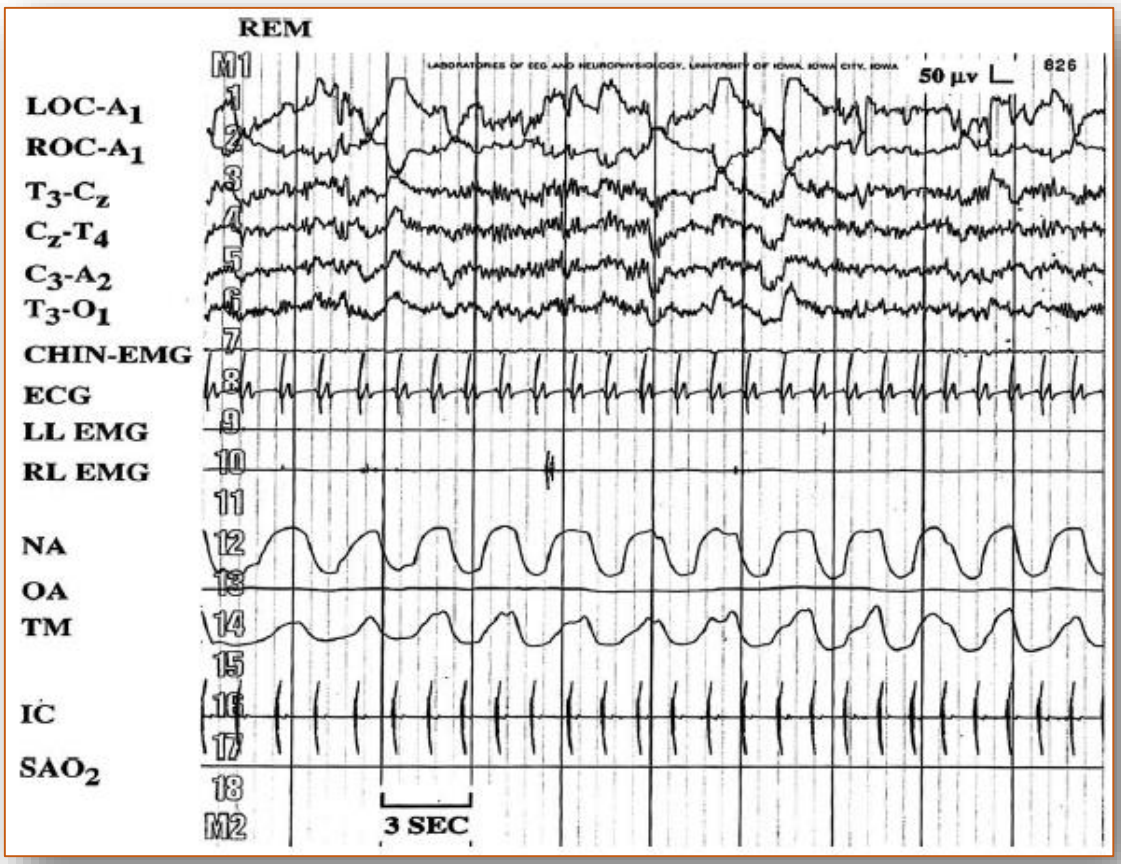


Figure 6: Stage REM

The EMG activity of a young adult in REM sleep is markedly reduced with occasional bursts of muscle activity, the bursts are associated with rapid eye movements (Figure 10). REM background has relatively low low-voltage, mixed-frequency activity with slow alpha/theta patterns and intermittent 3 Hz “saw-tooth” patterns in the anterior and the central regions (Berry *et al.* 2012a).

Older people tend to wake up more often throughout the night and are reported or known to sleep less deeply (Lavoie, Zeidler and Martin 2018). This is thought to be due to less production and secretion of melatonin, the hormone responsible for promoting sleep, the results include a decreased amount of slow-wave sleep, and this stage of

sleep is crucially needed as other sleep stages when scoring sleep stages (Pandi-Perumal *et al.* 2005). They may also be more sensitive to the changes in the environment, such as noise, and this may result in fragmented sleep (Berry *et al.* 2012a).

Early diagnosis of SDB prevents the incident of developing various conditions, which may include but are not limited to coronary artery disease and stroke, as nocturnal hypoxia that happens during SDB exacerbates ischaemic heart disease and therefore encourages the occurrence of pulmonary hypertension. Early diagnosis also improves the quality of life and neurocognitive dysfunction and reduces vehicle accidents as these patients tend to suffer from daytime hypersomnolence (Peppard *et al.* 2000; Ip *et al.* 2002; Arzt *et al.* 2005; Gami *et al.* 2005; Patil *et al.* 2007).

2.6 Sleep disorders

Sleep disorders are a wide range of conditions that are a result or cause of a disturbed ability to sleep well at night (Pavlova and Latreille 2019). These disorders may be a result of health-related issues or enormous stress. Sleep disorders have different effects on the body, depending on the type of the disorder, some individuals may have difficulty initiating sleep or keeping asleep throughout the night which then in turn results in extreme tiredness throughout the day. However, some people may wake up not complaining of sleepless nights but still complain about excessive day time sleepiness. The lack of sleep can affect energy, mood, concentration, and overall health (Kumar 2008). Signs and Symptoms differ as it depends on the type and severity of the sleep condition the individual is suffering from. These characteristics vary when the sleep disorder is not the primary problem but a result of an underlying condition. Common symptoms may include but are not limited to difficulty falling asleep or staying asleep, daytime tiredness, a strong urge to take multiple naps daily, lack of concentration, irritability, anxiety, and depression (Pavlova and Latreille 2019).

2.6.1 Categorisation of sleep abnormalities

The International Classification of Sleep Disorders-2 (ICSD-2) divides sleep disorders into insomnias, sleep-disordered breathing, hypersomnia of central origin, circadian

rhythm disorders, sleep-related movement disorders, and parasomnias (Kovacevic-Ristanovic and Kuzniar 2014). The sleep disorder of interest in this study is sleep-disordered breathing.

2.6.1.1 Insomnias

Insomnia is a true feeling of interrupted, inadequate, or non-restorative sleep regardless of sufficient sleeping duration. This is then further accompanied by daytime challenges such as falling asleep in places or under situations you normally would not fall asleep in (Worley 2018). Insomnia may be differentiated into three types: firstly, the sleep-onset delay or sleep onset insomnia, which is characterised by the struggle to initiate asleep at bedtime (Morin, Jarvis and Lynch 2007); secondly, the premature awakening or terminal insomnia, where there is early morning arousal; thirdly, sleep-maintenance insomnia, where sleep fragmentation with an inability to fall asleep again takes place (Worley 2018).

There are acute and chronic insomnias, less than three to four weeks, and more than four weeks, respectively. There are quite a number of factors that can trigger acute insomnia, namely, stressors of life, brief illness, rapid changes in time zones, drug withdrawal, use of central nervous system stimulants, and pain (Worley 2018). On the other hand, chronic insomnia may be related to other underlying conditions such are chronic psychophysiological arousal, psychiatric disorders, use of drugs and alcohol, and other medical, toxic, and environmental conditions (Worley 2018). Not forgetting that insomnia may also be a significant of a primary sleep apnoea syndrome, alveolar hypoventilation syndrome, periodic limb movements disorder, and development of restless leg syndrome. The major diagnostic phenomena of insomnia are difficulty getting to sleep or staying asleep, with associated daytime complaints of inability to perform certain daily life activities. (Worley 2018).

Intrinsic sleep disorders include psychophysiological insomnia, sleep-state misperception (paradoxical insomnia), restless leg syndrome, and idiopathic insomnia, all of which have insomnia as one of the complaints. Likewise, a lot of extrinsic sleep disorders such as inadequate sleep hygiene, environmental sleep disorder, altitude insomnia, adjustment sleep disorder, limit-setting sleep disorder, food allergy

insomnia, hypnotic-dependent sleep disorder, alcohol-dependant sleep disorder, and sleep-disordered breathing are likely to be accompanied by insomnia (Kovacevic-Ristanovic and Kuzniar 2014).

Insomnia is a commonly reported complaint in patients with sleep onset delay, as the latter has psychogenic causes where consciousness effort to fall asleep leads to central nervous system (CNS) arousal (Kovacevic-Ristanovic and Kuzniar 2014). Patients tent to describe themselves as light sleepers. Light sleepers usually have numerous somatic symptoms such as headaches, back pains, and palpitations that become better with the use of barbiturates, alcohol, minor tranquillisers, and hypnotics. This promotes the abuse of these substances (Kovacevic-Ristanovic and Kuzniar 2014).

Sleep-onset delay insomnia may be caused by restless legs syndrome (RLS), due to increased unpleasant creeping sensations in the calves whenever resting, especially during the night. This feeling creates an utmost irresistible urge to move the legs to relieve the sensation, which then interferes with sleep onset. Individuals with RLS mostly complain of tiredness and not feeling fully alert. RLS is reported to be more prevalent in women (Kovacevic-Ristanovic and Kuzniar 2014). Secondary causes of RLS include pregnancy, iron deficiency, and end-stage renal disease.

Neuropathies and radiculopathies have also been found to be associated with RLS, especially those caused by rheumatoid arthritis and diabetes mellitus (Mogavero *et al.* 2021). Dietary substances like caffeine and medication such as neuroleptics and tricyclic antidepressants can cause or exasperate RLS and periodic limb movements (PLMs) (Worley 2018). The medical conditions that sometimes lead to insomnia include alveolar hypoventilation, which in adults could be secondary to being overweight, chronic obstructive pulmonary disease, myopathy, cordotomy, and stroke (Brass *et al.* 2010).

2.6.1.2 Sleep-related breathing disorders

There are four types of sleep-related breathing disorders, namely, obstructive sleep apnoea (the cessation of breathing due to upper airway obstruction), central sleep

apnoea (cessation of breath due to absent respiratory effort or due to disturbance in the breathing drive), sleep-related hypoventilation disorders (shallow breathing due to a variety of medical or structural conditions), and sleep-related hypoxemia disorder (a condition that occurs when oxygen concentrations drop with increased levels of carbon dioxide that are not high enough to qualify for a diagnosis of sleep-related hypoventilation disorder (Roux, D'Ambrosio and Mohsenin 2000; Sateia 2014).

Sleep apnoea is an abnormal breathing pattern during sleep in a manner that there is the cessation of airflow at the level of the nostrils and the mouth, or due to disturbance in the breathing drive, lasting at least 10 seconds (Kovacevic-Ristanovic and Kuzniar 2014). Sleep apnoea is the most mentioned diagnosis among other sleep disorders, in sleep diagnostic institutes and the most common cause of excessive daytime sleepiness. Apnoeas are subdivided into three types, these include OSA secondary to an induced obstruction of the air pathway during sleep; central sleep apnoea (CSA) secondary to decrease respiratory muscle activity; and mixed apnoea which combines both conditions (Vgontzas, Bixler and Chrousos 2005; Kovacevic-Ristanovic and Kuzniar 2014). In most cases, mixed apnoea begins as a CSA (absent respiratory effort), and then builds up into an OSA with a crescendo-decrescendo pattern. Obstructive sleep apnoea is caused by pharyngeal collapse during muscle contraction (Ryan and Bradley 2005). Contributing factors to look out for, and usually pointed out by the ear-nose and throat specialists may include abnormal anatomical muscular or bony structures of the nasopharynx, oropharynx, or hypopharynx (for example, a short thick neck), macroglossia, micrognathia, retrognathia, a very small and low-positioned hyoid bone, or a narrow pharynx (Kovacevic-Ristanovic and Kuzniar 2014). Clinical presentations of OSA include snoring, pauses of breath at night, and excessive daytime sleepiness (Kovacevic-Ristanovic and Kuzniar 2014). Obesity and elevated blood pressure are frequently present. However, there is a misconception that only people significantly overweight experience sleep apnoea. Only up to 67% of individuals with sleep apnoea are overweight, the rest are not obese (Kushida *et al.* 2015). OSAs are understood to be worse and prevalent with increase in age and further worsen post alcohol or sedative drug intake (Kovacevic-Ristanovic and Kuzniar 2014). Patients with OSA usually report dryness in the mouth/throat upon awakening. On examination, a large neck circumference of ≥ 43.18 cm in males and ≥ 40.61 cm in females,

micrognathia, and retrognathia, and excessive oropharyngeal soft tissue should suggest the diagnosis of OSA (Kovacevic-Ristanovic and Kuzniar 2014).

In sleep apnoea, the cessation of breath during apnoeic periods causes sleep disruptions, and subsequent short awakenings (Kovacevic-Ristanovic and Kuzniar 2014). Occasionally, patients with sleep apnoea do realise that they wake up frequently at night; this presentation is more frequent in central sleep apnoea rather than in obstructive sleep apnoea, and usually women (Kovacevic-Ristanovic and Kuzniar 2014). Sleep apnoea increases the risk of ischaemic heart disease, cardiovascular death, congestive heart failure, and atrial fibrillation. Recent studies link OSA with the development or worsening of insulin resistance and type 2 diabetes (Nannapaneni, Ramar and Surani 2013). CSA results from any of the many processes that create the unsteadiness of the respiratory control centre (Kovacevic-Ristanovic and Kuzniar 2014).

With upper airway resistance syndrome (UARS), the passage of the upper airway is not fully compromised, nonetheless, symptoms of daytime sleepiness still occur (Kovacevic-Ristanovic and Kuzniar 2014). In a young healthy adult, the tidal volume is roughly 500 ml per inspiration or 7 ml/kg of body mass (Kovacevic-Ristanovic and Kuzniar 2014). When resistance is elevated, inspiratory muscles increase their effort to maintain normal tidal volume. This compensatory process triggers a temporary shift in the brain activity causing arousal, either alpha activity a 3-14 seconds duration of arousal, or other frequencies. This mechanism interrupts obstruction to prevent oxygen desaturation (Kovacevic-Ristanovic and Kuzniar 2014). These frequent arousals result in daytime sleepiness (Kovacevic-Ristanovic and Kuzniar 2014).

Medical and neurological disorders that may lead to the development of either CSA or OSA include among others brainstem infarction, lateral medullary syndrome, neuromuscular disorders (such as Myasthenia gravis), bulbar poliomyelitis, medullary neoplasms, syringomyelia and syringobulbia, olivopontocerebellar atrophy, Alzheimer disease, encephalitides, Creutzfeldt-Jakob disease, post-encephalitic parkinsonism, cervical cordotomy, Guillain-Barre syndrome, and myotonic dystrophy (Kovacevic-Ristanovic and Kuzniar 2014). Hypoventilation and daytime drowsiness are prominent in all of these disorders (Deak and Kirsch 2014; Kovacevic-Ristanovic and Kuzniar

2014). Poliomyelitis can cause atrophy of respiratory accessory muscles, leading to severe chest deformity such as kyphoscoliosis. Furthermore, impairment of cranial motor nerves may adversely affect the tongue and other upper airway muscles. This may result in the occurrence of all types of apnoeas (Deak and Kirsch 2014; Kovacevic-Ristanovic and Kuzniar 2014).

The evaluation of suspected sleep apnoea involves taking a full daytime and sleep history not only from the patient directory but also, most importantly, from their bed partner or someone they share the room with. A medical check-up should be more focused on excluding any blood pressure abnormalities, sign and symptoms of right-sided heart failure, and abnormal skeletal and muscle configurations of the face and neck. An ENT examination is important. Chest radiographs and ECGs are helpful when evaluating pulmonary hypertension, analysing the right and left ventricles condition, and ruling out coexisting cardiopulmonary disorders. A complete blood count may document the presence of polycythaemia in chronic hypoxemia or right-sided heart failure. The primary test that confirms the presence or absence of OSA is a PSG study. It gives a satisfactory estimate of sleep-disordered breathing severity, sleep fragmentation, and desaturation levels. PSG study guides the future of SDB treatment plan. The availability of affordable functional sleep centres will be the major driver of improved diagnosis of OSA (Leung and Douglas Bradley 2001).

Treatment of sleep apnoea may include but is not limited to weight loss, refraining from alcohol, and ceasing the use of hypno-sedative drugs. Due to the effect of gravity on the airway passage, sleep apnoea is typically worse in the supine than in the non-supine position of sleep. In some patients, sleep apnoea is only present in the supine position, while in the lateral sleep position, there are no breathing disturbances. In a case like this, restricting the supine position when sleeping may be of use in controlling apnoeas, by making supine sleep uncomfortable. A bulky object may be placed on the patient's back, such as a tennis ball t-shirt (Pevernagie, Sastry and Vanderveken 2014).

Pharmacologic approaches that are widely used may include protriptyline, mirtazapine, and fluoxetine, as they act directly on the upper airway muscle tone (Kovacevic-Ristanovic and Kuzniar 2014). The mechanism of action applied by CPAP as a mostly

used treatment for OSA is a pneumatic splint action to the upper airway. It produces pressurised air that results in elevated pressure in the oropharynx, which in turn results in transmural pressure gradient normalisation across the oropharyngeal airway, which then opens up the airway. However, some patients have poor compliance or do not use CPAP due to social reasons, discomfort from the mask, poor mask fit resulting in air leakage, and trouble breathing against the incoming air. Bilevel positive airway pressure (BiPAP) in a spontaneous mode (BiPAP-S) is an efficient substitute for individuals experiencing discomfort while expiring against the high air pressure delivered by CPAP (Kovacevic-Ristanovic and Kuzniar 2014). This device allows independent titration of expiratory (EPAP) and inspiratory (IPAP) airway pressure. BiPAP is usually used in cases of morbid obesity, intrinsic lung disease, and chest deformity, which are conditions associated with hypoventilation. BiPAP in spontaneous-timed mode (BiPAP-ST) is typically used in central sleep apnoea and conditions associated with hypoventilation. An adapt-servo ventilator device detects the patient-generated airflow and supplements it, breath by breath, with a variable, rather than constant pressure support. This modality has been shown to resolve a large proportion of idiopathic and opioid-related central sleep apnoeas, Cheyne-Stokes breathing problems, and complex sleep apnoeas (Kovacevic-Ristanovic and Kuzniar 2014). Surgical interventions involve correction of nasal or airway obstruction such as adenoidectomy or tonsillectomy. Uvulopalatoplasty involves the use of a laser for the partial excision of the uvula and the soft palate. Placement of implants within the soft palate reduces snoring but is unlikely to resolve apnoea. Lastly, surgical correction of maxillofacial anomalies is performed in patients that present with sleep apnoea and have craniofacial abnormalities that result in sleep apnoea (Kovacevic-Ristanovic and Kuzniar 2014). Oral appliances are recommended for patients with mild to moderate sleep apnoea who are not candidates for CPAP therapy or do not like using CPAP machines.

2.6.1.3 Hypersomnias of central origin

This condition involves excessive daytime sleepiness, however, not due to other sleep disorders. Central disorders of hypersomnolence include narcolepsy, idiopathic hypersomnolence, and insufficient sleep syndrome. In contrast, bipolar depression frequently is associated with hypersomnia, accompanied by shortened REM latency

and reduced stage N3 sleep (Young and Silber 2006; Harris, Monderer and Thorpy 2012; Worley 2018).

2.6.1.4 Narcolepsy

Narcolepsy is a syndrome that incorporates both excessive daytime sleepiness (EDS) and abnormal manifestations of the REM stage of sleep (Kovacevic-Ristanovic and Kuzniar 2014). These REM manifestations occur in a way of frequent sleep attacks with involvement of rapid eye movement (REM) periods, which are referred to as hypnagogic when occurring at sleep-onset, and hypnopompic when taking place at sleep-offset, hallucinations, cataplexy, and sleep paralysis (Cheyne 2006). The mostly documented symptoms of narcolepsy involve EDS, unexpected sleep attacks, and cataplexy. Sleep attacks typically last roughly 15 minutes. The ones affected awaken refreshed and it takes about 1 to 5 hours before the next episode happens. Cataplexy is an unanticipated decrease in or unexpected loss of muscle tone that either affects all the muscles or is limited to a particular muscle group (Dauvilliers, Arnulf and Mignot 2007). Cataplectic attacks are triggered by strong emotions such as laughter, surprise, outbursts of anger, or a feeling of exaltation. These attacks generally last for only a few seconds or as long as 30 minutes (Kovacevic-Ristanovic and Kuzniar 2014). Sleep paralysis occurs during sleep onset or when the patient is waking from sleep. Hallucinations are also in a similar manner, and they are usually frightening. Nocturnal sleep is also disrupted by frequent awakenings, frequent sleep-onset REM periods, and vivid dreams. The diagnosis is then confirmed by the Multiple Sleep Latency Test (MSLT), showing a mean sleep latency of 5 minutes or less, with two or more sleep-onset REM periods. However, the occurrence of REM sleep on three or more occasions within 10 minutes of sleep onset during evaluation is taken as evidence of narcolepsy (Kovacevic-Ristanovic and Kuzniar 2014). The presence of shortened nocturnal REM latency captured during the overnight PSG (< 20 minutes) may support the diagnosis (Kovacevic-Ristanovic and Kuzniar 2014). It is further advised that prior to MSLT testing, the medication that might affect the central nervous system be stopped 2-4 weeks in advance, including anxiolytics, sedatives, stimulants, and antidepressants. With that being said, clinical judgement should be used regarding changes to medications that could impair patient safety. The patient must have

sufficient sleep time 2 weeks before testing is commenced. A full night polysomnogram is then recorded the night before the MSLT.

Treating narcolepsy may involve improvement in sleep hygiene, while some patients may require stimulants, primarily dextroamphetamine or methylphenidate. Stimulants have the ability to boost the output of catecholamines as well as hamper their reuptake in the CNS, with serotonin being impacted somewhat less. Stimulants will likely diminish but not entirely eradicate the severity of excessive daytime drowsiness and performance impairments. Side effects often limit the use of stimulants. These may incorporate grumpiness, anxiety, neuralgia, tachycardia, and hypertension (Kovacevic-Ristanovic and Kuzniar 2014). Selective serotonin reuptake inhibitors and tricyclic antidepressants, among others are used to treat narcolepsy and cataplexy, as they tend to suppress REM. Cataplexy is treated by the use of imipramine, nortriptyline, and vivactil. Clomipramine is one of the efficacious drug in treating cataplexy, this is a tricyclic with influential serotonin-uptake inhibition (Kovacevic-Ristanovic and Kuzniar 2014). However, it has undesirable effects such as xerostomia, excessive perspiration, sexual impotence, putting on weight, hard stools, vague vision, and urinary retention (Kovacevic-Ristanovic and Kuzniar 2014).

2.6.1.5 Circadian rhythm sleep-wake disorders

This type of disorder involves aberration of sleep-walk cycles because of incorrect arrangement of the biological clock and customary sleep-wake time. These abnormalities are advanced or delayed sleep phase, shift work disorder, and jet lag (Abbott, Reid and Zee 2015). The delayed sleep-phase syndrome involves a delay in initializing sleep, whereas advanced sleep-phase syndrome entails waking up very early (Kovacevic-Ristanovic and Kuzniar 2014).

A diagnostic procedure used for early awakening and delayed sleep onset involves a PSG finding of a very short period of N3 and REM sleep latency, this may however also be taken as a biological marker of depression, stage N3 sleep also is reduced (Kovacevic-Ristanovic and Kuzniar 2014). Antidepressants are the drugs used for early morning awakening disorder; these include tricyclic antidepressants with sedative properties, such as amitriptyline and trimipramine, as they have the effect of shortening

the latency of sleep and further intensify sleep continuity (Kovacevic-Ristanovic and Kuzniar 2014).

2.6.1.6 Sleep-related movement disorders

Fragmented sleep occurs when there are frequent awakenings at night; these disturbances may be caused by a sleep disorder, primarily sleep apnoea or PLMs (Bonnet and Arand 2003; Kovacevic-Ristanovic and Kuzniar 2014). Kovacevic-Ristanovic and Kuzniar (2014) further state that periodic limb movement is a condition in which there are episodes of repetitive and highly stereotypical leg jerks during sleep. These are followed consistently by very short periods of arousal. Patients are usually not aware of these jerking movements, rather the matters reported are multiple disturbances of sleep with awakenings, and not feeling refreshed in the morning. A roommate or significant other usually can provide an accurate description of the movements (Kovacevic-Ristanovic and Kuzniar 2014).

2.6.1.7 Parasomnias

Parasomnias are abnormal behaviour or events arising from sleep (Wills and Garcia 2002). Parasomnias represent disorders of arousal, and sleep-stage transitions (Kovacevic-Ristanovic and Kuzniar 2014). Disorders of arousal include confusional arousal, sleepwalking, and sleep terrors. They all arise from NREM sleep, usually delta sleep, and can be triggered by forced arousal from delta sleep. These are mostly noted in childhood. Arousal from delta sleep (N3 sleep) is characterised by confusion, disorientation in time and space, and slow speech and mentation. These confusional may transform into sleepwalking, or sleep terror (Kovacevic-Ristanovic and Kuzniar 2014). It is uncommon for individuals to remember these events. The episodes of sleepwalking typically last anywhere from a brief moment up to several minutes in duration before the individual returns back to their state of restfulness. Sudden arousal from NREM slumber escorted by an ear-piercing scream or cry, coupled with behaviours that indicate excessive anxiety and heightened autonomic response is known as night terrors. Autonomic responses include sweating, dilated pupils, piloerection, fast breath, and tachycardia. Several groups of factors may take charge of the development of arousal disorders. These include genetic factors, factors causing

an extra amount of time spent in slow wave sleep or difficulty in returning to an alert state, the latter may be related to (age, recovering from previous sleep deprivation, intensive sickness, CNS depressant drugs, and more), and factors causing sleep fragmentation, such as pain, environmental stimuli, stimulants, stress, sleep apnoea, PLM, and more.

Somnambulism related disorders respond to benzodiazepines such as triazolam, clonazepam, and diazepam and to tricyclic drugs such as imipramine, desipramine, and clomipramine (Kovacevic-Ristanovic and Kuzniar 2014).

parasomnias associated with REM sleep encompasses REM sleep behaviour disorder (RBD), recurrent isolated sleep paralysis, and nightmare disorders. A sudden awakening from REM sleep with a recall of a troubling dream accompanied by anxiety with less noticeable autonomic arousal is known as a nightmare. Upon waking up, the patient is instantly well aware of the surrounding (Sateia 2014). RBD is a parasomnia characterised by a robust movement during REM, often resulting in injury. Episodes of RBD may involve laughter, chatting, chanting, singing, shouting, insulting, reaching for something, grabbing, punching, crawling, and running movements (Kovacevic-Ristanovic and Kuzniar 2014). Clonazepam is the drug of choice for the treatment of RBD.

Other sleep disorders are not classified anywhere as yet, and these are most notably environmental sleep disorders (Sateia 2014).

2.7 The effects of sleep-related breathing disorders

Beside the sleep fragmentation that have been mentioned multiple times in this paper, Yan *et al*. (2021) reported altered cerebral perfusion in OSA, with generalised and localised reduction of cerebral blood flow and volume predominantly in the parietal and prefrontal parts of the brain, the lesions are also multifocal in the cortical regions and deep brain structures. This was assessed using sleep parameters and neuropsychological deficits found using three Telsa Magnetic Resonance Imaging (3 T MRI), PSG, and neuropsychological tests in patients suffering from OSA, asymptomatic patients were used as reference subjects.

In a study done correlating regional hypoperfusion and PSG parameters, it was found that different controls of regional hypoperfusion were directly related to the intermittent hypoxia and sleep fragmentation variables which were notably in the parietal and orbitofrontal regions of the brain, respectively (Acet-Ozturk *et al*. 2022; Yan *et al.* 2021). Furthermore, instances of absolute or incomplete disturbance of airflow while asleep often result in both oxygen and carbon dioxide exchange irregularities and arousal which interrupts sleep (Paruthi, Chervin and Hoppin 2017). This further emphasises the importance of normalising the availability and performance of overnight PSGs in the public sector. These results may suggest an important pathomechanism for neurocognitive deficits in OSA due to impaired cerebral perfusion.

Many studies have been conducted on paediatric OSA. Research finds that paediatric OSA that is left to grow without intervention is highly related to behavioural and learning problems (Kaw *et al.* 2012; Singh *et al.* 2013; Amra *et al.* 2018; Christensson *et al.* 2018; Öztürk *et al.* 2019; Shredl 2021; Acet-Öztürk *et al.* 2022). When severe, it may take part in delayed growth and cardiovascular complications (Paruthi, Chervin and Hoppin 2017) There is a severity grading for OSA. Less than 5 AHI/RDI episodes per hour of sleep are within the normal range. Mild OSA is classified as more than 5 and less than 15 episodes of AHI/respiratory disturbance index (RDI) per hour of sleep; moderate is classified as AHI/RDI greater than 15 but less than 30 episodes per hour of sleep, and severe is classified as AHI/RDI greater than 30 episodes per hour of sleep (Kaw *et al.* 2012; Singh *et al.* 2013; Amra *et al.* 2018; Christensson *et al.* 2018; Öztürk *et al.* 2019; Shredl 2021; Acet-Öztürk *et al.* 2022).

In research done to find the effect of OSA on the transitional dynamics of sleep stages, it was noted that there was no significant percentage difference between sleep efficiency and N1. However, a significant difference was present in REM, N2, and N3 percentages where N2 had a slightly elevated percentage when looking at mild and severe apnoea, and REM and N3 indicated the opposite pattern (Bianchi *et al.* 2010). Other researchers report that higher respiratory disturbance index measurements and bradyarrhythmia occur mainly in REM sleep and may occur independently in the presence of a decrease in oxygen saturation (Koehler *et al.* 2000; Penzel *et al.* 2001).

Hypoxia in SDB appears to affect body functioning in different ways. The desaturation and re-oxygenation sequence is a typical problem coupled with the majority of sleep disorders (Lévy *et al.* 2008). This intermittent hypoxia is reported to lead to oxidative stress, which is defined as an occurrence caused by an imbalance between production and accumulation of reactive oxygen species (ROC) in cells and tissues and the ability of a biological system to detoxify these reactive products (Lévy *et al.* 2008; Pizzino *et al.* 2017). ROC is a type of unstable molecule and highly reactive chemical that contains oxygen as it is formed from diatomic oxygen, water, and hydrogen peroxide, it easily reacts with other molecules in a cell (Liou and Storz 2010; Pizzino *et al.* 2017). This study will not discuss the molecular and cellular mechanism part that occurs in SDB and hopoxia. Among other consequeces that may occur with hypoxia and sleep fragmentation caused by the recurrent arousals resulting from SDB, there may be fatigue, memory loss, muscle and joint pain, wrinkles (increasing in aging), decreased eye sight, headache, sensitivity to noise, and heart failure as a consequence of cardiac impairment due to the increase in muscle sympathetic nervous system activity (Lévy *et al.* 2008). Moreover, the severe the sleep apnoa, the worse the development of the consequences.

2.8 A glance into the risk factors for OSA

Obstructive sleep apnoea is more common in older age groups, specifically the adult male with elevated body mass index (BMI) (Rossi *et al.* 2021). BMI is calculated by taking the weight in kilograms divide it by the square of height in metres. Obstructive sleep apnoea is of significance, a likely life-threatening sleep illness that is not really taken into consideration in state hospitals, as it does not only affect sleep-related health but one's quality of life entirely (Garvey *et al.* 2015). Obstructive sleep apnoea apart from daytime sleepiness is associated with making the individual susceptible to diabetes, stroke, heart disease, hypertension, cognitive dysfunction, poor asthma management, increased road accidents, and affects one's productivity at work (Watson 2016; Shredl 2021).

The severity of OSA is assessed using AHI criteria. AHI is rated on a scale of events per hour of sleep, ≥ 5 (mild), ≥ 15 (moderate), and AHI ≥ 30 is regarded as severe. Garvey *et al.* (2015) estimated that 34% and 17% of men and women respectively

aged 30 to 70 have at least mild OSA. Furthermore, the occurrence rate of moderate to severe OSA was noted to have drastically increased by 10-20% over the past two decades (Chung, Abdullah and Liao 2016). The frequently coming up risk factors of OSA are obesity, male gender, and age above 65 years (Mannarino, Di Filippo and Pirro 2012). Men are reported to be more susceptible to OSA due to the tendency of body fat distribution in an androgenic manner, particularly on the neck ad trunk. Moreover, sex hormones have been found to influence neurologic control over muscle dilation as well as upper airway ventilation (Mannarino, Filippo and Pirro 2012). Alongside this finding lies another one: postmenopausal women not currently using hormone replacement therapy may be faced with a heightened threat of developing OSA when measured against premenopausal females or those already receiving hormone replacement treatment (Mannarino, Filippo and Pirro 2012). It should also be emphasised that some children can experience symptoms associated with OSA, hence early diagnosis is important regardless of age.

Overall, almost all research studies confirm that advancing age, male sex, being male, and higher body mass index increase the risk of developing OSA (Senaratna *et al.* 2017; Fietze *et al.* 2019). As Obstructive sleep apnoea worsens with weight gain the opposite is also proven to be true with the reduction of weight, hence patients are encouraged to work on weight reduction (Kim *et al.* 2014; Pahkala *et al.* 2014; Subramani *et al.* 2017). The actual relationship between OSA and obesity is not clearly interpreted, however fairly recognizable that fat deposits mainly narrow the upper airway by adding an extra tissue around the neck, this alters the opening and closing mechanism of the airway, reducing chest wall operating standards, the respiratory drive and blood compensation alliance gets compromised (Pahkala *et al*., 2014). Patients with a BMI ≥ 30 kg/m^2 are known to be at high-risk (Berry *et al*., 2017; Chung *et al*., 2013; Aurora and Quan 2016; Duarte *et al.* 2019), with the correlation studies between BMI and AHI are strongly suggesting sleep apnoea (Chung *et al*., 2014). The risk for OSA is increased over tenfold in people with a BMI of > 29 kg/m2 (Mergen *et al.* 2019). The mechanism whereby aging increases the risk of OSA is not fully understood. Aging being a risk factor of sleep apnoea may be driven by one or more of the physiological characteristics. Changes in how the brain controls breathing during sleep may raise the risk of sleep apnoea. Poor upper airway anatomy, as it becomes more prone to collapsibility; ineffective upper airway dilator muscle activity, as there is

reduced upper airway muscle reflex response to negative pressure in comparison with younger individuals; a low respiratory arousal threshold, as older individuals tend to have an increased frequency of spontaneous arousals, or an unstable ventilator control system (Edwards *et al.* 2014). Researchers strongly suggest that high-risk groups should be unquestionably assessed for the risk of OSA with a verified questionnaire (Berry *et al*., 2017; Aurora and Quan, 2016; Chung *et al*., 2013; Duarte *et al*., 2019; Kee *et al*., 2018; Mergen *et al*., 2019; Shredl 2021).

2.8.1 Treatment and therapeutic approaches for OSA

Different types of therapeutic approaches can be used to treat OSA. These include surgical and nonsurgical therapies (White 2006). Treatment decisions are individualised and are highly dependent on the clinical assessment of night-time sleep disruptions, either sudden or encountered changes in daily activities over a certain duration, and PSG study findings (Chang, Chen and Du 2020). Treatment plans differ between children and adults (Paruthi, Chervin and Hoppin 2017). With children, age is also considered the main part when making decisions about the treatment plan. If the patient is an adult, it then depends on the diagnosis and all other underlying factors and clinical symptoms. This is considered together with the results of PSG performed (Paruthi, Chervin and Hoppin 2017; Mansfield, Antic and McEvoy 2013).

The appropriate specialist may perform adenotonsillectomy, or advise on positive airway pressure therapy, watchful waiting for up to six months, rapid maxillary expansion (RME), mandibular advancement devices, corticosteroids, or anti-inflammatory therapy (Van den Berg *et al.* 1999; Paruthi, Chervin and Hoppin 2017). Referral for an adenotonsillectomy assessment is desirably recommended for a patient with physical examination and PSG-confirmed OSA and adenotonsillar hypertrophy (including ≥ 1 + tonsils) (Van den Berg *et al.* 1999; Paruthi, Chervin and Hoppin 2017). Informing the family about the results of conducted evaluations, taking into consideration the severity of symptoms, tonsil size, and severity of sleep apnoea is crucial (Paruthi 2015; Paruthi, Chervin and Hoppin 2017).

Positive airway pressure (PAP) therapy may be highly recommended for patients with a small potion of adenotonsillar tissue or those that are highly not recommended for

surgical approach. Stabilization of children with severe OSA may be necessary prior to adenotonsillectomy or another surgical procedure, or for those who continue to experience OSA after adenotonsillectomy. PAP may be administered as CPAP or BiPAP (Paruthi 2015). Other treatment options should be discussed with the patient. These may include RME, mandibular advancement devices, corticosteroids or anti-inflammatory therapy, and other therapies that may involve weight loss, and avoiding triggering situations such as environmental allergens or irritants (tobacco smoke). In addition, positional therapy, as simple as the elevation of the head of the bed can be considered.

Nutritional counselling with follow-up appointments to ensure reinforcement is of necessity for patients that need to lose weight. Severe obesity and associated morbidities, such as OSA, may warrant consideration of weight loss surgery as a viable option (Joosten, Hamilton and Naughton 2017). A mandibular advancement device may be used as it is also another type of oral appliance, it works by protruding the mandible thereby avoiding the tongue from falling backward and blocking air, this results in increased airway patency. This approach may be reasonable to consider when other treatment methods have failed (Schwartz *et al.* 2018).

Another treatment option is corticosteroids or anti-inflammatories considered for patients with mild or moderate OSA and obstruction of the nostrils resulting from adenoidal hypertrophy/ allergic rhinitis. A 2-4 weeks' intranasal corticosteroids or leukotriene modifier therapy may be used, which gives a desirable duration for a concrete decision to be made in determining whether an alternative therapy should be introduced (Grainger and Drake-Lee 2006; Park and Helman 2015; Liming *et al.* 2019).

Adenotonsillectomy or additional surgical procedures like uvulopalatopharyngoplasty, supraglottoplasty, tongue reduction, tongue base procedures, hypoglossal nerve stimulation, expansion sphincter pharyngoplasty, lateral pharyngoplasty, mandibular distraction osteogenesis may be included as surgical therapies. Tracheotomy is mostly used for children who had persistent OSA despite numerous treatment approaches (Mitchell 2007; MacKay and Chan 2016). Adenotonsillectomy is the excision of tonsils and adenoids (Boudewyns *et al.* 2017).

Positive airway pressure (PAP) is a nonsurgical therapy that involves the administration of air with pressure through a mask, preventing upper airway obstruction (Räsänen *et al.* 1985). PAP may not be feasible for all patients; for example, those with proper mask fitting, and cognitive behavioural (Zozula and Rosen 2001). Contraindications to CPAP include recent or currently active pneumothorax because higher airway pressures may worsen the air leak or cause recurrence. Hypoventilation primarily due to OSA may be treated effectively using CPAP. Hypoventilation is characterised by carbon dioxide (CO_2) retention detected on blood gas evaluation (elevated partial pressure of CO_2 [$PaCO_2$]) or polysomnography (elevated end-tidal CO_2). BiPAP and other non-invasive or invasive ventilation strategies can be used for patients with hypoventilation (Giles *et al.* 2006; Halbower 2020). CPAP and BiPAP are the two types of positive airway pressure. The difference between the two is that CPAP introduces air pressure t a constant level throughout the respiratory cycle, whereas during BiPAP there is more air introduced at a higher level during inspiration and the opposite is true during exhalation (Giles *et al.* 2006).

Another test that can be done to increase airway opening and reduce nocturnal obstruction is a rapid maxillary expansion (RME) which is an orthodontic technique that increases the airway by stretching out the palate and nasal passage (Figure 11) (Brunetto *et al.* 2022). REM can be used for children with narrow plate and small adenotonsillar tissue or those with residual OSA symptoms after adenotonsillectomy, all of this is successfully done before puberty (Pirelli, Saponara and Guilleminault 2015).

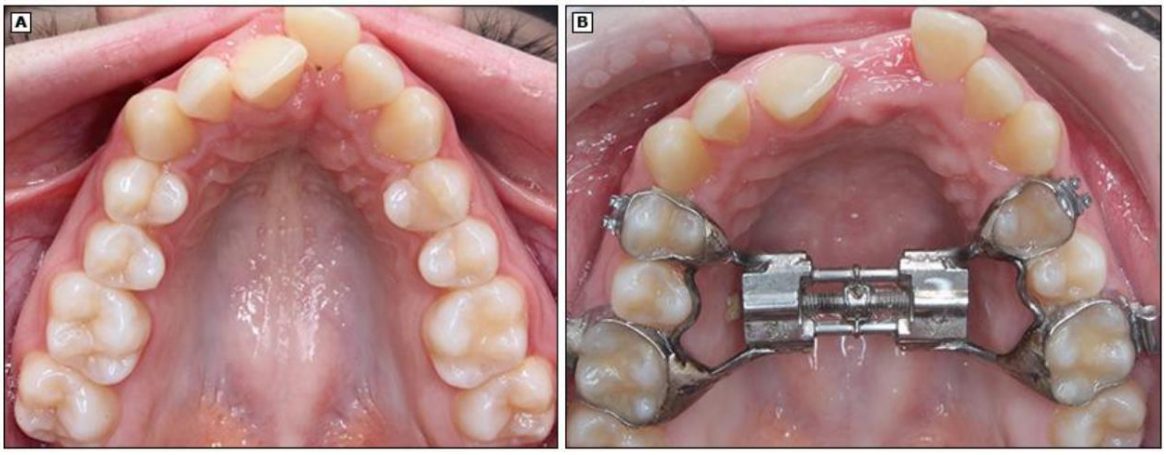


Figure 7: Rapid maxillary expansion
Source: Graphic 103338 Version 2.0 Photo courtesy of R.S. Conley, DDS

This dental appliance is placed in a way that it touches the palate and is held in positions by connecting to the molars. Its job is to expand the palate sideways n (Machado-Júnior, Zancanella and Crespo 2016).

Leukotriene modifier therapy is acclaimed for its use in reducing AHI and feasibly the size of adenotonsillar (Al-Ghamdi *et al.* 1997; Kheirandish, Goldbart and Gozal 2006). Nocturnal supplemental oxygen is useful for patients with severe hypoxemia associated with OSA, as well as patients who are not candidates for surgical treatment and those who are not able to tolerate PAP (Gottlieb *et al.* 2014). Antibiotics may only be useful in patients with recurrent throat infections They reduce the size of tonsils and adenoids, thereby temporarily improving OSA (Jazi, Barati and Kheradmand 2011).

Positional therapy is effective in OSA in a case where AHI is substantially higher when sleeping in a certain position compared to other positions. Pronation during sleep may make OSA worse in some patients (Zuberi, Rekab and Nguyen 2004; Richard *et al.* 2006). Therefore, positional therapy (avoiding certain position that appears to exacerbate apnoeas) may be a consideration. Tools to maintain a non-supine position may include t-shirts with a tennis-shaped bump at the back and the Sona Pillow. Studies in this area have been scarce and lack randomisation (Zuberi, Rekab and Nguyen 2004; Chan, Lee and Cistulli 2008).

Myofascial or myofunctional re-education involves physical therapy exercises that aim to strengthen the tongue and orofacial muscles, with a focus on retraining nose breathing, intending to reposition muscles to the appropriate position (McKeown, O'Connor-Reina and Plaza 2021). This approach is suggested for patients that could not succeed with adenotonsillectomy surgery or CPAP. However, information on efficacy is limited (McKeown, O'Connor-Reina and Plaza 2021).

2.9 The importance of using questionnaires prior to conducting the PSG

A body mass index (BMI) of ≥ 30 kg/m^2 is a substantial risk factor for obstructive-related apnoea (Garvey *et al*., 2015). The purpose of the screening tool is to assess the risk of developing sleep apnoea and further create an easy way to determine patients that are really in need of a PSG referral and increase the likelihood of diagnosis (Aurora

and Quan 2016). The four questionnaires used at IALCH can be laborious to fill out and the final score is calculated manually as this does not require special computer software to do. Soorujlall *et al*. (2021) conducted a study to determine the best screening questionnaire among the four. They concluded that the Berlin questionnaire matched best with the diagnostic PSG. The STOP-Bang questionnaire (SBQ) was reported as producing false positives hence it was ranked last. However, in other studies, SBQ was ranked to be the most suitable screening test due to its sensitivity in checking for OSA patients with obesity (Mergen *et al*. 2019).

It is also important to emphasise the point that questionnaires allow a much better chance of identifying those with undiagnosed sleep apnoea and save others from undergoing unnecessary and costly investigations. Therefore, using a questionnaire addresses the challenges of costs, a night spent in the sleep laboratory, suspense, and avoiding wasting important resources that could have saved someone's life (Kapur *et al*. 2017).

2.10 The effect of the change of adult scoring rules in scoring respiratory events

PSG data were initially scored according to AASM scoring manual version 2.0. However, to assess AHI, sleep stages, and respiratory events were then scored using the current recommended scoring rules. To score a respiratory event in the diagnostic study the peak signal excursion has to drop by greater than 90% and greater than 10 seconds in duration compared to the pre-event baseline when an oro-nasal thermal sensor or any other type of apnoea sensor used in the diagnostic study, PAP device flow in the titration study (Berry *et al.* 2012b).

All rules and notes were taken into consideration; for instance, when an event is partly hypopnoea and the other meets the standard for an apnoea, then the entire episode is scored as an apnoea (Berry *et al.* 2012b). The apnoea or hypopnoea events are scored in an epoch taken as sleep without the consideration of the stage of sleep (Berry *et al.* 2012b; Whitmore 2012). However, Berry *et al.* (2012b) and Whitmore (2012) state that apnoeas or hypopnoeas taking place during a page marked as an awake state should not be scored or included in the AHI due to the difficulty of determining the denominator in this situation. Exclusively, in the case of these events occurring multiple times,

disturbing sleep onset mentioning their presence in the report is important as this would be depriving the patient of peaceful sleep (Berry *et al.* 2012b; Whitmore 2012).

The use of supplemental oxygen during PSG is somewhat discouraged as it may affect desaturation. There is not even an available scoring guideline to support its use during the study. If for some significant reason, the diagnostic study had to be performed during the supplemental oxygen, this would need to be mentioned in the report, and the study would be of uncertain significance (Berry *et al.* 2012b).

Sleep apnoea is classified as obstructive, central, and mixed. Scoring an apnoea as OSA needs to meet the previously-mentioned criteria and also include elevated respiratory effort during the period of disturbed airflow (Berry *et al.* 2012; Rosenberg and Van Hout 2014). Scoring an apnoea as CSA needs to meet the criteria plus entirely no presence of respiratory effort during the period of completely ceased airflow (Berry *et al.* 2012b). Lastly, when scoring a mixed apnoea episode, the criteria need to be taken into consideration accompanied by the initial piece of the event composed of absent inspiratory effort accompanied by the resumption of inspiratory effort in the second part of the event (Berry *et al. 2012a;* Berry *et al.* 2012b). There has not been adequate supportive evidence in describing the CSA and OSA duration part of mixed apnoea, thus the duration of 10 seconds or way longer during an event is not essential in the definitions (Berry *et al.* 2012b).

An episode qualifies to be scored as a hypopnoea when the peak-to-peak waveform amplitude drop by greater than thirty percent in comparison with the baseline event when using either a nasal pressure in the diagnostic study, or a PAP machine during the titration study, or any other alternative hypopnea sensor. The duration of the event needs to be ≥ 10 seconds. The event has to be accompanied by either an oxygen desaturation of three percent or arousal, or both (Berry *et al.* 2012a).

Hypopneas can also be scored as either obstructive or central hypopneas. Hypopneas are scored as obstructive when the event is associated with either a snore, flat to nearly flat nasal pressure, or PAP machine flow signal when compared to that individual's normal breathing level, or in the presence of thoracoabdominal paradox (Berry *et al.*

2012a; Berry *et al.* 2017). Central hypopnoeas are scored when all that is present during obstructive hypopnoea is not occurring (Berry *et al.* 2012a).

The hypopnoea updates that came after version 2.0.1 make a big difference in assisting patients with a PAP compensation and other treatment plans as hypopnoeas are now scored using the above-mentioned criteria, including an oxygen desaturation of ≥ 3% which was previously not the case. The studies done have shown that there is a higher prevalence of sleep disordered breathing (SDB) with an oxygen desaturation threshold index of 3% when compared to when the 4% rule was used on its own (Berry *et al.* 2014; Berry *et al.* 2017).

Respiratory effort-related arousal (RERA) is scored in the presence of two occasions. Firstly, when there is an increased breathing effort or flattening of the nasal pressure line in the diagnostic study or a PAP machine flow in the titration study resulting in arousal. Secondly, RERA is scored when the disturbance of respiratory during sleep does not the criteria for an apnoea or hypopnoea (Berry *et al.* 2012a).

Cheyne-stokes breathing is a rare one and is characterised by the presence of ≥ 3 episodes of central apnoeas or central hypopnoeas in a row set apart by crescendo and decrescendo change in the respiratory amplitude lasting for a duration of ≥ 40 seconds (Berry *et al.* 2014; Khalid, Ayache and Auckley 2021; Berry *et al.* 2017). The apnoeas do not need to always occur consecutively all the time, sometimes the appearance of ≥ 5 central apnoeas or hypopnoeas per hour of sleep together with crescendo and decrescendo breathing pattern recorded in a ≥ 2 hour of sleep monitoring is enough to qualify a breathing pattern as a cheyne-stoke (Berry *et al.* 2014; Khalid, Ayache and Auckley 2021). The cycle length in Cheyne-Stokes breathing is measured from the beginning of a central apnoea to the end of the next crescendo-decrescendo pattern. More importantly, the central apnoeas that build the Cheyne stoke are also scored independently (Berry *et al.* 2014; Khalid, Ayache and Auckley 2021).

2.11 Effect of Stilnox/zolpidem in SDB

Stilnox, the brand name for the generic drug zolpidem, is a non-benzodiazepine drug that works on selective benzodiazepine receptor sites. It has more hypnotic action, which mean it works fast, with little or no anxiolytic or relaxant properties and a short half life of 2-3 hours depending on the dose taken (Jullian-Desayes *et al.* 2017). However, was found to be associated with loud snoring, RERAs, and hypopneas when given to patients during afternoon nap studies. This may be due to its effect on suppressing arousal (Keenan 2005). However, Abuan *et al.* (2020), reported the thickness of tongue muscle becoming thinner during the drug-induced sleep. Ahmad *et al*. (2022) found contrary results in their study which had 11 participants. The study was done to compare the variables when a drug-inducing sleep is used and when it is not, with the use of zolpidem resulting in reduced respiratory arousal index (39.7 ± 7.7 vs. 23.3 ± 4.4 events/h, P = 0.031). It also lowered the total AHI (55.6 ± 8.5 vs. 41.3 ± 7.5 events/hour, P = 0.033), and other clinical and physiological parameters were not affected (Ahmad *et al.* 2022).

Stilnox of 5-10 mg was used in all the patients that were recruited in this study as part of the routine when performing ANPSG at IALCH, as it assisted the patients to fall off to sleep without being severely affected by the external noise in the ward. It is mentioned in some articles and books that inadequate sleep or not sleeping and the use of any type of drug to activate sleep are not motivated when conducting sleep studies (Keenan 2005). However, it was reported that a single dose of 20 mg zolpidem on nocturnal breathing in patients with mild forms of sleep apnea syndrome, does not overcome the existing contraindications to the use of hypnotics in SBD (Cirignotta *et al.* 1988). A study conducted to compare ANPSG and OPSG had all the candidates take midazolam in ANPSG to induce sleep successfully, and no complications were observed (midazolam doses 6.2 ± 3.8 mg) (Gregório *et al.* 2011). Furthermore, Jullian-Desayes *et al.* (2017), gave a review on the hypnotics used in the study which one of the drugs was zolpidem, and there was no worsening of OSA or a significant increase in AHI. A study conducted in 2015, by Mason, Cates and Smith indicated no pharmacological compounds evidence suggesting deleterious effect on the severity of OSA as measured by AHI or RDI and there was a minimum overnight SpO_2 decrease (Mason, Cates and Smith 2015). Last but not least, upper airway patency and

collapsibility were found to be not affected by either zolpidem nor propofol, and this was done by measuring peak inspiratory flow and negative effort dependence (the marker of the dynamic compliance of the pharyngeal structure involved in collapse). These measurements remained unchanged during zolpidem-and propofol-induced sleep (Ordones *et al.* 2020).

Zolpidem is known for its tolerability and its effect on improving sleep quality but it might have variable effects on sleep. There is a need for more studies to incorporate more characteristics like arousal threshold, the pharyngeal passive critical closing pressure, and muscle responsiveness with different dosages. This will assist in understanding the inter-individual heterogeneity and the actual therapeutic potential of zolpidem for different types of people with OSA (Carberry, Grunstein and Eckert 2019).

2.12 Conclusion

Sleep disordered breathing is a serious matter and can affect peoples' lives severely. SDB can cause sleep deficiency which can impact the brain's functioning and that in turn interfere with work, school, driving, among other issues. As one can have trouble with concentrating, socialising as feeling frustrated and tired all the time takes over. Insufficient sleep can lead to obesity, diabetes and impaired glucose tolerance, cardiovascular disease, hypertension, anxiety symptoms, and depression.

It is widely known that in severe cases hypoxemia can interfere with heart and brain function. As SDB is left untreated lack of oxygen in the body's organs and tissues can occurs. There needs to be more states hospitals which can assist in diagnosis and treatment of SDB conditions in South Africa.

CHAPTER 3: RESEARCH DESIGN, METHODOLOGY AND METHODS

3.1 Introduction to this chapter

The basic set of assumptions that guided this research was the positivist paradigm comparing the AHI between the ANPSG and OPSG. The study was experimental, measurements were taken into consideration and sought sensitivity and specificity of each sleep study in the SDB diagnosis. The ontological philosophy was that SDB could manifest during sleep irrespective of the sleep duration (Gregório *et al.* 2011). The independent variable in this study was sleep, and the dependent variables were all the indices that were measured and calculated.

The diagnostic accuracy rate was measured and compared between ANPSG and OPSG in sleep-disordered breathing patients. The study hypothesised that ANPSG may yield similar results in 2-4 hours relative to OPSG in 6-10 hours. The controversial narrative was that ANPSG cannot be fully used as an alternative to OPSG (Gregório *et al.* 2011). The ANPSG is a standard operating procedure to diagnose sleep breathing disorder (SBD) at IALCH.

3.2 Research design

The research design was experimental. Different variables measured during ANPSG and OPSG were compared. The researcher utilised a positivist paradigm. This was a quantitative prospective study that analysed the medical history and PSG results. The same equipment and conditions were used for both PSG studies to make comparison easy, the only difference being the duration of sleep. Informed consent was obtained on the day of the ANPSG. PSG is a multi-channeled test specifically used in evaluating sleep stages and sleep-related breathing events to diagnose sleep disorders. During PSG, physiological parameters are measured simultaneously and continuously throughout the recording (Matheson, Singh and Packard 2007).

3.2.1 Aim of the study

The study aimed to compare the AHI and other polysomnography variables between the OPSG and ANPSG tests. As previously explained, ANPSG is the simplified form of sleep study, and OPSG is the sleep study done throughout the whole night. Different variables are scored and calculated manually to have an outcome of AHI. The dependent variable in this study is sleep, which includes sleep architecture and sleep duration recorded. The independent variables measured include sleep efficiency, arousal profile, disordered breathing profile, body position, oxygen saturation, heart rate, and restless leg profile.

3.3 The research setting

3.3.1 Study setting

The research study was conducted at Inkosi Albert Luthuli Central Hospital (IALCH) in Durban, KwaZulu-Natal, South Africa. The sleep laboratory, in the Department of Pulmonology ward A3 East, was where the sleep studies were conducted. IALCH management and the Department of Health, KwaZulu-Natal provincial government granted the approval to conduct the study (Annexures 1, 2, 3, 16, 21, and 22). All patients underwent the ANPSG and OPSG on consecutive days. The sleep recording times for ANPSG and OPSG were expressed as x-h PSG, where x = 2 to 4 hrs, and x = 6 to 10 hrs, respectively.

This study investigated the accuracy of the measured diagnostic results relative to the ANPSG and OPSG on the same patient (Gregório *et al.* 2011). IALCH uses ANPSG routinely because of its cost-effectiveness, and short duration, and it is performed in the afternoon during normal working hours.

3.3.2 Population and sampling

The patients recruited were between the ages of 18 and 70 years and had to satisfy the inclusion and exclusion criteria. Patients' responses to the screening questionnaires had to have been highly suggestive of SDB for the patients to be recruited into the study.

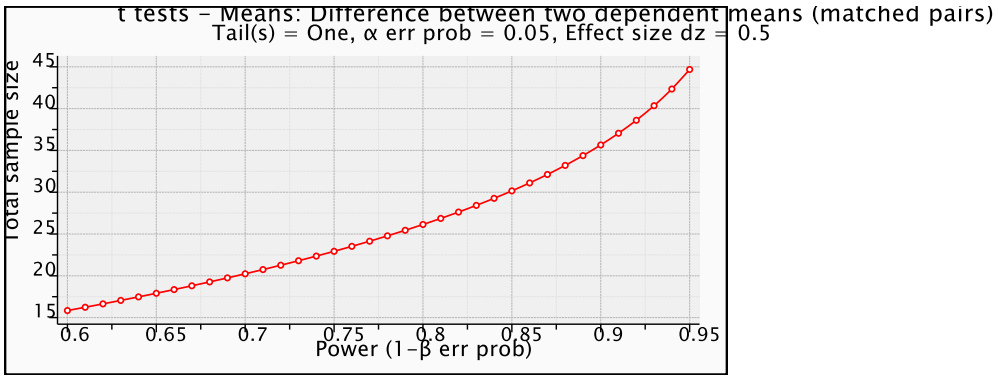
3.3.3 Sample size

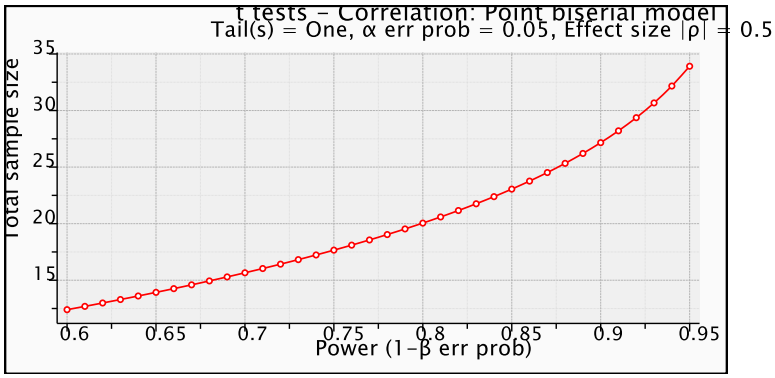
The researcher recruited the first 25 consecutive patients who met the inclusion criteria and agreed to take part in the study by signing informed consent.

The Cochran sample size formula ($Z^2pq/e^2 = N$) was used to estimate the sample size from the sample population where:
Z = area under the normal distribution curve was given as 1.96
P = probability that 10% of the sample population will be selected
Q = 1-p
E = margin of error given as 10%
N = estimated sample size
From the above formula, the estimated sample size was given N = 34.57, and the true sample size was estimated as follows:
$N = N_0/1+ (N_0-1)/N$
Where n = true sample size
No = estimated sample size
N = population size which is given as 80 (This was arrived at, accordibg to IALCH Neurophysiology log books/ database and DUT strategy)
Therefore, n = 24.35

A professional statistician was consulted for all statistical work, including the calculation of the sample size. Cochran's formula was deemed appropriate for sample size calculation in this study (Figure 12).

Figure 12: Calculation of sample size





3.3.4 Inclusion criteria

- Patients between the ages of 18 to 70 years old.
- Patients with symptoms suggestive of sleep-disordered breathing (SDB)

3.3.5 Exclusion criteria

- Patients older than 70 years or younger than 18 years.

- Patients with insomnia, to avoid inaccurate assessment of sleep-disordered breathing.

3.4 PSG procedure

Patients were recruited after being referred for sleep studies by physicians. The study was explained to the potential participant in detail after which informed consent was obtained (Annexures 4, 5, 6, and 7). OPSG was performed the night prior to the ANPSG or two days after the ANPSG. This was done to give time for the zolpidem to wash out and not affect the results of the ANPSG. A pamphlet with instructions was given to the patient on the day of the PSG booking (Annexure 23). It contained all the information about what was required from the patient on the day of the study.

On the day of the procedure, a PSG patient information sheet was filled in. There were four types of sleep questionnaires/scales used together with PSG to categorise sleep architecture and SDB. The architecture of sleep is a certain formulated temporal sequence of sleep stages, that contributes to the quantitative understanding of the night's sleep, table 1 lists the sleep indices measured in order to fairly understand the sleep architecture (Matheson, Singh and Packard 2007). These questionnaires are: Berlin, Epworth (ESS), Functional Outcome (FOSQ), and STOP-Bang. The total ESS score is compared on a scale of 0 to 24, with a score ≥ 16 taken as extreme sleepiness and warranting investigation (Annexure 11). The STOP-Bang total score is between 0 to 8, with a score ≤ 2 regarded as low risk, 3-4 is then considered to be the intermediate risk, and lastly a score equal to and above 5 is considered to be a high risk of developing sleep apnoea (Annexure 12).

Berlin questionnaire measures the risk of having sleep apnoea using 3 categories. Patients are then classified as either high-risk or low-risk based on their responses. A total score of two or more positive categories means a high risk of developing sleep apnoea (Annexure 9). FOSQ consists of five subscales and to obtain the total score, all subscale scores are added to calculate the mean. The mean is then multiplied by 5 regardless of the number of subscales scores used in the computation of the mean. A total score of 5 to 20, indicates bad functional status, with a higher score signifying better functional status. Total questionnaire scores along with PSG results are highly

useful in accurately diagnosing a sleep disorder (Annexure 10). An informed consent form for overnight PSG was requested and read to the patient on the day of the afternoon nap PSG (Annexures 4, 5, 6, and 7). A pill that induces sleep is sometimes used during the afternoon nap for patients that do not fall asleep within 30 minutes after lights have been switched off as part of IALCH standard protocol. For this study, all patients required the pill. Examples include Stilnox/Ziploxone usually administered as 1 pill (10 mg) adult dose or half a pill (5 mg) if you over 65 years of age or the physician suggested so. This is pre-approved by the physician examining and requesting the study. The doctor on call in the medical disciplines would be made aware of the presence of a patient undergoing PSG in the sleep laboratory in case unforeseen problems occurred and the patient required immediate medical attention (refer to Annexures 1 and 2).

Table 1: Indices derived from PSG

Category	**Names of indices**	**Explanations and some normal expectations**
Sleep-related category	Time in bed (TIB) Total bed time (TBT)	Minutes depend on the time spent in bed. Time from lights out to lights on.
	Total recording time (TRT)	Minutes depend on the total time of recording
	Total sleep time (TST)	Minutes of stages 1, 2, 3, and REM
	Sleep efficiency (SE)	> 90% (TST x 100)/ TBT
	Sleep latency	< 20 minutes from lights out until the first epoch of sleep
	REM latency	90-120 minutes
	Wake after sleep onset (WASO)	< 20 minutes
	Sleep period time (SPT)	Encompasses all sleep stages as well as periods of waking after sleep. TST + WASO
Arousals	Total arousal Index (AI)	Sleep disruption. < 10-25 (large variation by age)
	Respiratory arousal index	Total number of these arousals/ TST in hours
	Periodic limb movement (PLM) arousal index	< 15 /hr
	Respiratory effort or event-related arousals (RERA)	Arousals that disrupt sleep, but do not technically meet the definition of apnoeas or hypopnoeas. Flattening of inspiratory portion of nasal pressure (or PAP flow) with increasing respiratory effort

		leading to arousal. No associated desaturation.
Abnormal activity during the study	Periodic limb movement index (PLMI)	< 15
	Parasomnias	Sleepwalking, talking, bruxism, REM sleep behaviour disorders
	ECG signals	Need to be within the range of 60-100 beats per minute and QRS durations must not be outside the range of 0.08 sec to 0.12 sec.
	Apnoea hypopnoea index (AHI)	Number of all apnoeas/TST in minutes x 60
	RERA index	Total of RERA/ TST in hours
	Respiratory disturbance index (RDI)	(RERAS + Hypopnoeas + apnoeas) x 60 / TST (in minutes). RDI is the average number of episodes of apnoeas, hypopnoeas, and respiratory event-related arousal per hour of sleet
	Oxygen saturation indices	Oxygen desaturation index

Source: (Geyer, Talathi and Carney 2009)

During polysomnography (PSG), eighteen PSG channels are used (Annexure 13), including electro-encephalography (Fz-Cz, Cz-Oz, C3-A2, and C4-A1) (Figure 13), electro-oculography (LOC-reference, ROC-A2, and LOC2-A2) (Figure 14), submental and tibialis electromyography (active under the chin and at the belly of the tibial muscle, and reference will be 3 cm lateral to active and 3 cm distal from active, respectively) (Figures 14, 15 and 19, and 16 and 20 respectively). Electro-cardiography is expressed as (RA-LA, RA-LL, and LA-LL). Lead I, II, and Lead III configurations are used for ECG recording at IALCH (Figure 18) (Klabunde 2011). Table 2 provides an explanation of the use of each channel to measure the different parameters. Figure 20 shows an example of patient set-up. Machine settings can be found in Annexure 15. Khawaja *et al.* (2010) used a similar montage to conduct the tests in their study, i.e., for electroencephalography (Fz-Cz, Cz-Oz, C4-M1, C3-M2), and electrooculography (E1-Fpz and E2-Fpz). The process of conducting PSG at IALCH conforms to international standards and guidelines. Annexures 13 and 14 provide further clarity on the montage, channels, and placement, and Annexure 14 lists the calibration procures done at the beginning of the recording.

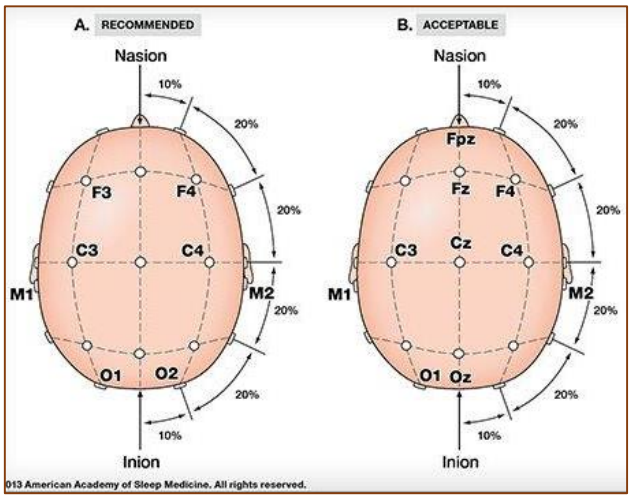


Figure 8: EEG electrode position determined by the International 10-20 system
Source: Berry *et al.* (2012a)

Figure 13 illustrates the electroencephalography (EEG) electrode placement used in polysomnography. (A) is recommended and (B) acceptable. The electrodes are placed according to the international 10-20 System.

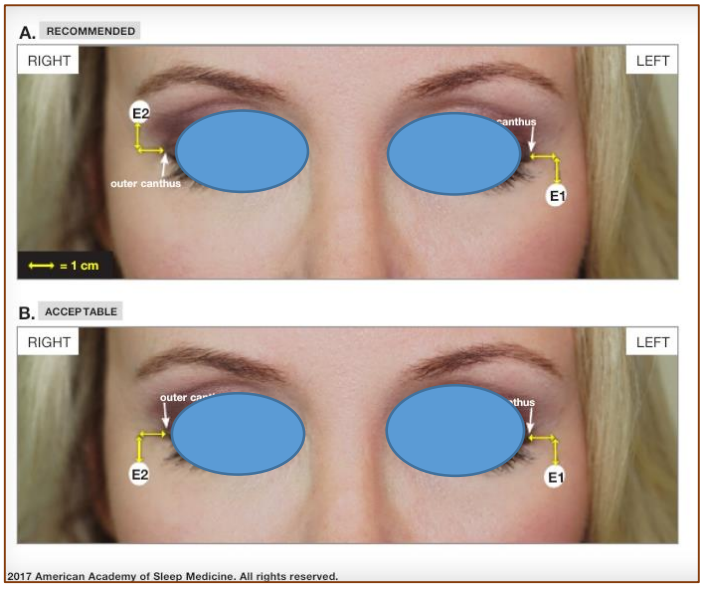


Figure 9: EOG derivation and electrode positions

Figure 14 shows the recommended EOG derivation and electrode positions.

A: The recommended EOG derivations are E1/ LOC – M2/A2 and E2/ ROC – M1/A1. E1 is placed at 1 cm below and 1 cm lateral to the left outer canthus and E2 is placed similarly on the right side, however, above the outer canthus (Berry *et al.* 2017).

B: Acceptable EEG derivations are E1/ LOC – Fpz and E2/ ROC – Fpz. Eye electrodes are placed in a similar fashion as above; the only difference is that both cup electrodes are placed below the outer canthus.

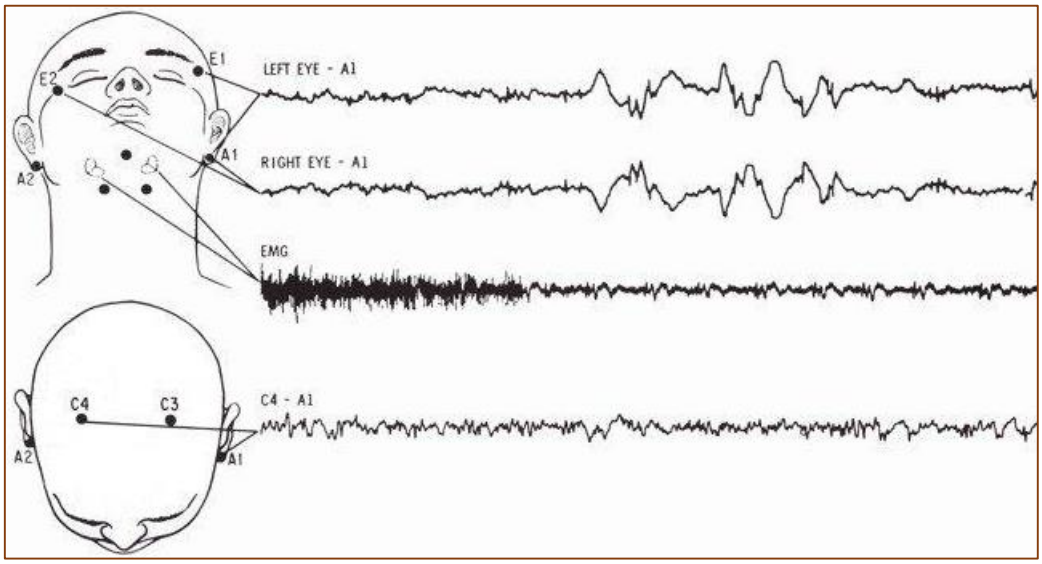


Figure 10: EOG, EMG and EEG derivation and electrode positions
Source Berry *et al.* (2012a).

Figure 15 is an example of the derivations of each trace from different placement regions.

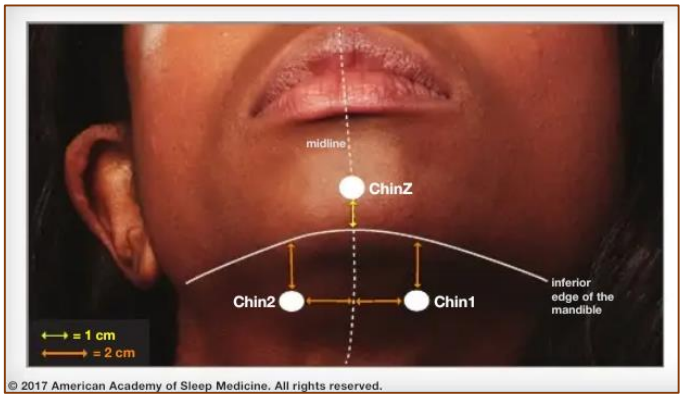


Figure 11: Chin EMG electrode position
Source Berry *et al.* (2012a).

The image in Figure 16 shows the total of three chin placements for a PSG study, the first placement is chinZ which is placed in the middle 1 cm above the lower edge of the mandible. The second placement is labeled as chin2 and is placed 2 cm below the inferior edge of the mandible and 2 cm to the right of the midline. The third is labeled as chin1 and placed 2 cm below the lower edge of the mandible and 2 cm to the left of the midline. ChinZ is the reference to both the electrodes placed below the mandible. Two mandible EMG placements are used for backup purposes in case one electrode happens to malfunction.

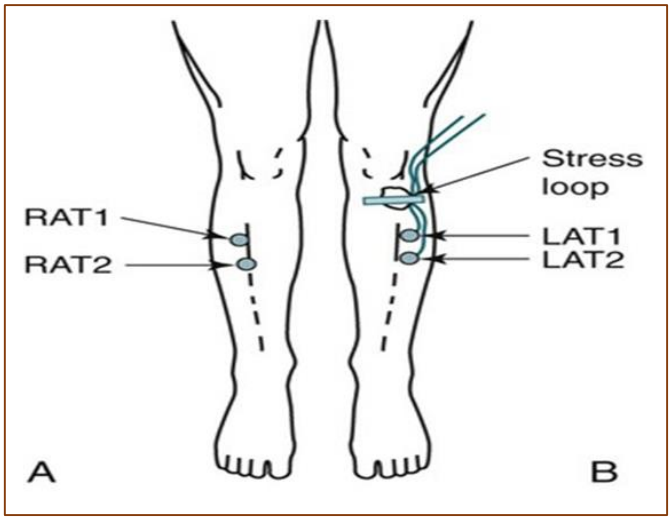


Figure 12: Leg EMG electrode position
RAT: right anterior tibialis muscle, LAT: left anterior tibialis muscle
Source: Neupsy Key. Fastest Neupsy Insight Engine. Monitoring of limb movements and other movements during sleep. Chapter 12. (neupsykey.com).

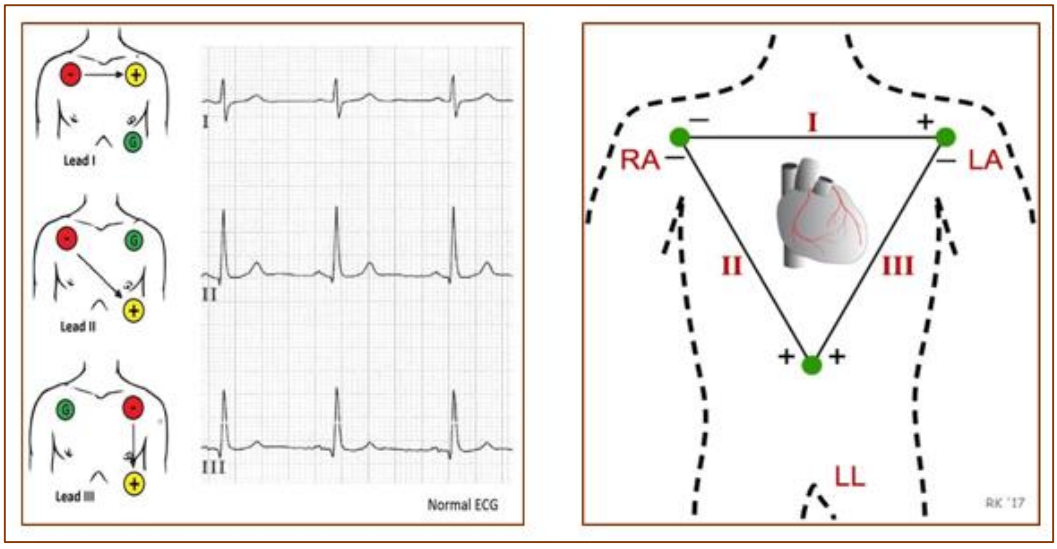


Figure 13: ECG electrode position
Source: Klabunde (2011)

Table 2: Parameters measured in ANPSG and OPSG

Parameters	Description
Electroencephalography (EEG)	Measures brain electrical activity.
Under the chin electromyography (EMG) and snore sensor.	Monitors and measures snoring.
Tibialis EMG (Figure 17)	Measures level of consciousness and restless leg movement.
Electrocardiography (ECG)	Measures heart rate.
Nasal cannula and thermistor	measures the airflow and breathing levels from the nose and mouth.
Inductive plethysmograph	Measure respiratory effort by detecting respiratory movement of the rib cage and abdomen.
Finger pulse oximeter	Measures percutaneous arterial oxygen saturation.
Body position	Entered by the researcher, looking via an audio-visual low-light camera.
Microphone	Measures the sound of snoring.

Level of consciousness and breathing changes were scored according to the American Academy of Sleep Medicine (AASM) version 2.0 in conjunction with the minor changes published on the updated versions for standardisation. Recordings are stored in a computer database using Nicolet software. Figure 19 summarises the steps taken when conducting a PSG study at IALCH:

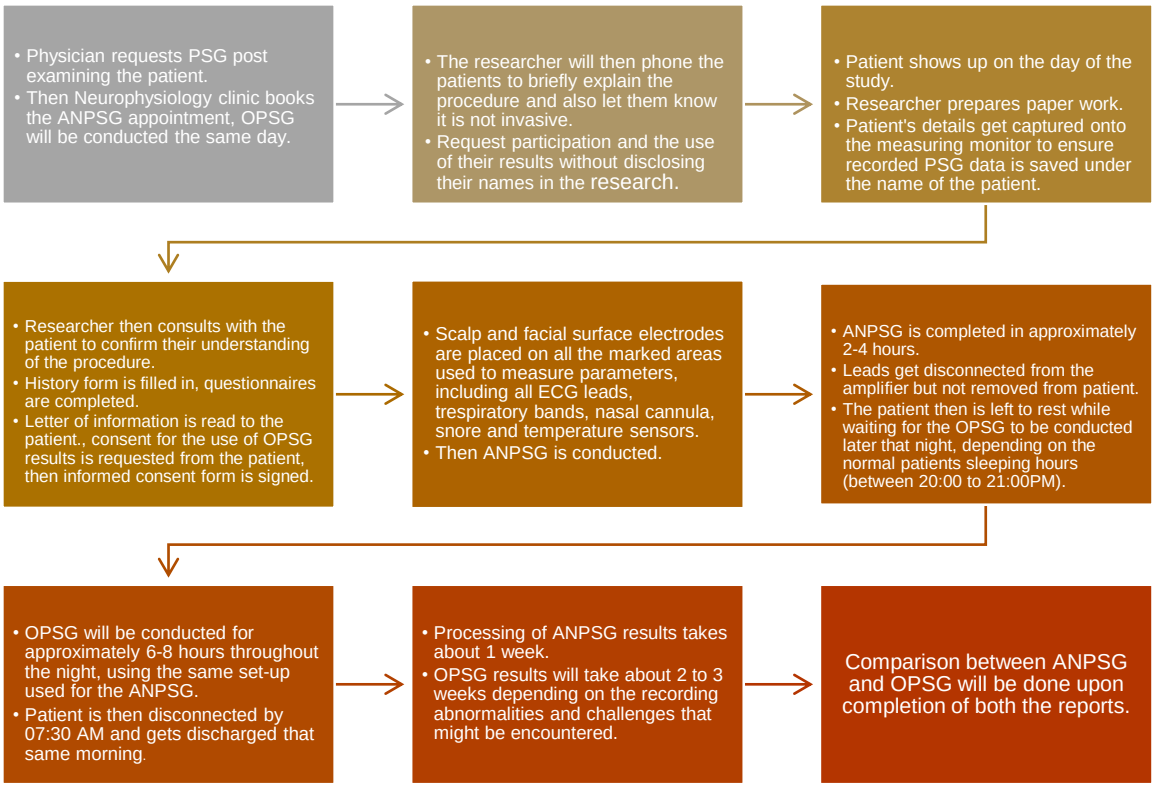


Figure 14: Procedure flow diagram

Figure 20: Head and face PSG set-up at IALCH

Figure 20 shows the head and face electrode placement.

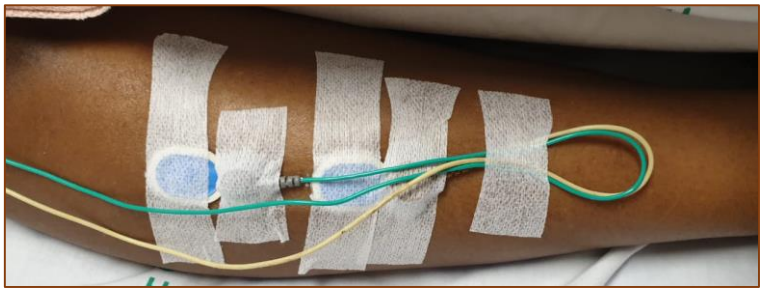


Figure 15: Leg PSG set-up at IALCH

Figure 21 shows the EMG placement with safety loops.

Figure 16: Respiratory effort belt PSG set-up at IALCH

Figure 22 shows the two respiratory effort belts (REB) – one for the thorax and the other for the abdominal region. These are placed when the patient is standing. There is a pulse oximeter on the thumb. Then the patient lies down. Respiratory inductive plethysmography (RIP) is the technology used in these REBs. Using two belts is crucial, among other reasons, to monitor the breathing pattern of a patient, as breathing occurs through two different mechanisms, namely, by the expansion of the chest wall and then by breathing into the abdomen. It makes it easier to precisely distinguish between central and obstructive sleep apnoea.

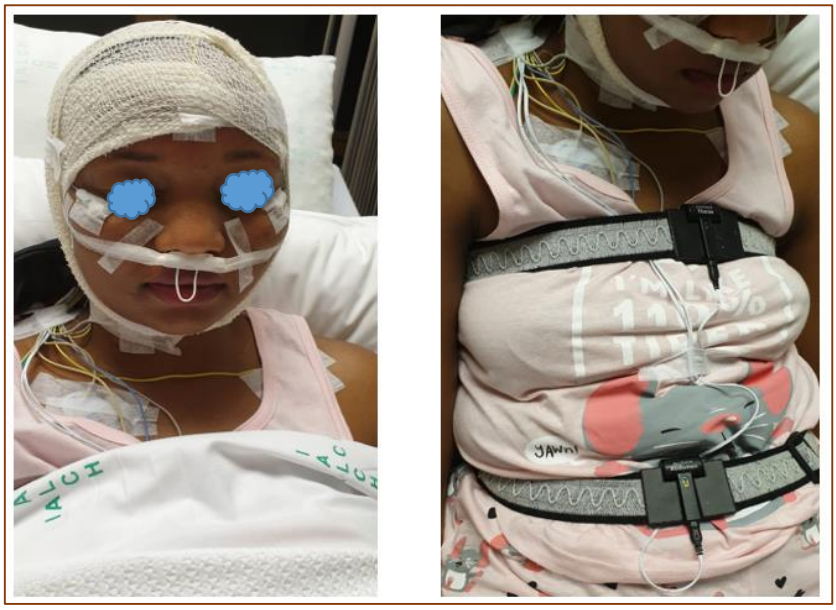


Figure 17: Head cover and safety loops PSG patient set-up at IALCH

Figure 23 shows the PSG electrodes and leads around the face secured with a bandage, and the sinusoidal shape of the respiratory inductance plethysmography respiratory effort belt. Note the safety loops between the belts to avoid the wires pulling out from the plug point during sleep.

Figure 18: PSG head-box and safety positioning set-up at IALCH

Figure 24 shows how the head box is placed; it is hooked on the side of the bed before putting the bed flat. and the leads are tightly secured for the patient's comfort and to protect the leads from breaking.

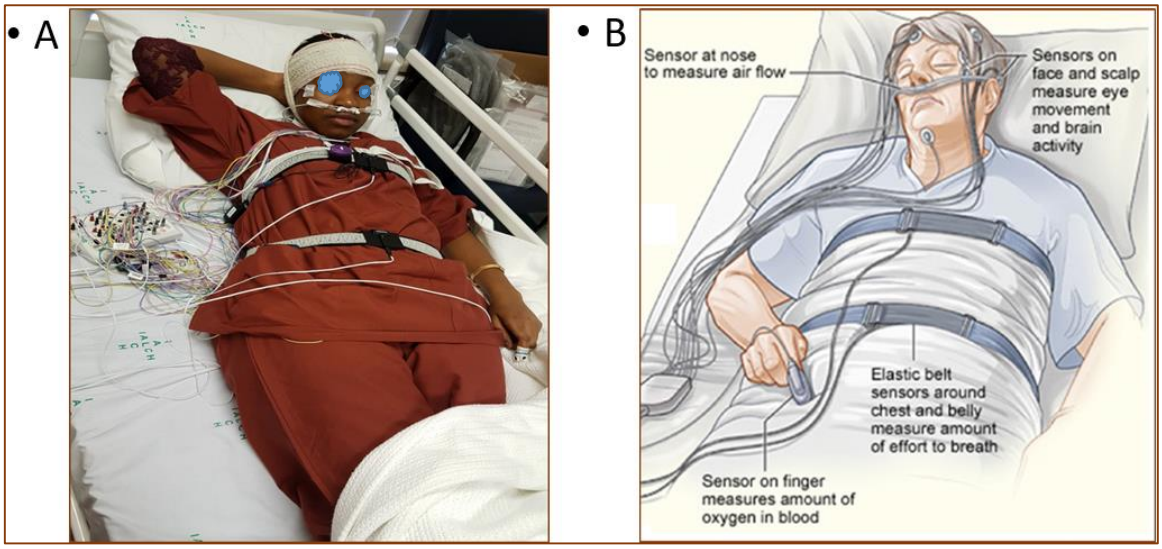


Figure 19: Full body PSG patient set-up at IALCH

Figure 25 shows what a fully placed PSG looks like. Image B on the right was adapted from: Fonpicbet.pw/eeg-in-sleep-research.html

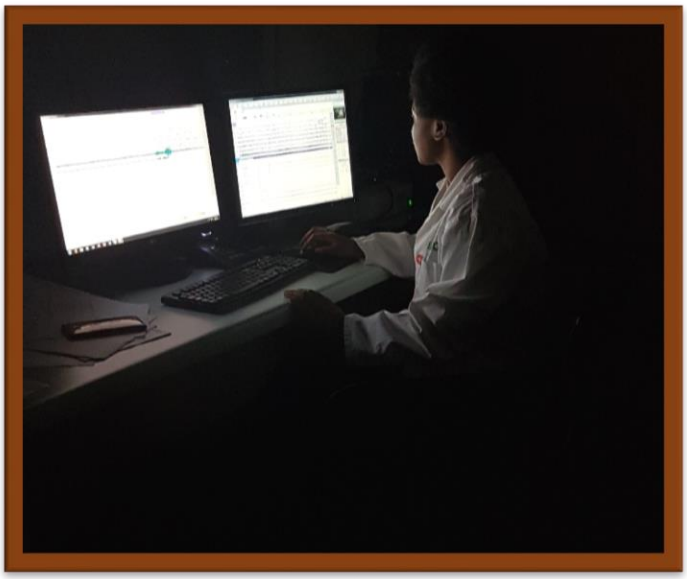


Figure 20: PSG scoring room at IALCH

Figure 26 shows a neurophysiologist sitting in the next room and scoring the sleep stages and apnoeas or RERAS while monitoring the patient and the quality of the recording.

3.5 Sleep study

3.5.1 The basic pattern of sleep

A healthy and normal adult usually falls asleep within a range of 10 minutes at night and goes through all sequences of sleep stages, namely three NREM stages and one REM stage. Izac (2006) states that NREM alternates with REM in recurring turns of 3-5 cycles at intervals of about 85 to 100 minutes. Kovacevic-Ristanovic and Kuzniar (2014), statement with the recurring cycle between these stages of sleep was rather slightly earlier with 90-minute intervals in adults and was noted to be in agreement with the findings of Matheson, Singh and Packard (2007), who further elaborated that NREM and REM sleep does take place recurring cycles of approximately 70-90 minutes for 4 to 6 cycles (Matheson, Singh and Packard 2007; Kovacevic-Ristanovic and Kuzniar 2014). The first REM sleep is of a short duration of approximately 10 minutes, which gradually advances in duration later in the night, hence OPSG is taken as the gold standard. In OPSG, the patient is given enough time to go through all of the stages of sleep during PSG recording, as some disorders progress or take place only during certain sleep stages (Matheson, Singh and Packard 2007).

3.5.2 The scoring summary of sleep stages in polysomnography

The terminology used for scoring adult sleep stages is structured as stage W for wakefulness, stage N1 for NREM 1, stage N2 for NREM 3, and stage R for REM. Stage N3 represents slow-wave sleep (Patel and Araujo 2018). Epochs are scored using set parameters such as a 30-second epoch for scoring sleep stages, commencing at the start of light off. Each epoch is assigned a state of patients' consciousness, depending on the stage it shows. In the case of two or more sleep stages occurring in one epoch, the longest stage of sleep on that epoch takes precedence and gets assigned (Patel and Araujo 2018).

3.5.3 The scoring summary of respiratory disturbance in polysomnography

Measuring respiratory disturbance during PSG recording involves nasal pressure, thermal sensors, pulse oximetry, and RIP. These need to be connected and properly calibrated before beginning the study. Thermal sensors include thermistors, thermocouples, and polyvinylidene fluoride (PVDF) (Berry *et al.* 2012a).

An apnoeic respiratory event is scored when there is a ≥ 10 seconds drop of ≥ 90% in comparison with the pre-event baseline using an oro-nasal thermal sensor. Desaturation may be present, but it is not a requirement (Berry *et al.* 2012a).

In the case of OSA, there also needs to be increased inspiratory effort throughout the entire event. In the case of CSA, there should be absent inspiratory effort throughout the entire episode. When scoring mixed apnoeas, the initial portion of the event should be composed of CSA features, followed by OSA characteristics in the second portion of the episode (Berry *et al.* 2012a). Respiratory disturbance index (RDI) is the average number of episodes of apnoeas, hypopnoeas, and respiratory event-related arousals per hour of sleep and is calculated as (RERAS + Hypopnoeas + apnoeas) x 60 / TST (in minutes).

3.5.3.1 Scoring of hypopnoeas

There are three criteria to be met to score an event as hypopnoea: firstly, the peak-to-peak drop by ≥ 30% from baseline using nasal pressure. Secondly, the drop need to last ≥ 10 seconds. Thirdly, there should be ≥3% oxygen desaturation (Berry *et al.* 2012a). Hypopnoeas can be scored as obstructive hypopnoeas, central hypopnoeas, or just hypopnoeas, this is completely optional. When electing to score obstruction hypopnoeas, snoring during the event has to be present; or an increased inspiratory flattening of the nasal pressure. The central hypopnoea is scored in the absence of all the obstruction hypopnoea criteria (Berry *et al.* 2012a).

3.5.3.2 Scoring respiratory effort-related arousals (RERAs)

RERA is scored when the breathing disturbance does not meet the criteria for either apnoeas or hypopnoeas. The increasing respiratory effort or flattening of the

inspiratory portion of the nasal pressure ≥ 10 in duration must lead to an arousal from sleep (Berry *et al.* 2012a).

3.5.3.3 Scoring Cheyne-Stokes breathing

During the scoring of Cheyne-Stokes breathing, there have to be episodes of ≥ 3 consecutive central apnoeas or hypopnoeas separated by a crescendo and decrescendo change in breathing amplitude with a cycle length of ≥ 40 seconds; or the presence of ≥ 5 central apnoeas or hypopnoeas per hour of sleep associated with the crescendo/decrescendo breathing pattern recorded over ≥ 2 hours of monitoring. Crescendo-decrescendo is the progressive increase of breath intensity, followed by a progressive decrease in intensity (Berry *et al.* 2012a).

3.5.3.4 Breathing related arousals

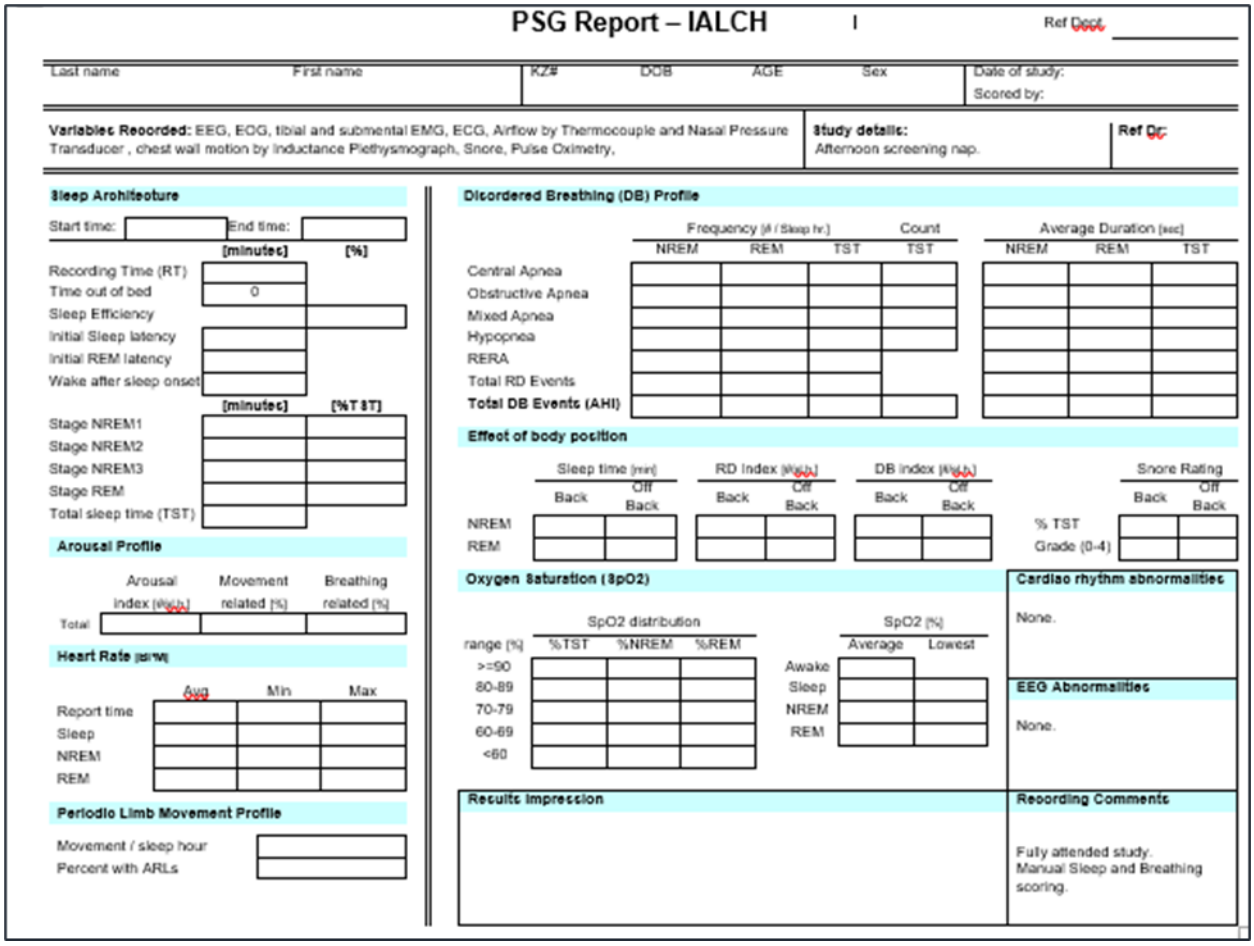
These arousals are the arousals that occur due to breathing disturbance, namely it may be during apnoeas, hypopnoeas, RERAs. Then, the percentage is calculated using the total of arousals, which include the spontaneous arousal. These arousals assist when writing a PSG report to support the severity of the apnoeas found.

3.5.3.5 Spontaneous related arousals

These arousals are not related to breathing disturbance, limb movements, snoring, and so on. Spontaneous arousals are all the other arousals that are not provoked by sleep related disorder. According to IALCH methods, the percentage is calculated using the total of arousals, which also include the breathing related arousals. These are included to also prove further that the patient sleep is affected mostly because of breathing related arousals, in most cases, spontaneous arousals have an extremely low percentage compared to that of breathing related arousals (Parrino, Grassi and Milioli 2014; Qian *et al.* 2021).

3.6 Data Collection

The data collected from the recruited patients included two types of PSG recordings and the four questionnaires previously listed – Berlin and STOP-Bang questionnaires to screen for the risk of developing sleep apnoea, functional outcome to rate functional status, and lastly Epworth sleep scale to rate the level of hypersomnia. The IALCH general patient information sheet included almost all useful patients' history, like medication, previous medical history, indication for the test, age, weight, neck circumference, BMI, and more (Annexure 8). PSG results were compiled by the researcher after scoring or analysing PSG recordings manually (Figure 27). A Table with the list of patients and the findings was created for ease of recognition of the data needed. However, all data collected were de-identified, and all were allocated a specific code for identification.


PSG Report – IALCH

Ref Doct.

Last name | First name | KZ# | DOB | AGE | Sex | Date of study: | Scored by:

Variables Recorded: EEG, EOG, tibial and submental EMG, ECG, Airflow by Thermocouple and Nasal Pressure Transducer , chest wall motion by Inductance Plethysmograph, Snore, Pulse Oximetry,

Study details: Afternoon screening nap.

Ref Dr:

Sleep Architecture

Start time: | End time: | [minutes] | [%]

Recording Time (RT)
Time out of bed | 0
Sleep Efficiency
Initial Sleep latency
Initial REM latency
Wake after sleep onset

[minutes] | [%TST]

Stage NREM1
Stage NREM2
Stage NREM3
Stage REM
Total sleep time (TST)

Arousal Profile

	Arousal index (#/hr)	Movement related (%)	Breathing related (%)
Total			

Heart Rate (bpm)

	Avg	Min	Max
Report time			
Sleep			
NREM			
REM			

Periodic Limb Movement Profile

Movement / sleep hour
Percent with ARLs

Disordered Breathing (DB) Profile

	Frequency (# / Sleep hr.) NREM	Frequency REM	Frequency TST	Count TST	Average Duration (sec) NREM	Average Duration REM	Average Duration TST
Central Apnea							
Obstructive Apnea							
Mixed Apnea							
Hypopnea							
RERA							
Total RD Events							
Total DB Events (AHI)							

Effect of body position

	Sleep time (min) Back	Sleep time Off Back	RD Index (#/hr) Back	RD Index Off Back	DB Index (#/hr) Back	DB Index Off Back	Snore Rating Back	Snore Rating Off Back
NREM							% TST	
REM							Grade (0-4)	

Oxygen Saturation (SpO2)

range (%)	SpO2 distribution %TST	%NREM	%REM
>=90			
80-89			
70-79			
60-69			
<60			

	SpO2 (%) Average	Lowest
Awake		
Sleep		
NREM		
REM		

Cardiac rhythm abnormalities

None.

EEG Abnormalities

None.

Results Impression

Recording Comments

Fully attended study.
Manual Sleep and Breathing scoring.

Figure 21: Data form to be filled upon completion of PSG recording

3.7 Reliability and validity of data collected

Filled in questionnaires and patient information sheets were scanned and kept on a laptop with a password only known by the researcher and a backup disk including all the PSG studies done for recruited patients. The researcher reviewed all the PSG studies done for the recruited patients. The participants underwent both afternoon nap PSG and overnight PSG to allow consistency and easy comparison.

Non-parametric tests using Kolmogorov-Smirnov helped to determine the normal distribution of the variables (Kinnear and Gray 2006). Regarding the inferential statistical analysis, the independent t-test was used to compare the mean difference between the AHI of overnight PSG and afternoon nap PSG. Barnes and Lewin (2011) stated that the independent t-test is the most suitable parametric test to pinpoint the mean differences, and for testing any significant differences between two variables. In addition, the Pearson correlation coefficient was used to determine the relationship between the demographic factors (age, race, neck and waist circumference, and weight), and the overnight versus afternoon nap PSG.

The thorough use of all the scoring rules in this research was another way of true validation in making sure none of the events were left unscored. It elevated the number of events, as none were missed or left out due to doubts about which rule they fall into.

3.8 Data analysis

All analysis was done using SPSS® (Version 28, 29). Sleep stages data were analysed, including NREM stages (1-3) and REM stages. PSG data were scored according to the AASM standard criteria (Berry *et al.* 2012a). Hypopnoeas were identified as greater than equal to thirty percent for at least ten seconds followed by three to four percent of desaturation or arousal. Cessation of airflow with respiratory efforts lasting at least ten seconds was identified as obstructive sleep apnoea, and the one without respiratory effort was classified as central sleep apnoea. Apnoea-hypopnoea index (AHI) was calculated and defined as the total number of hypopnoea, and apnoeas per hour of sleep. The severity of apnoeas was categorised according to AASM guidelines, as a

number of apnoeas per hour of sleep: normal if < 5, mild if 5 - 15, moderate if 15 - 30, and severe if > 30 (Epstein *et al.* 2009).

Quantitative data from participants were analysed in terms of comparing questionnaires, gender, age, race, neck circumference, waist circumference, and weight. Afternoon nap PSG and overnight PSG data were compared using SPSS® (Version 28), where both descriptive and inferential statistics were used to analyse the data. Univariate and bivariate analysis were most appropriate for descriptive statistics (Salkind and Frey 2019). Bar graphs and tables were used to present the data. Descriptive analysis was used in tables that presented mean and standard deviation, including median, and interquartile range for variables such as BMI, nech circumference, weight, and height. Pearson correlation analysis was used to correlate data between two grouped numbers using variables selected. Paired sample test was used to compare the afternoon and overnight PSG using the variables selected. To Determination of association between the parameters was done using Chi- square test for variables like demographic characteristic, and gender. The association between participants' selected variables and the PSG level of severity was calculated using ANOVA. Multiple regression was used to determine the predictors for higher or lower RDI or AHI in ANPSG and OPSG.

3.9 Ethical considerations

There was no harm to the patients. The procedure was non-invasive. A study information pamphlet was given to the patient on the day of the study and the informed consent was signed. A letter to request permission from the KZN Department of Health, and IALCH management was submitted to the relevant committees for approval to conduct the proposed study and this was granted. Ethical guidelines outlined in the Durban University of Technology, Faculty of Health Sciences policy documents were adhered to throughout this study. Ethical clearance was obtained with institutional research ethics committee (IREC) number 143/20 (Annexure 21) All data were de-identified, and all records got allocated a specific code for identification to ensure the confidentiality of the participants' results on the study

CHAPTER 4: RESEARCH FINDINGS AND ANALYSIS

This chapter presents the results obtained from analysis of the correlation of apnoea-hypopnoea index in afternoon polysomnography and overnight polysomnography at a health establishment located in KwaZulu-Natal. Clinical data were collected from 25 patients visiting the health establishment. The data collected from the responses were analysed with SPSS (version 28®). Test of Normality was performed for the data, which showed normal distribution (Annexure 27). Parametric statistical tests were therefore performed for all analysis.

4.1 Socio-demographic profile of the participants

4.1.1 Gender

Figure 28 and Table 4 reflect the gender of the participants. The data shows that most of the participants were females (56%) while males accounted for 44%.

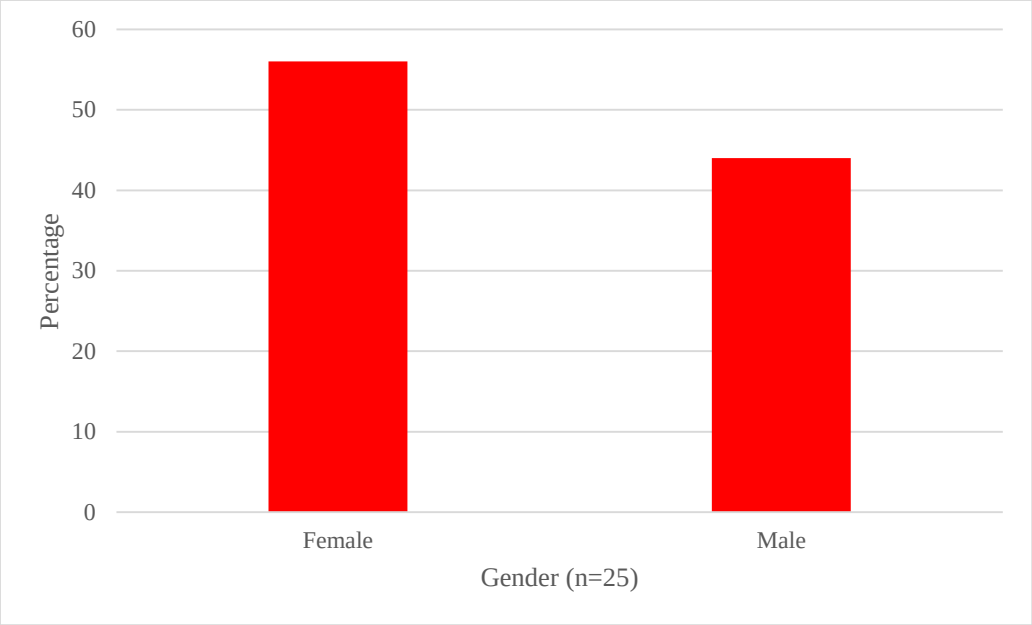


Figure 228: Gender

Table 3: Demographic data of the population

Variable	Categories	N (%), Statistics
Gender	*Female*	14 (56)
	Male	11 (44)
Age distribution	40 and below	5 (20)
	41-50	5 (20)
	51 and above	15 (60)
Age		
Mean and standard deviation		51.04 ± 10.65
Median		54
Interquartile range		16

4.1.2 Age distribution

Figure 29 and Table 3 show the distribution of the participants according to age. Most of the participants were 51 years of age and above which constituted 60% while those within 41-50 years and below 40 were 20% each. The mean age of the participants was 51.04 ± 10.65 years.

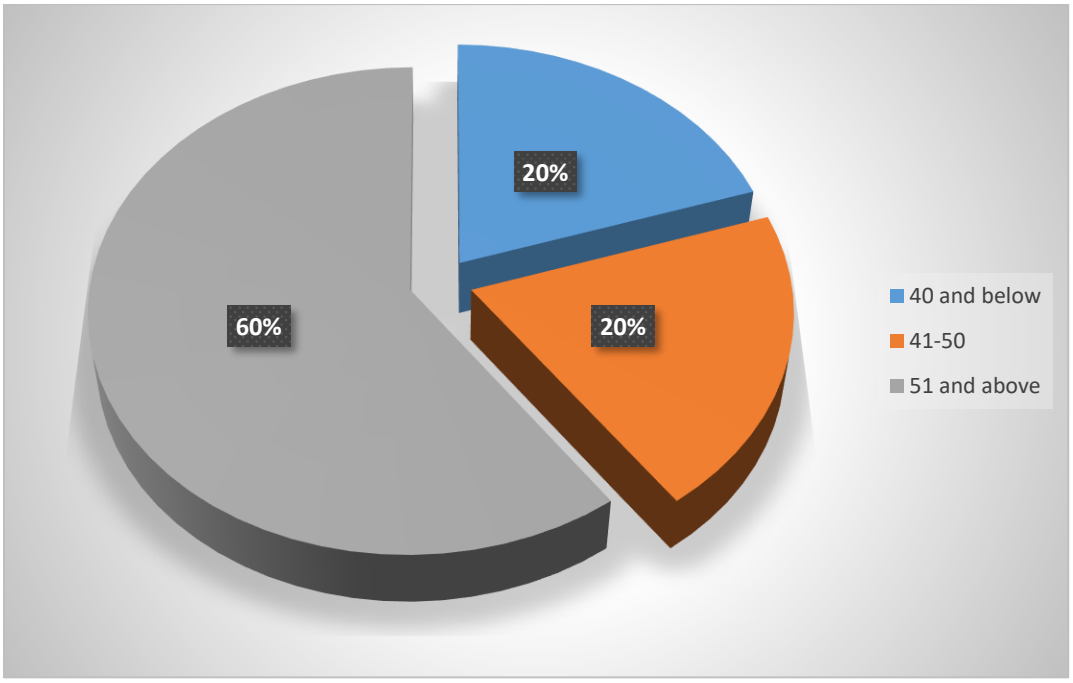


Figure 23: Age group distribution

4.1.3 Body mass index, neck circumference, weight, and height

Table 4 shows the physical profile of the participants according to their BMI, neck circumference, height, and weight. On average, the data shows that the participants had a mean BMI of 37.6 ± 10.9 (kg/ m^2), a neck circumference of 41.4 ± 4.6 cm, a weight of 103.1 ± 28.6 kg, and a height of 1.66 ± 0.1m, with median and IQR as expressed on the table.

Table 4: Physical profiles

Variable	Mean and standard deviation	Median	Interquartile range (IQR)
BMI (kg/m^2)	37.6 ± 10.9	38	16.88
Neck circumference (cm)	41.4 ± 4.6	42	6.50
Weight (kg)	103.1 ± 28.6	105.25	40.57
Height (m)	1.66 ± 0.1	1.64	0.15

4.2 Correlation of RDI, AHI, REM, and NREM between afternoon and overnight polysomnography

This section details the correlation between afternoon and overnight polysomnography measured for variables such as respiratory disturbance index (RDI), apnoea-hypopnea index (AHI), non-rapid eye movement (NREM), rapid eye movement (REM), total sleep time (TST), sleep efficiency (SE), sleep-related arousal, initial REM latency, and spontaneous related arousal.

4.2.1 Respiratory disturbance index (RDI)

The data in Table 5 show that the RDI measured for ANPSG correlates positively with the RDI measured for OPSG, and the association was significant ($r = 0.885$, $P < 0.001$). This explains that as the RDI for afternoon nap polysomnography (ANPSG) increased, the RDI for overnight polysomnography (OPSG) also increased, and *vice versa*.

Table 5: Correlation between RDI afternoon and RDI overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
ANPSG(RDI) - OPSG(RDI)	.885	<.001	.744	.946
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 30 further illustrates the association between RDI ANPSG and RDI OPSG. The correlation coefficient reveals a strong linear association between ANPSG and OPSG.

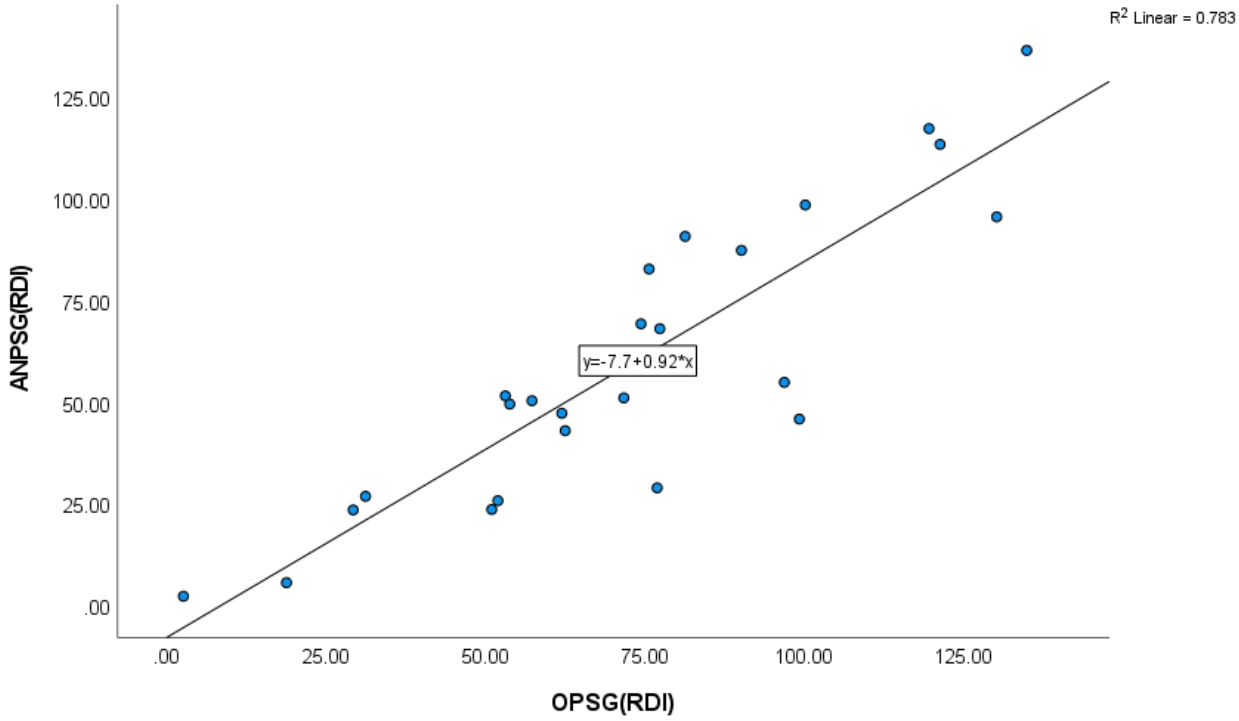


Figure 30: Association between RDI ANPSG and RDI OPSG

4.2.2 Apnoea hypopnea index (AHI)

Results on the correlation coefficient in Table 6 revealed a strong, positive linear association between ANPSG and OPSG ($r = 0.877$, $P < 0.001$). This means that as the AHI for ANPSG increased the AHI for OPSG also increased, and *vice versa*.

Table 6: Correlation between AHI afternoon and AHI overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
ANPSG(AHI) - OPSG(AHI)	.877	<.001	.728	.943
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 31 further illustrates the association between AHI ANPSG and AHI OPSG. The correlation coefficient reveals a strong linear association between ANPSG and OPSG.

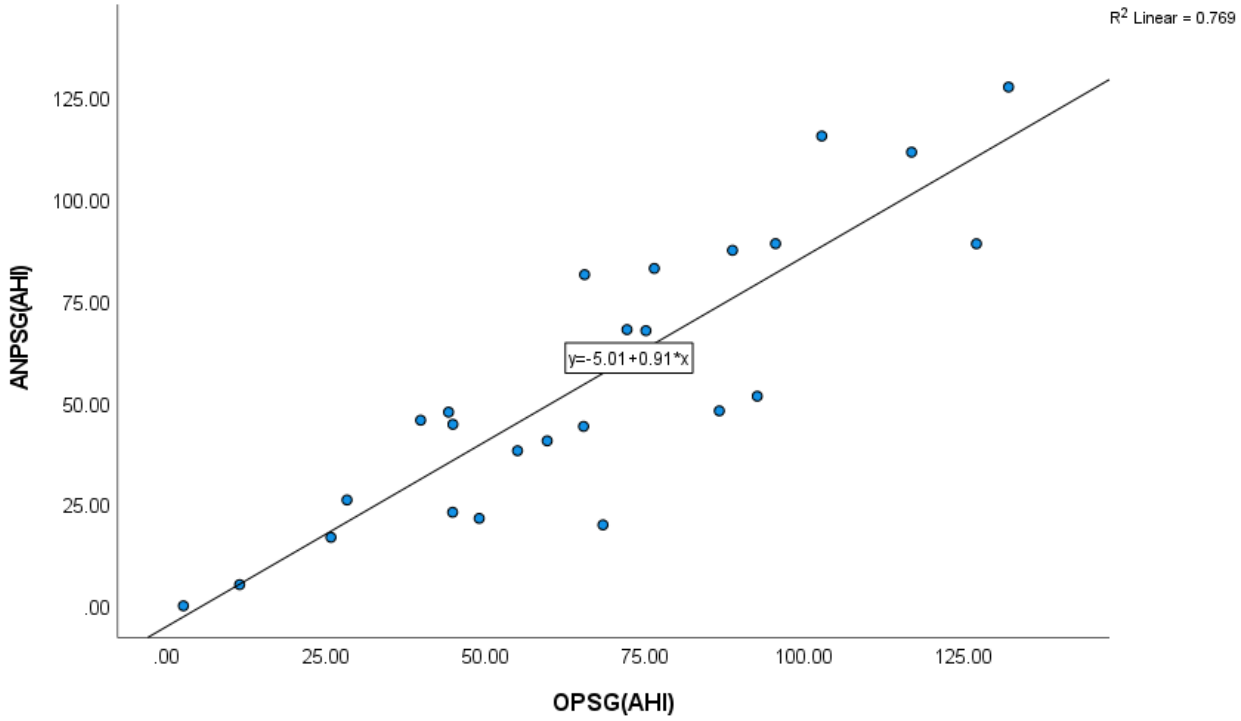


Figure 241: Association between AHI ANPSG and AHI OPSG

4.2.3 Non-rapid eye movement – NREM (RDI)

The data in Table 7 shows a significant connection between ANPSG and OPSG (r = 0.891, P < 0.001). This meant that as the NREM (RDI) for ANPSG increased, the NREM (RDI) for OPSG also increased, and *vice versa*.

Table 7: Correlation between NREM (RDI) afternoon and NREM (RDI) overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
NREMANPSG(RDI) - NREMOPSG(RDI)	.874	<.001	.723	.941
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 32 further illustrates the association between NREM (RDI) ANPSG and NREM (RDI) OPSG. The correlation coefficient reveals a strong linear association between ANPSG and OPSG.

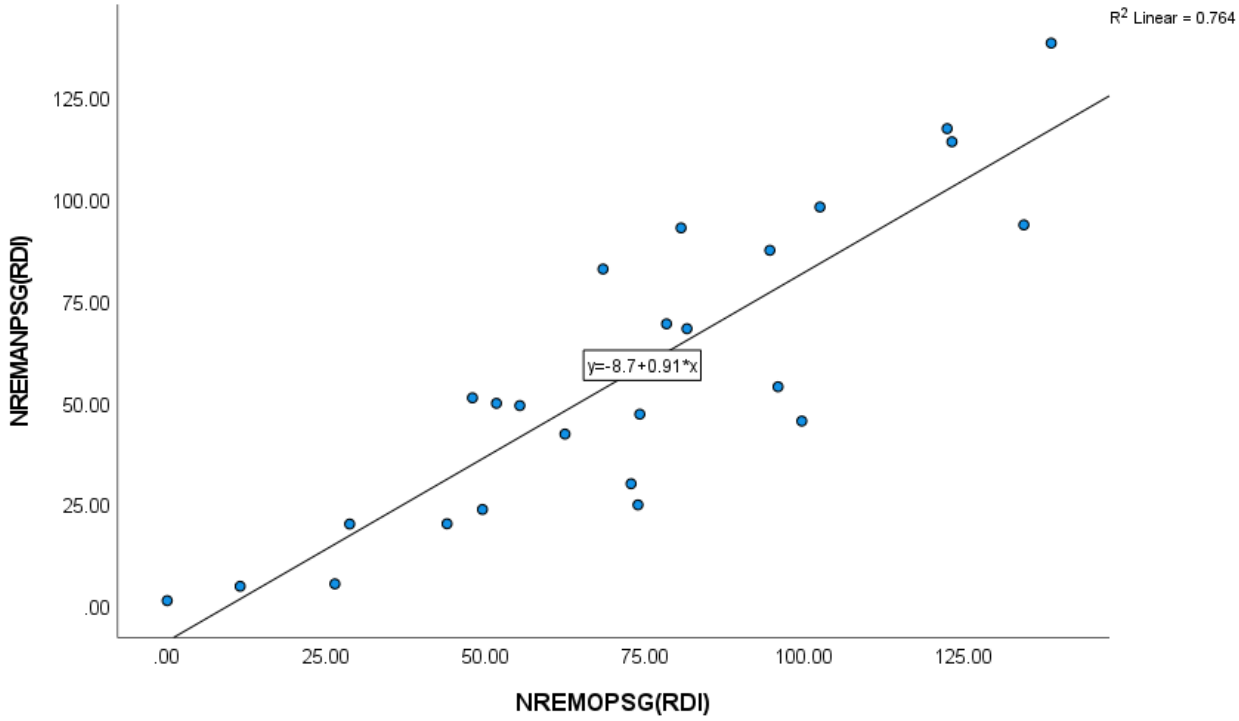


Figure 252: Association between NREM (RDI) ANPSG and NREM (RDI) OPSG

4.2.4 Non-rapid eye movement (NREM) apnoea hypopnea index (AHI)

The data in Table 8 show that there was a significant interconnection between ANPSG and OPSG (r = 0.937 P < 0.001). This means that as the NREM (AHI) for ANPSG increased, the NREM (AHI) for OPSG also increased, and *vice versa*.

Table 8: Correlation between NREM (AHI) afternoon and NREM (AHI) overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
NREMANPSG(AHI) - NREMOPSG(AHI)	.865	<.001	.704	.937
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 33 further illustrates the association between NREM (AHI) ANPSG and NREM (AHI) OPSG. The correlation coefficient reveals a strong linear association between ANPSG and OPSG.

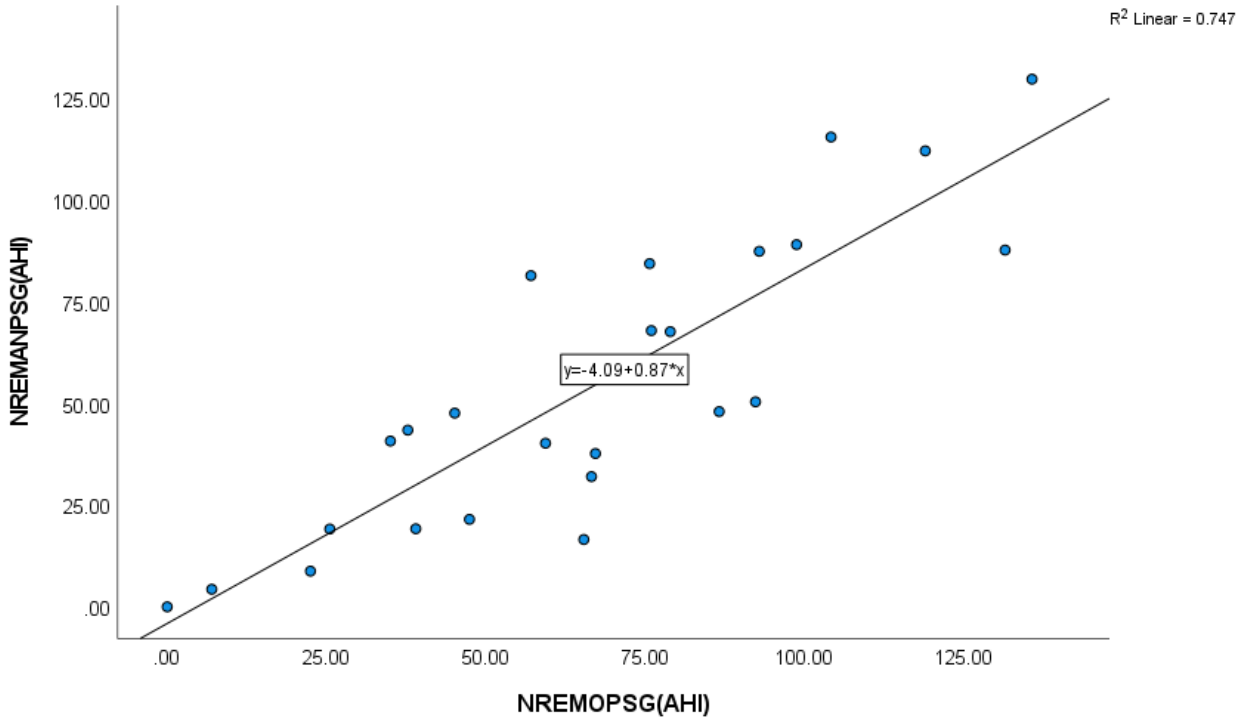


Figure 263: Association between NREM (AHI) ANPSG and NREM (AHI) OPSG

4.2.5 Rapid eye movement – REM (RDI)

The data in Table 9 show that there was a non-significant association between REM (RDI) measured for ANPSG and OPSG (P > 0.05).

Table 9: Correlation between REM (RDI) afternoon and REM (RDI) overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
REM ANPSG(RDI) - REM OPSG(RDI)	.171	.414	-.244	.528
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 34 further illustrates the association between REM measured for ANPSG and REM measured for OPSG. The data show no linear association between ANPSG and OPSG. More so, all values were outside the regression line, which suggests a poor association.

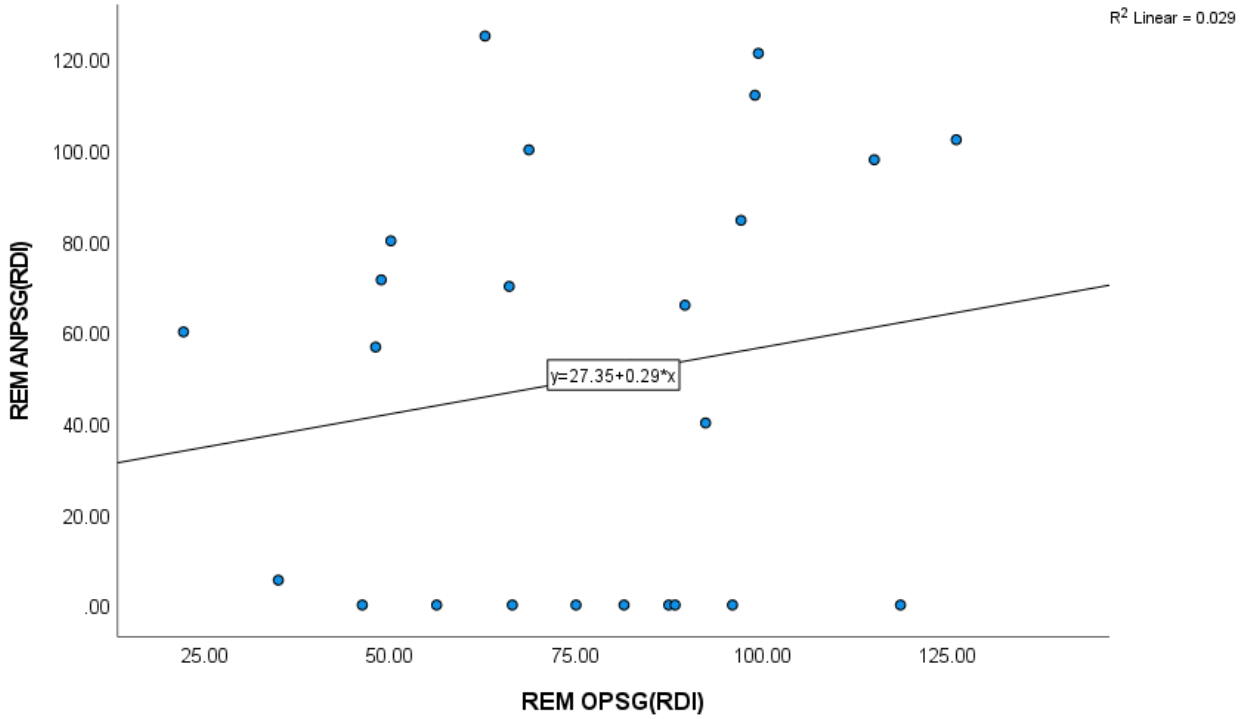


Figure 274: Association between REM(RDI) measured for ANPSG and REM(RDI) measured for OPSG

4.2.6 Rapid eye movement – REM (AHI)

The data in Table 10 show that there was no association between REM (AHI) measured for ANPSG and OPSG (P > 0.05).

Table 10: Correlation between REM (AHI) afternoon and REM (AHI) overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
REM ANPSG(AHI) - REM OPSG(AHI)	.140	.505	-.273	.505
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 35 further illustrates the association between REM (AHI) measured for ANPSG and REM (AHI) measured for OPSG. The data show no linear association between ANPSG and OPSG. More so, all values were outside the regression line, which suggests a poor association.

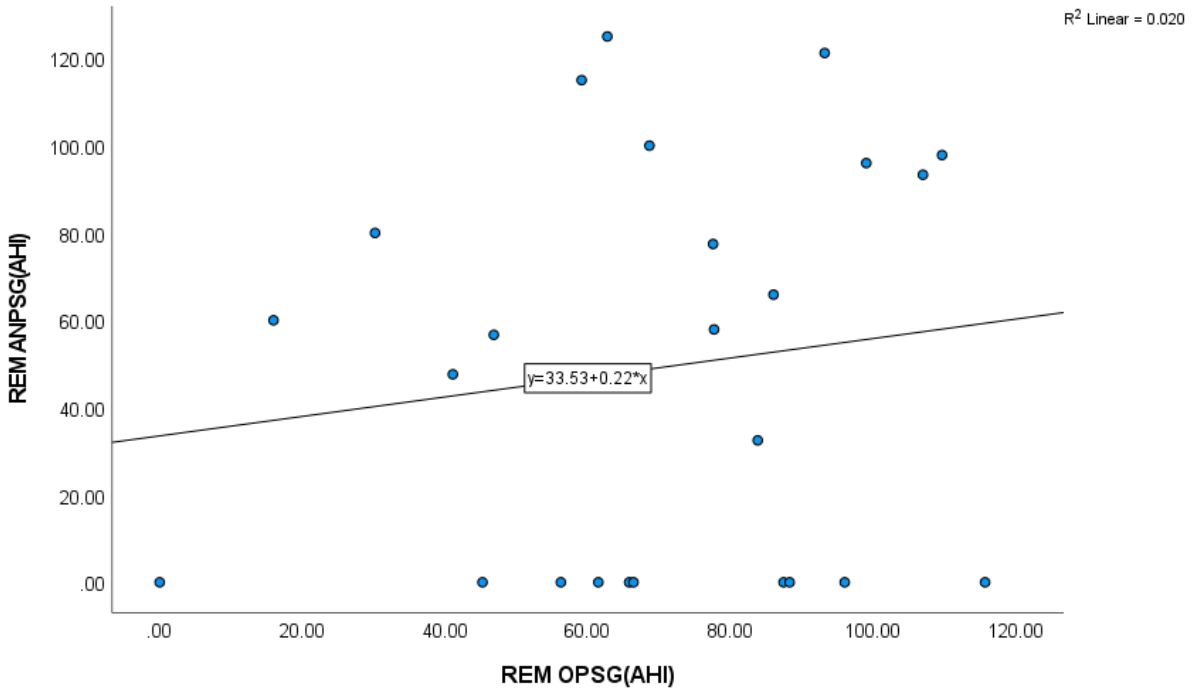


Figure 35: Association between REM (AHI) measured for ANPSG and REM (AHI) measured for OPSG

4.2.7 Total sleep time- TST

The data in Table 11 show that there was no association between TST measured for ANPSG and OPSG (P > 0.05).

Table 11: Correlation between total sleep time afternoon and overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
Total Sleep Time(ANPSG) – Total Sleep Time(OPSG)	.208	.317	-.208	.555
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 36 further illustrates the association between total sleep times measured for ANPSG with those measured for OPSG. The data show no linear association between ANPSG and OPSG. More so, all values were outside the regression line, which suggests a poor association.

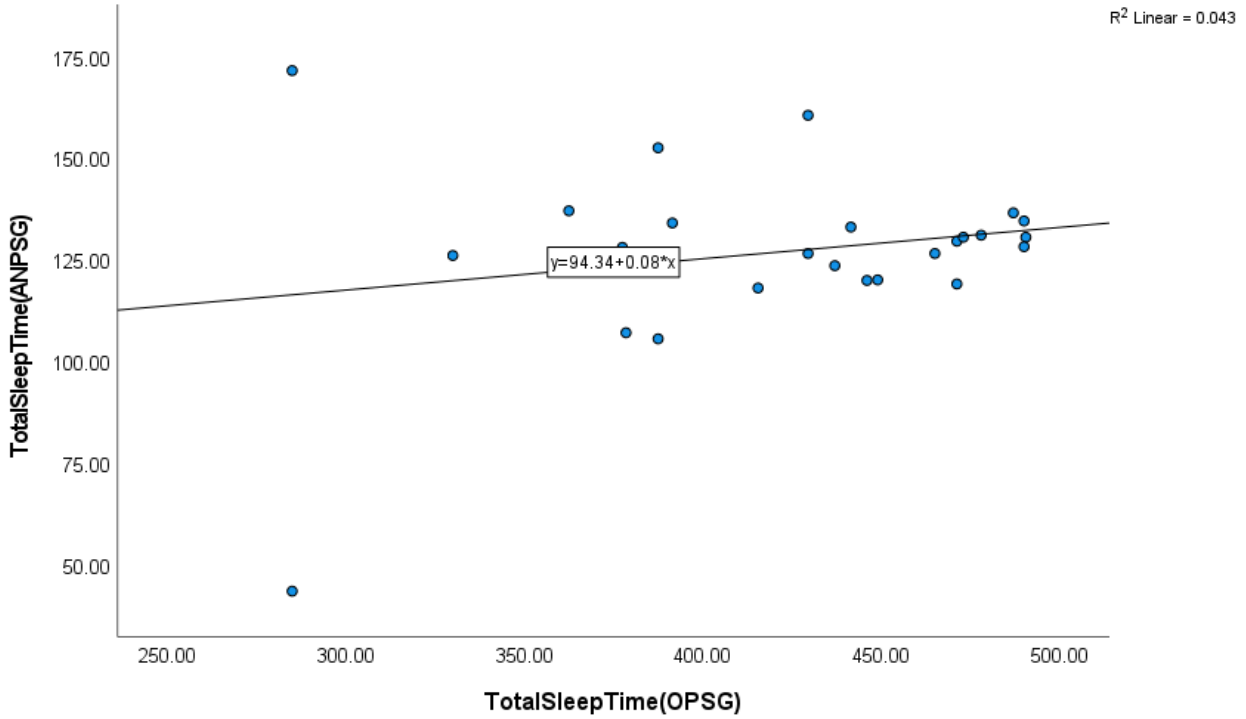


Figure 286: Association between total sleep times measured for ANPSG with those measured for OPSG

4.2.8 Sleep efficiency – SE

The data in Table 12 show that there was a significant correlation between ANPSG and OPSG (r = 0.687, P < 0.001)). This meant that as the sleep efficiency for ANPSG increased, the sleep efficiency for OPSG also increased, and *vice versa*.

Table 12: Correlation between sleep efficiency for the afternoon and overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
Sleep Efficiency(ANPSG) – Sleep Efficiency(OPSG)	.687	<.001	.389	.847
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 37 further illustrates the association between sleep efficiency measured for ANPSG and OPSG. The regression coefficient reveals a strong linear association between ANPSG and OPSG.

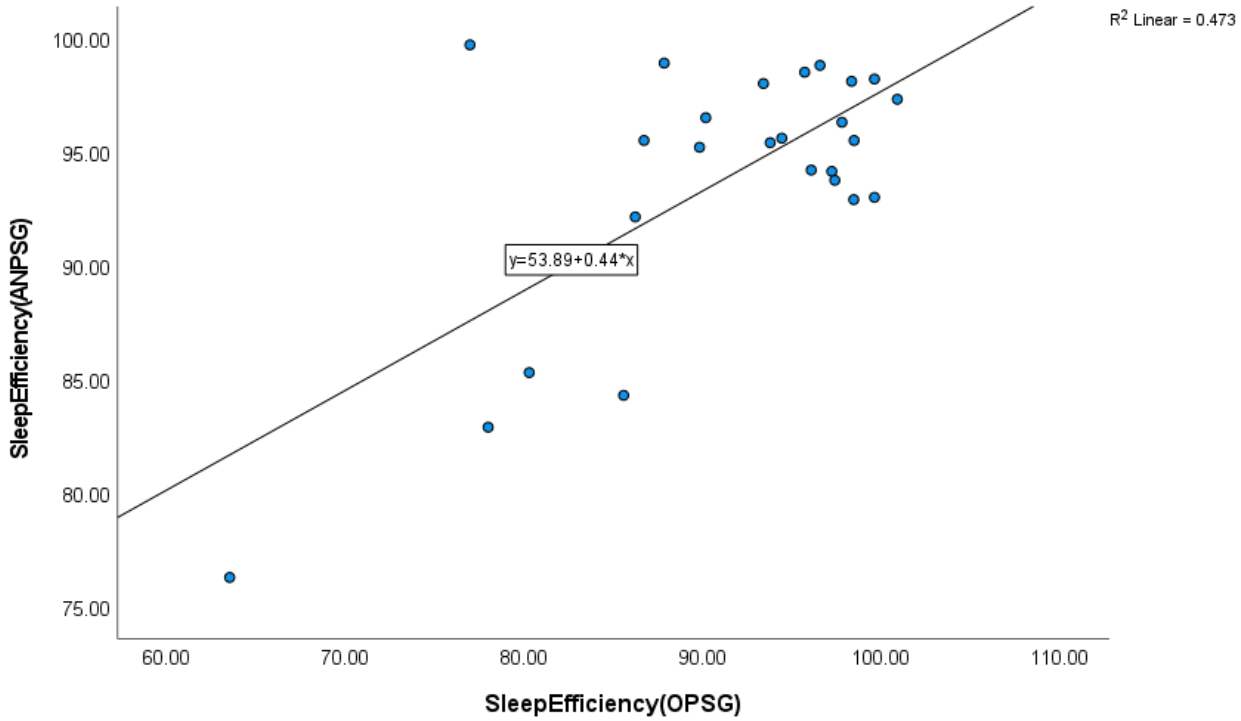


Figure 297: Association between sleep efficiency measured for ANPSG and OPSG

4.2.9 Initial REM latency (IRL)

The data in Table 13 show that there was no association between IRL measured for ANPSG and OPSG (P > 0.05).

Table 13: Correlation between initial REM latency for the afternoon and overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
Initial REM Latency(ANPSG) – Initial REM Latency(OPSG)	-.030	.885	-.420	.370
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 38 further illustrates the association between the initial REM latency measured for ANPSG and OPSG. The data show no linear association between ANPSG and OPSG. More so, all values were outside the regression line, which suggests a poor association.

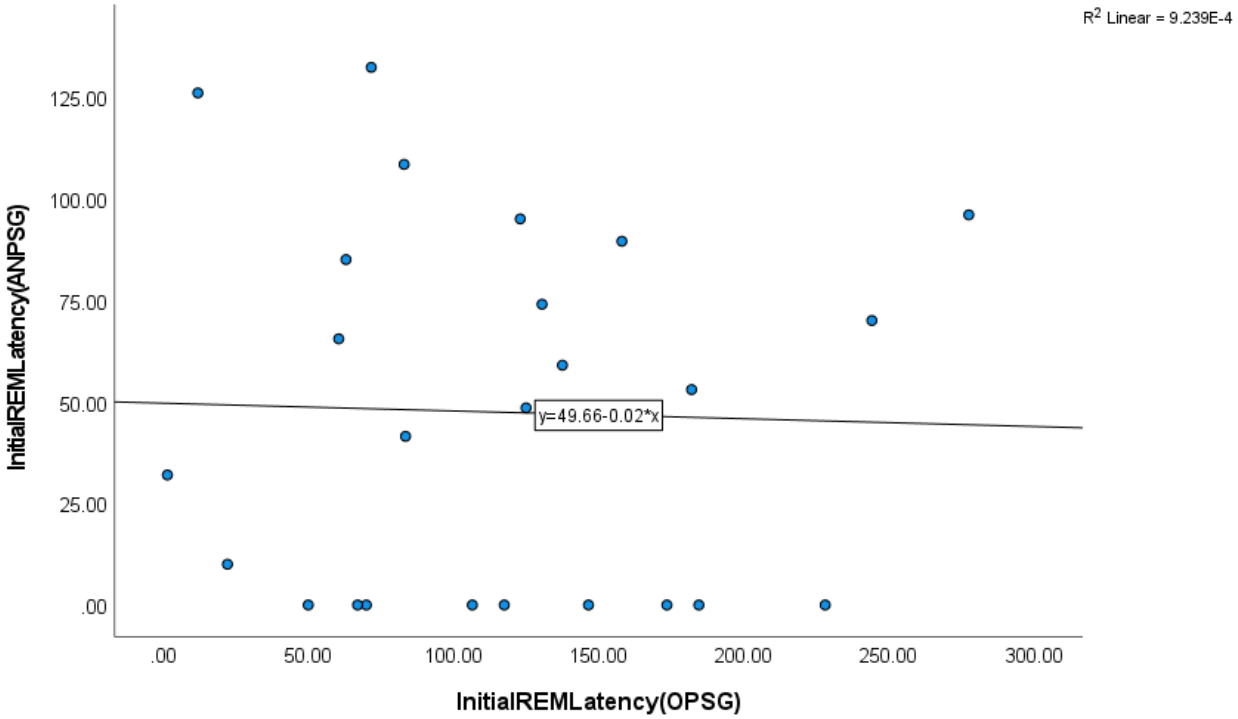


Figure 308: Association between initial REM latency measured for ANPSG with those measured for OPSG

4.2.10 Breathing-related arousal

The data in Table 14 show that there was a significant link between ANPSG and OPSG (r = 0.731, P < 0.001). This meant that as the breathing-related arousal for ANPSG increased, the breathing-related arousal for OPSG also increased, and *vice versa*.

Table 14: Correlation between breathing-related arousal for the afternoon and overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
BreathingRelatedArousals(ANPSG) - BreathingRelatedArousals(OPSG)	.731	<.001	.461	.870
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 39 further illustrates the association between sleep efficiency measured for ANPSG and OPSG. The regression coefficient reveals a strong linear association between ANPSG and OPSG.

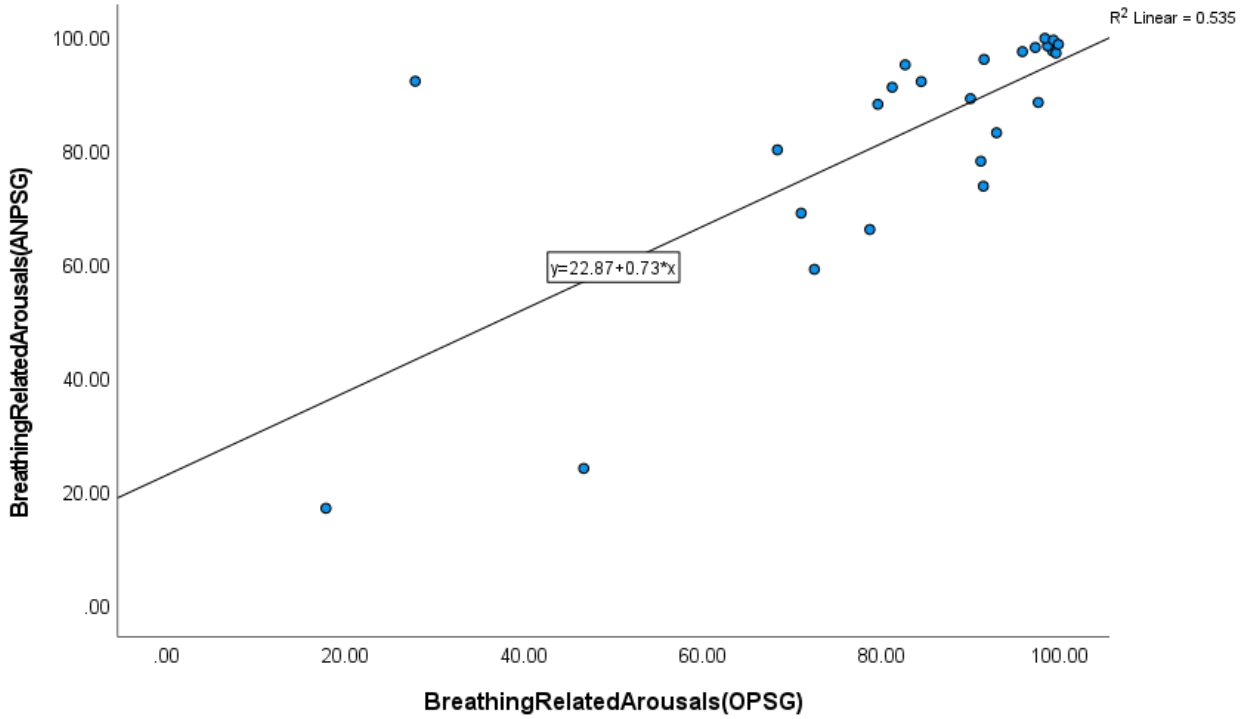


Figure 319: Association between breathing related arousal measured for ANPSG and OPSG

4.2.11 Spontaneous related arousal

The data in Table 15 show that there was a significant relation between ANPSG and OPSG (r = 0.457, p = 0.022). This meant that as the spontaneous-related arousal for ANPSG increased, the spontaneous-related arousal for OPSG also increased, and *vice versa*.

Table 15: Correlation between spontaneous related arousal for the afternoon and overnight polysomnography

	Pearson Correlation	Sig. (2-tailed)	95% Confidence Intervals (2-tailed)[a]	
			Lower	Upper
SpontaneousRelatedArousals (ANPSG) - SpontaneousRelatedArousals (OPSG)	.457	.022	.066	.717
a. Estimation is based on Fisher's r-to-z transformation with bias adjustment.				

Figure 40 further illustrates the association between spontaneous related arousal measured for ANPSG and OPSG. The regression coefficient reveals a weak linear association between ANPSG and OPSG.

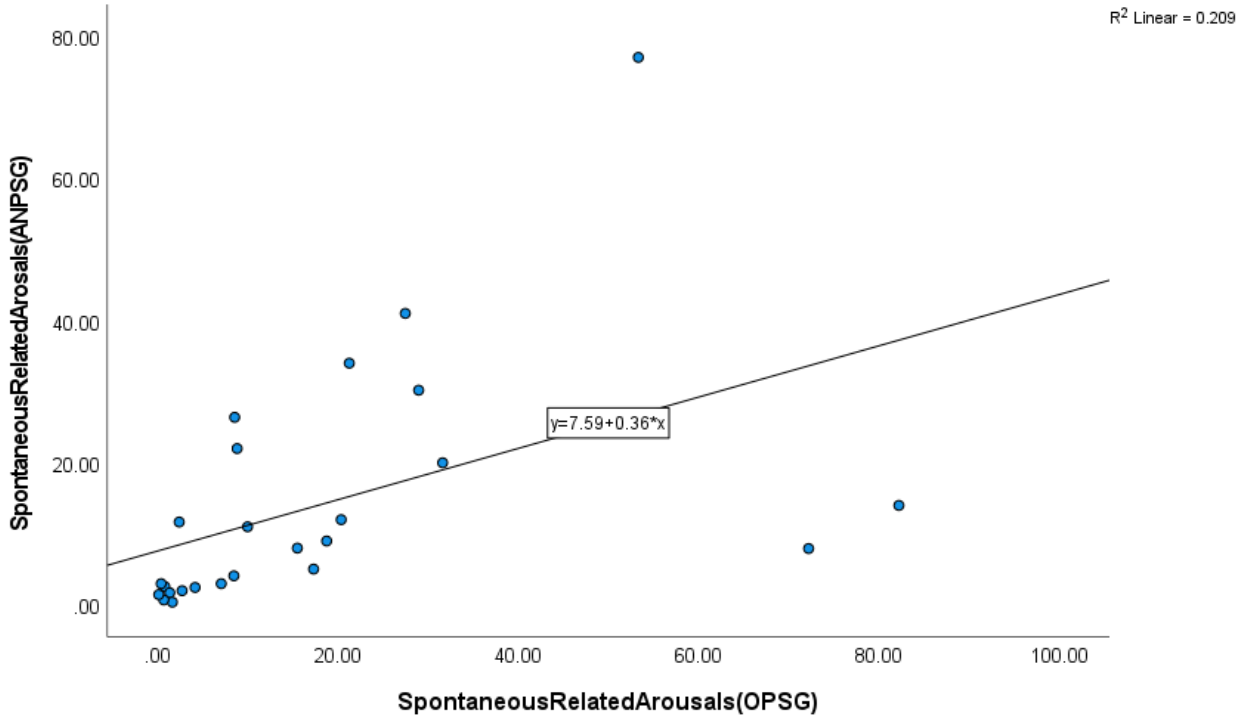


Figure 40: Association between spontaneous related arousal measured for ANPSG and OPSG

4.3 Comparison between afternoon and overnight polysomnography

Table 16 shows the mean, standard deviation, and the paired sample test for the afternoon (ANPSG), and overnight (OPSG) polysomnography measured for participants. The paired sample test shows that there was a statistically significant difference between afternoon and overnight polysomnography measured for RDI ($p < 0.001$), AHI ($p = 0.002$), NREM (RDI) ($p < 0.001$), NREM (AHI) ($p = 0.001$), REM (RDI) ($p = 0.005$), REM (AHI ($p = 0.027$), total sleeping time ($p < 0.001$), and initial REM latency ($p < 0.001$). The mean value measured for OPSG was higher when compared against the ANPSG for the following variables: RDI (72.85 ± 33.99), AHI (66.76 ± 33.73), NREM RDI (72.69 ± 36.48), NREM (AHI) (66.63 ± 36.18), REM (RDI) (76.72 ± 26.65), REM (AHI) (69.64 ± 28.97), total sleep time (421.96 ± 61.03), and initial REM latency (116.74 ± 71.46). No difference could be measured between ANPSPG and OPSG for the following variables: sleep efficiency ($p = 0.065$), breathing-related arousals ($p = 0.877$), and spontaneous-related arousal ($p = 0.368$).

Table 16: Paired sample test showing the comparison between afternoon and overnight polysomnography

Variables	Reference range	ANPSG	OPSG	P value
		Mean ± SD	Mean ± SD	
RDI	N = < 5, mild = 5-15, moderate = 15-30, Severe = > 30	59.61 ± 35.51	72.85 ± 33.99	< 0.001*
AHI	N = < 5, mild = 5-15, moderate = 15-30, Severe = >30	55.68 ± 34.97	66.76 ± 33.73	0.002*
NREM (RDI)	Severe = > 30	57.22 ± 37.83	72.69 ± 36.48	< 0.001*
NREM (AHI)	Severe = > 30	54.07 ± 36.53	66.63 ± 36.18	0.001*
REM (RDI)	Severe = > 30	47.79 ± 46.64	76.72 ± 26.65	0.005*
REM (AHI)	Severe = > 30	49.06 ± 46.15	69.64 ± 28.97	0.027*
Total sleep time	Minutes of stages 1, 2, 3, and REM	126.90 ± 22.59	421.96 ± 61.03	< 0.001*
Sleep efficiency	> 90%	93.86 ± 5.75	91.31 ± 9.04	0.065
Initial REM latency	90-120 minutes	47.43 ± 44.97	116.74 ± 71.46	< 0.001*
Breathing related arousals	See if % is more than the spontaneous arousals	82.64 ± 21.88	82.14 ± 21.99	0.877
Spontaneous related arousal	Arousals not related to breathing disturbance	14.03 ± 17.35	17.86 ± 21.99	0.368

* $p < 0.05$

4.4 Association between demographic characteristics and polysomnography

This section details the interrelationship between demographic characteristics such as age, gender, BMI, weight, height, neck circumference, and polysomnography (ANPSG and OPSG).

4.4.1 Association between gender and polysomnography

The association between participants' gender and polysomnography is given in Table 17. The data show that there was no statistically significant difference in the polysomnography measured at ANPSG ($p = 0.273$) and OPSG ($p = 0.109$).

Table 17: Gender and polysomnography

	ANPSG					OPSG				
Variable	**Normal**	**Mild**	**Moderate**	**Severe**	***P* value**	**Normal**	**Mild**	**Moderate**	**Severe**	***P* value**
Female (SD)	0 (0)	0 (0)	2 (14.3)	12 (85.7)	0.273	0 (0)	0 (0)	0 (0)	14 (100)	0.109
Male (SD)	1 (9.1)	1 (9.1)	3 (27.3)	6 (54.5)		1 (9.1)	1 (9.1)	2 (18.2)	7 (63.6)	

SD=standard deviation

4.4.2 Association between age, BMI, neck circumference, weight, height, and polysomnography

The association between participants' age, BMI, weight, height, and neck circumference and the polysomnography is given in Table 18. No statistically significant differences were found for the age, BMI, weight, and height, and the polysomnography measured for both ANPSG and OPSG ($p > 0.05$). However, there was a statistically significant association measured for the neck circumference for both the severity of ANPSG ($p = 0.017$) and severity of OPSG ($p = 0.025$). Participants with a neck circumference of 42.6 ± 3.8 had severe ANPSG while those with a neck circumference starting from 42.3 ± 4.0 had severe OPSG. It may be drawn from the data that the smaller the neck circumference the lower the risk of SAHS, possibly due to less tissue around the neck as the literature review states that men with neck circumference (NC) greater than 43.18 cm and above 38.1 cm for women are at risk of OSA (Kawaguchi *et al*. 2011; Kovacevic-Ristanovic and Kuzniar 2014).

Table 18: Age, BMI, neck circumference, weight, height, and polysomnography

	ANPSG					OPSG				
Variable	**Normal**	**Mild**	**Moderate**	**Severe**	***P* value**	**Normal**	**Mild**	**Moderate**	**Severe**	***P* value**
Age (years) (SD)	36 (0)	32 (0)	50.4 (14.3)	53.1 (8.5)	0.109	36 (0)	32 (0)	58 (4.2)	52 (10)	0.092
BMI (SD)	38.4 (0)	18.9 (0)	39.1 (13.2)	38.1 (10.3)	0.405	38.4 (0)	18.9 (0)	42.6 (6.0)	37.9 (11.0)	0.359
Neck circumference (SD)	36 (0)	30 (0)	40.5 (3.9)	42.6 (3.8)	0.017	36 (0)	30 (0)	40.5 (2.1)	42.3 (4.0)	0.025
Weight (SD)	106.6 (0)	45.6 (0)	96.7 (29.2)	108.0 (26.8)	0.184	106.6 (0)	45.6 (0)	101.1 (18.1)	105.9 (28.1)	0.239
Height (SD)	1.7 (0)	1.6 (0)	1.6 (0)	1.7 (0)	0.151	1.7 (0)	1.6 (0)	1.5 (0.0)	1.7 (0.1)	0.223

SD= Standard deviation

4.5 Predictors of respiratory disturbance index (RDI)

This section details the predictors of RDI. Multiple regression analysis was done to test which of the demographic variables predict RDI measured for ANPSG and OPSG. The results are summarised below.

4.5.1 Socio-demographic predictors of RDI measured for ANPSG and OPSG

The correlation of the RDI measured for ANPSG and OPSG is shown in Table 19. An inversely proportional correlation was found between the age group and RDI, however, the data suggested no correlation between neck circumference, height, weight, and RDI. This implies that as the age of the participants increased, the RDI measured for ANPSG and OPSG decreased.

Table 19: Correlation between socio-demographic predictors of RDI measured for ANPSG and OPSG

		ANPSG(RDI)	OPSG(RDI)
BMI	Pearson Correlation	.139	.208
	Sig. (2-tailed)	.509	.319
	N	25	25
Age group	Pearson Correlation	-.461*	-.396*
	Sig. (2-tailed)	.021	.050
	N	25	25
Gender	Pearson Correlation	.262	.167
	Sig. (2-tailed)	.205	.425
	N	25	25
Neck Circumference	Pearson Correlation	-.175	-.115
	Sig. (2-tailed)	.403	.584
	N	25	25
Weight	Pearson Correlation	-.022	.068
	Sig. (2-tailed)	.917	.747
	N	25	25
Height	Pearson Correlation	-.323	-.272
	Sig. (2-tailed)	.116	.188

4.5.2 Afternoon nap polysomnography (ANPSG)

The predictors of the RDI measured for ANPSG are shown in Table 20. The F-test suggests that the model was statistically significant. The regression coefficient ($r = 0.791$, $p = 0.004$) suggests a strong causal relationship in the predicted model. The beta coefficient for age was a negative predictor and significant ($\beta = -0.760$, $p < 0.001$).

This implies that as the age of the participant increased, the RDI measured at ANPSG decreased. The R^2 values measured suggest that there was a strong explanatory power (62.5%) for the predictors in the model. The data suggests that there is no collinearity in the measured independent variable (predictor).

Table 20: Multiple regression on socio-demographic predictors of RDI measured at ANPSG

Predictor	F-value	P-value	R	Beta Coefficients	Error	R Square	Significance	Collinearity statistics VIF
Gender	5.001	0.004	0.791	.186	10.904	0.625	.247	1.162
Age				-.760	.565		< .001	1.377
BMI				3.783	5.340		.033	129.630
Neck circumference				.113	1.679		.610	2.262
Weight				-3.354	1.969		.048	120.492
Height				1.088	248.395		.156	25.950

VIF = variance inflation factor

4.5.3 Overnight polysomnography (OPSG)

The predictors of the RDI measured for OPSG are shown in Table 21. The F-test suggests that the model was statistically significant. The regression coefficient ($r = 0.746$, $P = 0.013$) suggests a strong causal relationship in the predicted model. The beta coefficient for age was a negative predictor and significant ($\beta = -0.711$, $p < 0.001$). This implies that as the age of the participant increased, the RDI measured for OPSG decreased. The R^2 values measured suggest that there was a strong explanatory power (55.7%) for the predictors in the model. The data suggest that there is no collinearity in the measured independent variable (predictor).

Table 21: Multiple regression on socio-demographic predictors of RDI measured at OPSG

Predictor	F-value	P-value	R	Beta Coefficients	Error	R Square	Significance	Collinearity statistics VIF
Gender	3.768	0.013	0.746	.102	11.350	0.557	.555	1.162
Age				-.711	.588		.001	1.377
BMI				4.238	5.558		.029	129.630
Neck circumference				.063	1.748		.794	2.262
Weight				-3.733	2.050		.044	120.492
Height				1.333	258.550		.113	25.950

VIF = variance inflation factor

4.6 Predictors of the apnoea-hypopnea index – AHI

This section details the predictors of AHI. A multiple regression analysis was performed to test which of the demographic variables predicts AHI measured for ANPSG and OPSG. The results are summarised below.

4.6.1 Socio-demographic predictors of AHI measured for ANPSG and OPSG

The correlation of the AHI measured for ANPSG and OPSG is shown in Table 22. A correlation was found between the age group and AHI, however, the data suggested no correlation between neck circumference, height, and AHI. This implies that as the age of the participant increased, the AHI measured for ANPSG and OPSG increased as well.

Table 22: Correlation between socio-demographic predictors of AHI measured for ANPSG and OPSG

		ANPSG(AHI)	OPSG(AHI)
BMI	Pearson Correlation	.177	.198
	Sig. (2-tailed)	.396	.342
	N	25	25
Age group	Pearson Correlation	-.433*	-.406*
	Sig. (2-tailed)	.031	.044
	N	25	25
Gender	Pearson Correlation	.255	.126
	Sig. (2-tailed)	.218	.548
	N	25	25
Neck Circumference	Pearson Correlation	-.137	-.075
	Sig. (2-tailed)	.515	.720
	N	25	25
Weight	Pearson Correlation	.020	.064
	Sig. (2-tailed)	.923	.762
	N	25	25
Height	Pearson Correlation	-.312	-.252
	Sig. (2-tailed)	.129	.224
	N	25	25

4.6.2 Afternoon nap polysomnography – ANPSG

The predictors of the AHI measured for ANPSG are shown in Table 23. The F-test suggests that the model was statistically significant. The regression coefficient ($r = 0.791$, $p = 0.003$) suggests a strong causal relationship in the predicted model. The beta coefficient for age was a negative predictor and significant ($\beta = -0.753$ $P < 0.001$).

This implies that as the age of the participant increased, the AHI measured for ANPSG decreased. The R^2 values measured suggest that there was a strong explanatory power (62.6%) for the predictors in the model. The data suggest that there is no collinearity in the measured independent variable (predictor).

Table 23: Multiple regression on socio-demographic predictors of AHI measured at ANPSG

Predictor	F-value	P-value	R	Beta coefficients	Error	R Square	Significance	Collinearity statistics VIF
Gender	5.016	0.003	0.791	.190	10.729	0.626	.555	1.162
Age				-.753	.556		.001	1.377
BMI				3.947	5.254		.029	129.630
Neck circumference				.133	1.652		.794	2.262
Weight				-3.482	1.937		.044	120.492
Height				1.154	244.412		.113	25.950

4.6.3 Overnight polysomnography – OPSG

The predictors of the AHI measured for OPSG are shown in Table 24. The F-test suggests that the model was statistically significant. The regression coefficient ($r = 0.758$, $p = 0.009$) suggests a strong causal relationship in the predicted model. The beta coefficient for age was a negative predictor and significant ($\beta = -0.735$ $P < 0.001$). This implies that as the age of the participant increases, the AHI measured for OPSG decreases. The R^2 values measured suggest that there was a strong explanatory power (57.5%) for the predictors in the model. The data suggest that there is no collinearity in the measured independent variable (predictor).

Table 24: Multiple regression on socio-demographic predictors of AHI measured at OPSG

Predictor	F-value	P-value	R	Beta coefficients	Error	R Square	Significance	Collinearity statistics VIF
Gender	4.064	0.009	0.758	.060	10.729	0.575	.723	1.162
Age				-.735	.556		< .001	1.377
BMI				4.538	5.254		.018	129.630
Neck circumference				.111	1.652		.636	2.262
Weight				-4.053	1.937		.027	120.492
Height				1.445	244.412		.081	25.950

4.7 Dichotomisation of ANPSG and OPSG AHI and RDI

Table 25 tests the diagnostic potential of ANPSG against that of OPSG using SPSS analysis program. ANPSG AHI ≤15 had a sensitivity and specificity of 66.7% with a p-value of 0.021 which was statistically significant while that of OPSG had a sensitivity and specificity of 100% and significant, both with a 95% confidence interval. ANPSG AHI ≥15 predicted a 95.8% sensitivity with 100% specificity, while that of OPSG had sensitivity and specificity of 100%.

Table 25: Categorization of AHI and RDI as moderate to severe risk (AHI ≥15/hour) and less than moderate to low risk groups (AHI <15/hour) between ANPSG and OPSG.

AHI or RDI	Variable	<15	≥15
ANPSG AHI	Sensitivity	66.7%	95.8%
	Specificity	66.7%	100%
	Area under ROC curve	1.00	1.00
	Severity correctly classified	2/25(8%)	23/25(92%)
	(95% CI)	(1.00-1.00)	(1.00-1.00)
	Sig	0.021	0.021
OPSG AHI	Sensitivity	100%	100%
	Specificity	100%	100%
	Area under ROC curve	(0.00-0.00)	(1.00-1.00)
	Severity correctly classified	2/25 (8%)	23/25 (92%)
	(95% CI)	(0.00-0.00)	(1.00-1.00)
	Sig	0.021	0.021
ANPSG RDI	Sensitivity	100%	100%
	Specificity	100%	100%
	Area under ROC curve	(0.00-0.00)	(1.00-1.00)
	Severity correctly classified	2/25 (8%)	23/25(92%)
	(95% CI)	(0.00-0.00)	(1.00-100)
	Sig	0.021	0.021
OPSG RDI	Sensitivity	100%	100%
	Specificity	100%	100%
	Area under ROC curve	(0.00-0.00)	(1.00-1.00)
	Severity correctly classified	1/25 (4%)	24/25(96%)
	(95% CI)	(0.00-0.00)	(1.00-1.00)
	Sig	0.096	0.096

Figure 41 and 42 further illustrate the association between ANPSG and OPSG measured for AHI and RDI, respectively. The regression coefficient reveals a strong linear association between ANPSG and OPSG.

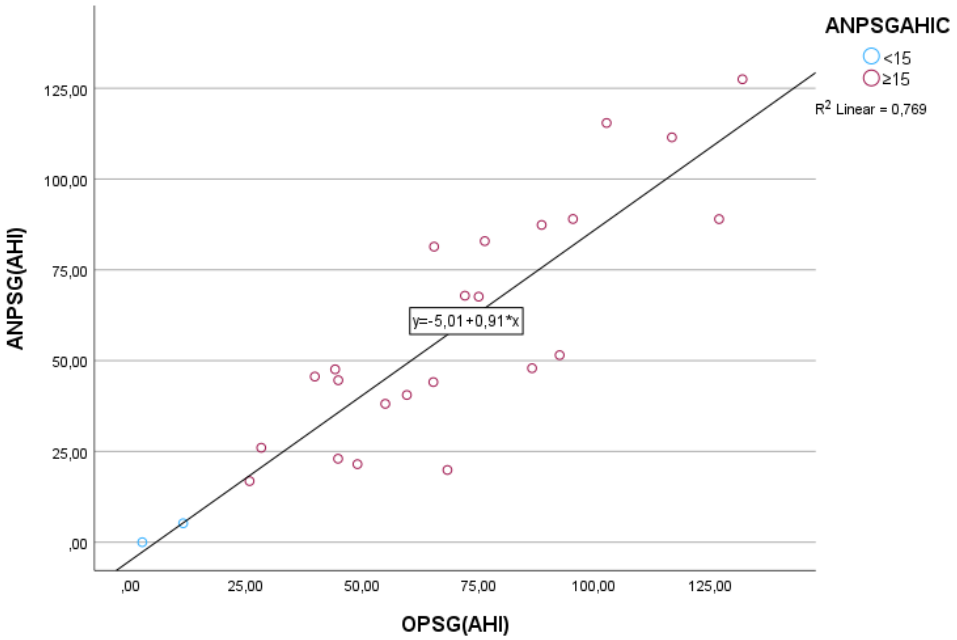


Figure 41: Association between ANPSG and OPSG measured for AHI

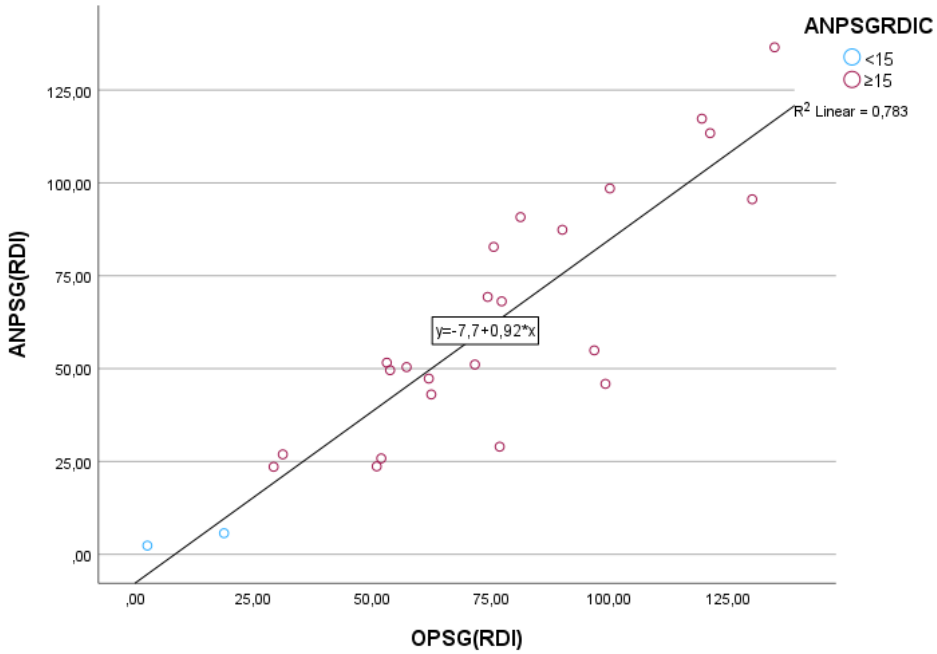


Figure 42: Association between ANPSG and OPSG measured for RDI

Figure 43 below, illustrates the difference in RDI and AHI between the ANPSG and OPSG for each individual participant.

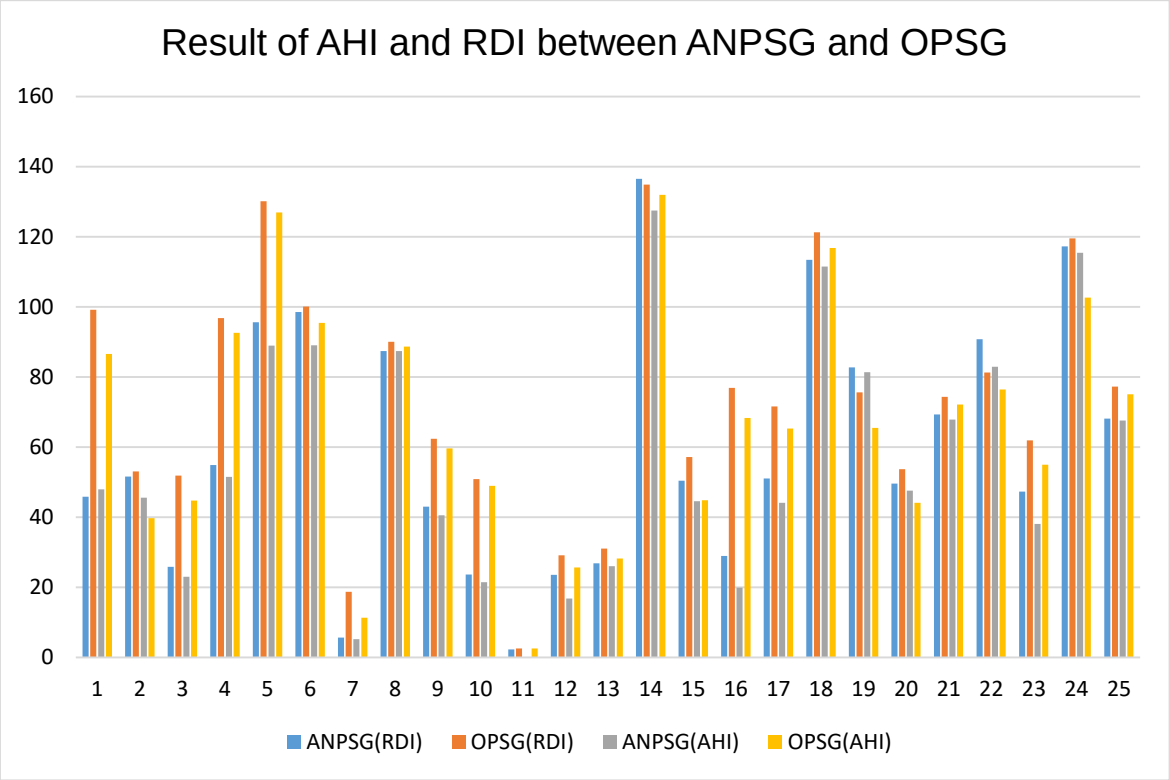


Figure 43: Results of RDI and AHI between ANPSG and OPSG

CHAPTER 5: DISCUSSION, STRENGTHS AND LIMITATIONS

5.1 Discussion of results

Obstructive sleep apnoea (OSA) is reported to be much more noticeable around the age of ≥ 65 years and above compared to people aged 30-64 (Mannarino, Filippo and Pirro 2012). It also appears that there are racial disparities between Caucasians and African Americans with the latter affected more by OSA at a younger age (Mannarino Filippo and Pirro 2012). This was not explored in this study. Obesity is the most commonly reported risk factor for OSA; it is clarified that a weight gain of 10% from a person's current weight, increases the risk of developing OSA sixfold (Mannarino, Filippo and Pirro 2012). The challenge is that fat deposits alter the normal upper airway mechanism in many ways (Garvey *et al.* 2015; Shredl 2021).

This study aimed to compare the apnoea-hypopnoea index (AHI) and other polysomnography variables between the overnight polysomnography (OPSG) and the afternoon-nap polysomnography (ANPSG) tests. The objective of this study was to demonstrate that the OPSG and ANPSG yield similar results or have similar sensitivity in diagnosing sleep-disordered breathing (SDB) when the measurements for ANPSG and OPSG are performed on the same patients. The null hypothesis of this research was that any duration of sleep ≥ 2 hours does not affect the diagnostic value of AHI and other variables. Therefore, the significance of polysomnography (PSG) in measuring SDB using different sleep duration periods was tested. The controversial narrative is that ANPSG cannot be fully used as an alternative to OPSG (Gregório *et al*., 2011: 1098). Universally, OPSG is the gold standard when it comes to evaluating SDB (Epstein *et al*., 2009: 265). However, the standard diagnostic procedure for the diagnosis of sleep-disordered breathing (SDB) at Inkosi Albert Luthuli Central Hospital, Durban, is the ANPSG.

In this study, there were more female than male participants (56% versus 44%) (see Figure 28, and Table 4). This demographic information finding is unlike most studies which have shown that OSA is more common in males. However, another study done in South Africa had a higher number of females than male, 29% and 71%, respectively which led them to conclude that there is a necessity for actively promoting health

education and systematic screening and treatment of OSA to prevent future cardiovascular morbidity, especially among women (Ozoh *et al.* 2014). Rossi *et al.* (2021) further noted that OSA prevalence was more prominent in older ages, especially in males, and specifically those with higher body mass index. In this study, 60% of the participants were older than 51 years of age and 20% were in the age range of 41-50 years. The mean age of the participants was 51.04 ± 10.65 years (see Figure 29). This finding is consistent with studies from other parts of the world. As previously hinted and explained that older age is a risk factor for OSA or sleep apnoea and this may be driven by one or more physiological characteristics, like unstable ventilator control system or ineffective upper airway dilator muscle activity (Edwards *et al.* 2014; Rossi *et al.* 2021).

It was indicated that a BMI of 32 (most importantly above 40), and neck circumference (NC) greater than 43.18 cm for men and above 38.1 cm for women are useful predictors of OSA (Kawaguchi *et al*. 2011; Kovacevic-Ristanovic and Kuzniar 2014). In this study, participants with a neck circumference of 42.6 ± 3.8 had severe ANPSG while those with a neck circumference of 42.3 ± 4.0 had severe OPSG (Table 4, Figure 29). It can be drawn from the data that the lower the neck circumference the lower the risk of abnormal ANPSG and OPSG.

Mannarino, Filippo and Pirro (2012) published that patients at risk of OSA are usually obese, especially males older than 65 years old. It is further explained that the susceptibility of men to OSA occurs as a consequence of an androgenic pattern of body fat dissemination, specifically on the trunk and neck (Mannarino, Filippo and Pirro 2012).

The literature shows that the interrelation between OSA and obesity involves a mechanical factor, such as excessive fat deposits in the upper airway (Kawaguchi *et al.* 2011). In this study, the mean BMI was 37.6 ± 10.9 (kg/m^2), mean neck circumference 41.4 ± 4.6 m, mean weight 103.1 ± 28.6 kg, and mean height of 1.66 ± 0.1 m, with median 38, 42, 105,25, 1.64, and interquartal range of 16.88, 16.88, 40.57, 0.15, respectively (Table 4). An addendum of table of normality was also added (Annexure 27). Martinho *et al.* 2008 referred to the larger neck circumference and soft tissue deformities of the upper airway as being pointers to the conception of and

severity of OSAHS (Martinho *et al.* 2008). Weight loss has been shown to be the first line of treatment in obese patients and has shown improvement in OSA severity and cardiometabolic abnormalities (Romero-Corral *et al.* 2010).

It is impossible to scientifically explain the exact interconnection between OSA and obesity as at this point is not fully understood; however, it is plainly apparent that obesity may narrow the upper airway structure, altering the airway collapsibility, reducing the chest wall's ability to stretch and expand, resulting in a change of the relationship between respiratory drive and load compensation, causing reductions in functional residual capacity (Pahkala *et al*., 2014). Carneiro *et al*. (2012) and Chung *et al*. (2014) state that obesity is the most significant risk factor for OSA as it is remarkably reversible. Furthermore, the risk of developing OSA is amplified over tenfold in people with a BMI of > 29 kg/m^2, this pushes for a nutritious and healthier diet within society (Mergen *et al.* 2019). Patients with a BMI of ≥ 30 kg/m^2 are regarded as high-risk of being diagnosed with sleep-disordered breathing (Berry *et al.*, 2017; Chung *et al*., 2013; (Aurora and Quan 2016; Duarte *et al.* 2019), and correlation between BMI and AHI is strong (Chung *et al*. 2014). The researcher strongly suggests that high-risk groups in both in-patient and out-patient facilities should be screened for OSA with a validated questionnaire of choice (Berry *et al.* 2017; Aurora and Quan. 2016; Chung *et al*. 2013; Duarte *et al*. 2019; Kee *et al*. 2018; Mergen *et al*. 2019; Shredl 2021) hence Neurophysiology in IALCH uses four sleep questionnaires.

The demographic characteristics such as age, gender, BMI, weight, height, neck circumference, and polysomnography (ANPSG and OPSG) are outlined in Table 17 and Table 18. The association between the participants' gender and the polysomnography showed that there was no statistically substantial difference in the polysomnography measured at ANPSG (P = 0.273) and OPSG (P = 0.109), refer to Table 17. No statistically significant differences were found for age, BMI, weight, and height, and the polysomnography measured for both ANPSG and OPSG (P>0.05). There was a statistically significant association measured for the neck circumference for both ANPSG (P=0.017) and OPSG (P=0.025), refer to Table 18.

Annexure 26 shows the results from the questionnaires that indicate the majority of participants had problems with daily life skills due to the complications of sleep apnoea.

For example, some participants had scores that indicated a high risk of sleep apnoea with the Berlin questionnaire; very poor functional status with the functional outcome of sleep questionnaire; excessive hypersomnia with the Epworth sleep scale questionnaire; and a high risk of OSA with the STOP-Bang questionnaire (Annexure 26). Hypertension and excessive daytime sleepiness were common findings in our cohort. A study looking at daytime sleepiness considered a significant risk factor of hypertension in patients with obstructive sleep apnea (OSA), concluded OSA with objective daytime sleepiness is independently associated with hypertension (Ren *et al.* 2016). Another study looking at the association between the phenotype of excessive daytime sleepiness (EDS) and blood pressure (BP) in patients with obstructive sleep apnoea hypopnea syndrome (OSAHS), found that patients with more severe OSAHS showed more severe EDS and had more high BP (Wang *et al.* 2014).

This study (Table 5) showed that the RDI measured during ANPSG correlates positively with the RDI measured during OPSG and the association was significant ($r = 0.885$, $P < 0.001$). Figure 30, further illustrates the association between RDI ANPSG and RDI OPSG, with the regression coefficient revealing a strong linear association between ANPSG and OPSG. Therefore, as the RDI for ANPSG increased, the RDI for OPSG increased as well, and *vice versa*. The RDI is a similar measure to AHI, the difference being that it also includes the number of respiratory effort-related arousals per hour of sleep, in addition to the total number of apnoea and hypopnea events.

This study showed a significant association between ANPSG and OPSG ($r = 0.877$, $P < 0.001$). As the AHI for ANPSG increased the AHI for OPSG increased as well, and *vice versa* (Table 6). The regression coefficient reveals a strong linear association between ANPSG and OPSG (Figure 31).

In this study, the RDI data during NREM showed that there was a significant relation between ANPSG and OPSG ($r = 0.891$, $P < 0.001$). This means that as the NREM (RDI) for ANPSG increased, the NREM (RDI) for OPSG also increase, and *vice versa* (Table 7). The regression coefficient revealed a strong linear association between ANPSG and OPSG (Figure 32). The observation of having more apnoea during REM sleep and light sleep may be related more to the lack of stability in the control of

breathing. REM sleep is a fluctuating sleep stage during which upper airway collapsibility may vary from breath to breath (Penzel *et al.* 2001).

The data in Table 8 further illustrates that there was a significant association between ANPSG and OPSG ($r = 0.937$, $P < 0.001$). The regression coefficient in Figure 33 revealed a stronger linear association between ANPSG and OPSG. This meant that as the NREM apnoea-hypopnoea index (AHI) for ANPSG increased the NREM apnoea hypopnoea index (AHI) for OPSG increased as well, and *vice versa*. However, the data in Table 9 and Figure 34 indicating a correlation between REM (RDI) afternoon and REM (RDI) overnight polysomnography illustrated no linear association between REM measured for ANPSG and REM measured for OPSG ($P > 0.05$). More so, all values were outside the regression line, which suggests a poor association. During sleep, non-rapid eye movement (NREM) and rapid eye movement (REM) alternate in 4 to 6 cycles of approximately 70 to 90 minutes. This makes it important for the patient to be given enough time to go through all of the stages of sleep during PSG recording, as some sleep disorders are exacerbated by or occur only during certain sleep stages (Matheson, Singh and Packard 2007). The first REM period is short lasting approximately 10 minutes, and only later in the night or early morning do REM sleep episodes become progressively longer (Matheson, Singh and Packard 2007). Absent or only one REM sleep underestimates AHI since apnoeas and hypopnoeas tend to be worse in REM sleep as respiratory muscles are more hypotonic during this stage of sleep (de los Reyes 2014).

The correlation between REM apnoea-hypopnoea index (AHI) in the afternoon and REM (AHI) in overnight polysomnography showed no linear association ($P > 0.05$). More so, all values were outside the regression line, which suggests a poor association, see the data in Table 10 and Figure 35. Therefore, all values captured were variable and show that in some of the ANPSG, the REM stages of sleep could not be measured. Similar results were obtained for initial REM latency measured for the afternoon and overnight polysomnography. The data showed that there was no linear association between ANPSG and OPSG with this parameter ($P > 0.05$) (Table 13 and Figure 38). More so, all values were outside the regression line, which suggested poor association. This may have happened due to insufficient REM sleep obtained during the ANPSG as absent or only one REM sleep which underestimated

AHI since apnoeas and hypopnoeas tend to be worse in REM sleep as respiratory muscles are more hypotonic during this stage of sleep (de los Reyes 2014).

This study does not allow the comparison of sleep stages due to limitation of sleep cycles in ANPSG, intead the TST was compared. Patients were also not restricted to only sleep in supine position, therefore the sleep apnoeas recorded were not position specific. The association between total sleep times measured for ANPSG in comparison with that measured for OPSG showed no linear association ($p > 0.05$) (Table 11, Figure 36). More so, all values were outside the regression line, which suggested poor association. This showed that the durations of sleep between ANPSG and OPSG were very different as none of the durations correlated. This was expected due to the different sleep durations used for ANPSG and OPSG.

The calculation method used for sleep efficiency (SE) is total sleep time divided by the total time in bed, all multiplied by one hundred (TST x 100)/TBT and, this needs to be > 90% for it to qualify as within normal range. There was a significant correlation between sleep efficiency measured during ANPSG and OPSG ($r = 0.687$, $P < 0.001$). Both tests had SE that complemented each other (Table 12). The regression coefficient revealed a strong linear association between SE of ANPSG and OPSG (Figures 37). This further means that the majority of participants had more or less similar types of sleeping patterns when comparing the sleep pattern in ANPSG with that in OPSG. This proves that both PSGs were conducted in a similar environment and same properties except for duration. Not much could be found in the literature about this topic.

The study assessed the frequent awakening caused by apnoeas during sleep measuring breathing-related arousal for the afternoon and overnight polysomnography. The results (Table 14) indicated that there was a significant association between ANPSG and OPSG ($r = 0.731$, $P < 0.001$). The regression coefficient revealed a strong linear association between ANPSG and OPSG (Figure 39). This meant that the breathing disturbances were similar between ANPSG and OPSG. This was hypothetically expected for this study as it was previously mentioned that regardless of the duration of the test, the presence of SDB is not determined by

the longest or shortest hours of sleep because the apnoeas should occur in a similar manner and these are apnoeic arousals. These arousals are believed to occur as a result of escaping mechanism (Younes 2004).

A significant association between ANPSG and OPSG (r = 0.457, P = 0.022) was found for spontaneous-related arousal (Table 15). However, the regression coefficient revealed a weak linear association between ANPSG and OPSG (Figure 40). Therefore, general awakenings that were not evoked by breathing disturbance could not correlate. This was hypothetically expected due to different durations between the studies and different times of the day with different triggers such as the level of noise during the day in comparison with the night. Not much could be found in the literature on this agender.

However, the paired sample test shows that there were statistically significant differences between afternoon and overnight polysomnography measured for RDI (P < 0.001), AHI (P = 0.002), NREM (RDI) (p < 0.001), NREM (AHI) (P = 0.001), REM (RDI) (p = 0.005), REM (AHI (p = 0.027), total sleeping time (p < 0.001), and initial REM latency (p < 0.001), refer to Table 16.

The mean value measured for OPSG was higher when compared against the ANPSG for the following variables: RDI (72.85 ± 33.99), AHI (66.76 ± 33.73), NREM RDI (72.69 ± 36.48), NREM (AHI) (66.63 ± 36.18), REM (RDI) (76.72 ± 26.65), REM (AHI) (69.64 ± 28.97), total sleep time (421.96 ± 61.03), and initial REM latency (116.74 ± 71.46). table 16.

No difference could be measured between ANPSPG and OPSG for the variables sleep efficiency (p = 0.065), breathing-related arousals (P = 0.877), and spontaneous-related arousal (p = 0.368), refer to Table 16. This all points to the underestimation of the severity of sleep-disordered breathing by ANPSG when compared with OPSG (Nixon and Brouillette 2002).

Table 19 details the correlation between the socio-demographic predictors of RDI measured for ANPSG and OPSG where an inversely proportional correlation was

detected between age and RDI, when age increased, the RDI decreased. Furthermore, No correlation was found between NC, height, weight, and RDI.

The researcher then proceeded to the section detailing the predictors of RDI, where the predictors of the RDI measured for ANPSG and OPSG were explained, the results are summarised in Table 20 for ANPSG RDI and Table 21 for OPSG RDI. The multiple regression analysis was performed to test which of the demographic variables predict RDI measured for ANPSG and OPSG. With regards to ANPSG, the F-test suggested that the model was statistically significant. The correlation coefficient ($r = 0.791$, $p = 0.004$) suggested a strong causal relationship in the predicted model (Table 20). The beta coefficient for age was a negative predictor and significant ($\beta = -0.760$, $P < 0.001$). This implied that as the age of the participants increased, the RDI measured at ANPSG decreased (Table 20). This finding is not in keeping with other studies as previously mentioned that as age increases, the risk of developing OSA also increases. This opposing finding may be due to the scattered age difference between the participants. Obstructive sleep apnoea prevalence is much higher in older age groups, in males, and in those with higher body mass index (BMI) (Rossi *et al.* 2021). The R^2 values measured suggested that there was a strong explanatory power (62.5%) for the predictors in the model (Table 20). Overall, the data suggested that there is no collinearity in the measured independent variable (predictor).

Multiple regression on socio-demographic predictors of the RDI measured for OPSG is shown in Table 21. The regression coefficient ($r = 0.746$, $p = 0.013$) suggested a strong causal relationship in the predicted model. Age was a negative predictor and significant ($\beta = -0.711$, $p < 0.001$). This implied that as the age of the participants increased, the RDI measured at OPSG decreased. This finding is not in keeping with other studies as previously mentioned that as age increases, the risk of developing sleep apnoea also increases. This opposing finding may be due to the scattered age difference between the participants, or it may mean that the population in this study was variable in a sense indicating that any individual can be affected by SDB regardless of age.

Looking at the section detailing the socio-demographic predictors of AHI measured for ANPSG and OPSG. The correlation of the AHI measured for ANPSG and OPSG is

shown in Table 22. A correlation was found between the age group and AHI, however, the data suggested no correlation between neck circumference, height, and AHI. This implies that as the age of the participant increased, the AHI measured for ANPSG and OPSG increased as well. This finding is in keeping with other researchers' findings as age is widely listed as one of the risk factors (Rossi *et al.* 2021). The mechanism whereby aging increases the risk of OSA is not fully understood. Aging being a risk factor of sleep apnoea may be driven by one or more of the physiological characteristics. These may include changes in how the brain controls breathing during sleep. Poor upper airway anatomy, as it may become more prone to collapsibility; ineffective upper airway dilator muscle activity, as there may be reduced upper airway muscle reflex response to negative pressure in comparison with younger individuals; a low respiratory arousal threshold, as older individuals tend to have an increased frequency of spontaneous arousals, or an unstable ventilator control system (Edwards *et al.* 2014).

The section detailing the predictors of the apnoea hypopnoea index (AHI) measured for ANPSG is shown in Table 23 using multiple regression analysis The regression coefficient ($r = 0.746$, $p = 0.003$) suggested a strong causal relationship in the predicted model. This further supports the discussion of increased weight, BMI, and larger neck circumference in increasing the likelihood of developing SDB that was outlined at the beginning of this chapter. Obesity is defined as a BMI≥ 30, whereas a BMI ≥ 25.0 indicates the person is overweight. Compared to men, women have lower rates of being overweight or obese. The major contributing factors to obesity include environment, eating behavior, and physical inactivity. Psychosocial circumstances and genetics also play important roles in obesity(Naidoo ; Jehan *et al.* 2017). It is reported that obese individuals with shorter sleep duration have twice as many subjective sleep problems compared to non-obese people. Being obese or overweight is associated with decreased amount of sleep compared to non-obese patients; thus weight reduction can lessen sleep problems (Jehan *et al.* 2017). As a result preventing weight gain had a positive effect on sleep quality and duration in adult Black women(Jehan *et al.* 2017). The actual relationship between OSA and obesity is not clearly interpreted, although fairly understood that fat deposits add extra tissue around the neck narrowing the upper airway, and in turn this alters the opening and closing mechanism of the

airway, reducing chest wall operating standards, the respiratory drive and blood compensation alliance gets compromised (Pahkala *et al*., 2014).

The multiple regression analysis on socio-demographic predictors of AHI measured at OPSG was performed to test which of the demographic variables predicts AHI measured for OPSG, Table 24. The F-test suggested that the model was statistically significant. The regression coefficient ($r = 0.758$, $P = 0.009$) indicated a strong causal relationship in the predicted model. The beta coefficient for age was a negative predictor and significant ($\beta = -0.735$, $p < 0.001$). This implies that as the age of the participant increased, the AHI measured at OPSG decreased. Garvey *et al*. (2015) estimated that 34% of men and 17% of women aged 30-70 have at least mild OSA. The R^2 values measured suggested that there was a strong explanatory power (57.5%) for the predictors in the model. The data suggest that there is no collinearity in the measured independent variable (predictor).

The diagnostic potential of ANPSG was tested against that of OPSG using SPSS analysis program (version 28, 29) outlined in Table 25 with regression coefficient revealing a strong linear association (Figure 41 and 42) between ANPSG and OPSG, AHI at 0,769 and RDI at 0,783. Categorizing AHI and RDI as moderate to severe risk (AHI/RDI ≥15/hour) and less than moderate to low risk groups (AHI/RDI <15/hour) revealed a good correlation between ANPSG and OPSG. The results indicated a sensitivity and specificity for ANPSG (AHI,RDI <15) 66.7%, 100% and 66.7%, 100%, respectively; with p-value of 0.021 which was statistically significant. Sensitivity and specificity for OPSG (AHI,RDI <15) was 100% and 100%, respectively, and significant both with a 95% confidence interval less than 0.05, indicating a more precise estimate. ANPSG (AHI,RDI ≥15) had a sensitivity and specificity 95.8%, 100%, and 100%, 100% respectively; with a p-value of 0.021. Lastly OPSG (AHI,RDI ≥15) revealed similar outcome of 100% in sensivity and specifity, with a confidence interval of 0.021, and 0.096 indicating a more precise estimate. In overall, OPSG had slightly higher total percentages which supports and indicating ANPSG to be a good screening test for SDB, this is further supported by the bar chart (Figure 43) plotted to clearly exhibit the number of AHI and RDI for ANPSG and OPSG for each individual participant.

The sensitivity and specificity for the diagnosis of OSA (AHI>15 events/hour) by induced sleep ANPSG done in a similar fashion as IALCH compared to that of OPSG were 0.83 and 0.72, respectively. This in their study suggested that the ANPSG method may be useful screening tool for patient requiring OPSG or to assist in selecting which patient may require such treatment, and therefore a promising alternative method in the diagnosis of OSA (Gregório *et al.* 2011). In this current study AHI and RDI was rated separately for clear understanding. The best values for sensitivity and specificity were obtained through the receiver operating characteristic (ROC) curve Some of the few reasons to define 15/hour as a cut-off point for less than moderated to low and moderate to severe risk include but not limited to prevalence of moderate to severe OSA in middle-aged adults of approximately 13% in men and 6% found in woman (Lyons *et al.* 2015). Mahakit (2012) reports that, to be considered a candidate for surgical intervention (UPPP), a patient has to have less than 20 events/hour (RileyPowell-Stanford Surgical Protocol) (Mahakit 2012). Mild snoring intensity (loudness) was rated and defined as in bed annoyance (~ 25 dB) (Mahakit 2012). The sensitivity, and specificity of 92%, and 91.3%, in a study to compare daytime PSG with overnight PSG demonstrated that daytime PSG can be acceptable as a screening test and can also be used as an assessment tool for the outcome of surgical interventions, as the sensitivity, and specificity values were found to be high (Mahakit 2012).

Studies closely related to the current study compared split-night with overnight PSG. In a split-night study comparing 2 hours and 3 hours AHI in comparison with that of OPSG a concordance correlation coefficient of 0.93 and 0.97, respectively was found (Khawaja *et al.* 2010). Urgeijo et al demonstrated that the ANPSG was a technique for rapid diagnosis of SDB and found that 70% of ANPSG were diagnosed as SDB patients (JI, Gastan and Landa 2001). Mizuma et al compared two-hour daytime PSG with overnight PSG and found no significant differences between apnea index and mean oxygen saturation (SaO2). A positive correlation was found between daytime PSG and OPSG, so as to suggest that daytime PSG was useful not only in diagnosing sleep apnoea sysdrome but also in evaluating the severity (MIZUMA, SONNENSCHEIN and MEIER-EWERT 1996). Sergi et al (1998) found similar results of no difference in AHI and SaO2 between OPSG and ANPSG. The sensitivity was 91% and specificity was 100% (Sergi *et al.* 1998). Suzuki et al (2000), found not

difference between the two tests, as the researcher studied two hour nap PSG in long distance drivers and compared it with OPSG (Suzuki *et al.* 2000).

The researcher had hoped to obtain a diverse range of participants in terms of gender, race, and age. This was important as it provides an opportunity to analyse data in terms of gender, age, and race. Exclusion criteria excluded patients older than 70 years or younger than 18 years and patients with insomnia, to prevent inaccurate assessment of sleep-disordered breathing. The purpose of this study was to demonstrate that the ANPSG and OPSG yield similar (no significant difference) results in the diagnosis of SDB when the measurements for ANPSG and OPSG when performed on the same patients. The chief goal was to improve the diagnosis of sleep disorders in IALCH. Furthermore, the questions that were to be clarified by this research included the difference in PSG values obtained during ANPSG when compared to OPSG in the diagnosis of SDB. This study has provided the answers. ANPSG underestimates the severity parameters of OSA. However, the question of whether the diagnosis of SDB can be made confidently with ANPSG instead of OPSG at IALCH has been answered. ANPSG may confirm the AHS, however may not grade the true nocturnal severity, and when SDB is at a mild stage, ANPSG may not rule out that sleep disordered breathing will be or will not be present in the OPSG.

5.2 Strengths

This is the first study of sleep apnoea from KwaZulu-Natal and according to the researcher's knowledge, the first study to compare ANPSG to OPSG in South Africa. The cohort was composed of all racial groups: Africans, Indians, and Asians. The methodology was rigorous and the laboratory setup conforms to international standards.

Most of the studies in Africa were about the awareness and the risk of OSA (Roche *et al.* 2021). A study conducted in South Africa looking at the prevalence of OSA in older adults, and its association with cardiometabolic diseases (CMR) in a rural, low-income areas. The outcome was that older adults (≥ 40) with obesity, and hypertension within the South African community where the study was conducted in Mpumalanga, OSA prevalence is alarming and associated with CMR. The results highlighted the necessity

for actively promoting health education and systematic screening and treatment of OSA in this population to prevent future cardiovascular morbidity, especially among women, as there were 22 males and 53 females that took part in the study (Ozoh *et al.* 2014; Roche *et al.* 2021).

5.3 Limitations

The research was performed in the midst and worst part of the COVID-19 pandemic. This affected participant recruitment as all resources were channeled toward fighting the pandemic. The sample size ideally should have been larger but was affected by the COVID-19 restrictions which made it difficult to recruit subjects. The researcher had to continue to contribute one hundred percent to service delivery work, particularly considering the challenges which were posed by COVID-19 to all healthcare workers.

Future studies should incorporate more technology collaboration between Neurophysiology and Pulmonology in measuring characteristics like arousal threshold, the pharyngeal passive critical closing pressure, the sleep stage cycles, AHI, RDI, and muscle responsiveness with different stilnox dosages. This will assist in understanding the inter-individual heterogeneity and the actual therapeutic potential of stilnox/ zolpidem for different types of people with SDB (Carberry, Grunstein and Eckert 2019).

CHAPTER 6: SUMMARY, CONCLUSION, AND RECOMMENDATIONS

6.1 Summary

This study was performed to determine the correlation of the apnoea-hypopnoea index (AHI) and other polysomnography variables measured during afternoon nap polysomnography (ANPSG) and overnight polysomnography (OPSG) on the same patients at Inkosi Albert Luthuli Central Hospital (IALCH). The results showed a gender distribution of 56% females and 44% males. Most of the participants were above the age of 51 years of age (60%) with a mean age of 51.04 ± 10.65.

The participants in this study had a mean BMI of 37.6 ± 10.9 kg/m^2, a mean neck circumference of 41.4 ± 4.6 cm, an average weight of 103.1 ± 28.6 kg, and a mean height of 1.66 ± 0.1 m. The study confirms the overwhelming evidence that there is a very strong association between BMI and neck circumference with OSA. Martinho *et al*. (2008) described larger neck circumference and soft tissue abnormalities of the upper airway as being markers of the presence and severity of OSAS. Further research found that a larger neck circumference (p = 0.02), posterior soft palate (p = 0.03), and thick soft palate (p = 0.04) were all associated with OSAS severity (Martinho *et al.* 2008). Participants in this study with a neck circumference of 42.6 ± 3.8 had severe OSA as measured by ANPSG while those with a neck circumference of 42.3 ± 4.0 had severe OSA as measured by OPSG.

Both ANPSG and OPSG showed strong correlation in diagnosing SAHS, the ROC indicated similar percentages except when RDI ≥15 was compared, then OPSG showed slightly higher percentage, this indicated that ANPSG is an excellent screening test for SDB. Annexure 20 indicates the brief outcomes of the reports. Thus, the ANPSG is as good as the OPSG in diagnosing OSA in our setting. The paired sample test showed that there were statistically significant differences between afternoon and overnight polysomnography. The mean value measured for OPSG was higher when compared against the ANPSG for variables; RDI (72.85 ± 33.99), AHI (66.76 ± 33.73), NREM RDI (72.69 ± 36.48), NREM (AHI) (66.63 ± 36.18), REM (RDI) (76.72 ± 26.65),

REM (AHI) (69.64 ± 28.97), total sleep time (421.96 ± 61.03), and initial REM latency (116.74 ± 71.46). No differences could be detected between ANPSPG and OPSG for the variables sleep efficiency (p = 0.065), breathing-related arousals (p = 0.877), and spontaneous-related arousal (p = 0.368).

Therefore, although ANPSG is as good as OPSG at diagnosing and documenting abnormal variables for RDI, AHI, NREM RDI, REM RDI, REM AHI, NREM AHI, and initial REM latency, the values obtained during the OPSG are significantly higher than those obtained during ANPSG. This is an important finding for clinicians to remember when interpreting the results of sleep studies performed during ANPSG.

6.2 Conclusion

Overall, our results show that ANPSG underestimates the severity of sleep apnoea parameters. ANPSG may confirm the AHS, however may not grade the true nocturnal severity. When SDB is at a mild stage, ANPSG may not rule out that sleep disordered breathing will be or will not be present in the OPSG. As ANPSG is not as good as OPSG at grading the severity of some of the measured variables, it underestimates the severity of the indexes. This study is in agreement with the statement raised by Foldvary-Schaefer et al, (2019) when they stated that an ANPSG may confirm sleep breathing disorder but may not grade its true nocturnal severity and does not rule out that sleep-disordered breathing will be found on an OPSG (Foldvary-Schaefer, Grigg-Damberger and Mehra 2019).

6.3 Recommendations

The researcher makes the following recommendations to clinicians, specialist physicians, pulmonologists, and management at IALCH and the DOH KwaZulu-Natal:

1. Promote the use of ANPSG for the diagnosis of sleep apnoea. This will greatly facilitate the quicker assessment of suspected cases while reducing costs both financially and in terms of human resources. The proviso is that ANPSG is not as accurate as OPSG at severity grading.
2. The backlog of patients requiring investigations for OSA will be reduced if we ramp up ANPSG.

3. Actively campaign for the education of the general population regarding the strong association between obesity and neck circumference with OSA and other forms of sleep-disordered breathing.
4. Establish sleep centres of excellence at every regional hospital in KwaZulu-Natal to promote advocacy, diagnosis, and treatment of OSA and other forms of sleep-disordered breathing.

REFERENCES

Abbott, S. M., Reid, K. J. and Zee, P. C. 2015. Circadian rhythm sleep-wake disorders. *Psychiatric Clinics*, 38 (4): 805-823.

Acet-Öztürk, N. A., Dilektasli, A. G., Aydın-Güçlü, Ö., Demirdöğen, E., Coşkun, F., Ursavaş, A., Karadağ, M. and Uzaslan, E. 2022. Long-term oxygen treatment need is less frequent in eosinophilic COPD patients. *The Clinical Respiratory Journal*, 16 (1): 49-56.

Ahmad, B., Sankari, A., Eshraghi, M., Aldwaikat, A., Yarandi, H., Zeineddine, S., Salloum, A. and Badr, M. S. 2022. Effect of Zolpidem on nocturnal arousals and susceptibility to central sleep apnea. *Sleep and Breathing*. doi: 10.1007/s11325-022-02593-3

Al-Ghamdi, S. A., Manoukian, J. J., Morielli, A., Oudjhane, K., Ducharme, F. M. and Brouillette, R. T. 1997. Do systemic corticosteroids effectively treat obstructive sleep apnea secondary to adenotonsillar hypertrophy? *The Laryngoscope*, 107 (10): 1382-1387.

Alomri, R. M., Kennedy, G. A., Wali, S. O., Ahejaili, F. and Robinson, S. R. 2021. Differential associations of hypoxia, sleep fragmentation, and depressive symptoms with cognitive dysfunction in obstructive sleep apnea. *Sleep*, 44 (4): zsaa213.

Altevogt, B. M. and Colten, H. R. 2006. *Sleep disorders and sleep deprivation: an unmet public health problem*. Washington D.C.: National Academies Press.

Amra, B., Rahmati, B., Soltaninejad, F. and Feizi, A. 2018. Screening questionnaires for obstructive sleep apnea: an updated systematic review. *Oman Medical Journal*, 33 (3): 184.

Arzt, M., Young, T., Finn, L., Skatrud, J. B. and Bradley, T. D. 2005. Association of sleep-disordered breathing and the occurrence of stroke. *American Journal of Respiratory and Critical Care Medicine*, 172 (11): 1447-1451.

Abuan, M. R. A., Lin, W.-N., Hsin, L.-J., Lee, L.-A., Fang, T.-J., Chen, N.-H., Lo, Y.-L. and Li, H.-Y. 2020. Tongue imaging during drug-induced sleep ultrasound in obstructive sleep apnea patients. *Auris Nasus Larynx*, 47 (5): 828-836.

Aung, H., Sivakumar, A., Gholami, S., Venkateswaran, S. and Gorain, B. 2019. An overview of the anatomy and physiology of the lung. In: *Nanotechnology-based targeted drug delivery systems for lung cancer*. Amsterdam: Elsevier, 1-20.

Aurora, R. N. and Quan, S. F. 2016. Quality measure for screening for adult obstructive sleep apnea by primary care physicians. *Journal of Clinical Sleep Medicine*, 12 (8): 1185-1187.

Barber, L. K., Munz, D. C., Bagsby, P. G. and Powell, E. D. 2010. Sleep consistency and sufficiency: are both necessary for less psychological strain? *Stress and Health*, 26 (3): 186-193.

Barnes, S. and Lewin, C. 2011. Differences and relationships in quantitative data. In: Somekh, B. and Lewin, C. eds. *Theory and methods in social research*. London: Sage.

Benca, R. M. 2005. Diagnosis and treatment of chronic insomnia: a review. *Psychiatric services*, 56 (3): 332-343.

Berry, R., Brooks, R., Gamaldo, C., Jarding, S., Marcus, C. and Vaughn, B. 2014. *authors. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Version 2.1*. Darien, IL: American Academy of Sleep Medicine

Berry, R. B., Brooks, R., Gamaldo, C., Harding, S. M., Lloyd, R. M., Quan, S. F., Troester, M. T. and Vaughn, B. V. 2017. *AASM scoring manual updates for 2017 (version 2.4)*. Darien, IL: American Academy of Sleep Medicine.

Berry, R. B., Brooks, R., Gamaldo, C. E., Harding, S. M., Marcus, C. and Vaughn, B. V. 2012a. The AASM manual for the scoring of sleep and associated events. *Rules, Terminology and Technical Specifications, Darien, Illinois, American Academy of Sleep Medicine*. Darien, IL: American Academy of Sleep Medicine.

Berry, R. B., Budhiraja, R., Gottlieb, D. J., Gozal, D., Iber, C., Kapur, V. K., Marcus, C. L., Mehra, R., Parthasarathy, S. and Quan, S. F. 2012b. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events: deliberations of the sleep apnea definitions task force of the American Academy of Sleep Medicine. *Journal of Clinical Sleep Medicine*, 8 (5): 597-619.

Bianchi, M. T., Cash, S. S., Mietus, J., Peng, C.-K. and Thomas, R. 2010. Obstructive sleep apnea alters sleep stage transition dynamics. *PLoS One*, 5 (6): e11356.

Bonnet, M. H. and Arand, D. L. 2003. Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep medicine reviews*, 7 (4): 297-310.

Boudewyns, A., Abel, F., Alexopoulos, E., Evangelisti, M., Kaditis, A., Miano, S., Villa, M. and Verhulst, S. 2017. Adenotonsillectomy to treat obstructive sleep apnea: is it enough? *Pediatric Pulmonology*, 52 (5): 699-709.

Boyd, S. B. and Walters, A. S. 2013. Effectiveness of treatment apnea-hypopnea index: a mathematical estimate of the true apnea-hypopnea index in the home setting. *Journal of Oral and Maxillofacial Surgery*, 71 (2): 351-357.

Brass, S. D., Duquette, P., Proulx-Therrien, J. and Auerbach, S. 2010. Sleep disorders in patients with multiple sclerosis. *Sleep medicine reviews*, 14 (2): 121-129.

Brindle, R. C. and Conklin, S. M. 2012. Daytime sleep accelerates cardiovascular recovery after psychological stress. *International Journal of Behavioral Medicine*, 19 (1): 111-114.

Brunetto, D. P., Moschik, C. E., Dominguez-Mompell, R., Jaria, E., Sant'Anna, E. F. and Moon, W. 2022. Mini-implant assisted rapid palatal expansion (MARPE) effects on adult obstructive sleep apnea (OSA) and quality of life: a multi-center prospective controlled trial. *Progress in Orthodontics*, 23 (1): 3.

Carberry, J. C., Grunstein, R. R. and Eckert, D. J. 2019. The effects of admidem in obstructive sleep apnea–an open-label pilot study. *Journal of Sleep Research*, 28 (6): e12853.

Carley, D. W. and Farabi, S. S. 2016. Physiology of sleep. *Diabetes Spectrum*, 29 (1): 5-9.

Carneiro, G., Flório, R. T., Zanella, M. T., Pradella-Hallinan, M., Ribeiro-Filho, F. F., Tufik, S. and Togeiro, S. M. 2012. Is mandatory screening for obstructive sleep apnea with polysomnography in all severely obese patients indicated? Sleep and Breathing, 16 (1): 163-168.

Cassano, P., Gelardi, M., Cassano, M., Fiorella, M. and Fiorella, R. 2003. Adenoid tissue rhinopharyngeal obstruction grading based on fiberendoscopic findings: a novel approach to therapeutic management. *International Journal of Pediatric Otorhinolaryngology*, 67 (12): 1303-1309.

Chai-Coetzer, C. L. and McEvoy, R. D. 2017. *The debate should now be over: simplified cardiorespiratory sleep tests are a reliable, cost-saving option for diagnosing obstructive sleep apnea* New York, NY: American Thoracic Society.

Chan, A. S., Lee, R. W. and Cistulli, P. A. 2008. Non-positive airway pressure modalities: mandibular advancement devices/positional therapy. *Proceedings of the American Thoracic Society*, 5 (2): 179-184.

Chang, H. P., Chen, Y. F. and Du, J. K. 2020. Obstructive sleep apnea treatment in adults. *The Kaohsiung journal of medical sciences*, 36 (1): 7-12.

Chang, J.-W. R., Akemokwe, F. M., Marangu, D. M., Chisunkha, B., Irekpita, E., Obasikene, G., Kagima, J. W. and Obonyo, C. O. 2020. Obstructive sleep apnea awareness among primary care physicians in Africa. *Annals of the American Thoracic Society*, 17 (1): 98-106.

Cheyne, J. A. 2006. Sleep paralysis, state transition disruptions, and narcolepsy. In: *Narcolepsy and hypersomnia*. CRC Press, 147-156.

Chokroverty, S. 2010. Overview of sleep & sleep disorders. *Indian Journal of Medical Research*, 131 (2): 126-140.

Chou, K. T., Chang, Y. T., Chen, Y. M., Su, K. C., Perng, D. W., Chang, S. C. and Shiao, G. M. 2011. The minimum period of polysomnography required to confirm a diagnosis of severe obstructive sleep apnoea. *Respirology*, 16 (7): 1096-1102.

Christensson, E., Franklin, K. A., Sahlin, C., Palm, A., Ulfberg, J., Eriksson, L. I., Lindberg, E., Hagel, E. and Fagerlund, M. J. 2018. Can STOP-Bang and pulse oximetry detect and exclude obstructive sleep apnea? *Anesthesia & Analgesia*, 127 (3): 736-743.

Chung, F., Yang, Y. and Liao, P. 2013. Predictive performance of the STOP-Bang score for identifying obstructive sleep apnea in obese patients. *Obesity surgery*, 23: 2050-2057.

Chung, F., Yang, Y., Brown, R. and Liao, P. 2014. Alternative scoring models of STOP-bang questionnaire improve specificity to detect undiagnosed obstructive sleep apnea. *Journal of clinical sleep medicine*, 10 (9): 951-958.

Chung, F., Abdullah, H. R. and Liao, P. 2016. STOP-Bang questionnaire: a practical approach to screen for obstructive sleep apnea. *Chest*, 149 (3): 631-638.

Cirignotta, F., Mondini, S., Zucconi, M., Gerardi, R., Farolfi, A. and Lugaresi, E. 1988. Zolpidem-polysomnographic study of the effect of a new hypnotic drug in sleep apnea syndrome. *Pharmacology Biochemistry and Behavior*, 29 (4): 807-809.

Copinschi, G. 2005. Metabolic and endocrine effects of sleep deprivation. *Essential Psychopharmacology*, 6 (6): 341-347.

Dauvilliers, Y., Arnulf, I. and Mignot, E. 2007. Narcolepsy with cataplexy. *The Lancet*, 369 (9560): 499-511.

de los Reyes, G. 2014. *Sleep study interpretation*. Available: https://www.apsresp.org/pdf/esap/esap-201408-lectures/gp-1-3.pdf (Accessed 2020 06 07).

Deak, M. C. and Kirsch, D. B. 2014. Sleep-disordered breathing in neurologic conditions. *Clinics in Chest Medicine*, 35 (3): 547-556.

Dingman, M. 2017. Know your brain: association areas. Available: https://neuroscientificallychallenged.com/ (Accessed 2020 10 13).

Duarte, R. L., Magalhães-da-Silveira, F. J., Oliveira-e-Sa, T. S., Rabahi, M. F., Mello, F. C. and Gozal, D. 2019. Predicting obstructive sleep apnea in patients with insomnia: a comparative study with four screening instruments. *Lung*, 197 (4): 451-458.

Duckrow, R. and Zaveri, H. 2005. Coherence of the electroencephalogram during the first sleep cycle. *Clinical Neurophysiology*, 116 (5): 1088-1095.

Edwards, B. A., Wellman, A., Sands, S. A., Owens, R. L., Eckert, D. J., White, D. P. and Malhotra, A. 2014. Obstructive sleep apnea in older adults is a distinctly different physiological phenotype. *Sleep*, 37 (7): 1227-1236.

Epstein, L., Kristo, D., Strollo Jr, P., Friedman, N., Malhotra, A., Patil, S., Ramar, K., Rogers, R., Schwab, R. and Weaver, E. 2009. Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *Journal of Clinical Sleep Medicine*, 5 (3): 263-276.

Fietze, I., Laharnar, N., Obst, A., Ewert, R., Felix, S. B., Garcia, C., Gläser, S., Glos, M., Schmidt, C. O. and Stubbe, B. 2019. Prevalence and association analysis of obstructive sleep apnea with gender and age differences–Results of SHIP-Trend. *Journal of Sleep Research*, 28 (5): e12770.

Foldvary-Schaefer, N., Grigg-Damberger, M. and Mehra, R. 2019. *Sleep disorders: a case a week from the Cleveland Clinic*. Oxford: Oxford University Press.

Gami, A. S., Howard, D. E., Olson, E. J. and Somers, V. K. 2005. Day–night pattern of sudden death in obstructive sleep apnea. *New England Journal of Medicine*, 352 (12): 1206-1214.

Garvey, J. F., Pengo, M. F., Drakatos, P. and Kent, B. D. 2015. Epidemiological aspects of obstructive sleep apnea. *Journal of Thoracic Disease*, 7 (5): 920.

Geyer, J. D., Talathi, S. and Carney, P. R. 2009. *Introduction to sleep and polysomnography. Reading EEGs: a practical approach.* Philadelphia Lippincott Williams & Wilkins. ,

Giles, T. L., Lasserson, T. J., Smith, B., White, J., Wright, J. J. and Cates, C. J. 2006. Continuous positive airways pressure for obstructive sleep apnoea in adults. *Cochrane Database of Systematic Reviews*, (1)

Gildner, T. E., Liebert, M. A., Kowal, P., Chatterji, S. and Josh Snodgrass, J. 2014. Sleep duration, sleep quality, and obesity risk among older adults from six middle-income countries: Findings from the study on global ageing and adult health (SAGE). *American Journal of Human Biology*, 26 (6): 803-812.

Glenn, C. 2012. Technology of polysomnography. In: Yamada, T. and Meng, E. eds. *Practical guide for clinical neurophysiologic testing: EEG.* Philadelphia: Lippincott Williams & Wilkins, 220-245.

Gottlieb, D. J., Punjabi, N. M., Mehra, R., Patel, S. R., Quan, S. F., Babineau, D. C., Tracy, R. P., Rueschman, M., Blumenthal, R. S. and Lewis, E. F. 2014. CPAP versus oxygen in obstructive sleep apnea. *New England Journal of Medicine*, 370 (24): 2276-2285.

Grainger, J. and Drake-Lee, A. 2006. Montelukast in allergic rhinitis: a systematic review and meta-analysis. *Clinical Otolaryngology*, 31 (5): 360-367.

Gregório, M. G., Jacomelli, M., Inoue, D., Genta, P. R., de Figueiredo, A. C. and Lorenzi-Filho, G. 2011. Comparison of full versus short induced-sleep polysomnography for the diagnosis of sleep apnea. *The Laryngoscope*, 121 (5): 1098-1103.

Halbower, A. C. 2020. *Continuous positive airway pressure (CPAP) for pediatric obstructive sleep apnea.* Waltham, MA: UpToDate.

Harris, S. F., Monderer, R. S. and Thorpy, M. 2012. Hypersomnias of central origin. *Neurologic Clinics*, 30 (4): 1027.

Ho, V., Crainiceanu, C. M., Punjabi, N. M., Redline, S. and Gottlieb, D. J. 2015. Calibration model for apnea-hypopnea indices: impact of alternative criteria for hypopneas. *Sleep*, 38 (12): 1887-1892.

Ip, M. S., Lam, B., Ng, M. M., Lam, W. K., Tsang, K. W. and Lam, K. S. 2002. Obstructive sleep apnea is independently associated with insulin resistance. *American Journal of Respiratory and Critical Care Medicine*, 165 (5): 670-676.

Izac, M. S. M. 2006. Basic anatomy and physiology of sleep. *American Journal of Electroneurodiagnostic Technology*, 46 (1): 18-38.

Jazi, S. M. H., Barati, B. and Kheradmand, A. 2011. Treatment of adenotonsillar hypertrophy: a prospective randomized trial comparing azithromycin vs. fluticasone. *Journal of Research in Medical Sciences*, 16 (12): 1590.

Jehan, S., Zizi, F., Pandi-Perumal, S. R., Wall, S., Auguste, E., Myers, A. K., Jean-Louis, G. and McFarlane, S. I. 2017. Obstructive sleep apnea and obesity: implications for public health. *Sleep medicine and disorders: international journal*, 1 (4)

JI, A. U., Gastan, M. and Landa, U. 2001. Nap polysomnography: sufficient grounds for initiating CPAP treatment? *Archivos de Bronconeumologia*, 37 (8): 302-306.

Joosten, S. A., Hamilton, G. S. and Naughton, M. T. 2017. Impact of weight loss management in OSA. *Chest*, 152 (1): 194-203.

Jullian-Desayes, I., Revol, B., Chareyre, E., Camus, P., Villier, C., Borel, J. C., Pepin, J. L. and Joyeux-Faure, M. 2017. Impact of concomitant medications on obstructive sleep apnoea. *British journal of clinical pharmacology*, 83 (4): 688-708.

Kahlke, P. E., Witmans, M. B., Alabdoulsalam, T., Young, R., MacLean, J. E. and Mandhane, P. J. 2013. Full-night versus 4 h evening polysomnography in children less than 2 years of age. *Sleep Medicine*, 14 (2): 177-182.

Kapur, V. K., Auckley, D. H., Chowdhuri, S., Kuhlmann, D. C., Mehra, R., Ramar, K. and Harrod, C. G. 2017. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American Academy of Sleep Medicine clinical practice guideline. *Journal of Clinical Sleep Medicine*, 13 (03): 479-504.

Kaw, R., Chung, F., Pasupuleti, V., Mehta, J., Gay, P. and Hernández, A. V. 2012. Meta-analysis of the association between obstructive sleep apnoea and postoperative outcome. *British Journal of Anaesthesia*, 109 (6): 897-906.

Kawaguchi, Y., Fukumoto, S., Inaba, M., Koyama, H., Shoji, T., Shoji, S. and Nishizawa, Y. 2011. Different impacts of neck circumference and visceral obesity on the severity of obstructive sleep apnea syndrome. *Obesity*, 19 (2): 276-282.

Keenan, S. A. 2005. An overview of polysomnography. *Handbook of Clinical Neurophysiology*. Available: https://doi.org/10.1016/S1567-4231(09)70028-0 (Accessed 2022 12 01)

Kee, K., Dixon, J., Shaw, J., Vulikh, E., Schlaich, M., Kaye, D. M., Zimmet, P. and Naughton, M. T. 2018. Comparison of commonly used questionnaires to identify obstructive sleep apnea in a high-risk population. Journal of clinical sleep medicine, 14 (12): 2057-2064.

Kenny, P. P., Christine, G. G. and David, J. T. 2007. A comparison of polysomnography and the WatchPAT in the diagnosis of obstructive sleep apnea. *Otolaryngology—Head and Neck Surgery*, 137 (4): 665-668.

Khalid, F., Ayache, M. and Auckley, D. 2021. The differential impact of respiratory event scoring criteria on CPAP eligibility in women and men. *Journal of Clinical Sleep Medicine*, 17 (12): 2409-2414.

Khawaja, I. S., Olson, E. J., Van Der Walt, C., Bukartyk, J., Somers, V., Dierkhising, R. and Morgenthaler, T. I. 2010. Diagnostic accuracy of split-night polysomnograms. *Journal of Clinical Sleep Medicine*, 6 (04): 357-362.

Kheirandish, L., Goldbart, A. D. and Gozal, D. 2006. Intranasal steroids and oral leukotriene modifier therapy in residual sleep-disordered breathing after tonsillectomy and adenoidectomy in children. *Pediatrics*, 117 (1): e61-e66.

Kim, M. K., Lee, W.-Y., Kang, J.-H., Kang, J.-H., Kim, B. T., Kim, S. M., Kim, E. M., Suh, S.-H., Shin, H. J. and Lee, K. R. 2014. 2014 clinical practice guidelines for overweight and obesity in Korea. *Endocrinology and Metabolism*, 29 (4): 405-409.

Kinnear, P. and Gray, C. 2006. *SPSS 12 made simple*. London: Psychology Press.

Klabunde, R. 2011. *Cardiovascular physiology concepts*. Philadelphia, PA: Lippincott Williams & Wilkins.

Kleinstreuer, C. and Zhang, Z. 2010. Airflow and particle transport in the human respiratory system. *Annual Review of Fluid Mechanics*, 42: 301-334.

Koehler, U., Becker, H. F., Grimm, W., Heitmann, J., Peter, J. H. and Schäfer, H. 2000. Relations among hypoxemia, sleep stage, and bradyarrhythmia during obstructive sleep apnea. *American Heart Journal*, 139 (1): 142-148.

Kovacevic-Ristanovic, R. and Kuzniar, T. 2014. Sleep disorders. In: Weiner, W. J., Goetz, C. G., Shin, R. K., & Lewis, S. L. eds. *Neurology for the non-neurologist.* . Philadelphia, PA: Lippincott Williams & Wilkins, 156-191.

Krimsky, W. R. and Leiter, J. C. 2005. Physiology of breathing and respiratory control during sleep. In: Proceedings of *Seminars in respiratory and critical care medicine*. New York, NY: Thieme Medical Publishers, 5-12.

Kryger, M. H. and Malhotra, A. 2019. *Management of obstructive sleep apnea in adults* Waltham, MA: UpToDate.

Kryger, M. H., Roth, T. and Dement, W. C. 2017. *Principles and practice of sleep medicine*. Amsterdam: Elsevier.

Kumar, V. M. 2008. Sleep and sleep disorders. *Indian Journal of Chest Diseases and Allied Sciences*, 50 (1): 129.

Kumar, V. M. 2013. Physiology of normal sleep: from young to old. *Ann Natl Acad Med Sci (India)*, 49 (3&4): 81-91.

Kushida, C., Chediak, A., Berry, R., Brown, L., Gozal, D., Iber, C., Parthasarathy, S., Quan, S. and Rowley, J. 2008. Positive Airway Pressure Titration Task Force; American Academy of Sleep Medicine. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. *Journal of Clinical Sleep Medicine*, 4 (2): 157-171.

Kushida, C. A., Nichols, D. A., Holmes, T. H., Miller, R., Griffin, K., Cardell, C.-Y., Hyde, P. R., Cohen, E., Manber, R. and Walsh, J. K. 2015. SMART DOCS: a new patient-centered outcomes and coordinated-care management approach for the future practice of sleep medicine. *Sleep*, 38 (2): 315-326.

Lamberg, L. 2006. Sleeping poorly while pregnant may not be "normal". *JAMA*, 295 (12): 1357-1361.

Lavoie, C. J., Zeidler, M. R. and Martin, J. L. 2018. Sleep and aging. *Sleep Science and Practice*, 2 (1): 3.

Leung, R. S. and Douglas Bradley, T. 2001. Sleep apnea and cardiovascular disease. *American Journal of Respiratory and Critical Care Medicine*, 164 (12): 2147-2165.

Lévy, P., Pépin, J., Arnaud, C., Tamisier, R., Borel, J., Dematteis, M., Godin-Ribuot, D. and Ribuot, C. 2008. Intermittent hypoxia and sleep-disordered breathing: current concepts and perspectives. *European Respiratory Journal*, 32 (4): 1082-1095.

Liming, B. J., Ryan, M., Mack, D., Ahmad, I. and Camacho, M. 2019. Montelukast and nasal corticosteroids to treat pediatric obstructive sleep apnea: a systematic review and meta-analysis. *Otolaryngology–Head and Neck Surgery*, 160 (4): 594-602.

Lin, C. M., Davidson, T. M. and Ancoli-Israel, S. 2008. Gender differences in obstructive sleep apnea and treatment implications. *Sleep Medicine Reviews*, 12 (6): 481-496.

Lloberes, P., Rodríguez, B., Roca, A., Sagales, M. T., de la Calzada, M. D., Giménez, S., Romero, O. and Sampol, G. 2004. Comparison of conventional nighttime with automatic or manual daytime CPAP titration in unselected sleep apnea patients: study of the usefulness of daytime titration studies. *Respiratory Medicine*, 98 (7): 619-625.

Loft, M. and Cameron, L. 2014. The importance of sleep: relationships between sleep quality and work demands, the prioritization of sleep and pre-sleep arousal in day-time employees. *Work & Stress*, 28 (3): 289-304.

Liou, G.-Y. and Storz, P. 2010. Reactive oxygen species in cancer. *Free radical research*, 44 (5): 479-496.

Machado-Júnior, A.-J., Zancanella, E. and Crespo, A.-N. 2016. Rapid maxillary expansion and obstructive sleep apnea: a review and meta-analysis. *Medicina Oral, Patologia Oral y Cirugia Bucal*, 21 (4): e465.

MacKay, S. G. and Chan, L. 2016. Surgical approaches to obstructive sleep apnea. *Sleep Medicine Clinics*, 11 (3): 331-341.

Mannarino, M. R., Di Filippo, F. and Pirro, M. 2012. Obstructive sleep apnea syndrome. *EuropeanJjournal of Internal Medicine*, 23 (7): 586-593.

Mansfield, D. R., Antic, N. A. and McEvoy, R. D. 2013. How to assess, diagnose, refer and treat adult obstructive sleep apnoea: a commentary on the choices. *Medical Journal of Australia*, 199: S21-S26.

Mason, M., Cates, C. J. and Smith, I. 2015. Effects of opioid, hypnotic and sedating medications on sleep-disordered breathing in adults with obstructive sleep apnoea. *Cochrane Database of Systematic Reviews*, (7)

Marcus, C. L., Carroll, J. L., Bamford, O., Pyzik, P. and Loughlin, G. M. 1995. Supplemental oxygen during sleep in children with sleep-disordered breathing. *American Journal of Respiratory and Critical Care Medicine*, 152 (4): 1297-1301.

Marcus, C. L., Chapman, D., Ward, S. D., McColley, S. A., Herrerias, C. T., Stillwell, P. C., Howenstine, M., Light, M. J., McColley, S. A., Schaeffer, D. A., Wagener, J. S. and Laskosz, L. N. 2002. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*, 109 (4): 704-712.

Martinho, F. L., Tangerina, R. d. P., Moura, S., Gregório, L. C., Tufik, S. and Bittencourt, L. R. A. 2008. Systematic head and neck physical examination as a predictor of obstructive sleep apnea in class III obese patients. *Brazilian Journal of Medical and Biological Research*, 41: 1093-1097.

Matheson, J. K., Singh, R. and Packard, A. 2007. Polysomnography and sleep disorders. In: *The Clinical Neurophysiology Primer*. Berlin: Springer, 393-445.

McKeown, P., O'Connor-Reina, C. and Plaza, G. 2021. Breathing re-education and phenotypes of sleep apnea: A review. *Journal of Clinical Medicine*, 10 (3): 471.

Mbena, B. P. 2022. Assessing Knowledge of Obstructive Sleep Apnoea among 4th Year and 6th Year Medical Students, Internal Medicine Registrars and Medical Consultants at the University of Cape Town.

McNicholas, W. T. 2008. Diagnosis of obstructive sleep apnea in adults. *Proceedings of the American Thoracic Society*, 5 (2): 154-160.

Medic, G., Wille, M. and Hemels, M. E. 2017. Short-and long-term health consequences of sleep disruption. *Nature and Science of Sleep*, 9: 151.

Medicine, A. A. o. S. 2005. International Classification of Sleep Disorders–Second Edition (ICSD-2), 298 pages. *ISBN*, 978: 0965722025.

Mergen, H., Altındağ, B., Uçar, Z. Z. and Kılıçaslan, I. K. 2019. The predictive performance of the STOP-Bang questionnaire in obstructive sleep apnea screening of obese population at sleep clinical setting. *Cureus*, 11 (12)

Mitchell, R. B. 2007. Adenotonsillectomy for obstructive sleep apnea in children: outcome evaluated by pre-and postoperative polysomnography. *The Laryngoscope*, 117 (10): 1844-1854.

Mogavero, M. P., Mezzapesa, D. M., Savarese, M., DelRosso, L. M., Lanza, G. and Ferri, R. 2021. Morphological analysis of the brain subcortical gray structures in restless legs syndrome. *Sleep Medicine*, 88: 74-80.

Morrell, M. J., Finn, L., Kim, H., Peppard, P. E., Safwan Badr, M. and Young, T. 2000. Sleep fragmentation, awake blood pressure, and sleep-disordered breathing in a population-based study. *American Journal of Respiratory and Critical Care Medicine*, 162 (6): 2091-2096.

Morin, A. K., Jarvis, C. I. and Lynch, A. M. 2007. Therapeutic options for sleep-maintenance and sleep-onset insomnia. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, 27 (1): 89-110.

Naidoo, K. 2015. Occurrence of obesity and risk of obstructive sleep apnoea syndrome in adult patients presenting for elective surgery at an academic hospital.

Nannapaneni, S., Ramar, K. and Surani, S. 2013. Effect of obstructive sleep apnea on type 2 diabetes mellitus: a comprehensive literature review. *World Journal of Diabetes*, 4 (6): 238.

Nixon, G. M. and Brouillette, R. T. 2002. Diagnostic techniques for obstructive sleep apnoea: is polysomnography necessary? *Paediatric respiratory reviews*, 3 (1): 18-24.

Norra, C., Richter, N. and Juckel, G. 2011. Sleep disturbances and suicidality: a common association to look for in clinical practise and preventive care. *EPMA Journal*, 2 (3): 295-307.

Ordones, A. B., Grad, G. F., Cahali, M. B., Lorenzi-Filho, G., Sennes, L. U. and Genta, P. R. 2020. Comparison of upper airway obstruction during zolpidem-induced sleep and propofol-induced sleep in patients with obstructive sleep apnea: a pilot study. *Journal of clinical sleep medicine*, 16 (5): 725-732.

Öztürk, N. A. A., Dilektaşlı, A. G., Çetinoğlu, E. D., Ursavaş, A. and Karadağ, M. 2019. Diagnostic accuracy of a modified STOP-BANG questionnaire with national anthropometric obesity indexes. *Turkish Thoracic Journal*, 20 (2): 103.

Ozoh, O. B., Okubadejo, N. U., Akinkugbe, A. O., Ojo, O. O., Asoegwu, C. N., Amadi, C., Odeniyi, I. and Mbakwem, A. C. 2014. Prospective assessment of the risk of obstructive sleep apnea in patients attending a tertiary health facility in Sub-Saharan Africa. *Pan African Medical Journal*, 17 (1)

Pahkala, R., Seppä, J., Ikonen, A., Smirnov, G. and Tuomilehto, H. 2014. The impact of pharyngeal fat tissue on the pathogenesis of obstructive sleep apnea. *Sleep and Breathing*, 18 (2): 275-282.

Pandi-Perumal, S., Zisapel, N., Srinivasan, V. and Cardinali, D. 2005. Melatonin and sleep in aging population. *Experimental Gerontology*, 40 (12): 911-925.

Parrino, L., Grassi, A. and Milioli, G. 2014. Cyclic alternating pattern in polysomnography: what is it and what does it mean? *Current Opinion in Pulmonary Medicine*, 20 (6): 533-541.

Park, S. Y. and Helman, S. N. 2015. The Nose and Sleep Disorders. *Sataloff's comprehensive textbook of otolaryngology: head & neck surgery: rhinology/allergy and immunology*, 2: 285. New Delhi: Jaypee Brothers Medical Publishers

Paruthi, S. 2015a. Common causes, effects, and solutions to sleep deprivation in children. Available: https://sleepreviewmag.com/sleep-health/demographics/age/common-causes-effects-solutions-sleep-deprivation-children/ (Accessed 2022 09 04).

Paruthi, S. 2015b. Management of obstructive sleep apnea in children. *UptoDate.* March 31, 2015.

Paruthi, S., Chervin, R. D. and Hoppin, A. G. 2017. Evaluation of suspected obstructive sleep apnea in children. Available: *Uptodate. com* (Accessed 2022 09 04).

Patel, A. K. and Araujo, J. F. 2018. Physiology, sleep stages. In: *StatPearls [Internet]*. Treasure Island, FL: StatPearls Publishing.

Patil, S. P., Schneider, H., Schwartz, A. R. and Smith, P. L. 2007. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*, 132 (1): 325-337.

Pavlova, M. K. and Latreille, V. 2019. Sleep disorders. *The American Journal of Medicine*, 132 (3): 292-299.

Penzel, T., Möller, M., Becker, H. F., Knaack, L. and Peter, J.-H. 2001. Effect of sleep position and sleep stage on the collapsibility of the upper airways in patients with sleep apnea. *Sleep*, 24 (1): 90-95.

Peppard, P. E., Young, T., Palta, M. and Skatrud, J. 2000. Prospective study of the association between sleep-disordered breathing and hypertension. *New England Journal of Medicine*, 342 (19): 1378-1384.

Pevernagie, D., Sastry, M. and Vanderveken, O. 2014. Treatment of respiratory sleep disorders. In: *Sleep medicine textbook*. European Sleep Research Society (ESRS), 259-274.

Pirelli, P., Saponara, M. and Guilleminault, C. 2015. Rapid maxillary expansion (RME) for pediatric obstructive sleep apnea: a 12-year follow-up. *Sleep Medicine*, 16 (8): 933-935.

Pierre ESCOURROU, S. L. and Marlène REHEL, H. N. 2000. Needs and costs of sleep monitoring. *European Neurological Network: ENN*, 78: 69.

Pizzino, G., Irrera, N., Cucinotta, M., Pallio, G., Mannino, F., Arcoraci, V., Squadrito, F., Altavilla, D. and Bitto, A. 2017. Oxidative stress: harms and benefits for human health. *Oxidative medicine and cellular longevity*, 2017

Qian, X., Qiu, Y., He, Q., Lu, Y., Lin, H., Xu, F., Zhu, F., Liu, Z., Li, X. and Cao, Y. 2021. A review of methods for sleep arousal detection using polysomnographic signals. *Brain sciences*, 11 (10): 1274.

Radtke, R. A. 1990. Sleep disorders: laboratory evaluation. In: *Current practice of clinical electroencephalography*. Raven Press New York, 561-592.

Ramachandran, S. K. and Josephs, L. A. 2009. A meta-analysis of clinical screening tests for obstructive sleep apnea. *Anesthesiology*, 110 (4): 928-939.

Räsänen, J., Heikkilä, J., Downs, J., Nikki, P., Väisänen, I. and Viitanen, A. 1985. Continuous positive airway pressure by face mask in acute cardiogenic pulmonary edema. *The American Journal of Cardiology*, 55 (4): 296-300.

Ren, R., Li, Y., Zhang, J., Zhou, J., Sun, Y., Tan, L., Li, T., Wing, Y.-K. and Tang, X. 2016. Obstructive sleep apnea with objective daytime sleepiness is associated with hypertension. *Hypertension*, 68 (5): 1264-1270.

Redline, S., Kirchner, H. L., Quan, S. F., Gottlieb, D. J., Kapur, V. and Newman, A. 2004. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Archives of Internal Medicine*, 164 (4): 406-418.

Richard, W., Kox, D., den Herder, C., Laman, M., van Tinteren, H. and de Vries, N. 2006. The role of sleep position in obstructive sleep apnea syndrome. *European Archives of Oto-Rhino-Laryngology and Head & Neck*, 263 (10): 946-950.

Roche, J., Rae, D. E., Redman, K. N., Knutson, K. L., Von Schantz, M., Gómez-Olivé, F. X. and Scheuermaier, K. 2021. Impact of obstructive sleep apnea on cardiometabolic health in a random sample of older adults in rural South Africa: building the case for the treatment of sleep disorders in underresourced settings. *Journal of clinical sleep medicine*, 17 (7): 1423-1434.

Romero-Corral, A., Caples, S. M., Lopez-Jimenez, F. and Somers, V. K. 2010. Interactions between obesity and obstructive sleep apnea: implications for treatment. *Chest*, 137 (3): 711-719.

Rosenberg, R. 2010. *A technologist's introduction to sleep disoders*. Westchester, NY: American Academy of Sleep Medicine.

Rosenberg, R. S. and Van Hout, S. 2014. The American Academy of Sleep Medicine inter-scorer reliability program: respiratory events. *Journal of Clinical Sleep Medicine*, 10 (4): 447-454.

Rosen, I. M., Kirsch, D. B., Carden, K. A., Malhotra, R. K., Ramar, K., Aurora, R. N., Kristo, D. A., Martin, J. L., Olson, E. J. and Rosen, C. L. 2018. Clinical use of a home sleep apnea test: an updated American Academy of Sleep Medicine position statement. *Journal of clinical sleep medicine*, 14 (12): 2075-2077.

Rossi, A., Lo Giudice, A., Di Pardo, C., Valentini, A. T., Marradi, F., Vanacore, N. and Grippaudo, C. 2021. Clinical evidence in the treatment of obstructive sleep apnoea with oral appliances: a systematic review. *International Journal of Dentistry*, 2021

Roux, F., D'Ambrosio, C. and Mohsenin, V. 2000. Sleep-related breathing disorders and cardiovascular disease. *The American Journal of Medicine*, 108 (5): 396-402.

Ryan, C. M. and Bradley, T. D. 2005. Pathogenesis of obstructive sleep apnea. *Journal of Applied Physiology*, 99 (6): 2440-2450.

Sa, J., Choe, S., Cho, B.-y., Chaput, J.-P., Kim, G., Park, C.-H., Chung, J., Choi, Y., Nelson, B. and Kim, Y. 2020. Relationship between sleep and obesity among US and South Korean college students. *BMC Public Health*, 20: 1-11.

Salkind, N. J. and Frey, B. B. 2019. *Statistics for people who (think they) hate statistics*. Thousand Oaks, CA: Sage.

Sateia, M. J. 2014. International classification of sleep disorders. *Chest*, 146 (5): 1387-1394.

Schwartz, M., Acosta, L., Hung, Y.-L., Padilla, M. and Enciso, R. 2018. Effects of CPAP and mandibular advancement device treatment in obstructive sleep apnea patients: a systematic review and meta-analysis. *Sleep and Breathing*, 22 (3): 555-568.

Senaratna, C. V., Perret, J. L., Lodge, C. J., Lowe, A. J., Campbell, B. E., Matheson, M. C., Hamilton, G. S. and Dharmage, S. C. 2017. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Medicine Reviews*, 34: 70-81.

Sharma, S. and Srijithesh, P. 2013. Sleeping over a sleep disorder - awareness of obstructive sleep apnoea as a modifiable risk factor for hypertension and stroke: A survey among health care professionals and medical students. *Annals of Indian Academy of Neurology*, 16 (2): 151.

Shredl, B. P. 2021. Increasing identification of risk for obstructive sleep apnea in adults with obesity by utilizing the STOP-Bang questionnaire in primary care. Doctoral thesis, University of Missouri--Kansas City.

Shrivastava, D., Jung, S., Saadat, M., Sirohi, R. and Crewson, K. 2014. How to interpret the results of a sleep study. *Journal of Community Hospital Internal Medicine Perspectives*, 4 (5): 24983.

Singh, M., Liao, P., Kobah, S., Wijeysundera, D., Shapiro, C. and Chung, F. 2013. Proportion of surgical patients with undiagnosed obstructive sleep apnoea. *British Journal of Anaesthesia*, 110 (4): 629-636.

Soorujlall, K., Prakaschandra , R., Mdluli, D.,Kibi-Makusha, Y. 2021. Dianostic accuracy of screening for sleep disorders using sleep questionnaires in correlation with polysomnography study. Available: *Durban university of technology* (Accessed 2022 09 15).

Subramani, Y., Singh, M., Wong, J., Kushida, C. A., Malhotra, A. and Chung, F. 2017. Understanding phenotypes of obstructive sleep apnea: applications in anesthesia, surgery, and perioperative medicine. *Anesthesia and Analgesia*, 124 (1): 179.

Suzuki, M., Saigusa, H., Chiba, S., Yagi, T., Shibasaki, K., Hayashi, M., Suzuki, M., Moriyama, K. and Kodera, K. 2005. Discrepancy in polysomnography scoring for a patient with obstructive sleep apnea hypopnea syndrome. *The Tohoku Journal of Experimental Medicine*, 206 (4): 353-360.

Taylor, D. J., Lichstein, K. L. and Durrence, H. H. 2003. Insomnia as a health risk factor. *Behavioral Sleep Medicine*, 1 (4): 227-247.

Van den Berg, A., Montoya-Pelaez, L., Halliday, E., Hassan, I. and Baloch, M. 1999. Analgesia for adenotonsillectomy in children and young adults: a comparison of tramadol, pethidine and nalbuphine. *European Journal of Anaesthesiology*, 16 (3): 186-194.

Victor, M., Ropper, A. H. and Adams, R. D. 2001. *Principles of Neurology*. New York, NY: McGraw-Hill .

Vgontzas, A. N., Bixler, E. O. and Chrousos, G. P. 2005. Sleep apnea is a manifestation of the metabolic syndrome. *Sleep medicine reviews*, 9 (3): 211-224.

Walker, M. and Stickgold, R. 2009. Sleep disorders medicine; basic science, technical consideration, and clinical aspects. (Available: https://link.springer.com/book/10.1007/978-1-4939-6578-6 (Accessed 2020 06 05)

Wang, Q., Zhang, C., Jia, P., Zhang, J., Feng, L., Wei, S., Luo, Y., Su, L., Zhao, C. and Dong, H. 2014. The association between the phenotype of excessive daytime sleepiness and blood pressure in patients with obstructive sleep apnea-hypopnea syndrome. *International journal of medical sciences*, 11 (7): 713.

Watson, N. F. 2016. Health care savings: the economic value of diagnostic and therapeutic care for obstructive sleep apnea. *Journal of Clinical Sleep Medicine*, 12 (8): 1075-1077.

Whitmore, H. 2012. Adult Sleep Scoring. In: Mattice, C., Brooks, R., Lee-Chiong, T. eds.*Fundamentals of Sleep Technology*. Philadelphia, PA: Wolters Kluwer Health, 360.

White, D. P. 2006. Sleep apnea. *Proceedings of the American Thoracic Society*, 3 (1): 124-128.

Whitten, T. A., Hughes, A. M., Dickson, C. T. and Caplan, J. B. 2011. A better oscillation detection method robustly extracts EEG rhythms across brain state changes: the human alpha rhythm as a test case. *Neuroimage*, 54 (2): 860-874.

Wills, L. and Garcia, J. 2002. Parasomnias. *CNS Drugs*, 16 (12): 803-810.

Worley, S. L. 2018. The extraordinary importance of sleep: the detrimental effects of inadequate sleep on health and public safety drive an explosion of sleep research. *Pharmacy and Therapeutics*, 43 (12): 758.

Xu, M., Chung, S., Zhang, S., Zhong, P., Ma, C., Chang, W.-C., Weissbourd, B., Sakai, N., Luo, L. and Nishino, S. 2015. Basal forebrain circuit for sleep-wake control. *Nature Neuroscience*, 18 (11): 1641-1647.

Yamada, T. and Meng, E. 2012. *Practical guide for clinical neurophysiologic testing: EEG*. Phladelhia, PA: Lippincott Williams & Wilkins.

Yan, L., Park, H. R., Kezirian, E. J., Yook, S., Kim, J.-H., Joo, E. Y. and Kim, H. 2021. Altered regional cerebral blood flow in obstructive sleep apnea is associated with sleep fragmentation and oxygen desaturation. *Journal of Cerebral Blood Flow & Metabolism*: 41 (10): 2712-2724.

Young, T., Skatrud, J. and Peppard, P. 2004. Risk factors for obstructive sleep apnea in adults. *JAMA*, 291 (16): 2013-2016.

Young, T. J. and Silber, M. H. 2006. Hypersomnias of central origin. *Chest*, 130 (3): 913-920.

Younes, M. 2004. Role of arousals in the pathogenesis of obstructive sleep apnea. *American journal of respiratory and critical care medicine*, 169 (5): 623-633.

Zozula, R. and Rosen, R. 2001. Compliance with continuous positive airway pressure therapy: assessing and improving treatment outcomes. *Current Opinion in Pulmonary Medicine*, 7 (6): 391-398.

Zuberi, N. A., Rekab, K. and Nguyen, H. V. 2004. Sleep apnea avoidance pillow effects on obstructive sleep apnea syndrome and snoring. *Sleep and Breathing*, 8 (4): 201-207.

ANNEXURES

Annexure 1: Approval to conduct research project: Department of Neurophysiology and Respiratory

health
Department:
Health
PROVINCE OF KWAZULU-NATAL

DIRECTORATE: NEUROPHYSIOLOGY

Physical Address 800 Vusi Mzimela Road Mayville 4091
Postal Address: Private Bag X03, Mayville , 4058
Tel. Phone: 031 240 1634 Fax 031 240 1005
Email : yabanathi.kibi @ialch.co.za

Inkosi Albert Luthuli Central Hospital
Department of Neurophysiology

IALCH Management Committee

Inkosi Albert Luthuli Central Hospital

800 Bellair Road

Mayville

Durban

4091

RE: Approval to conduct research project: Department of Neurophysiology and Respiratory

Dear Sir/Madam,

My name is Miss Yabanathi Kibi, I am a student at Durban University of Technology (DUT), and my student number is 20908471. I hereby ask for permission to conduct a prospective research project in sleep studies, in order to obtain a Master of Health Sciences in neurophysiology clinical technology through the Department of Biomedical and Clinical Technology at DUT. The area of research includes patients suspected to have sleep-disordered breathing seen at Inkosi Albert Luthuli Central Hospital. Dr Rosaley Prakaschandra (PhD (Clin Medicine - Cardio)), Mr Dalincebo Mdluli (Masters (Clinical Technology – Neurophysiology)), and Professor Kennedy Nyamande (Head of Respiratory Department) are the supervisors for this research.

Topic: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

This is a non-invasive study comparing the Apnoea-Hypopnoea Index (AHI) between the afternoon nap and overnight Polysomnography (PSG) at Inkosi Albert Luthuli Central Hospital (IALCH), to rate their diagnostic value, and verify that afternoon nap PSG can be fully used as an alternative to overnight PSG. At IALCH only the afternoon nap is used to diagnose sleep apnoea hypopnoea syndrome (SAHS) or sleep-disordered breathing (SDB). There is a controversy that an afternoon nap PSG cannot be fully used as an alternative to overnight PSG, as the latter is the gold standard method for evaluating individuals with sleep disorders, below there is a letter of information summarising the study for the management committee.

Please find attached a copy of the research proposal, together with the ethical clearance approval certificate from the Durban University of Technology (DUT).

Please feel free to contact me or my supervisor should you require additional information regarding my study.

Thank you for your consideration.

Kind regards.

Mrs Yabanathi Kibi
(Research student, 062 480 6187)

Annexure 2: Information letter regarding agreement of recruiting patients for the study

health
Department:
Health
PROVINCE OF KWAZULU-NATAL

DIRECTORATE; NEUROPHYSIOLOGY

Physical Address 800 Vusi Mzimela Road Mayville 4091
Postal Address: Private Bag X03, Mayville , 4058
Tel. Phone: 031 240 1634 Fax 031 240 1005
Email : yabanathi.kibi @ialch.co.za

Inkosi Albert Luthuli Central Hospital
Department of Neurophysiology

INKOSI ALBERT LUTHULI CENTRAL HOSPITAL INFORMATION LETTER REGARDING AGREEMENT OF RECRUITING PATIENTS FOR THE STUDY

Title of the Research Study: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Principal Investigator/s/researcher: Yabanathi Kibi, B-Tech: Neuro-clinical technology

Co-Investigator/s/supervisor/s: Prof Kennedy Nyamande ((MB ChB (UZ), FCP (SA), certificate of Pulmonology (College of Physicians of South Africa), Ph.D. (UKZN)), Dr. Rosaley Prakaschandra (Ph.D. (Clin Medicine - Cardio)), Mr. Dalincebo Mdluli (Masters (Clinical Technology – Neurophysiology)).

Dear Hospital Management Committee

I am Miss Yabanathi Kibi, I am registered at the Durban University of Technology for a Master of Health Sciences in neurophysiology clinical technology. I kindly request approval to conduct a prospective research project in sleep studies. The title of the research study is The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Dr. Rosaley Prakaschandra (Ph.D. (Clin Medicine - Cardio)) and Mr. Dalincebo Mdluli (Masters (Clinical Technology – Neurophysiology)) are the supervisors and Prof Kennedy Nyamande, MB ChB (UZ), FCP (SA), Certificate Pulmonology (SA) Phys, Ph.D. (UKZN)) is the co-supervisor for this study.

This study compares the Apnoea-Hypopnoea Index (AHI) between the afternoon nap and overnight Polysomnography (PSG) at Inkosi Albert Luthuli Central Hospital (IALCH), to rate their diagnostic value, and evaluate if afternoon PSG can be fully used as an alternative to overnight PSG. At IALCH only the afternoon nap is used to diagnose sleep apnoea hypopnoea syndrome (SAHS) or sleep-disordered breathing (SDB).

There is a controversy that an afternoon nap PSG cannot be fully used as an alternative for overnight PSG, as the latter is the gold standard method for evaluating individuals with sleep disorders. In this study, the researcher will investigate whether afternoon nap PSG has similar sensitivity and specificity to overnight PSG in diagnosing sleep apnoea or any sleep-related disorder that may be found within the recruited patients.

This research will provide evidence of whether IALCH apneic patients are under-diagnosed or not and provide recommendations that may be utilised to improve the quality of PSG in KwaZulu-Natal, South Africa.

On doing the procedure, the sleep study is requested by a Physician, then an appointment is set by a Neurophysiology Clinical Technologist to do an afternoon nap PSG and overnight PSG. Attended afternoon and overnight sleep studies are conducted for patients suspected to have sleep-disordered breathing, these patients usually have symptoms of trouble sleeping at night, trouble keeping themselves awake during the day, cessation of breathing at night, snoring, and too many awakenings at night.

On the day of the procedure, PSG patient information sheet will be filled in. The 4 types of sleep questionnaires/scales used together with PSG are used to categorize sleep architecture and sleep-disordered breathing. These include Berlin, Epworth (ESS), Functional Outcome (FOSQ), and Stop-Bang. The total ESS score is based on a scale of 0 to 24, with a score equal to and above 16 considered to be very sleepy and warranting further investigation. The Stop-Bang total score ranges from 0 to 8, with a score less than and equal to 2 considered to be low risk, 3-4 considered the intermediate risk, and a score equal to and above 5 considered to be a high risk of developing sleep apnoea. Berlin questionnaire consists of 3 categories related to the risk of having sleep apnoea. 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. A total score of 2 or more positive categories means a high risk of developing sleep apnoea. FOSQ consists of 5 subscales, to obtain a total score, all subscale scores are added to calculate the mean, mean is then multiplied by 5 regardless of the number of subscale scores used in the computation of the mean. A total score of 5 to 20, indicates bad functional status, with a higher score signifying

better functional status. Total questionnaires score along with PSG testing are highly useful in diagnosing sleep disorders.

The researcher will conduct both the afternoon nap PSG and the overnight PSG studies. Seventeen PSG channels are used, including electro-encephalography for the scalp (Fz-Cz, Cz-Oz, C3-A2, and C4-A1), electro-oculography for the eyes (LOC-reference, ROC-A2, and LOC2-A2), submental and tibialis electromyography (active under the chin and at the belly of the tibial muscle, and reference will be 3 cm lateral to active and 3 cm distal from active, respectively), and electro-cardiography or the heart (RA-LA, RA-LL, and LA-LL).

In conducting the Polysomnography (PSG), Electroencephalography (EEG) is used to measure brain electrical activity, Electromyography (EMG) and snore sensor to measure snoring on the neck under the chin, level of consciousness and restless leg movement with tibialis EMG, and Electrocardiography (ECG) to measure heart rate. The nasal cannula and thermistor for both the nose and the mouth will be used to measure the airflow and breathing levels. Respiratory effort is measured by looking at the respiratory movement of the rib cage and abdomen using an inductive plethysmograph, and percutaneous arterial oxygen saturation using a finger pulse oximeter. Body position is typed in by the Neurophysiology Clinical Technologist scoring the study, looking at an audio-visual low-light camera. Scoring will be done according to an updated American Academy of Sleep Medicine (AASM) version 2.0. Recordings will be stored in a computer database using Nicolet software.

The researcher is further requesting assistance from respiratory ward Doctors, Nurses in the A3E ward, and Neurologists including management in taking care of the patient in the case of any challenges or needs that may arise towards the patient’s health on the night of the study. OPSG is requested to be free of charge as this will be done mainly for the research, although the patients will get both the results, and this is done to improve the health and care of the patient, including the hospital standard.

This study will include a number of 25 adult patients from 18 years to 70 years old with symptoms suggestive of sleep-disordered breathing (SDB). This age group is chosen specifically to minimise anything that may lead to false results. Among other factors, older people often sleep less deeply and wake up more often throughout the night, which may mislead the PSG results. Patients with insomnia will not be participating to prevent inaccuracy assessment of sleep-disordered breathing.

There is no risk of any kind of infection. There is no pain to be expected as there are no needles used. Although, minimal discomfort is to be expected as there will be cleaning of the scalp with Nuprep and ear-bud to remove oil and superficial dead skin. During the removal of electrodes, acetone may be used to remove the ones stuck with medical glue (collodion).

Participants may be withdrawn from the study if they are already using a certain treatment or some appliances for sleep-disordered breathing. Patients that are not comfortable with sleeping at the hospital for one night, patients with heart problems or insomnia.

The proposed study and the results will help Neurophysiology Clinical Technologists know which of the two PSG tests is accurate, specific, and sensitive enough in diagnosing patients with sleep-related disorders, as this is the only public hospital in KwaZulu-Natal conducting sleep studies for patients and would be appropriate to do right by those who cannot afford to go to Private sectors.

The study will also contribute to providing valuable evidence-based information depending on the results from the study. The results of the study will be shared during presentations at conferences, workshops, and seminars. In the case of overnight results being more informative, in regards to changing patients' diagnosis and management, then the sleep study reports will have to be sent to the referring physician with a recommendation for treatment once the dissertation is complete and submitted.

Confidentiality is guaranteed during the study as only the researcher will have access to the study profile. When questions are asked, only the researcher and the patient will be in the test room and every piece of information taken will be kept in a password laptop, and there won't be names used to describe the patients, only codes that will not be able to be traced back to patients' names. There is assurance that there won't be any research-related injury.

Persons to contact in the event of any problems or queries:

Please contact the researcher (062 480 6187), the supervisor (031 373 5291), the co-supervisor (031 240 1344), or the Institutional Research Ethics administrator at 031 373 2375. Complaints can be reported to the Director: of Research and Postgraduate Support Dr. L Linganiso at 031 373 2577 or researchdirector@dut.ac.za.

Annexure 3: Approval to conduct research project

Provincial Health Research Committee

Health Research and Knowledge Management Unit

KwaZulu-Natal Department of Health

Natalia Building

330 Langalibalele Street

Pietermaritzburg

3201

RE: APPROVAL TO CONDUCT RESEARCH PROJECT

Attention: Dr E Lutge
Chairperson of Provincial Health and Research Ethics Committee

I hereby request approval to conduct a prospective research project in sleep studies, to obtain a Master of Health Sciences in neurophysiology clinical technology through the Department of Biomedical and Clinical Technology at the Durban University of Technology (DUT).

The area of research includes patients suspected to have sleep-disordered breathing seen at Inkosi Albert Luthuli Central Hospital. The provisional title is as follows:
The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.
The following attachments include all supporting documents:

1. Research proposal
2. Ethical clearance approval certificate from the Durban University of Technology (DUT).

My supervisor's details are as follows:

- Dr Rosaley Prakaschandra (rosaleypra@dut.ac.za), 031 373 3252.
- Mr Dalincebo Mdluli (dalincebom@dut.ac.za), 061 512 5047.
- Professor Kennedy Nyamande (kennedynyamande@gmail.com), 082 771 9737.

Please feel free to contact the researcher or the supervisor should you have any queries.

Thank you for your consideration.

Kind regards.

Mrs Yabanathi Kibi
(Research student)
062 480 6187

Annexure 4: Patient's letter of information

PATIENT'S LETTER OF INFORMATION

Title of the Research Study: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Principal Investigator/s/researcher: Mrs. Yabanathi Kibi, B-Tech: Neuro-clinical technology

Co-Investigator/s/supervisor/s: Prof Kennedy Nyamande ((MB ChB (UZ), FCP (SA), certificate of Pulmonology (College of Physicians of South Africa), Dr. Rosaley Prakaschandra (Ph.D. (Clin Medicine - Cardio)), Mr. Dalincebo Mdluli (Masters (Clinical Technology – Neurophysiology)).

Brief Introduction and Purpose of the Study:

This study compares the Apnoea-Hypopnoea Index (AHI) between the afternoon nap and overnight Polysomnography (PSG) at Inkosi Albert Luthuli Central Hospital (IALCH), to rate their diagnostic value, and evaluate if afternoon PSG can be fully used as an alternative to overnight PSG. At IALCH only the afternoon nap is used to diagnose sleep apnoea hypopnoea syndrome (SAHS) or sleep-disordered breathing (SDB).

There is a controversy that an afternoon nap PSG cannot be fully used as an alternative for overnight PSG, as the latter is the gold standard method for evaluating individuals with sleep disorders. In this study, the researcher will investigate whether afternoon nap PSG has similar sensitivity and specificity to overnight PSG in diagnosing sleep apnoea or any sleep-related disorder that may be found within the recruited patients.

This research will provide evidence of whether IALCH apneic patients are under-diagnosed or not and provide recommendations that may be utilized to improve the quality of PSG in KwaZulu-Natal, South Africa.

Dear Participant

I am Miss Yabanathi Kibi, I am registered at the Durban University of Technology for a Master of Health Sciences in neurophysiology clinical technology. I kindly request your approval in participating in the research project on sleep studies. In this study, as a participant, you will be required to undergo two PSG studies for the researcher to be able to rate their diagnostic value. You are allowed to ask as many questions as you wish because it is important for you to fully understand the study. You are entitled to discuss the study with your family and friends. Furthermore, you are under no obligation to respond to the researchers' request on the same date of request. For this purpose, a copy of the letter of Information document will be given to you to take home for better understanding. Research is a systematic search or inquiry for generalized new knowledge, and is mainly done to establish facts and reach a new conclusion that will assist the next generation.

Outline of the Procedure:

On doing the procedure, the sleep study is requested by a Physician, then an appointment is set by a Neurophysiology Clinical Technologist to do an afternoon nap PSG and overnight PSG. Attended afternoon and overnight sleep studies are conducted for patients suspected to have sleep-disordered breathing, these patients usually have symptoms of trouble sleeping at night, trouble keeping themselves awake during the day, cessation of breathing at night, snoring, and too many awakenings at night.

On the day of the procedure, PSG patient information sheet will be filled in. The 4 types of sleep questionnaires/scales used together with PSG are used to categorize sleep architecture and sleep-disordered breathing. These include Berlin, Epworth (ESS), Functional Outcome (FOSQ), and Stop-Bang. The total ESS score is based on a scale of 0 to 24, with a score equal to and above 16 considered to be very sleepy and warranting further investigation. The Stop-Bang total score ranges from 0 to 8, with a score less than and equal to 2 considered to be low risk, 3-4 considered the intermediate risk, and a score equal to and above 5 considered to be a high risk of developing sleep apnoea. Berlin questionnaire consists of 3 categories related to the risk of having sleep apnoea. 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. A total score of 2 or more positive categories means a high risk of developing sleep apnoea. FOSQ consists of 5 subscales, to obtain a total score, all subscale scores are added to calculate the mean, mean is then multiplied by 5 regardless of the number of subscale scores used in the computation of the mean. A total score of 5 to 20, indicates bad functional status, with a higher score signifying better functional status. Total questionnaires score along with PSG testing are highly useful in diagnosing sleep disorders.

The researcher will conduct both the afternoon nap PSG and the overnight PSG studies. Seventeen PSG channels are used, including electro-encephalography for the scalp (Fz-Cz, Cz-Oz, C3-A2, and C4-A1), electro-oculography for the eyes (LOC-reference, ROC-A2, and LOC2-A2), submental and tibialis electromyography (active under the chin and at the belly of the tibial muscle, and reference will be 3 cm lateral to active and 3 cm distal from active, respectively), and electro-cardiography or the heart (RA-LA, RA-LL, and LA-LL).

In conducting the Polysomnography (PSG), Electroencephalography (EEG) is used to measure brain electrical activity, Electromyography (EMG) and snore sensor to measure snoring on the neck under the chin, level of consciousness and restless leg movement with tibialis EMG, and Electrocardiography (ECG) to measure heart rate. The nasal cannula and thermistor for both the nose and the mouth will be used to measure the airflow and breathing levels. Respiratory effort is measured by looking at the respiratory movement of the rib cage and abdomen using an inductive plethysmograph, and percutaneous arterial oxygen saturation using a finger pulse oximeter. Body position is typed in by the Neurophysiology Clinical Technologist scoring the study, looking at an audio-visual low-light camera. Scoring will be done according to an updated American Academy of Sleep Medicine (AASM) version 2.0. Recordings will be stored in a computer database using Nicolet software.

OPSG will be free of charge as this will be done mainly for the research, although the patients will get both the results, and this is done to improve the health and care of the patient, including the hospital standard.

This study will include a number of 25 adult patients from 18 years to 70 years old with symptoms suggestive of sleep-disordered breathing (SDB). This age group is chosen specifically to minimise anything that may lead to false results. Among other factors, older people often sleep less deeply and wake up more often throughout the night, which may mislead the PSG results. Patients with insomnia will not be participating to prevent inaccuracy assessment of sleep-disordered breathing.

Risks or Discomforts to the Participant:

There is no risk of any kind of infection. There is no pain to be expected as there are no needles used. Although, minimal discomfort is to be expected as there will be cleaning of the scalp with Nuprep and ear-bud to remove oil and superficial dead skin. During the removal of electrodes, acetone may be used to remove the ones stuck with medical glue (collodion).

Reasons for withdrawal of participation in the study:

Participants may be withdrawn from the study if they are already using a certain treatment or some appliances for sleep-disordered breathing. Patients that are not

comfortable with sleeping at the hospital for one night, patients with heart problems or insomnia. Participation is voluntary, therefore this allows you as a patient to withdraw at any stage preferably before starting the procedure. This means that you are entitled to withdraw from the study at any time you wish to do so and withdrawal will not contradict the appropriate standard care you are to receive.

Benefits:

The proposed study and the results will help Neurophysiology Clinical Technologists know which of the two PSG tests is accurate, specific, and sensitive enough in diagnosing patients with sleep-related disorders, as this is the only public hospital in KwaZulu-Natal conducting sleep studies for patients and would be appropriate to do right by those who cannot afford to go to Private sectors.

The study will also contribute to providing valuable evidence-based information depending on the results from the study. The results of the study will be shared during presentations at conferences, workshops, and seminars. In the case of overnight results being more informative, in regards to changing patients' diagnosis and management, then the sleep study reports will have to be sent to the referring physician with a recommendation for treatment once the dissertation is complete and submitted.

Therefore, in this regard, the benefits will be both directly and indirectly, as a sleep study printout will be given to you for your personal use. Another copy of the results will be uploaded into the system to assist physicians with your health management.

Remuneration:

You will get to have a better understanding of the study and also get to take home a full overnight PSG report that will be printed once the calculations have been completed and the report finalized. This report will further assist your Doctor with your health management as it will be saved on the system.

Cost of the Study:

You will not be expected to contribute any money towards this research as the overnight PSG will be done on the same date as your routine afternoon nap PSG study.

Confidentiality:

Confidentiality is guaranteed during the study as only the researcher will have access to the study profile. When questions are asked, only the researcher and the patient will be in the test room and every piece of information taken will be kept in a password laptop, and there won't be names used to describe the patients, only codes that will

not be able to be traced back to patients' names. There is assurance that there won't be any research-related injury.

Results:

The results of the study will be shared with other Neurophysiology Clinical Technologists and Doctors in IALCH during presentations as this will be open for discussion in order to know which of the two PSG tests is accurate, specific, and sensitive enough in diagnosing patients with sleep-related disorders, or if the two methods have similar accuracy

IALCH is the only public hospital with the resources to perform sleep studies. The results of the study from the dissertation will therefore inform provincial policy, as the information will be presented at conferences, workshops, and seminars. This study may be published, as this is the first proposed study in KZN at a public institute.

Research-related Injury:

There is no risk of any kind of infection. There is no pain to be expected as there are no needles used. Although, minimal discomfort is to be expected as there will be cleaning of the scalp with Nuprep (this is similar to face scrub) and earbud to remove oil and superficial dead skin cells. During the removal of electrodes, acetone may be used to remove the ones stuck with medical glue (collodion). The smell of acetone is similar to that of nail polish remover.

Storage of all electronic and hard copies including tape recordings:

All data will be de-identified, and all records will be allocated a specific code for identification. The data obtained specifically for the research (OPSG data) will be stored on a personal laptop that is password protected and only the researcher will have access to this document. The data will be kept for 5 years, and thereafter be deleted. The hard copies of the data collected will be taken for shredding.

Persons to contact in the event of any problems or queries:

Please contact the researcher (062 480 6187), the supervisor (031 373 5291), the co-supervisor (031 240 1344), or the Institutional Research Ethics administrator at 031 373 2375. Complaints can be reported to the Director: of Research and Postgraduate Support Dr. L Linganiso at 031 373 2577 or researchdirector@dut.ac.za.

Annexure 5: Consent

CONSENT

Full Title of the Study: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Name of the Researcher: Mrs. Yabanathi Kibi

Statement of Agreement to Participate in the Research Study:

- I hereby confirm that I have been informed by the researcher, **Yabanathi Kibi**, about the nature, conduct, benefits, and risks of this study - Research Ethics Clearance Number: _____,
- I have also received, read, and understood the above-written information (Participant Letter of Information) regarding the study.
- I am aware that the results of the study, including personal details regarding my sex, age, date of birth, initials, and diagnosis will be anonymously processed into a study report.
- Given the requirements of research, I agree that the data collected during this study can be processed in a computerized system by the researcher.
- I may, at any stage, without prejudice, withdraw my consent and participation in the study.
- I have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the study.
- I understand that significant new findings developed during this research which may relate to my participation will be made available to me.

_____ _____
_____

Full Name of Participant **Date & Time** **Signature/R-Thumbprint**

I, **Yabanathi Kibi** herewith confirm that the above participant has been fully informed about the nature, conduct, and risks of the above study.

_____ _____ _____

Full Name of Researcher **Date & Time** **Signature**

_____ _____ _____

Full Name of Witness (If applicable) **Date & Time** **Signature**

_____ _____ _____

Full Name of Legal Guardian(If applicable) **Date & Time** **Signature**

Annexure 6: Incwadi yolwazi

INCWADI YOLWAZI

Title of the Research Study: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Principal Investigator/s/researcher: Mrs. Yabanathi Kibi, B-Tech: Neuro-clinical technology

Co-Investigator/s/supervisor/s: U-Prof Kennedy Nyamande ((MB ChB (UZ), FCP (SA), certificate of Pulmonology (College of Physicians of South Africa), Ph.D. (UKZN)), Kanye no- Dr. Rosaley Prakaschandra (Ph.D. (Clin Medicine - Cardio)), no- Mr. Dalincebo Mdluli (Masters (Clinical Technology – Neurophysiology)). amasekela alolucwaningo.

Isingeniso esifushane Kanye nenhloso yocwaningo:

Lolucwaningo luqhathanisa i-Apnoea-Hypopnoea Index (AHI) phakathi kwe-Polysomnography yasemini ne-Polysomnography yasebusuku e-Inkosi Albert Luthuli Central Hospital (IALCH), ukuze kukalekiswe izinga loxilongo phakathi kwezifundo ze-Polysomnography (PSG), bese kubonakala ukuthi i-PSG yasemini ingasetshenziswa ngokuphelele ngaphandle kwesidingo sokwenza i-PSG yasebusuku. E-IALCH i-PSG yasemini iyona kuphela esetshenziswayo ukuxilonga ubukhona, nobunzima be-sleep apnoea hypopnoea syndrome (SAHS) or sleep disordered breathing (SDB).

Kunempikiswano yokuthi i-PSG yasemini ayivumelekile ukusetshenziswa ngokuphelelele esikhundleni se-PSG yasebusuku, i-PSG yasebusuku iyonandlela esemqoka elandelwayo ekuhlolweni kwalabo abasolakala ngathi banesifo sokungaphefumuli ngendlela uma belele. Kulesisifundo, umcwaningi uzophenya ukuthi i-PSG yantambama ikhipha imiphumela ecishe ifane uma ngabe kuqhathaniswa nale-PSG yasebusuku uma kuxilogwa i-sleep apnoea noma iyiphi inkinga ephathelene nokuphazamiseka kobuthongo uma isiguli silele, kulaba ababambe iqhaza.

Lolucwaningo luzonikeza ubufakazi bokuthi iziguli ezisolakala ukuthi ziphethwe i-sleep apnoea azihlolisisiwe kahle noma cha, bese inikeza iseluleko esingase sisetshenziswe ekwenzeni ikhwalithi ye-PSG ibengcono lapha esibhedlela sase-IALCH, KwaZulu-Natal, eNingizimu Africa

Mbambi iqhaza othandekayo

Igama ngingu-Yabanathi wakwa-Kibi, ngibhalisiwe enyuvesi ebizwa nge- Durban University of Technology ukwenza izifundo ze- Master of Health Sciences kwi-neurophysiology clinical technology. Bengicela ngomusa imvume yakho yokubamba iqhaza kulolucwaningo lokuphenya kabanzi kezifundo zesifo sokulala. Isihloko socwaningo: Ukuqhathaniswa kwe-Apnoea-Hypopnoea Index phakathi kwe-Polysomnography yantambama i ne-Polysomnography yasebusuku ngakuhlangothi lwakwezempilo, KwaZulu-Natal. Kulolucwaningo, njengombamb'iqhaza, uyacelwa ukuthi wenze izivivinyo ezimbili ze-PSG, lesi santambama kanye nalesi sasebusuku, lokhu kunikeza ithuba lokuqhathanisa indlela yoxilongo kulezinhlobo ezimbili. Uvumelekile ukubuza yonke imibuzo ofisa ukuyibuza ngalolucwaningo ngoba kubalulekile ukuthi usiqonde ngokugcwele isifundo. Unelungelo lokuxoxa ngalolucwaningo nomdeni wakho Kanye nabangane bakho. Ngaphezu kwalokho, awukho ngaphansi kwesibopho sokuphendula isicelo somcwaningi ngosuku olufanayo lwesicelo. Ngale njongo, uzonikezwa ikhophi yencwadi yedokhumende yolwazi ozohamba nayo uma usuya ekhaya ukuze uyiqondisise kangcono. Ucwaningo ukusesha okuhlelekile noma ukubuza noma ukwazi kabanzi ngolwazi olusha, futhi lwenzelwa ikakhulukazi ukuthola amaqiniso kanye nokuzama ukuthola isiphetho esisha esizosiza esizukulunwani esizayo.

Uhlaka Lwenqubo:

Isicelo sokuhlolwa kwesifo esiphathelene nokuphazamiseka kokulala siyaye sifakwe uDokotela, bese usuku lokuza luhlelwa i-Neurophysiology Clinical Technologists ozokwenza i-PSG yantambama Kanye neyasebuseku. I-PSG yantambama Kanye neyasebusuku yenziwa kwiziguli ezisolakala sengathi zinezimpawu zesifo sokungaphefumuli kahle uma zilele, lezi ziguli zibika izimpawu zokungalali kahle ebusku, ukuvuka bekhathele nokuzela okuyisimanga emini, ukunqanyukelwa umoya ebusuku, ukuhonqa, Kanye nokuvuka njalo emajukujukwini obusuku.

Ngosuku lokuhlolwa, ifomu lokwazi kabanzi ngokuhlupha/ ngokuphethe isiguli, lizogcwaliswa. Kunezinhlobo ezine zezikali noma zemibuzo ehlukahlukene esetshenziswa kanye ne-PSG ukuhlela indlela yokulala Kanye nesifo sokuphazamiseka okungabekezeleki uma ulele. Lapho kubalwa, I-Berlin, Epworth (ESS), Functional Outcome (FOSQ), and Stop-Bang yenhlobo zemiqulu yemibuzo. Inani eliphelele le-ESS isekelwe esilingiswaneni esingu-0 kuya k-24, The total ESS score is based on a scale of 0 to 24, isilinganiso esilingana, noma, esingaphezulu kuka-16 sithathwa njengesilinganiso sokuzela okungabekezeleki, kanti futhi lokho kudinga ucwaningo olunzulu. Inani eliphele le-Stop-Bang likalwa suka ku-0 kuya ku-8, isilinganiso esilingana, noma, esingaphansi kuka-2 sithathwa njengesilinganiso esinengozi encane; isilinganiso noma esilingana no-3-4 sithathwa njengesilinganiso esinengozi ephakathi nendawo; isilinganiso esilingana, noma, esingaphezulu kuka-5 sithathwa njengesilinganiso esinengozi enkulu yokuthola i-sleep apnoea. Iqulu

lemibuzo ebizwa nge-Berlin inezigaba ezintathu ephathelene nobungozi bokuba nesifo sokunqanyukelwa umoya uma ulele. Iziguli lapha zihhukaniswa njengalowo osengozini ephezulu noma engozini encane ngokusekelwe izimpendulo zabo kulezo zinhlelo zemibuzo Kanye nezibalo zazo zonke izigaba zesibonakaliso. Uma ngabe isibalo esiphelele singu-2 nangaphezulu kuchaza ukuba sengozini enkulu yokuphathwa isifo sokunqanyukelwa umoya uma ulele. FOSQ iqukethe izigaba ezingu-5, ukuthola amaphuzu aphelele, wonke amaphuzu ezigaba ayahlanganiswa ukuze kubalwe i-mean, bese i-mean yandiswa ngo-5, ngaphandle kokukhathaliseka kokuthi inani lezigaba ezisetshenzisiwe ekubaleni i-mean zingaki. Uma isibalo esiphelele singu-5 kuya ku-20, lokho kuchaza isimo esibi sokusebenza, amaphuzu aphezulu achaza isimo esingcono sokusebenza. Inani eliphelele lemikhakha yemibuzo kanye nokuhlolwa kwe-PSG kuyasiza kakhulu ekutholeni izifo eziphathelene nokuphazamisa ubuthongo.

Umcwaningi uzokwenza i-PSG yantambama kanye neyasebusuku. Kusetshenziswa iziteshi ezingu-17 ze-PSG, lapho kubalwa i-electro-encephalography efakwa esikhumbeni sasekhanda (Fz-Cz, Cz-Oz, C3-A2, kanye no-C4-A1), i-electro-oculography efakwa eduze kwamehlo (LOC-reference, ROC-A2, Kanye ne-LOC2-A2), i-submental Kanye ne-tibialis electromyography (u-active ofakwa ngaphansi kwesilevu, naphezu komsipha obizwa nge-tibialis. I-reference izoba kude ne-active ngo-3 cm), i-electro-cardiography ezofakwa eduze nalapho kushaya khona inhliziyo (RA-LA, RA-LL, and LA-LL).

Ekwenzeni le-Polysomnography (PSG); i-electroencephalography (EEG) isetshenziswa ekukaleni umsebenzi wogesi wobuchopho, i-electromyography (EMG) Kanye ne-snore sensor zisetshenziswa ekukaleni ukuhongqa lapha entanyeni ngaphansi kwesilevu, izinga lokuqapha kanye nokunyakaza komlenze okungapheli kukalwa nge-tibialis EMG, bese i-Electrocardiography (ECG) ikala isilinganiso senhliziyo. The nasal cannula and thermistor kubekwa ngasekhaleni nasemulonyeni ukuze kukalwe ukuhamba komoya kanye nezinga lokuphefumula. Umzamo wokuphefumula ukalwa ngokubuka ukunyakaza kokuphefumula kwezimbambo kanye nesisu kusetshenziswa i-inductive plethysmograph, kanye ne-finger pulse oximetry esetshenziswa ekukaleni izinga le-oxygen egazini. Icele lomzimba isiguli elele ngalo lifakwa i-Neurophysiology Clinical Technologist esuke imaka i-PSGs, isebenzisa i-audio-visual low-light camera. Ukumaka kuzokwenziwa kulandelwa imigomo ye-American Academy of Sleep Medicine (AASM) version 2.0 evuselelwe ngonyaka ka-2012. Izifundo ze-PSG zizogcinwa kwikhompuyutha kusetshenziswa i-nicolet software.

I-OPSG izokwenziwa mahhala njengoba izobe yenziwelwa ucwaningo ngenhloso yokwenza ngcono impilo yeziguli, nanjengoba iziguli zizothola imiphula yalezivivinyo zombili. Enye inhloso yalolucwaningo ukunyusa izinga lesibhedlela.

Lolucwaningo luzobandakanya inani leziguli esezikhulile ezingu-25 ezineminyaka sukela ku-18 weminyaka kuya eminyakeni engaphansi kwengu-70. Iziguli ezinbabamba iqhaza ezibika izimpawu ezibukeka sengathi i-sleep disordered breathing (SDB). Leziguli eziphakathi kwaleminyaka zikhethwa ngesizathu sokunciphisa noma iyiphi into engaholela emiphumeleni yamanga. Ngaphakathi kwezinye yezizathu, abantu abadala kakhulu imvamisa balala kancane ngokujulile kanti futhi baphaphama kaningi uma kubalwa ubusuku bonke, ngakhoke lokhu kungadukisa imiphumela ye-PSG. Iziguli ezinesifo sokuqwasha ebusuku (insomnia) angeke zilibambe iqhaza ukuvikela imiphumela engaqondile ye-sleep disordered breathing.

Izingozi noma ukungaphatheki kahle kobamb'iqhaza:

Abukho ubungozi bokutheleleka ngamagciwane. Abukho ubuhlungu obungase bulindeleke njengoba kungekho mijovo ezosetshenziswa. Ukungakhululeki kahle okuncane kungalindeleka njengoba kuzobe kuhlanzwa isikhumba nge-Nuprep ne-ear-bud ukususa amafutha nesikhumba esingaphezulu esingasasebenzi. Ngesikhathi sokususa ama-electrodes, ingase isetshenziswe i-acetone ukususa lokho okuzobe kunamathele ngamadla ngalesikhathi kusetshenziswa i-medical glue ebizwa nge-collodian.

Izizathu zokuhoxa kokubamba iqhaza ocwaningweni:

Iziguli ezibambe iqhaza zingakhishwa kucwaningo uma sezake zaxilongwa noma zisebenzisa izinto ezithize ngenhloso yokwenza isifo se-sleep disordered breathing ngcono. Uma ngabe iziguli zingakhululekile ngokulala usuku olunye esibhedlela. Uma ngabe iziguli zinesifo senhliziyo, noma isifo sokuqwasha ebusuku. Ukubamba iqhaza kungokuzithandela, lokho kukunikeza imvume njengesiguli, yokuhoxa kunoma yisiphi isigaba ngaphambi kokuqala inqubo. Lokhu kusho ukuthi unelungelo lokuhoxa oncwaningweni noma kunini lapho ufisa ukewnza kanjalo futhi ukuhoxa ngeke kuphikisane nokunakekelwa okujwayelekile okufanele ukuthole.

Izinzuzo:

Ucwaningo oluhlongozwayo kanye nemiphumela izosiza ama-Neurophysiology Clinical Technologists ukuze azi ukuthi ulona luphi ucwaningo lwe-PSG olushaya esikhonkosini ngokuphelele ekutholeni ama-disorders aphathelene nokuphazamisa ubuthongo. Lokhu kuzobe sekuchaza ukuthi, leyo ehlabe esikhonkosini, iyona ekumele isetshenziswe ekucwaningeni iziguli, njengoba i-ALCH kuyisona sodwa isibhedlela sikahulumeni lapha e-KZN esenza ama-sleep studies. Kanti futhi kungakuhle ukwenza ngendlela elungile nakulabo abangakwazi ukufinyelela ezibhedlela ekungasizona ezikahulumeni.

Ucwaningo luzofaka isandla ekuhlinzekeni ubufakazi obubalulekile, nolwazi olusekelwe kepha kuzoya ngemiphumela yocwaningo. Imiphumela yocwaningo izokwabiwa ngesikhathi sesethulo ezinkomfeni, nakuma-workshop, Kanye nakuma-seminars. Uma ngabe imiphumela ye-PSG yasebusuku ikhipha ulwazi oluthe xaxa, ngokuphathelene nokuguqula uxilongo kanye nenqubekela phambili yempilo yesiguli, lapho ke imiphumela ye-PSG yasebuku kuyomele ithunyelwe kudokotela wesiguli kanye neziphakamiso zokwelashwa uma ngabe zikhona.

Ngakho-ke, kulokhu, izinzuzo zizoba ngandlela zombili, ngokuqondile nangokungaqondile, njengoba uzonikezwa nawe ikhophi yesifundo sokulala ukuze uyisenzise wena buqu lapho udinga ukuyisebenzisa khona. Elinye ikhophi lizifakwa ohlelweni lwesibhedlela ngaphansi kwefayela lakho ukusiza odokotela ngokuqhubekela phambili ngakwezempilo yakho.

Remuneration:

Uzothola ukuqondisisa kangcono lolucwaningo lwesifo sokulala uphinde uthole ukuhamba nekhophi yemiphumela ye-PSG yasebusuku leyo ezobe ephirintiwe uma sekuqedwe izibalo nombiko usuphoyhuliwe. Lombiko uzophinda usize udokotela wakho ekusizeni impilo yakho kangcono njengoba uzogcinwa ohlelweni.

Izindleko zesifundo:

Ngeke ulindeleke ukuthi unikele nganoma iyiphi imali kulolucwaningo njengoba i-PSG yasebusuku izokwenziwa ngosuku olufanayo nesifundo sakho se-PSG yantambama.

Imfihlo:

Imfihlakalo iqinisekisiwe ngesikhathi socwaningo, ngumcwaningio kuphela ozobe enemvume yokufinyelela kwizincwadi zocwan ingo. Ngesikhathi semibuzo, ngumcwaningi kuphela kanye nesiguli abazobe besendlini yokuxilonga, kanto lonke ulwazi oluzobe luthanthwa kwisiguli luzobekwa kwi-compuyutha ene-password, kanti amagama eziguli angeke abhalwe phansi lapho, kungaba izinombolo ezithile ezaziwa umcwaningi kuphela ukuthi zichaza ukuthini. Kuzoba nocophelelo lokuthi akukho ukulimala okuhlobene nocwaningo.

Imiphumela:

Imiphumela yocwaningo izokwabiwa namanye amaNeurophysiology Clinical Technologists kanye no Dokotela e-IALCH ngesikhathi sezethulo njengoba lokhu kuzobe kuvulelwe ukuxoxisana ukuze kwaziwe ukuthi yikuphi ukuhlowa kew-PSG okuyikhona okunembile, okucacile nokuzwela ngokwanele ekutholeni iziguli ezinenkinga yesifo esihlobene nokulala, noma ngabe lezindlela zombili zinokunemba okufanayo.

IALCH yisona kuphela isibhedlela sikahulumeni esinezinsiza zokwenza izifundo zokulala. Imiphumela yocwaningo ezobe iphothuliwe izokwaziswa kwinqubomgomo yesifundazwe, njengoba ulwazi luzokwethulwa ezingqungqutheleni, komasifundisane nasemihlanganweni. Lolucwaningo lungashicilelwa, nje ngoba lesi kuyisifundo kuyifundo sokuqala esihlongozwayo la e-KZN esikhungweni sikahulumeni.

Ukulimala okuhlobene nocwaningo:

Ayikho ingozi yanoma yiluphi uhlobo lokutheleleka. Abukho ubuhlungu obungalindeleka njengoba kungekho zinaliti ezizosetyenziswa. Yize kungalindeleka ukungakhululeki njengoba kuzoba nokuhlanzwa kwesikhumba sekhanda ngeNuprep (lokhu kufana nokhilimu ojwayelekile wokukhuhla nokuhlanza ubuso) kanye nokotini wendlebe ukuze kusuke uwoyela namaseli esikhumba afile angaphezulu. Ngesikhathi sokususwa kwama-electrodes, i-acetone ingasetshenziswa ukuze kusuke lawa ma-electrodes ananyathiselwe ngengcina yezokwelapha ebizwa phecelezi nge collodian. During the removal of electrodes, acetone may be used to remove the ones stuck with medical glue (collodian). Iphunga le-acetone (uketshezi olungenambala) licishe lifane nephunga loketshezi olusetshenziswa uma ususa upende ofakwa kuzinzipho zeminwe noma ezinzwaneni.

Ukugcinwa kwazo zonke izinto zikagesi namakhophi aqinile kanye namateyipu aqoshiwe:

Yonke imininingwane nolwazi luzobhalawa ngendlela yokuthi omunye umuntu ongasiye umcwaningi walesi sifundo angakwazi ukuchaza ukuthi le miphumela eyalo mthathiqhaza. Wonke amarekhodi azonikezwa ikhodi ethile yokuhlonza. Imininingwane etholelwe ngqo lolucwaningo (i-PSG yasebusuku) izogcinwa kwi-laptop yomcwaningi siqu ezobe ivikelwe nge-password kanti futhi ngumcwaningi kuphela ozokwazi ukufinyelela kulemibhalo. Imininingwane nolwazi luzogcinwa iminyaka emihlanu, bese ngemumva kwalokho iyasuswa. Amakhophi alukhuni wolwazi oluqoqiwe azothathwa ukuze ahlukaniswe acoboshiswe. Imibiko ebhaliwe ye-PSG yantambama kanye ne-PSG yasebusuku izofakwa ohlelweni (Meditech Production ALS.Live615. 15F.Ring) ngaphansi kwe-Neurophysiology Imaging and Therapeutic Services drive ngezinhloso sokubukeza kanye nokuqhathanisa kwenqubekela phambili yempilo yesiguli esikhathini esizayo.

Umuntu ongaxhumana naye noma ngabe unayiphi inkinga noma imibuzo:

Sicela uxhumane nomcwaningi (062 480 6187), umphathi woncwaningi wasesikoleni (031 373 5291), umphathi womcwaningi wasemsebenzini (031 240 1344), noma ngabe i-Institutional Research Ethics administrator on 031 373 2375. Izikhalazo zingabikwa kwi-Director: Research and Postgraduate Support Dr L Linganiso on 031 373 2577 or researchdirector@dut.ac.za.

Annexure 7: Isivumelwano

ISIVUMELWANO

Isihloko sesifundo esigcwelwe: The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.

Igama lomcwaningi: Mrs. Yabanathi Kibi

Isitatimende sesivumelwano sokuhlanganyela esifundweni socwaningo:

- Ngiyaqinisekisa ukuthi ngitsheliwe umcwaningi, **Yabanathi Kibi**, mayelana nemvelo, ukuziphatha, izinzuzo kanye nezingozi zalolucwaningo - Research Ethics Clearance Number: _____,
- Ngiphinde ngathola, ngafunda futhi ngayiqonda imininingwane ebhaliwe ngenhla (Incwadi yolwani) mayelana nalolucwaningo.
- Ngiyazi ukuthi imiphumela yocwaningo, kufaka phakathi imininingwane mayelana nobulili bami, iminyaka, usuku lokuzalwa, amagama ami kanye nokuxilogwa kuzobikwa ngokuthi kungabhalwa amagama abantu eqiniso uma ngabe sekukhishwa imiphumela.
- Ngokubuka izidingo zocwaningo, ngiyavuma ukuthi ulwazi oluqoqwe ngalesisifundo kungafakwa kuhlelo olu-computerised ngumcwaningi.
- Ngingakwazi, nganoma yisiphi isigaba, ngaphandle kokubandlulula, ngihoxise imvume yami kanye nokuhlanganyela esifundweni.
- Benginethuba elanele lokubuza imibuzo (ngokuzithandela kwami) futhi ngazinikela ukuthi ngilungele ukuhlanganyela kulolucwaningo.
- Ngiyaqonda ukuthi ukutholakala okusha okuphawulekayo okutholiwe ngalesikhathi kwenziwa ucwaningo okungase kuhlobanise nokuhlanganyela kwami kuzovumeleka ukuthi kufinyelele kimi.

_____	_____	_____
Amagama aphelele	**Usuku nesikhathi**	**Sayina/isithupha**

omhlanganyeli **sakwesokudla**

Mina, **Yabanathi Kibi** lapha ngiqinisekisa ukuthi umhlanganyeli ongenhla waziswe ngokuphelele ngohlobo, ukwenziwa kanye nobungozi besifundo esingenhla.

_____	_____	_____
Amagama aphelele omcwaningi	**Usuku nesikhathi**	**Sayina**
_____	_____	_____
Amagama aphelele ofakazi	**Usuku nesikhathi**	**Sayina**
_____	_____	_____
Amagama aphelele omnakekeli osemthethweni	**Usuku nesikhathi**	**Sayina**

Annexure 8: Polysomnography general patient information

POLYSOMNOGRAPHY GENERAL PATIENT INFORMATION

Base Hospital: …….....

Hospital No.: KZ……..…….. D.O.B.: ……....

Patient's Name: ……..…… Age: …… Gender: …..……..…..

Telephone Number…….Occupation……..……..….

Previous Weight…..……Kg: Height……..…m: BMI……..Kg/m^2 Neck Circumference……cm

Previous PSG: Yes/No ……

Current Weight……....Kg: Height……..…m: BMI……..Kg/m^2 Neck Circumference……cm

Referring Doctor: ……..… Dept/Ward: ……

CLINICAL SUMMARY:

1. GENERAL HISTORY RELATED TO THE STUDY

1.1. Reason for Visiting the Referring Doctor:

	Sleep during the day while performing an activity.			Feeling of suffocation/ choking during sleep.
	Restless sleep.			Constantly waking up while you are sleeping.
	Approximate frequency of waking up per night.			Snoring A. Constant, B. Intermittent, or C. Seldom
	Approximate hours of sleep per night.			
	Unrefreshed mornings(moody, lethargic, do not want to talk to anyone or do anything)			
	Waking up at night gasping for air.			

1.2. Other illnesses the patient is suffering from: …….....

1.3. Medication: ……..……..….

1.4. Patients sleep rating: Good ☐ Moderate ☐

Bad ☐ Very Bad ☐

Comments on what is ticked (√) on 1.4: ……

1.5. Headaches in the morning: Yes ☐ No ☐

2. SYMPTOMS OF POSSIBLE NARCOLEPSY

	Irresistible Sleep Attacks
	Hypnagogic Hallucinations
	Cataplexy
	Sleep Paralysis

3. SYMPTOMS OF RESTLESS LEG SYNDROME (DURING AWAKE)

Experience of weird, uncomfortable, irritating, strange sensations on the legs or feet when sitting or lying still?

Yes ☐ No ☐

Comments……..……

4. SIGNS AND SYMPTOMS OF PERIODIC LEG MOVEMENT DISORDER (DURING SLEEP)

4.1. Repetitive leg movements in one or both legs. ☐

4.2. Sleep disruption or restless sleep, due to frequent leg movements causing disturbed sleep and multiple brief awakenings. (awakenings are easily missed by patients captured on the sleep test). ☐

4.3. Daytime sleepiness ☐

4.4. Behavioural and school performance problems, for example, irritability, hyperactivity, and impulsivity. ☐

Other……

5. SLEEP QUESTIONNAIRES COMPLETED: Yes ☐ No ☐

Select which questionnaires were completed:

Epworth sleep scale questionnaire ☐

Functional outcome of sleep questionnaire (FOSQ) ☐

Berlin questionnaire (for sleep apnoea) ☐

Stop-bang questionnaire ☐

Further Technologist Remarks/Impressions

……

……

Date: ……. Neurophysiologist: ……..

Annexure 9: Questionnaires (Part 1-4)

NB: The researcher will complete the questionnaire

Berlin Questionnaire (for sleep apnoea) /Uhlu lwemibuzo

KZ Number: _____

Full name/: _____ Gender: Male ☐ Female/ ☐

Amagama aphelele Ubulili: Owesilisa Owesifazane

Date of Birth/: ____/_____/_____

Usuku lokuzalwa

Today's date/: _____/_____/_____

Usuku lwanamuhla

Weight/Isisisindo: _____ (kg) Height/Ubude: _____ (cm)

Neck circumference/Isikali somqala jikelele: _____ (cm)

1. **Has your weight changed in the last 5 years? / Ngabe isisindo sakho sishintshile uma uqhathanisa neminyaka eyisihlanu edlule?**
   - ☐ Increased/ Senyukile
   - ☐ Decreased/ Sehlile
   - ☐ No change/ Asishintshile

2. **Do you snore?/ Ingabe uyahhonqa?**
   - ☐ Yes/ Yebo
   - ☐ No/ Cha
   - ☐ Don't know/ Angazi

If you snore:

3. **Your snoring is? / Ukuhhonqa kwakho ku?:**
   - ☐ Slightly louder than breathing / Kungaphezulu kancane kunendlela ophefumulu ngayo
   - ☐ As loud as talking / Kulingana nendlela ophefumula ngayo
   - ☐ Louder than talking/ Ngaphezulu kwendlela ophefumula ngayo

- ☐ Very loud / Uhhonqa kakhulu
- ☐ Can be heard in adjacent rooms / Kuzwakala ekamelweni eliseduze nalapho ulele khona

4. **How often do you snore? Uhhongqa kangaki?**
   - ☐ Nearly every day / Cishe nsuku zonke
   - ☐ 3-4 times a week / 3-4 yezikhathi evikini
   - ☐ 1-2 times a week /1-2 yezikhathi evikini
   - ☐ 1-2 times a month / 1-2 yezikhathi enyangeni
   - ☐ never or nearly never / Akwenzeki neze

5. **Has your snoring ever bothered other people? Ukuhhongqa kwakho kuyake kuphazamise abanye abantu.**
   - ☐ Yes / Yebo
   - ☐ No / Cha
   - ☐ Don't know/ Angazi

6. **Has anyone noticed that you quit breathing during your sleep? / Ingabe ukhona ini oseyake abone ukuthi unqanyukelwa umoya uma ulele?**
   - ☐ Nearly every day/ Cishe nsuku zonke
   - ☐ 3-4 times a week / 3-4 yezikhathi evikini
   - ☐ 1-2 times a week / 1-2 yezikhathi evikini
   - ☐ 1-2 times a month / 1-2 yezikhathi enyangeni
   - ☐ never or nearly never / Akwenzeki neze

7. **How often do you feel tired or fatigued after your sleep? Awukalekise indlela okhathala ngayo uma uvuka ebuthingweni.**
   - ☐ Nearly every day / Cishe nsuku zonke
   - ☐ 3-4 times a week / 3-4 yezikhathi evikini
   - ☐ 1-2 times a week / 1-2 yezikhathi evikini

- ☐ 1-2 times a month / 1-2 yezikhathi enyangeni
- ☐ never or nearly never/ Akwenzeki neze

8. **During your waketime, do you feel tired, fatigued or not up to par? / Uma uvuka, uzizwa ukhathele, ucobekile, noma uzizwa ungafuni kuzwa lutho?**
   - ☐ Nearly every day/ Cishe nsuku zonke
   - ☐ 3-4 times a week/ 3-4 yezikhathi evikini
   - ☐ 1-2 times a week / 1-2 yezikhathi evikini
   - ☐ 1-2 times a month/ 1-2 yezikhathi enyangeni
   - ☐ never or nearly never/ Akwenzeki neze

9. **Have you ever nodded off or fallen asleep while driving a vehicle? / Ingabe wake wangquphazela noma walala ushayela imoto?**
   - ☐ Yes/ Yebo (1)
   - ☐ No/ Cha

If yes / Uma uthe yebo:

9a. How often does it occur/ Kwenzeka kangaki lokhu?
- ☐ nearly every day (1) / Cishe nsuku zonke
- ☐ 3-4 times a week (1)/3-4 yezikhathi evikini
- ☐ 1-2 times a week / 1-2 yezikhathi evikini
- ☐ 1-2 times a month / 1-2 yezikhathi enyangeni

10. **Do you have high blood pressure?/** Ingabe unaso ini isifo somfutho wegazi ophakeme?
    - ☐ Yes / Yebo
    - ☐ No /Cha
    - ☐ Don't know / Angazi

Scoring Berlin Questionnaire

Adapted from: Table 2 from Netzer, et al., 1999. (Netzer NC, Stoohs RA, Netzer CM, Clark K,
Strohl KP. Using the Berlin Questionnaire to identify patients at risk for sleep apnea syndrome. Ann
Intern Med. 1999 Oct 5;131(7):485-91).

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:

Categories:

Category 1: items 1, 2, 3, 4, 5.

Item 1: if 'Yes', assign 1 point
Item 2: if 'c' is the response, assign 1 point
Item 3: if 'a' or 'b' is the response, assign 1 point
Item 4: if 'a' is the response, assign 1 point
Item 5: if 'a' is the response, assign 2 points

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

Category 2: items 6, 7, 8 (item 9 should be noted separately).

Item 6: if 'a' or 'b' is the response, assign 1 point
Item 7: if 'a' or 'b' is the response, assign 1 point
Item 8: if 'a' 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 the patient is greater than 30kg/m2.
(BMI must be calculated. BMI is defined as the weight (kg) divided by height (m) squared, i.e., kg/m2).

Scoring:

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.

Additional question: item 9 should be noted separately,
IALCH Neurophysiology clinic.2019.

Annexure 10: Functional Outcome of Sleep Questionnaire

KZ Number: _____

Full name/: _____ Gender: Male☐ Fema☐

Amagama aphelele Ubulili Owesilisa Owesifazane

Date of Birth/: ____/_____/_____

Usuku lokuzalwa

Today's date/: _____/_____/_____

Usuku lwanamuhla

Weight/Isisisindo: _____ (kg) Height/Ubude: _____ (cm)

Neck circumference/Isikali somqala jikelele: _____ (cm)

Note: In this questionnaire, when the words "sleepy" or "tired" are used, it describes the feeling that you can't keep your eyes open, your head is droopy, that you want to nod off, or you feel the urge to take a nap. These words do not refer to the tired or fatigued feeling you may have after you have exercised.

Inothi: Kuloluhlu lwemibuzo, uma amagama "ukulala" noma "ukukhathala" esetshenzisiwe, kuchaza indlela ozizwa ngayo ukuthi awukazi ngisho ukugcina amehlo akho evuliwe, ikhanda lingqathuzela, ufuna ukulala manje. Ngakhoke lokho akusho indlela ozizwa ngayo uma uqeda ukuzivocavoca.

Please use the following key when answering the questions/Cela usebenzinze lokhu okulandelayo uma ngabe uphendula lohlu lwemibuzo olulandelayo :

0 = I don't do this activity for other reasons/ Angikwenzi lokhu ngenxa yezinye izizathu
1 = Yes, extreme/ Yebo, kakhulu
2 = Yes, moderate, Yebo, kakhudlwana
3 = Yes, a little / Yebo kancane
4 = No/ Cha

#	Question	Answer
1	Do you generally have difficulty concentrating on the things you do because you are sleepy or tired? Ingabe kuvamile ukuthi kubenzima ukugxila kulokho osuke ekwenza, ngenxa yokuthi ufuna ukulala noma ukhathele?	
2	Do you generally have difficulty remembering things because you are too sleepy or tired? Ingabe kuvamile ukuthi kubenzima ukukhumbula izinto ngoba usuke uzela kakhulu noma ukhathele?	
3	Do you have difficulty finishing a meal because you become sleepy or tired? Ingabe unabo ubunzima bokuqeda ukudla khwakho ngoba uphazanyiswa ukuthi usuyalala noma ukhathele?	
4	Do you have difficulty working on a hobby (for example sewing, collecting, gardening) because you are sleepy or tired? Ingabe unabo ubunzima bokuqhubeka wenze izinto othanda ukuzenza ngoba nakhu usuyazela noma ukhathele (izinto ezifana nokuthunga, noma ukusebrnza engadini)?	
5	Do you have difficulty doing work around the house (for example: cleaning the house, doing laundry, taking out the trash, or repairing work) because you are sleepy or tired? Ingabe unabo ubunzima bokwenza imisebenzi yasendlini (isilinganiso: ukuhlanza endlini, ukuwasha izingubo, ukukhipha udoti, noma ukulungisa lapha nalaphaya okuphukile) nogoba nakhu usufuna ukulala noma ukhathele?	
6	Do you have difficulty operating a motor vehicle for short distances (*less* than 100 miles) because you become sleepy or tired? Ingabe unabo ubunzima bokushayela imoto okwesikhashana (ngaphansi kuka 100 miles) ngoba uvele uzele noma ukhathale?	
7	Do you have difficulty operating a motor vehicle for long distances (*greater* than 100 miles because you become sleepy or tired?	

	Ingabe unabo ubunzima bokushayela imoto umgama omude (ngaphezului kuka 100 miles) ngoba uvele uzele noma ukhathale?	
8	Do you have difficulty getting things done because you are too sleepy or tired to drive or take public transportation? Ingabe unabo ubunzima bokwenza lokhu okumele ukwenze ngoba uzela lento engabekezeleki noma ukhathrele ngisho ukushayela noma ukugibela zokuthutha zomphakathi?	
9	Do you have difficulty taking care of financial affairs and doing paperwork (for example writing checks, paying bills, keeping financial records, filling out tax forms, etc.) because you are sleepy or tired? Ingabe unabo ubunzima bokunakekela ezezimali nokwenza umsebenzi wephepha (isilinganiso: ukubhala amatsheke, ukukhokhela izikweletu, ukugcina amarekhodi azezimali, ukugcwalisa nokugcina amafomu entela, nokunye nje..) ngoba uyazela noma ukhathele?	
10	Do you have difficulty performing employed or volunteer work because you are sleepy or tired? Ingabe unabo ubunzima bokwenza umsebenzi owuqashelwe noma owuvolontiyele ngoba uyazela noma ukhathele?	
11	Do you have difficulty maintaining a telephone conversation because you become sleepy or tired? Ingabe unabo ubunzima bokungcina ingxoxo ocingweni noma uvele uzele ngoba ufuna ukulala noma ukhathele?	
12	Do you have difficulty when your family or friends visit in *your* home because you become sleepy or tired? Ingabe unabo ubunzima uma izihlobo zakho noma abangani bakho bekuvakashela ekhayeni lakho ngoba uyazela noma ukhathele?	
13	Do you have difficulty visiting with your family or friends in *their* homes because you become sleepy or tired? Ingabe unabo ubunzima bokuvakasha nezihlobo zakho noma abangani bakho ekhayeni labo ngoba uyazela noma uyakhathala?	

14	Do you have difficulty doing things with your family or friends because you are too sleepy or tired? Ingabe unabo ubunzima bokwenxza izinto nomndeni wakho noma abangani bakho ngoba uyazela kakhulu noma ukhathele?	
15	**<u>For question 15 only</u>, answer using only 1, 2, 3, or 4.** **<u>Kulombuzo weshumi nanhlanu kuphela</u>, phendula usebenzise 1, 2, 3, noma 4 kuphela.** Has your relationship with family, friends, or work colleagues been affected because you are sleepy or tired? Ingabe ubudlelwane bakho nomndeni wakho, abangani bakho noma labo osebenza nabo sebuchaphazelekile ngoba uyazela noma ukhathele?	
16	Do you have difficulty exercising or participating in sporting activities because you are sleepy or tired? Ingabe unabo ubunzima bokuzivocavoca noma ukuzibandakanya kwezemidlalo ngoba uyazela noma ukhathele?	
17	Do you have difficulty watching a movie or videotape because you become sleepy or tired? Ingabe unabo ubunzima bokubuka ibhayisikobho ngoba uvele uzele noma ukhathale?	
18	Do you have difficulty enjoying the theater or a lecture because you become sleepy or tired? Ingabe unabo ubunzima bokunamela ishashalazi noma inkulumo ngoba uyakhathala noma uvele ufune ukulala?	
19	Do you have difficulty enjoying a concert because you become sleepy or tired? Ingabe unabo ubunzima bokujabulela ikhonsathi ngobga uyazela noma ukhathele?	
20	Do you have difficulty watching television because you are sleepy or tired?	

	Ingabe unabo ubunzima bokubuka umabona-kude ngoba uyazela noma ukhathele?	
21	Do you have difficulty participating in religious services, meetings, or a group or club because you are sleepy or tired? Ingabe unabo ubunzima bokuzibandakanya emihlanganenwi yezokholo, emihlanganenwi noma iyiphi, noma emaqenjini ngoba uyazela noma ukhathele?	
22	Do you have difficulty being as active as you want to be in the *evening* because you are sleepy or tired? Ingabe unabo ubunzima bokuzigcinina usemadleni ngokugcwele nangengendlela othanda ngayo ngezinkhathi zasebuku noma uyake uzele noma ukhathale?	
23	Do you have difficulty being as active as you want to be in the *morning* because you are sleepy or tired? Ingabe unabo ubunzima bokuzigcinina usemadleni ngokugcwele nangengendlela othanda ngayo ngezinkhathi zasekuseni noma uyake uzele noma ukhathale?	
24	Do you have difficulty being as active as you want to be in the *afternoon* because you are sleepy or tired? Ingabe unabo ubunzima bokuzigcinina usemadleni ngokugcwele nangengendlela othanda ngayo ngezinkhathi zantambama noma uyake uzele noma ukhathale?	
25	Do you have difficulty keeping pace with others your age because you are sleepy or tired? Ingabe unabo ubunzima bokuzigcina usesimeni esifanayo nalabo olingana nabo ngeminyaka, noma uyahluleka ngonga uyazela noma ukhathele?	
26	**For question 26 only, answer using this scale (1=very low; 2=low; 3=medium; 4=high).** How would you rate your general level of activity?	

	<u>Kulombuzo wamashumi amabili nesithupha kuphela</u>, phendula usebenzise 1= phansi kakhulu, 2= phansi, 3= phakathi nendawo, noma 4= phezulu.	
27	Has your intimate or sexual relationship been affected because you are sleepy or tired? Ingabe ukuhlangana ngokocansi nalowo osendelene naye kuye kwaphazamiseka noma sekuyaphazamiseka ngoba uyazela noma ukhathele.	
28	Has your desire for intimacy or sex been affected because you are sleepy or tired? Ingabe isifiso sokusondelana ngokocansi siye saphazamiseka ngoba uyazela noma ukhathele.	
29	Has your ability to become sexually aroused been affected because you are sleepy or tired? Ingabe ukukwazi ukuvuswa ngokocansi kuye kwaphazamiseka ngoba uyazela noma ukhathele.	
30	Has your ability to have an orgasm been affected because you are sleepy or tired? Ingabe ukufinyelela esigabeni sokuba ne-orgasm keye kwaphazamiseka ngoba ukhathele noma uyazela.	

*** END OF QUESTIONNAIRE ***

<u>To be calculated by the researcher</u>

SCORING INSTRUCTIONS OF FOSQ

SUBSCALES	**QUESTIONS**	**ITEM #**
General productivity	8 questions	1-4, 8-11
Social outcome	2 questions	12, 13
Activity level	9 questions	5, 14-16, 22-26
Vigilance	7 questions	6, 7, 17-21
Intimate relationships and sexual activity	4 questions	27-30

A response score of 0 for an item should be scored as N/A or missing response.

Thus, the potential range of scores for any item is 1-4.
Calculate the mean of the answered items with responses equal to or greater than 1 for each subscale. This is the weighted mean item-total or subscale score.
For example: if a subscale has six questions and one question has a missing response and one with a N/A response. Then you would not include those two questions as to why you added the responses and you would divide by four instead of six when calculating the mean. This prevents a score biased due to missing answers or skipped questions because an individual doesn't engage in a particular activity due to reasons other than disorders of excessive sleepiness.
The potential range of scores for each subscale is 1-4.

FOR TOTAL SCORE
To obtain the total score, take all the subscale scores and calculate the mean of these scores and then multiply that mean by five. Multiply by five regardless of the number of subscales scores used in the computation of the mean.
For example, if you have a subscale score for all subscales, then you multiply the mean of those scores by 5; f you have subscale scores for only 4 of the 5 subscales. Then you would also multiply the mean by five.
The potential range of scores for the total score is 5-20, with higher scores indicating better functional status.

IALCH Neurophysiology clinic.2019

Annexure 11: Epworth Sleep Scale Questionnaire

KZ Number: _____

Full name/: _____ Gender: Male Female

Amagama aphelele

Date of Birth/: ____/_____/_____

Usuku lokuzalwa

Today's date/: _____/_____/_____

Usuku lwanamuhla

Weight/Isisisindo: _____ (kg) Height/Ubude: _____ (cm)

Neck circumference/Isikali somqala jikelele: _____ (cm)

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you.

Ingabe kujwayeleke kangakanani ukuthi uzele ude ulale kulezi zimo ezilandelayo, uma kubhekwa ukuzizwa nje ukuthi ukhathele? Lokhu kubhekiswe endleleni oyijwayele yempilo yezikhathi zamuva nje. Ngisho noma ungakaze uzenze ezinye zalezi zinto muva nje, zama indlela yokubheka ukuthi ngabe zikuthinte ngayiphi indlela.

Use the following scale to choose the most appropriate number for each situation/ Sebenzisa lezi zikali ezilandelayo ukukhetha eyona nombolo efanelekile kusimo ngasinye:

0 = Would never doze/Angisoze ngazela ngalala

1 = Slight chance of dozing/Mancane amathuba wokuthi ngingalala

2 = Moderate chance of dozing/Kungezeka ngizele ngilale

3 = High chance of dozing/Maningi amathuba wokuthi ngizele ngilale

#	Situation	Answer
1	Sitting and reading Ngihlezi, ngifunda	
2	Watching TV Ngibuka umabona kude	
3	Sitting, inactive in a public place (e.g. a theatre or a meeting) Ngihlezi, ngingenzi lutho endaweni yomphakathi (isibonelo, eshashalazini noma emhlanganweni)	
4	As a passenger in a car for an hour without a break Njengomgibeli esithuthwini cishe ihora elilodwa ngaphandle kwekhefu.	
5	Lying down to rest in the afternoon when circumstances permit Uma ucambalele ngezikhathi zantambama, nesimo sikuvumela.	
6	Sitting and talking to someone Uhlezi, ukhuluma nomuntu	
7	Sitting quietly after lunch without alcohol Uzihlalele kuthulekile ngemva kwesidlo sasemini ngaphadle kotshwala.	
8	In a car, while stopped for a few minutes in traffic Emotweni, usamile okwemizuzu embalwa ekucinanaeni kwezithuthi.	

To be calculated by the researcher

Scoring Epworth Sleep Scale Questionnaire

< 10: Normal

10 – 16: Mild Hypersomnia

>16: Excessive Hypersomnia

IALCH Neurophysiology clinic. 2019.

Annexure 12: Stop-Bang questionnaire

KZ Number: _____

Full name/: _____ Gender: Male Female

Amagama aphelele

Date of Birth/: ____/_____/_____

Usuku lokuzalwa

Today's date/: _____/_____/_____

Usuku lwanamuhla

Weight/Isisisindo: _____ (kg) Height/Ubude: _____ (cm)

Neck circumference/Isikali somqala jikelele: _____ (cm)

Yes/Yebo	No/Cha	Do you snore loudly (loud enough to be heard through closed doors, or your bed partner elbows you for snoring at night)? Ingabe uhhonqa kakhulu, kuze kuzwakale kuvalwe izicabha, noma umuntu olala eceleni kwakho uze akudshaye ngendololwane ngoba usuke uhhonqa ebusuku nilele?
Yes/Yebo	No/Cha	Do you often feel tired, fatigued, or sleepy during the daytime (such as falling asleep during driving)? Ingabe uzizwa ukhathele isikhathi esiningi, ucobekile, noma uzela ngesikhathi sasemini (isilinganiso, ukulala ushayela)?
Yes/Yebo	No/Cha	Has anyone observed you stop breathing or choking/gasping during your sleep? Ingabe ukhona owake wakubona unqanyukelwa umoya noma ukinyeka ulele ebusuku?
YesYebo	No/Cha	Do you have or are being treated for high blood pressure? Ingabe unaso isifo somfutho wegazi ophakeme noma uyayithola imishanguzo yaso?

Yes/Yebo	No/Cha	What is body mass index more than 35 kg/m^2? Ingabe inkoma yomzimba omkhulu ongaphezulu kuka-35 kg/m^2?
Yes/Yebo	No/Cha	Is your age older than 50 years old? Ingabe iminyaka yakho ingaphuzulu kwengu 50 years old?
Yes/Yebo	No/Cha	Is the neck size large? (measured around Adam's apple) Ingabe isikali sentamo sikhulu? (ikalwa kwi-Adam's apple) For male, is your shirt collar 17 inches or larger? Kubantu besilisa, ingabe ikhola yehembe lakho lingu-17 inches noma ngaphezulu? For females, is your shirt collar 16 inches or larger? Kubantu besifazane, ingabe ikhola yehembe lakho lingu-16 inches noma ngaphezulu.
Yes/Yebo	No/Cha	Is your gender male? Ingabe ubulili bakho bungobesilisa?

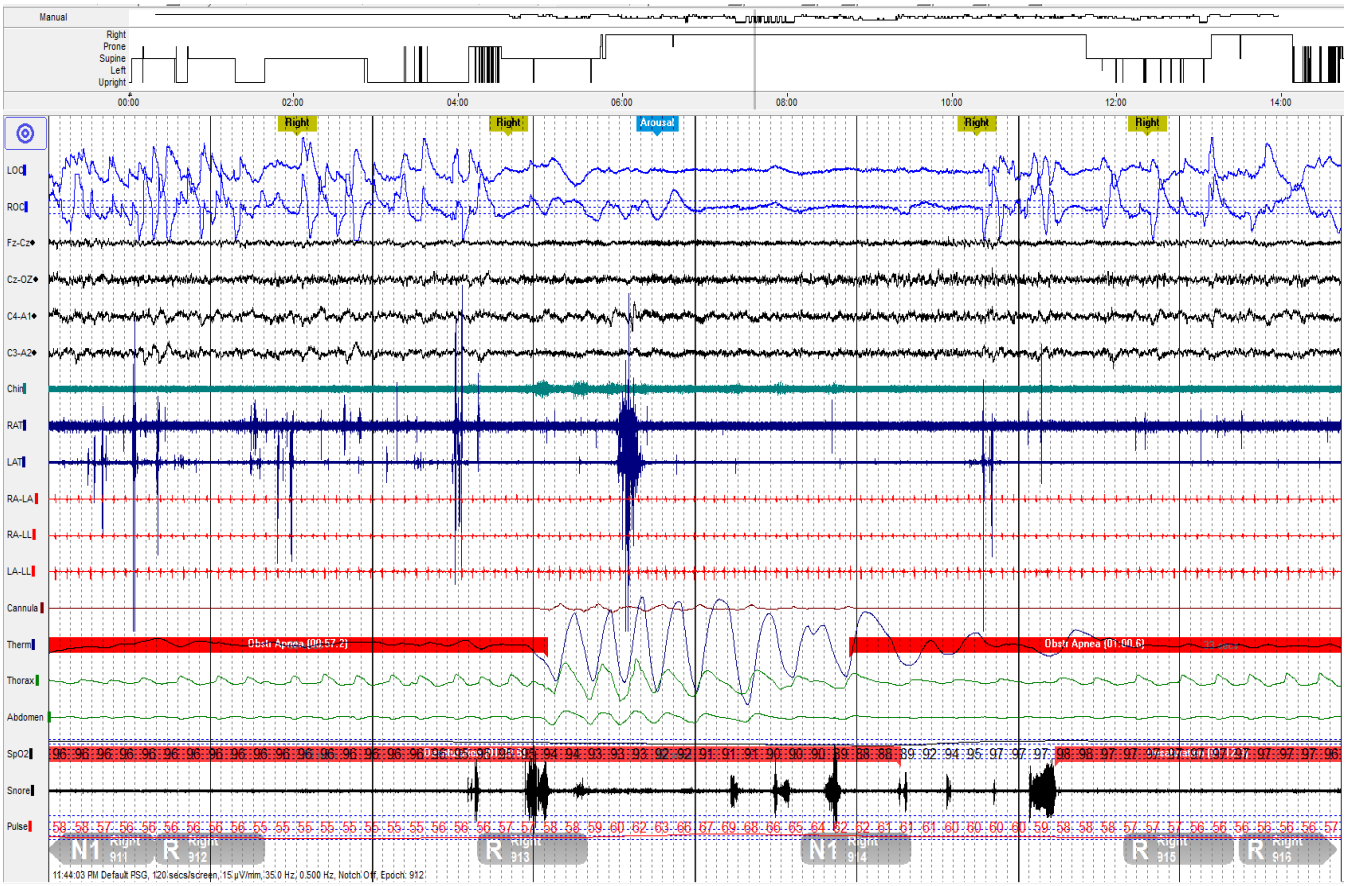
<u>To be calculated by the researcher</u>

Scoring instructions for Stop-Bang Questionnaire

Low risk of OSA: Yes to 0 to 2 questions
Intermediate risk of OSA: Yes to 3 to 4 questions
High risk of OSA: Yes to 5 to 8 questions

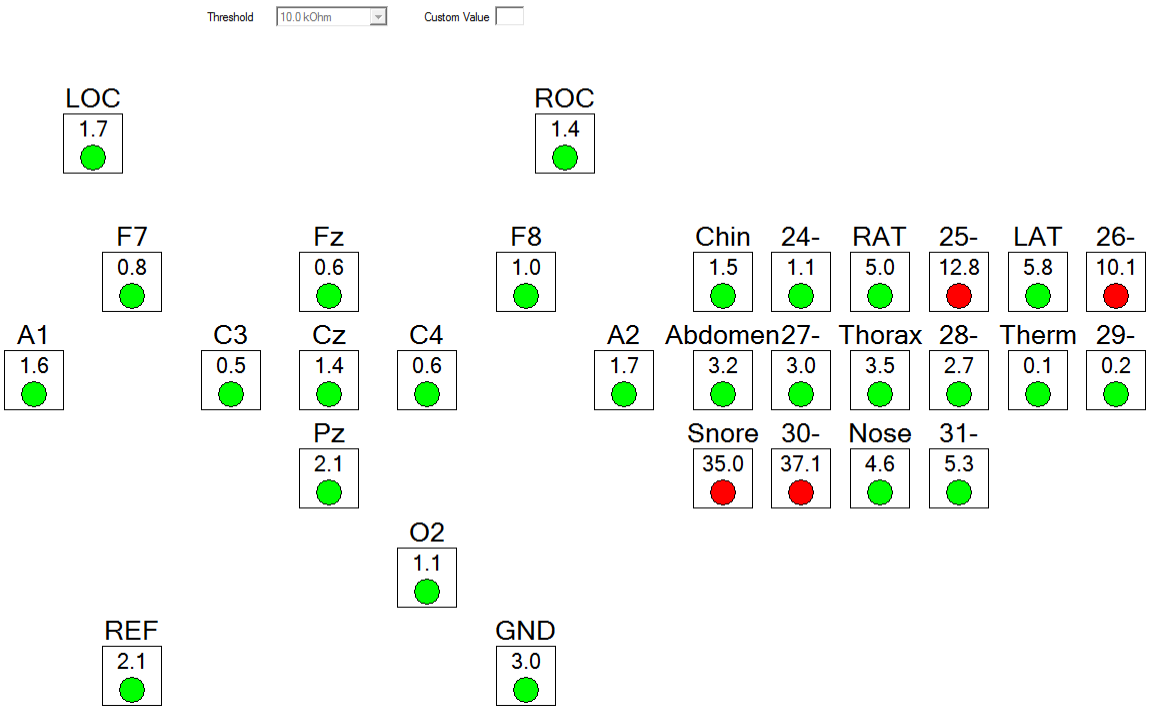
IALCH Neurophysiology clinic.2019.

Annexure 13: A sample of polysomnogram (PSG)18 channels: the generation of PSG waveform



Adapted from IALCH Neurophysiology clinic.

Annexure 14: The placement position of the PSG electrodes



Adapted from IALCH Neurophysiology clinic.

Annexure 15: Technical and digital specifications

III. Technical and Digital Specifications

A. Digital Specifications for Routine PSG Recordings[N1]

1. **Maximum Electrode Impedances: 5 K Ω**[N2] RECOMMENDED

2. **Minimum Digital Resolution: 12 bits per sample** RECOMMENDED

3. **Sampling Rates**

	Desirable	Minimal	
EEG[N3,N4]	500 Hz	200 Hz	RECOMMENDED
EOG[N5]	500 Hz	200 Hz	RECOMMENDED
EMG[N6]	500 Hz	200 Hz	RECOMMENDED
ECG[N7]	500 Hz	200 Hz	RECOMMENDED
Airflow	100 Hz	25 Hz	RECOMMENDED
Oximetry, Transcutaneous PCO_2[N8]	25 Hz	10 Hz	RECOMMENDED
Nasal Pressure, End-Tidal PCO_2, PAP Device Flow[N9]	100 Hz	25 Hz	RECOMMENDED
Esophageal Pressure	100 Hz	25 Hz	RECOMMENDED
Body Position[N10]	1 Hz	1 Hz	RECOMMENDED
Snoring Sounds[N11]	500 Hz	200 Hz	RECOMMENDED
Rib Cage and Abdominal Movements[N12]	100 Hz	25 Hz	RECOMMENDED

4. **Routinely Recorded Filter Settings**

	Low-Frequency Filter	High-Frequency Filter	
EEG[N4,N13]	0.3 Hz	35 Hz	RECOMMENDED
EOG[N13]	0.3 Hz	35 Hz	RECOMMENDED
EMG[N6]	10 Hz	100 Hz	RECOMMENDED
ECG[N14]	0.3 Hz	70 Hz	RECOMMENDED
Oronasal Thermal Flow, Thoracoabdominal Belt Signals	0.1 Hz	15 Hz	RECOMMENDED
Nasal Pressure	Direct current (DC) or ≤0.03 Hz	100 Hz	RECOMMENDED
PAP Device Flow	DC	DC	RECOMMENDED
Snoring	10 Hz	100 Hz	RECOMMENDED

Bio-Cal Procedure

- Allow at least 3 -5 minutes to complete bio-cal
- Start with patient relaxed and sitting up on bed
- Instruct patient to look straight, and remain looking straight until instructed to do otherwise
- Instruct to only move EYES, not head, upon instruction
- Instruct not to talk or move spontaneously during bio-cal procedure
- Give instructions at rate of 1 – 2 seconds to allow for end of movement

1. Close eyes 10 – 30 seconds
2. Open eyes 10 – 30 seconds without blinking

Keep eyes open: only moving your eyes not your head

3. Look Straight
4. Look up
5. Look down
6. Look up
7. Look down
8. Look straight
9. Look left
10. Look right
11. Look left
12. Look right
13. Blink 5 times fast
14. Look straight
15. Close your eyes

With eyes closed: only moving your eye inside your closed lids

16. Roll your eyes up
17. Roll your eyes down
18. Roll your eyes up
19. Roll your eyes down

Look straight still with eyes closed

20. Roll your eyes left
21. Roll your eyes right
22. Roll your eyes left
23. Roll your eyes right
24. Blink 5 times fast

Open your eyes and look straight

25. Bite on your teeth
26. Relax
27. Stretch jaw open
28. Relax
29. Count to ten
30. Say lalalala

Additional breathing procedures

32. Breath normal (At least 10 sec until all bands and airflow channels stable)
33. Tense Left Leg (by dorsiflexion)
34. Tense Right Leg (by dorsiflexion)

Repeat leg calibrations

35. Test snore sensor (Cough and snore and talk/count, lalala)
36. Test EMG (Cough, Tense Jaw by biting and stretching, swallow)
37. Test Thermocouple (Breath through MOUTH ONLY for at least 10sec, until trace has stabilized and cannula trace flat)
38. Breath OUT through mouth
39. Hold breath out 10 seconds: all airflow and effort traces must be flat
40. Breath IN Fast through mouth

May be necessary to hold patient nose closed

This may be repeated several times to ensure you know direction of inhalation deflection: Must be DOWN

41. Test Cannula/PTAF (Breath through nose ONLY for at least 10sec, until trace has stabilized. Thermocouple trace will be active)
42. Breath OUT through NOSE
43. Hold breath out 10 seconds
44. Breath IN Fast through NOSE

This may be repeated several times to ensure you know direction of inhalation deflection: Must be DOWN

45. Breath out and hold out while moving tummy in and out to test breathing bands and SUM
46. Breath normal until you can adjust all effort and airflow polarities to reflect inhalation DOWN

Annexure 16: Application for permission for private/part-time study

ANNEXURE A

health

Department:
Health
PROVINCE OF KWAZULU-NATAL

Inkosi Albert Luthuli Central Hospital
Department: Human Resources
Postal Address : Private Bag x03 Mayville
Physical Address: 800 Vusi Mzimela Road Mayville
Tel.: 031-240 1019 Fax.: 031-240 1089
Email.: cebilehle@ialch.co.za
www.kznhealth.gov.za

APPLICATION FOR PERMISSION FOR PRIVATE/PART-TIME STUDY

APPLICANT'S FULL NAME: JABANATHI KIBI

PERSAL NUMBER: 65 34 9334

JOB TITLE: Junior Neuro-Clinical Technologist

WARD / DEPARTMENT: NEUROPHYSIOLOGY

QUALIFICATION APPLYING FOR: Master of health Clinical Technology

SUBJECTS / COURSES / MODULES: Thesis (1st registration).

EDUCATIONAL INSTITUTION: Durban University of Technology.

ANTICIPATED LEAVE:

STUDY/RESEARCH: 2 DAYS PER EXAMINATION, LIMITED TO 10 DAYS PER ANNUM: 10 days.

8 - HOUR (ATTENDANCE OF CLASSES) LEAVE:

EXAMINATION DATES:

PREPARATION DATES:

SIGNATURE OF APPLICANT — DATE: 29/04/2019

APPLICATION RECOMMENDED / NOT RECOMMENDED

SIGNATURE OF IMMEDIATE SUPERVISOR — DATE: 29/4/19.

APPLICATION APPROVED / NOT APPROVED

SIGNATURE OF SENIOR MANAGER — DATE: 29/04/2019

Annexure 17: Introduction to research ethics certificate

Zertifikat
Certificat
Certificado
Certificate

TRREE

Promouvoir les plus hauts standards éthiques dans la protection des participants à la recherche biomédicale
Promoting the highest ethical standards in the protection of biomedical research participants

Clinical Trials Centre
The University of Hong Kong

Certificat de formation - Training Certificate

Ce document atteste que - this document certifies that

Yabanathi Kibi

a complété avec succès - has successfully completed

Introduction to Research Ethics

du programme de formation TRREE en évaluation éthique de la recherche
of the TRREE training programme in research ethics evaluation

Release Date: 2020/11/30

CID : MnlwbmBtuM

Professeur Dominique Sprumont
Coordinateur TRREE Coordinator

FMH Continuing Education Program (5 Credits)
Programme de Formation continue (5 Crédits)

Foederatio Pharmaceutica Helvetiae FPH Programmes de formation continue Continuing Education Programms
Programmes de formation continue

Ce programme est soutenu par - This program is supported by :

European and Developing Countries Clinical Trials Partnership (EDCTP) (www.edctp.org) - Swiss National Science Foundation (www.snf.ch) - Canadian Institutes of Health Research (http://www.cihr-irsc.gc.ca/e/2891.html) - Swiss Academy of Medical Science (SAMS/ASSM/SAMW) (www.samw.ch) - Commission for Research Partnerships with Developing Countries (www.kfpe.ch)

[REV : 20170310]

Annexure 18: Research ethics evaluation certificate

TRREE

Zertifikat
Certificat
Certificado
Certificate

Promouvoir les plus hauts standards éthiques dans la protection des participants à la recherche biomédicale
Promoting the highest ethical standards in the protection of biomedical research participants

Clinical Trials Centre
The University of Hong Kong

Certificat de formation - Training Certificate

Ce document atteste que - this document certifies that

Yabanathi Kibi

a complété avec succès - has successfully completed

Research Ethics Evaluation

du programme de formation TRREE en évaluation éthique de la recherche
of the TRREE training programme in research ethics evaluation

Release Date: 2020/12/03
CID : zjAjLkZBGc

Professeur Dominique Sprumont
Coordinateur TRREE Coordinator

FMH Continuing Education Program (5 Credits)
Programme de Formation continue (5 Crédits)

Foederatio Pharmaceutica Helvetiae FPH Programmes de formation continue
Continuing Education Programms
Programmes de formation continue

Ce programme est soutenu par - This program is supported by :
European and Developing Countries Clinical Trials Partnership (EDCTP) (www.edctp.org) - Swiss National Science Foundation (www.snf.ch) - Canadian Institutes of Health Research (http://www.cihr-irsc.gc.ca/e/2891.html) - Swiss Academy of Medical Science (SAMS/ASSM/SAMW) (www.samw.ch) - Commission for Research Partnerships with Developing Countries (www.kfpe.ch)

[REV : 20170310]

Annexure 19: Informed consent certificate

TRREE

Zertifikat
Certificat
Certificado
Certificate

Promouvoir les plus hauts standards éthiques dans la protection des participants à la recherche biomédicale
Promoting the highest ethical standards in the protection of biomedical research participants

Clinical Trials Centre
The University of Hong Kong

Certificat de formation - Training Certificate

Ce document atteste que - this document certifies that

Yabanathi Kibi

a complété avec succès - has successfully completed

Informed Consent

du programme de formation TRREE en évaluation éthique de la recherche
of the TRREE training programme in research ethics evaluation

Release Date: 2020/12/10
CID : cmlDWD6S0I

Professeur Dominique Sprumont
Coordinateur TRREE Coordinator

FMH Continuing Education Program (5 Credits)
Programme de Formation continue (5 Crédits)

Foederatio Pharmaceutica Helvetiae FPH Programmes de formation continue Continuing Education Programms
Programmes de formation continue

Ce programme est soutenu par - This program is supported by :
European and Developing Countries Clinical Trials Partnership (EDCTP) (www.edctp.org) - Swiss National Science Foundation (www.snf.ch) - Canadian Institutes of Health Research (http://www.cihr-irsc.gc.ca/e/2891.html) - Swiss Academy of Medical Science (SAMS/ASSM/SAMW) (www.samw.ch) - Commission for Research Partnerships with Developing Countries (www.kfpe.ch)

[REV : 20170310]

Annexure 20: Questionnaires, weight versus ANPSG and OPSG conclusions

Questionnaires, Weight versus ANPSG and OPSG conclusions

Number of Candidates	4 Questionnaires Conclusion	Weight (kg)	ANPSG Conclusion(RDI&AHI)	OPSG Conclusion
	A= Berlin Questionnaire, B= Functional outcome of sleep questionnaire, C= Epworth sleep scale questionnaire, D= Stop-bang questionnaire			
1	A= High risk of sleep apnoea, B = Poor functional status, C = Normal, D= High risk of OSA.	157.6	Severe non-positional OSAHS with abnormal sleep architecture	Severe non positional OSAHS with abnormal sleep architecture
2	A= High risk of sleep apnoea, B = Poor functional status, C = Mild hypersomnia, D= High risk of OSA.	146.95	Severe non positional OSAHS with abnormal sleep architecture	Severe non-positional OSA with abnormal sleep architecture HS
3	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= Intermediate risk of OSA.	72	Moderate non-positional OSAHS with abnormal sleep architecture	Severe non-positional OSAHS with abnormal sleep architecture
4	A= High risk of sleep apnoea, B= Very poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	113.30	Severe non-positional OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
5	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	128.45	Severe non-positional OSAHS with abnormal sleep architecture	Severe non-positional OSAHS with abnormal sleep architecture
6	A= High risk of sleep apnoea, B= Poor functional status, C=Normal, D= Intermediate risk of OSA	70.60	Moderate UAOS and OSAHS with abnormal sleep architecture	Severe UAOS and OSAHS with abnormal sleep architecture
7	A= High risk of sleep apnoea, B= Poor functional status, C=Normal, D= Intermediate risk of OSA	120.05	Severe UAOS and OSAHS with abnormal sleep architecture	Severe UAOS and OSAHS with abnormal sleep architecture
8	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= High risk of OSA	124.65	Severe UAOS and OSAHS with abnormal sleep architecture	Severe UAOS and OSAHS with abnormal sleep architecture
9	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= High risk of OSA	96.60	Severe non-positional OSAHS with abnormal sleep architecture	Severe non-positional OSAHS

10	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= High risk of OSA	126.35	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
11	A= High risk of sleep apnoea, B= Very poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	83.45	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
12	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	90.20	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
13	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	114.55	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
14	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= High risk of OSA.	86.40	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
15	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= low risk of OSA.	45.60	Mild OSAHS	Mild to moderate OSAHS
16	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= Intermediate risk of OSA.	62.25	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
17	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= High risk of OSA.	105.25	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
18	A= High risk of sleep apnoea, B= Poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	91.20	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
19	A= High risk of sleep apnoea, B= Very poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	61.95	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
20	A= High risk of sleep apnoea, B= Very poor functional status, C=Mild hypersomnia, D= High risk of OSA.	137.35	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
21	A= High risk of sleep apnoea, B= Very poor functional status, C=Mild hypersomnia, D= High risk of OSA.	138.45	Moderate OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
22	A= Low risk of sleep apnoea, B= Moderate functional status, C= Normal, D= Low risk of OSA.	106.55	No sleep apnoea	No Sleep apnoea
23	A= High risk of sleep apnoea, B= Moderate functional status, C= Mild hypersomnia, D= High risk of OSA.	88.30	Moderate OSAHS	Moderate OSAHS

24	A= High risk of sleep apnoea, B= Poor functional status, C= Mild hypersomnia, D= High risk of OSA.	113.95	Moderate OAHS	Moderate OSAHS
25	A= High risk of sleep apnoea, B= Very poor functional status, C= Excessive hypersomnia, D= High risk of OSA.	96.55	Severe OSAHS	Severe OSAHS

Annexure 21: DUT ethics clearance certificate

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

P O Box 1334, Durban, South Africa, 4001

Tel: 031 373 2375
Email: lavishad@dut.ac.za
http://www.dut.ac.za/research/institutional_research_ethics

www.dut.ac.za

13 May 2021

Ms Y Kibi
3A Kinloch Avenue
Atholl Heights
Westville
Durban
3629

Dear Ms Kibi

The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal
Ethics Clearance number: IREC 143/20

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

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

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

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

Yours Sincerely

Dr K Padayachy
Deputy Chairperson: IREC

ENVISION2030 transparency • honesty • integrity • respect • accountability
fairness • professionalism • commitment • compassion • excellence

Annexure 22: KZN Department of Health approval

KWAZULU-NATAL PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

DIRECTORATE:
Health Research & Knowledge Management Unit

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

NHRD Ref: KZ_202105_004

Dear Ms Y Kibi
(DUT)

Approval of research

1. The research proposal titled **'The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal.'** was reviewed by the KwaZulu-Natal Department of Health (KZN-DoH).

   The proposal is hereby **approved** for research to be undertaken at Inkosi Albert Luthuli Central hospital.

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

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

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

Yours Sincerely

Dr E Lutge
Chairperson, Health Research Committee
Date: 14/07/2021.

GROWING KWAZULU-NATAL TOGETHER

Annexure 23: Out patient sleep information

health
Department:
Health
PROVINCE OF KWAZULU-NATAL

DIRECTORATE: NEUROPHYSIOLOGY

Physical Address: 800 Vusi Mvelase Road
Postal Address: Private Bag X03, Mayville, 4058
Tel: 031 2401030 Fax: 031 2401089 Email: christelle.vanderwalt@ialch.co.za
www.kznhealth.gov.za

Inkosi Albert Luthuli Central Hospital
Neurophysiology Department

Dear Mr/Mrs/Ms/Dr

Below are the instructions regarding your upcoming SLEEP STUDY on the .....

1. → You are scheduled for a SLEEP STUDY TEST (PSG).
   → The Sleep study department will contact you with a day per SMS
   → **PLEASE COME ON THE INDICATED DATE**
2. → You MUST arrive at IALCH at 8:00 am to register, then proceed to Neurophysiology department, no later than 10:00 am.

YOUR DOCTOR WILL PROVIDE AN OUT-PATIENT PRESCRIPTION FOR A SLEEP MEDICATION. YOU MUST BRING MEDICATION WITH, BUT NOT TAKE IT UNTIL INSTRUCTED BY THE SLEEP STUDY TECHNOLOGISTS.

- → PATIENT TO REGISTER AS NEUROPHYSIOLOGY OUTPATIENT, BUT WILL BE SEEN IN A3E SLEEP LAB
- → PATIENT TO ARRIVE AT NEUROPHYSIOLOGY DEPARTMENT **NO LATER THAN 10:00**.
- → **MUST BRING OWN LUNCH/SNACKS**
- → **NO CAFFEINE ON DAY OF STUDY AFTER 06:00**
- → **NO SMOKING AFTER 10:00**

MUST COME WITH CLEAN DRY, NATURAL HAIR -- NO WEAVES, ATTACHMENTS OR PLATING AS THIS INTERFERES WITH THE STUDY (NO SHINE SPRAY, HAIRSPRAY, GELS OR CREAM TREATMENTS)

MUST BRING COMFORTABLE CLOTHES TO SLEEP IN

MUST ARRANGE ACCOMPANIED TRANSPORT AS SEDATIVE MAY BE ADMINISTERED.

REPORT TAKES MIMIMUM OF 14 DAYS FOR FINALISATION.

Annexure 24: Editing certificate

DR RICHARD STEELE
BA HDE MTech(Hom)
HOMEOPATH
Registration No. A07309 HM
Practice No. 0807524
Freelance academic editor
Associate member: Professional Editors' Guild, South Africa

154 Magenta Place
Morgan Bay
5292
Eastern Cape

082-928-6208
rsteele@vodamail.co.za

EDITING CERTIFICATE

Re: **Yabanathi Kibi**
DUT master's thesis: **The correlation of apnoea-hypopnoea index in afternoon nap polysomnography and overnight polysomnography at a health establishment in KwaZulu-Natal**

I confirm that I have edited this dissertation and the references for clarity and language. I returned the document to the author with track changes so correct implementation of the changes and clarifications requested in the text and references is the responsibility of the author. I am a freelance editor specialising in proofreading and editing academic documents. My original tertiary degree which I obtained at the University of Cape Town was a B.A. with English as a major and I went on to complete an H.D.E. (P.G.) Sec. with English as my teaching subject. I was a part-time lecturer in the Department of Homoeopathy at the Durban University of Technology for 13 years and supervised many master's degree dissertations during that period.

Dr Richard Steele
18 December 2022
per email

Annexure 25: Turnitin similarity index report

Turnitin Originality Report

MH Clinical Technology by Yabanathi Kibi

From Yabanathi Kibi (Kibi Yabanathi Masters Dissertation)

- Processed on 24-Apr-2023 09:58 SAST
- ID: 2073809305
- Word Count: 23368

Similarity Index
15%
Similarity by Source

Internet Sources:
12%
Publications:
11%
Student Papers:
N/A

Annexure 26: BMI, neck circumference, weight, and height versus questionnaires, indexes, and PSG conclusions

Number	Parameters		Questionnaires		Indexes				Conclusion	
					ANPSG		OPSG		ANPSG	OPSG
					RDI (#/sleep hr.)	AHI(#/sleep hr.)	RDI(#/sleep hr.)	AHI(#/sleep hr.)		
1	BMI	61.6 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	45.89	47.91	99.15	86.60	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	43 cm	Functional outcome of sleep questionnaire	Poor functional Status						
	Weight	157.6 kg	Epworth sleep scale questionnaire	Normal						
	Height	1.60 m	Stop-bang questionnaire	High risk of OSA						
2	BMI	45.9 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	38.81	44.60	57.21	44.81	Severe OSAHS with abnormal sleep architecture.	Severe OSAHS with abnormal sleep architecture.
	Neck Circumference	44 cm	Functional outcome of sleep questionnaire	Poor functional Status						
	Weight	146.95 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.79 m	Stop-bang questionnaire	High risk of OSA						
3	BMI	28.5 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	28.98	19.89	76.85	68.35	Moderate non-positional OSAHS with abnormal sleep	Severe non-positional OSAHS with abnormal sleep

									architecture.	architecture.
	Neck Circumference	42 cm	Functional outcome of sleep questionnaire	Poor functional Status						
	Weight	72 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.59 m	Stop-bang questionnaire	Intermediate risk of OSA						
4	BMI	32 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	51.09	44.09	71.62	65.31	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	46.5	Functional outcome of sleep questionnaire	Very poor functional Status						
	Weight	113.30	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.9 m	Stop-bang questionnaire	High risk of OSA						
5	BMI	49 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	51.59	45.61	53.04	39.75	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	41 cm	Functional outcome of sleep questionnaire	Poor functional Status						
	Weight	128.45 kg	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.62 m	Stop-bang	High risk of OSA						

			questionnaire							
6	BMI	25.0 kg/m^2	Berlin questionnaire	High risk of OSA	25.83	22.99	51.87	44.77	Moderate UAOS and OSAHS with abnormal sleep architecture.	Severe UAOS and OSAHS with abnormal sleep architecture.
	Neck Circumference	34.5 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	70.60 kg	Epworth sleep scale questionnaire	Normal						
	Height	1.68 m	Stop-bang questionnaire	Intermediate risk of OSA						
7	BMI	43.6 kg/m^2	Berlin questionnaire	High risk of OSA	54.91	51.51	96.80	92.55	Severe UAOS and OSAHS with abnormal sleep architecture.	Severe UAOS and OSAHS with abnormal sleep architecture.
	Neck Circumference	39 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	120.05 kg	Epworth sleep scale questionnaire	Normal						
	Height	1.66 m	Stop-bang questionnaire	Intermediate risk of OSA						
8	BMI	46.9 kg/m^2	Berlin questionnaire	High risk of sleep apnea	95.58	88.97	130.11	126.93	Severe UAOS and OSAHS with abnormal sleep architecture.	Severe UAOS and OSAHS with abnormal sleep architecture.
	Neck Circumference	40 cm	Functional outcome of sleep questionnaire	Poor functional status						

	Weight	124.65kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.63 m	Stop-bang questionnaire	High risk of OSA						
9	BMI	33.6 kg/m^2	Berlin questionnaire	High risk of sleep apnea	113.38	111.48	121.23	116.76	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	46cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	96.60 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.695 m	Stop-bang questionnaire	High risk of OSA						
10	BMI	38.8 kg/m^2	Berlin questionnaire	High risk of sleep apnea	82.76	81.38	75.58	65.47	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	49cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	126.35kg	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.805 m	Stop-bang questionnaire	High risk of OSA						
11	BMI	26.3 kg/m^2	Berlin questionnaire	High risk of sleep apnea	49.57	47.60	53.73	44.13	Severe non-positional OSAHS with	Severe non-positional OSAHS with

									abnormal sleep architecture.	abnormal sleep architecture.
	Neck Circumference	43cm	Functional outcome of sleep questionnaire	Very poor functional status						
	Weight	83.450kg	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.78m	Stop-bang questionnaire	High risk of OSA						
12	BMI	34.8 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	69.29	67.88	74.32	72.12	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	41cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	90.20 kg	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.61m	Stop-bang questionnaire	High risk of OSA.						
13	BMI	37.4 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	90.78	82.91	81.24	76.40	Severe non-positional OSAHS with abnormal sleep architecture.	Severe non-positional OSAHS with abnormal sleep architecture.
	Neck Circumference	47 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	114.55 kg	Epworth sleep scale questionnaire	Excessive hypersomnia						

	Height	1.75	Stop-bang questionnaire	High risk of OSA.						
14	BMI	40.5 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	98.5	89	100.09	95.42	Severe OSAHS with abnormal sleep architecture.	Severe OSAHS with abnormal sleep architecture.
	Neck Circumference	40 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	86.40 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.46 m	Stop-bang questionnaire	High risk of OSA.						
15	BMI	18.98 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	5.69	5.21	18.71	11.36	Mild OSAHS	Mild to moderate OSAHS
	Neck Circumference	30 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	45.60 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.55 m	Stop-bang questionnaire	Low risk of OSA.						
16	BMI	23.41 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	87.36	87.36	90.70	88.67	Severe OSAHS with abnormal sleep architectures	Severe OSAHS with abnormal sleep architectures
	Neck Circumference	33cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	62.25 kg	Epworth sleep scale questionnaire	Excessive hypersomnia						

	Height	1.63 m	Stop-bang questionnaire	Intermediate risk of OSA.						
17	BMI	32 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	47.31	38.11	61.90	54.95	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
	Neck Circumference	46 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	105.25 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.82 m	Stop-bang questionnaire	High risk of OSA.						
18	BMI	30.47 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	117.27	115.44	119.49	102.67	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
	Neck Circumference	46 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	91.20 kg	Epworth sleep scale questionnaire	Excessive hypersomnia						
	Height	1.73 m	Stop-bang questionnaire	High risk of OSA.						
19	BMI	21 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	68.10	67.62	77.27	75.09	Severe OSAHS with abnormal sleep architecture	Severe OSAHS with abnormal sleep architecture
	Neck Circumference	43cm	Functional outcome of sleep questionnaire	Very poor functional status						
	Weight	61.95kg	Epworth sleep scale	Excessive hypersomnia						

			questionnaire							
	Height	1.73 m	Stop-bang questionnaire	High risk of OSA.						
20	BMI	50.45 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	43.04	40.54	62.42	59.60	Severe non-positional OSAHS	Severe non-positional OSAHS
	Neck Circumference	41 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	137.35 kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.64 m	Stop-bang questionnaire	High risk of OSA.						
21	BMI	57 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	23.68	21.49	50.91	48.94	Moderate OSAHS with abnormal sleep architecture	Severe non-positional OSAHS
	Neck Circumference	45 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	138.45kg	Epworth sleep scale questionnaire	Mild hypersomnia						
	Height	1.57m	Stop-bang questionnaire	High risk of OSA.						
22	BMI	38.42 kg/m^2	Berlin questionnaire	Low risk of sleep apnoea	2.35	0	2.54	2.54	No sleep apnoea noted	No sleep apnoea noted
	Neck Circumference	36 cm	Functional outcome of sleep questionnaire	B= Moderate functional status						
	Weight	106.55 kg	Epworth sleep scale questionnaire	Normal						

	Height	1.665 m	Stop-bang questionnaire	Low risk of OSA.						
23	BMI	38.33kg /m^2	Berlin questionnaire	High risk of sleep apnoea	23.56	16.83	29.17	25.68	Moderate non-positional OSAHS	Moderate non-positional OSAHS
	Neck Circumference	42cm	Functional outcome of sleep questionnaire	Moderate functional status						
	Weight	88.30 kg	Epworth sleep scale questionnaire	Mild hypersomnia, D						
	Height	1.52 m	Stop-bang questionnaire	High risk of OSA.						
24	BMI	46.82 kg/m^2	Berlin questionnaire	High risk of sleep apnoea	26.91	26.01	31.11	28.20	Moderate non-positional OSAHS	Moderate non-positional OSAHS
	Neck Circumference	39 cm	Functional outcome of sleep questionnaire	Poor functional status						
	Weight	113.95 kg	Epworth sleep scale questionnaire	Mild hypersomnia, D						
	Height	1.56 m	Stop-bang questionnaire	Intermediate risk of OSA.						
25	BMI	38kg/ m^2	Berlin questionnaire	High risk of sleep apnoea	136.49	127.48	134.82	131.97	Severe non-positional OSAHS	Severe non-positional OSAHS
	Neck Circumference	39 cm	Functional outcome of sleep questionnaire	Very poor functional status						
	Weight	96.55 kg	Epworth sleep scale questionnaire	Excessive hypersomnia, D						
	Height	1.585	Stop-bang questionnaire	High risk of OSA.						

Annexure 27: Tests of normality for table 4

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Agegroup	,141	25	,200*	,943	25	,177
BMI	,095	25	,200*	,981	25	,913
Neck Circumference	,138	25	,200*	,954	25	,315
Weight	,079	25	,200*	,988	25	,985
Height	,106	25	,200*	,981	25	,913

*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction