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The Relationship Between Methods of Calculating the AHI and Daytime Sleepiness, Severity of Sleep Apnea, and CPAP Pressure Settings

Kathryn J. Kane
Walden University

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Walden University
2019

Abstract

The Relationship Between Methods of Calculating the AHI and Daytime Sleepiness,

Severity of Sleep Apnea, and CPAP Pressure Settings

by

Kathryn J. Kane

MA, Walden University, 2006

BS, Loyola University, 2002

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Clinical Psychology

Walden University

March 2019

Abstract

Sleep apnea impacts one's mental and physical health, and has social consequences which affect all of society. Not only is there is a weak correlation between the severity of sleep apnea and daytime sleepiness as reported on the Epworth Sleepiness Scale (ESS), but there is also a weak correlation between the severity of sleep apnea and prescribed treatment as indicated by 95th percentile pressure on a continuous positive airway pressure (CPAP) machine. This poses difficulty for providers as well as patients throughout screening and treatment. The purpose of this study was to use a biopsychosocial approach to perform a within-subjects analysis in order to quantitatively investigate methods of calculating the severity of sleep apnea. The study included historical data of 75 participants, each diagnosed with obstructive sleep apnea, prescribed Automatic CPAP therapy, and compliant with treatment. The Apnea Hypopnea Index (AHI) was calculated in the current fashion and also while considering the length of a respiratory event, type of respiratory event, and the combination of these two factors. Linear regression was used to determine if there is a significant difference in the relationships between the AHI and the ESS as well as the AHI and the 95th percentile CPAP pressure. Results endorsed neither a strong relationship between the AHIs and the ESS nor a strong relationship between the AHIs and 95th percentile CPAP pressure either. However, the relationships were somewhat stronger when considering the length and type of the respiratory event. Findings support a need for future research to explore these relationships and offer more accurate screening and treatment of individuals with sleep apnea. Ultimately, those with sleep apnea will experience an improvement in mental, physical and social functioning which may positively impact those without sleep apnea.

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Dedication

This dissertation is dedicated to my husband, children, and my staff. All of you have been supportive throughout my education. To the Dream Team: I could not have completed my education without the hard work each of you has displayed throughout your years with me. I sincerely appreciate each and every one of you.

To my husband, Bill: I am so thankful for the support you have given me while I was off working, writing, reading, and not sleeping! Thanks for all the snacks and meals you prepared during these difficult years; I will never take you for granted.

And to my children, Bishop and Iris: You will never know how much I appreciate your patience with me during this busy time. I wish I could have taken more breaks and played more games with you both, but I hope you will appreciate the sacrifices we all made to help me achieve this goal. I love you both infinity!

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At this time, I wish to acknowledge Dr. Jessica Tischner for her direction and guidance through the process. Life has a tendency to get in the way, but Dr. Tischner was able to keep me going and help me finally get to the end. Thank you.

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Chapter 1: Introduction to the Study

Though the exact function of sleep is still unknown, studies have shown that sleep is a vital human function (Kryger, Roth, & Dement, 2000). Sleep is characterized by 4 stages; Stages N1 through N3 comprise non-rapid eye movement (NREM) sleep, while the final stage is known as Stage R, or rapid eye movement (REM). One sleep cycle consists of light sleep, slow-wave sleep (SWS; also known as deep sleep), and REM sleep. Individuals experience multiple sleep cycles each night (Kryger et al., 2000). There are many factors that affect the quality of sleep a person has including age, genetic disposition, general medical health, medications used, alcohol consumption, psychiatric health, sleep hygiene and/or the presence of a sleep disorder, to name a few (Aldrich, 1999). One such sleep disorder that affects the quality of one's sleep includes obstructive sleep apnea (OSA).

Perhaps the most commonly discussed sleep disorder, snoring and OSA has become increasingly publicized in the media. OSA is a respiratory disorder during sleep that is characterized by the cessation of airflow for periods of time. Many patients with OSA experience disturbed sleep throughout the night. A symptom of both snoring and OSA is an abundance of neurological arousals. These arousals awaken the individual from continuous sleep progression and typically prohibit the person from entering or maintaining deep sleep periods in which the throat muscles are more relaxed. Though a person may not physically wake up (or remember waking), the brain wakes from deep sleep and re-enters light sleep in which the throat muscles are less susceptible to collapsing (Aldrich, 1999).

In the field of sleep medicine, practitioners conduct overnight sleep studies to detect sleep disorders such as OSA in patients (Boone, 2004; de Souza et al., 2003). These studies allow for a thorough assessment of physiological parameters such as breathing and respiratory effort (Kryger et al., 2000). Medical practitioners must rely on screening tools to help determine which patients should seek further diagnostic care such as a sleep study. Many healthcare practitioners administer the Epworth Sleepiness Scale (ESS), to screen individuals for symptoms of daytimes sleepiness (Johns, 1992). An elevated score on this measure (ESS > 10) may result in a healthcare practitioner ordering a formal diagnostic test, such as a sleep study, to determine if sleep apnea (or another sleep disorder) exists. Although researchers have conducted multiple studies to support the validity of the ESS, it is not strongly correlated with the presence and severity of sleep apnea, which leaves many healthcare practitioners without a useful way of screening for OSA (Guimaraes, Martins, Vaz Rodriguez, Teixira & Moutinho dos Santos, 2012). Instead, the severity of OSA is determined during an overnight diagnostic sleep study.

The severity of sleep apnea is defined by counting the total number of apneas (90% or greater reduction in respiratory flow, for 10 seconds or longer) plus hypopneas (30% or greater reduction in respiratory flow, for 10 seconds or longer, and an associated desaturation in oxygen saturation by 3% or more, and/or an arousal or fragmentation of sleep) and dividing by the TST (TST) (Berry et al., 2017). This yields the apnea-hypopnea index (AHI). This number is often utilized by healthcare practitioners to make

decisions about the individual's current and future healthcare (Dempsey, Veasey, Morgan, & O'Donnell, 2010).

In the current rules for the calculation of the AHI, both apneas and hypopneas are weighed equally as respiratory events, and the length of time each event occurrs is not a factor in the calculation. Thus, a 50-second apnea is seen as equivalent to a 10-second hypopnea (Berry et al., 2017). Consequently, it appears that the sleep apnea diagnosis of one patient with an AHI of 20 would be equivalent to any other patient with an AHI of 20 events per hour of TST. However, when comparing the raw data, the patient with 50-second apneas seems to have a more compromised respiratory system than the patient with 10-second hypopneas. As such, if the two patients had significantly different severity levels of sleep apnea, they may have proportionately different daytime symptoms as well as treatment plans. Since the AHI calculation does not consider the length and severity of the respiratory events, it would seem as though the correlation between AHI and ESS is not strong.

Methods of treatment for OSA and snoring include the following: surgical options, continuous positive airway pressure (CPAP), and dental appliances. The most common method is CPAP, also known as CPAP therapy. CPAP therapy involves a mask worn over the nose and/or mouth which is attached to a machine that delivers pressurized air to the patient to hold the airway open during the night. This treatment is very successful in relieving nearly all symptoms of OSA as well as snoring (Carswell et al., 2004). Unfortuantely, Sawyer et al. (2011) indicate that anywhere between 29 and 83% of individuals diagnosed with OSA do not adhere to CPAP therapy. Because the

prescribed pressure setting will determine the success of treatment, it is a critical factor when considering adherence. If an individual's diagnosed level of sleep apnea severity is not accurate, the prescribed pressure settings may also not be appropriate. Furthermore, treatment of OSA can be difficult to predict with the current method of AHI calculation. Cancelo et al. (2013) found a very poor relationship between AHI and the prescribed CPAP pressure. Because of the low predictive value of the AHI when determining CPAP pressure (Oksenberg, Arons & Froom, 2006), there is a need to explore the way in which the AHI is calculated.

Background

In the field of sleep medicine, polysomnography (PSG) is considered to be the gold standard in detecting sleep disorders (Boone, 2004; de Souza et al., 2003). PSG studies allow for a thorough assessment of physiological parameters such as brain waves (EEG), breathing, respiratory effort, muscle movement, heart rate and rhythm, and more (Kryger et al., 2000). Standard procedures include the application of surface electrodes to the head to monitor neural patterns, a pressure sensitive device that measures airflow, elastic belts on the torso to monitor respiratory effort, an SaO_2 probe to measure oxygen saturation levels in the blood, and electrocardiogram electrodes to monitor cardiac activity. PSG studies are typically performed overnight in a clinical setting and involve numerous wires being attached to the patient. The data retrieved from PSG is analyzed to determine the presence of respiratory disturbances such as apneas and hypopneas. In conjunction with the other parameters of sleep, the AHI is calculated by determining the number of times an individual has a respiratory disturbance per hour of TST.

If an individual frequently experiences these respiratory disturbances during sleep, a diagnosis of OSA may be given. OSA is a disease in which a person suffers from respiratory difficulties while sleeping due to a physical obstruction in one's airway. This disease progresses from snoring to periods of time where one ceases to breathe while sleeping. It results from a physical obstruction in the airway, often caused by a collapsing pharyngeal airway due to extra tissue growth or relaxed throat muscles in sleep. As one falls asleep and the muscles begin to relax, the extra tissue will continue to move closer together until the airway is obstructed. Snoring is precursor and sign of OSA. Snoring is the noise that results from a partial obstruction in the air passages, while OSA is a complete obstruction. Snoring and OSA are usually accompanied by a decrease in blood oxygen levels, frequently seen as low as 70%, due to the decrease in airflow. Furthermore, many patients with severe OSA wake up gasping for air when the airway collapses completely. This is the body's defense mechanism against death or other medical consequences. Similarly, a symptom of both snoring and OSA is an abundance of neurological arousals. These arousals "wake" a patient from continuous sleep progression and typically prohibit the person from entering or maintaining deep sleep periods in which the throat muscles are more relaxed. Though a person may not physically wake up (or remember waking), the brain wakes from deep sleep and re-enters light sleep in which the throat muscles are less susceptible to collapsing. (Aldrich, 1999)

Symptoms of OSA as well as sleep fragmentation include excessive daytime sleepiness, irritability, reduced libido, dry mouth and sore throat, frequent nocturnal awakenings, and heavy nighttime sweating. Morning headaches are also a classic sign

and symptom of OSA resulting from the lack of oxygen to bodily tissues including the brain. Recent research has shown that OSA may be a leading contributing factor in the development of other medical diseases such as heart disease, lung disease, strokes, and diabetes (Leung & Bradley, 2001). Neural, cardiac, and pulmonary activity can be greatly affected by the cessation of airflow resulting in low oxygen levels; it is easy to see how heart and lung disease can accumulate over the years. Other associated conditions include diabetes, obesity, hypertension, gastroesophageal reflux disease (GERD), and more. Frequently, the diagnosis and subsequent treatment of OSA will improve the affiliated conditions (Aldrich, 1999; Carswell et al., 2004).

One form of treatment for OSA is CPAP in which a machine delivers pressurized air to hold the airway open like an air splint. The individual wears a mask that covers the nose and/or mouth; this mask is connected to the machine with six-foot long tubing to allow the machine a medium through which to deliver the pressurized air. A commonly prescribed version of CPAP is called automatic CPAP. In this mode of treatment, the machine is charged with recognizing when the airway is compromised and adjusting accordingly. The 95th percentile pressure represents the pressure that the machine is at or below approximately 95% of the time in order to effectively respond to respiratory events and minimize airway narrowing. After continuous usage, data from the automatic CPAP is downloaded to determine compliance and the 95th percentile of CPAP machine pressure.

Unfortunately, the current methods of screening, diagnosis, and treatment are flawed. Severity of diagnosis is not correlated with the reported symptomology,

prescribed treatment is not correlated with severity of diagnosis, and compliance with treatment methods is weak. More accurate methods of diagnosis are needed in order to improve the screening and treatment processes.

Problem Statement

There is a high prevalence of sleep apnea in American society. It is estimated that 4% of men and 2% of women suffer from this disorder, which translates to approximately 22 million Americans. Despite the increase in public awareness, some researchers believe that only 10 to 20% of those affected actually seek diagnosis and treatment (American Sleep apnea Association, 2018). Untreated individuals may not only face enormous health risks, but may also be contributing to an epidemic with negative social consequences for even those who do not suffer from sleep apnea.

Sleep apnea has been shown to contribute significantly to motor vehicle accidents, substandard grades/learning, ADHD, work accidents, mood changes, poor social relations, and other adverse social interactions (Kim & Young, 2005; Maquet, 2001; Miller, 2005; Punjabi, 2008). Furthermore, there are numerous medical complications linked to sleep apnea such as hypertension, heart failure, atrial fibrillation, stroke, depression, diabetes, arrhythmias, and other heart disease (American Sleep apnea Asociation, 2018; Park, Nasser & Mallari, 2001). In order to minimize these consequences, medical practitioners must determine the likelihood that their patients have sleep apnea and then send those with a high probability to specialists for diagnosis and treatment. Currently, the screening tools available to medical practitioners are not accurate at predicting the level and severity of sleep apnea (Guimaraes et al., 2012). One

unexplored issue involves the inferior relationship between the currently accepted screening tool and the severity of sleep apnea. Perhaps it may be inferior relationship due to an incorrectly calculated level of apnea severity.

Since 1991, many healthcare practitioners have been using the ESS, an 8-item Likert survey that yields a total score of up to 24 points, to screen individuals for symptoms of daytimes sleepiness (Johns, 1992). An elevated score on this measure (ESS > 10) may result in a healthcare practitioner ordering a formal diagnostic test, such as a sleep study, to determine if sleep apnea (or another sleep disorder) exists. This tool has been widely accepted since its inception and is still recommended to this day (Epstein et al., 2009).

The gold standard in sleep testing is an overnight polysomnogram in which the individual's respiratory flow and effort are monitored along with electroencephalography (brain waves), oxygen saturation, heart rate, electromyography (muscle activity), and more (Kryger et al., 2000). The severity of the disorder is defined by counting the total number of apneas (90% or greater reduction in respiratory flow, for 10 seconds or longer) plus hypopneas (30% or greater reduction in respiratory flow, for 10 seconds or longer, and an associated desaturation in oxygen saturation by 3% or more, and/or an arousal or fragmentation of sleep) and dividing by the TST (TST; Berry et al., 2017). This yields the AHI. This number is often utilized by healthcare practitioners to make decisions about the individual's current and future healthcare (Dempsey et al., 2010). In addition, health insurance companies utilize the AHI and ESS to determine coverage for treatment of

sleep apnea and related conditions. Consequently, much rests on the accuracy of this number.

In the current rules for the calculation of the AHI, both apneas and hypopneas are weighed equally as a respiratory event, and the length of time each event occurs is not a factor in the calculation; thus, a 50-second apnea is seen as equivalent to a 10-second hypopnea (Berry et al., 2017). Consequently, it appears that the sleep apnea diagnosis of one patient with an AHI of 20 would be equivalent to any other patient with an AHI of 20 events per hour of total sleep time (TST). For example, Patient A has an AHI of 20, which is comprised of 75% obstructive apneas and 25% hypopneas, and the average length of time for the respiratory events is 50 seconds. Patient B has an AHI of 20 comprised 100% of hypopneas and the average respiratory event lasts 15 seconds. These patients are considered equally severe in their diagnosis of sleep apnea; however, when comparing the raw data of these two people, Patient A seems to have a more compromised respiratory system than Patient B. The total length of time that Patient A is spending not breathing is significantly longer than Patient B. Furthermore, Findley, Wilhoit, and Suratt (1985) determined that the apnea duration is inversely proportional to the oxygen saturation level in patients with sleep apnea. Park et al. (2001) determined that this factor results in a proportional amount of cardiac damage. Consequently, Patient A's and Patient B's conditions may not be equally severe.

Furthermore, treatment of OSA is difficult to predict with the curent method of AHI calculation. The most common form of treatment, CPAP therapy, involves the delivery of pressurized air to hold open the airway of an individual during sleep. Cancelo

et al. (2013) have revealed a very poor relationship between AHI and the prescribed CPAP pressure. Because of the low predictive value of the AHI when determining CPAP pressure (Oksenberg et al., 2006), there is a need to explore the way in which the AHI is calculated.

Clearly, there is a need to more accurately screen for, diagnose, and treat sleep apnea. While the ESS is a highly respected tool and is often used to determine the need for formal sleep testing, the correlation between the total ESS and the severity of sleep apnea has been inconsistent (Guimaraes et al., 2012). Similarly, past research has revealed a poor correlation between the AHI and the prescribed CPAP pressure (Cancelo et al., 2013). Some researchers have considered new screening tools while others have considered new ways to calculate the severity of sleep apnea, such as measuring the length of the respiratory event (Park et al., 2001). In this study, I explored the impact of using the length of respiratory events as well as the nature of the event to calculate the AHI on the total score on the current screening tool for sleep apnea, the ESS. Similarly, the impact of using the length of respiratory events as well as the nature of the event to calculate the AHI on the 95th percentile pressure of the automatic CPAP machine used to treat the individual's sleep apnea was explored.

Purpose

The purpose of this study was to quantitatively investigate methods of calculating the severity of sleep apnea and to determine the relationship of each method to both the score on the current screening tool, the ESS, used to identify patients who may have sleep apnea and the 95th percentile of pressure from the patient's automatic CPAP machine.

First, I caluculated the AHI in the currently accepted format as determined by the American Academy of Sleep Medicine (2015). Then, I calculated the AHI with consideration given to the length of the events experienced. Next, the type of events were weighted and the AHI was recalculated. Finally, I determined the AHI once again with both the length of the events considered along with weight given to the nature of the events experienced. The correlation between the reported score on the ESS and each of these options was explored to determine which option might accurately represent the patient's reported symptoms. Furthermore, I conducted a statistical analysis of the relationship between the 95th percentile of the automatic CPAP machine and the four AHI calculations.

Theoretical Constructs

I used biopsychosocial theory (Engel, 1977) to assess the biological, psychological, and social implications of sleep apnea. This theory posits that mind and body cyclically affect one another and are not mutually exclusive (Halligan, 2006). In addition, both the mind and body have an impact on the social aspect of life. An individual's perception of health can affect his/her willingness to address those health concerns. Therefore, it can be hypothesized that the subjective level of sleepiness, *the mind*, along with the actual severity of sleep apnea, *the body*, can bear significant weight on the individual's reaction to the diagnosis of the disorder, *the social*. Similarly, how society views sleep apnea may affect the way an individual views/reports the severity of symptoms, which can impact whether or not s/he seeks medical care (Borrell-Carrio, Suchman, & Epstein, 2004). All three aspects of the biopsychosocial theory are

intertwined and highly applicable to the screening, diagnosis, and treatment of sleep apnea.

Research Questions and Hypotheses

The proposed research was designed to test the following null and alternative hypotheses which have been derived from the review of the existing literature in the area of sleep, sleep apnea, and subjective sleepiness:

RQ1: Is there a significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods?

$H_0 1$: There is no significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

$H_a 1$: There is a significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

RQ2: Is there a significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated

using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods?

$H_0$2: There is no significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

*H*a2: There is a significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

Independent and Dependent Variables

When comparing the AHI within the subjects, the independent variable was the method of AHI calculation, while the dependent variables included the AHI as calculated by the current method, a time-based method, a weighted method, and a combination of both time-based and weighted methods. When exploring the relationship between the methods of AHI calculation to the ESS score, the independent variable was the ESS score while the dependent variables were the four calculated AHIs. For the relationship between the methods of AHI calculation to the 95th percentile CPAP pressure, the dependent variable is the CPAP pressure while the independent variables are the four calculated AHI.

Nature of Study

The quantitative method assisted me in understanding the accuracy of calculating the severity of sleep apnea, or AHI, when compared with measures of subjective daytime sleepiness such as the ESS as well as the pressure of the corresponding automatic CPAP machine.

In order to document the presence and calculate the severity of OSA, I calculated the AHI in both the currently accepted manner as well as the proposed alternatives: time-based, event-weighted, and a combination of both time-based and event-weighted. The ESS scores along with the 95th percentile pressures were correlated to all four options respectively.

I retrieved participant data from archived data from a private sleep disorders clinic in Northern Illinois. The pool of participants includes approximately 70 individuals, presumably representative of the general adult population between the ages of 22 and 71 years. Participants who completed testing for the purposes of employment/insurance requirements were excluded because their reports of daytime sleepiness may not be accurate; those who were actively prescribed sedative medication were also excluded from the trial.

I used SPSS statistical software to analyze the data. Descriptive statistics were performed on the demographics of the participants. A within-subjects repeated measures ANOVA was used to analyze the AHIs in order to determine if there was statistical significance between each of the outcomes. I performed a linear regression to analyze the ESS and each of the AHI data to determine which method represented the most

statistically significant relationship between the level of daytime sleepiness and the severity of sleep apnea. I used a second repeated measures ANOVA to analyze the CPAP pressure amongst the four groups of data to determine if there was statistical significance in the difference between each of the outcomes. A linear regression was used to analyze which method of AHI calculation represented the strongest relationship between the CPAP pressure and the severity of sleep apnea.

Significance

There is a definite need to more accurately determine the severity of sleep apnea. First, patients could be prioritized more accurately for treatment or other medical care if the level of severity was well established. Similarly, the severity of one's sleep apnea could impact an individual's motivation, or sense of urgency, to seek appropriate medical care including weight loss, initiation of CPAP therapy, or other tangential medical care (Punjabi, 2008). Another impact this could have involves the coverage of medical care by third-party payors. Medical insurance companies write policies and procedures for the coverage of diagnostic procedures, treatment, and incidental medical expenses based on the severity of sleep apnea. A more accurate AHI would provide patients suitable care and coverage by their insurance companies. And finally, physicians would benefit from this change as well. Not only would a more accurate diagnosis help a health care practitioner understand the extent to which their patient's health has declined, but this research can also help the healthcare practitioner make better decisions for the patient's overall healthcare. A more accurate method of calculating the AHI is essential to medicine and the lives of those who suffer from this disorder.

In recent years, sleep has come to be recognized as a crucial vital sign (Grandner & Malhotra, 2015). Consequently, the significance of this study lies in its ability to bring awareness to useful screening of sleep disorders by medical practitioners as well as accurate diagnosis of the 5 to 10% of the population estimated to experience sleep apnea (Weaver, 2005). Prioritizing medical care and appropriately treating patients may help prevent complicated medical conditions, ease current comorbid conditions, and improve

quality of life. In turn, this could bring about positive social benefits such as a decrease in long-term costs of medical care, a reduction in automobile or work-related accidents due to sleepiness, improvement in academic or workplace performance, and an improvement in social interactions between all. Further, this study contributes to the research literature by providing evidenced-based research regarding the correlation between daytime sleepiness and the severity of sleep apnea when considering the length of respiratory events as well as the classification.

Definition of Terms

Apnea: A reduction in breathing by greater than 90% of the baseline flow for 10 seconds or more.

Arousal: An abrupt change from sleep to wakefulness, or from a "deeper" stage of non-REM sleep to a "lighter" stage of sleep.

Biological clock: The brain process responsible for 24-hour fluctuations in body temperature, hormone secretion, other bodily activities as well as the daily alternation of sleep and wakefulness. The biological clock is found in a pair of tiny bilateral brain areas called the suprachiasmatic nuclei.

Circadian rhythm: The innate, daily, fluctuation of behavioral and physiological functions, including sleep waking, generally tied to the periodicity of light/dark cues.

Deep sleep: Also known as stage N3 sleep and slow wave sleep. This is scored when greater than or equal to 20% of an epoch contains EEG waves of frequency 0.5-2 Hz and peak-to-peak amplitude greater than 75 µV, measured over the frontal regions referenced to the contralateral ear or mastoid.

Epoch: A section of 30 seconds of recording time during a polysomnogram.

Fragmentation: Brief arousals occurring throughout one's sleep, reducing the quality of sleep and/or the total amount of time spent in the deeper levels of sleep.

Hypopnea: A reduction in breathing by 30 to 90% of the baseline flow for 10 seconds or more. The event must be accompanied by an EEG arousal or a greater than or equal to 3 percent oxygen desaturation (or greater than or equal to 4 percent desaturation with no EEG arousal).

Light sleep: Stages N1 and N2 sleep.

Serotonin: Neurotransmitter in the brain that regulates mood, appetite, sexual activity, aggression, body temperature, and sleep cycle.

Sleep architecture: The structure of sleep which is generally composed of a cyclical pattern of the various NREM and REM sleep stages.

Sleep debt: The accumulated amount of sleep loss from insufficient sleep, regardless of cause.

Sleep deprivation: Acute or chronic lack of sufficient sleep.

Sleep efficiency: The percentage or ratio of time spent asleep divided by time allotted for sleep.

Sleep hygiene: The habits, environmental factors, and practices that may influence the length and quality of one's sleep. This includes bedtime, wake time, nighttime rituals, and disruptions to one's sleep.

Sleep inertia: Sleepiness and cognitive / psychomotor impairment that can occur immediately after awakening. This may persist for minutes to more than an hour after

awakening and is more commonly experienced when awakening from slow-wave sleep or when sleep efficiency is insufficient.

Sleep latency: The amount of time it takes to fall asleep once one attempts to fall asleep.

Assumptions and Limitations

Assumptions

I assumed the following:

1. The ESS had been completed by the participants in an accurate and truthful manner to provide precise results and relationships.

2. The polysomnogram was performed and analyzed according to the current AASM guidelines to provide consistently accepted medical standards of diagnosis.

3. The electrodes used during the acquisition of the polysomnogram were of similar quality and condition to eliminate the possibility of interference.

4. The data from the polysomnogram comprehensively represents the severity of the participants' daily sleep apnea symptoms and did not signify results that would represent an outlier within the data.

5. The scoring technician accurately followed the most recent guidelines for scoring sleep stages and respiratory events to signify consistency in the industry.

6. The participants did not have any unidentified medical or psychological disorders that would interfere with the findings.

7. Statistical sphericity was acceptable since there was no change to the data collected or the conditions of obtaining the data. Instead, the identification of the sleep events was the only factor to change; therefore, power to correctly reject null should also be acceptable.

Limitations

The following were potential limitations of this study:

1. Sleep apnea severity can vary night-to-night depending on the patient's daily sleep patterns. The polysomnogram that was performed may or may not accurately depict the average severity of sleep apnea in each participant.

2. Each polysomnogram may have been performed and/or analyzed by different technicians. The subjective nature of performing sleep studies along with the interpretation of electrical signals when analyzing the data may vary slightly from one person to the next due to the subjective nature of identifying signals.

3. Each polysomnogram may have been performed with a different set of electrodes; the age, condition and brand of each electrode may cause slightly different signals during the acquisition of data.

4. The ESS is a self-report measure which is subjective in nature. Individuals may experience symptoms differently and consider sleepiness as a relative measure.

5. The results of the ESS depend on the ability of all individuals to be honest in reporting.

6. The sampling strategy of participant selection is limited to the individuals who have sought medical care. This may result in difficulty applying the results to populations of individuals who tend not to seek medical care due to financial, social, religious, or other factors.

7. The participants in this study may be of similar socioeconomic background, race, and education levels. This may result in an inability to generalize the results to dissimilar populations.

Summary

As I have discussed in this chapter, there is a high prevalence of sleep apnea in American society, which is correlated with medical comorbidities and social consequences. Medical practitioners currently rely on screening tools such as the ESS to determine which patients should undergo a sleep study (Johns, 1992). However, the score on the ESS is not strongly correlated with the presence and severity of sleep apnea (Guimaraes et al., 2012). Instead, overnight sleep studies are completed to document the presence and/or severity of OSA (Boone, 2004; de Souza et al., 2003). The AHI is the average number of times a person has an abnormal respiratory event; it is synonymous with the severity of sleep apnea. This number is often used by healthcare practitioners to make decisions about the individual's current and future healthcare (Dempsey et al., 2010). In addition, health insurance companies use the AHI and ESS to determine coverage for treatment of sleep apnea and related conditions. Consequently, much rests on the accuracy of this number. Furthermore, Cancelo et al. (2013) identified a very poor relationship between the AHI and the CPAP pressure. Because of the low predictive

value of the AHI when determining CPAP pressure (Oksenberg et al., 2006), there is a need to explore the way in which the AHI is calculated.

In Chapter 2, I present the history of sleep medicine, the structure and facets of normal sleep, and an in-depth review of the causes, consequences, and treatment of sleep apnea. In it, I explore whether or not the relationship between the ESS and 95th CPAP percentile pressure and the severity of sleep apnea may be lower due to an incorrectly calculated level of apnea severity. The current rules for the calculation of the AHI are also presented along with a discussion of the correlation between the AHI and the screening and treatment of sleep apnea (see Berry et al., 2017). I detail proposed methods of calculating the AHI differently are detailed in Chapter 2 as well.

Chapter 2: Literature Review

Perhaps the most commonly discussed sleep disorder, OSA has become

increasingly publicized in the media. This disorder is characterized by the cessation of

airflow for periods of time due to a blockage or collapsing of the airway during sleep,

often resulting in arousals from sleep. These sleep disruptions prevent continuous sleep

progression, and typically prohibit entering or maintaining deep sleep periods in which

the throat muscles are more relaxed allowing for a more compromised airway. Though a

person may not physically wake up (or remember waking), the brain wakes from deep

sleep and re-enters light sleep in which the throat muscles are less susceptible to

collapsing (Aldrich, 1999).

Currently, medical practitioners administer the ESS to screen individuals for

symptoms of daytimes sleepiness (Johns, 1992). An elevated score on this measure (ESS

> 10) may result in a healthcare practitioner ordering a sleep study to determine if a sleep

disorder exists. Although multiple studies have shown the validity of the ESS, it is not

strongly correlated with the presence and severity of sleep apnea, which leaves many

healthcare practitioners without a useful way of screening for OSA (Guimaraes et al.,

2012). Instead, OSA is screened for during an overnight diagnostic sleep study.

Overnight sleep studies are used to diagnose the presence and severity of sleep

apnea (Boone, 2004; de Souza et al., 2003), and they allow for a thorough assessment of

physiological parameters including brain waves, oxygen saturation, breathing, and

respiratory effort (Kryger et al., 2000). The severity of sleep apnea is defined by counting

the total number of apneas, otherwise known as the cessation of airflow, plus hypopneas,

a significant reduction in airflow, and then dividing by the TST (Berry et al., 2017). This yields the AHI. Healthcare practitioners often use this number to make decisions about the individual's current and future healthcare (Dempsey et al., 2010).

The most common method of treatment is CPAP, also known as CPAP therapy. CPAP therapy consists of a mask worn over the nose and/or mouth which is attached to a machine that delivers pressurized air to the patient to hold the airway open during the night. This treatment is very successful in relieving nearly all symptoms of OSA as well as snoring (Carswell et al., 2004). With automatic CPAP, the machine is able to recognize when the airway is compromised and will adjust the pressure accordingly. Therefore, the pressure may fluctuate throughout the period of usage; the 95th percentile pressure is calculated by the machine to represent the most appropriate pressure needed to eliminate apneic events at least 95% of the time (Prasad, Carley, & Herdegen, 2010).

In the current rules for the calculation of the AHI, both apneas and hypopneas are weighed equally as respiratory events, and the length of time each event occurrs is not a factor in the calculation; consequently, it appears that the sleep apnea diagnosis of one patient with an AHI of 20 would be equivalent to any other patient with an AHI of 20 events per hour of TST. However, when comparing raw data, a patient with 50-second apneas seems to have a more compromised respiratory system than a patient with the same number of 10-second hypopneas. As such, if the two patients had significantly different severity levels of sleep apnea, they may have proportionately different daytime symptoms as well as treatment plans. Since the AHI calculation does not consider the length and severity of the respiratory events, this may impact the relationship between

AHI and the score on the ESS. In truth, the ESS score is not strongly correlated with the presence and severity of sleep apnea (Guimaraes et al., 2012).

Furthermore, researchers have also revealed a very poor relationship between the AHI and the 95th percentile CPAP pressure (Cancelo et al., 2013). This, too, may be impacted by the nature of the AHI calculation. The inability to predict the severity of sleep apnea along with the low predictive value of the AHI when determining CPAP pressure (Oksenberg et al., 2006) suggests an urgent need to explore the way in which the AHI is calculated.

Description of the Literature Search

I used multiple search methods to gather literature for this review. First, I used the EBSCOhost research database to search for background information as well as relevant research studies. Databases I used include Academic Search Premier, Health Source Nursing/Academic Edition, Clinical Pharmacology, Medline, PsycARTICLES, and PsycINFO. In addition, I used search engines such as Google and Yahoo to search for articles and information. Critical search terms included *sleep, apnea, severity, calculation, apnea-hypopnea index, respiratory disturbance index, awareness, subjective, sleepiness, ESS, survey, sleep studies, polysomnography, accuracy, correlation, research, diagnosis, treatment*, and combinations of related words. Other sources of information include clinical sleep manuals, such as *Principles and Practice of Sleep Medicine* (4th ed.), as well as the journal *Sleep* which publishes current research in sleep medicine. The primary sources I used to investigate the issue were generally published within the last 5

years. However, some of the sources used for background information in each area were 10 to 25 years old. All reference articles were published in peer-reviewed journals.

Normal Sleep

In 1928, a German psychiatrist named Hans Berger became the first man to record the electrical activity of the human brain. This discovery allowed experts to differentiate the brain waves of sleep from those of wakefulness (Aldrich, 1999). Consequently, the invention of the diagnostic procedure used to identify brain waves, the EEG, is considered to be the turning point in the history of sleep research. Since then, scientists have spent a great deal of time studying sleep medicine. Not only have scientists learned about the basic sleep cycle, circadian rhythms, and sleep disorders, but they have also discovered the impact of sleep on many physical and psychological functions such as growth and development (Kryger et al., 2000).

Sleep Architecture

Sleep is characterized by 4 stages. Stages N1 through N3 comprise NREM sleep, while the final stage is known as Stage R, or REM. Stage N1, the first stage of sleep, is commonly called the sleep-wake transition phase. This stage of sleep is very light and is characterized by slow, rolling-eye movements. Stage N1 sleep generally lasts for approximately 5 minutes before the next stage is entered. The next stage, Stage N2, is a continuation of light sleep and the EEG patterns are characterized by theta waves and sleep spindles. Theta waves represent consistent electrical peaks of activity in the cerebral cortex in the range of 4 to 8 cycles (peaks) per second; spindles tend to be short, intermittent bursts of electrical activity in the range of 12 to 14 cycles per second

(Rechtschaffen & Kales, 1968). Although scientists are still uncertain of the exact purpose of sleep spindles, they are used to identify Stage N2 sleep (Aldrich, 1999).

Stage N3 is classified as deep sleep which is also known as SWS. The frequencies of the electrical waves in these stages of sleep are much slower than that of light sleep, falling in the 1 to 3 cycles per second range called delta. Similarly, delta waves are also much higher in amplitude than other frequencies. Stage N3 is characterized by epochs containing greater than 20% of EEG activity consisting of delta waves (Berry et al., 2017).

The final stage is REM sleep, which transitions to a faster frequency of electrical activity with brain waves occurring in the 3 to 12 cycles per second range. The frequencies in Stage R are called mixed-frequency as there is no apparent pattern or consistency throughout the stage. REM sleep is characterized by rapid eye movements that appear as sharp, sudden jerks of the eye muscles, usually in the same direction (Rechtschaffen & Kales, 1968). Scientists have associated Stage R sleep with dreaming via investigations in which subjects are awakened during this stage and asked to report whether dreams were occurring. Results show that 80% of people studied report dreaming while in Stage R sleep, while only 40% report dreams occurring in other stages (Pagel, 2000). Therefore, REM sleep is sometimes interchangeably referred to as dream sleep. Though the purpose of dreaming is still uncertain, it is well accepted that this stage of sleep is an intrinsic part of normal sleep (Pagel, 2000).

One complete sleep cycle is normally composed of light sleep, deep sleep (SWS), and REM sleep; individuals experience several sleep cycles each night (Kryger et al.,

2000). While a typical night of sleep consists of all stages of sleep, the proportion and organization of sleep cycles do fluctuate with age. While newborn infants require approximately 16 to 17 hours of sleep each day, TST requirements begin to decline to 13 to 14 hours of sleep a day by 6 to 8 months of age (Sheldon, Riter & Detrojan, 1999). Furthermore, sleep at this age occurs in frequent, periodic intervals. At 3 to 6 weeks of age, the average length of the longest sleep period is about 3 to 6 hours with 1 or 2 hours of wake in between. By 16 weeks to 6 months of age, most infants have developed a consistent diurnal sleep-wake pattern with the most extensive wake period following the lengthiest sleep period. Sleep slowly begins to consolidate into a single nocturnal sleep period of about 8 to 9 hours with brief, well-defined naps occurring during the daytime (Sheldon et al., 1999).

By the preschool years, there is gradual development of a consolidated nocturnal sleep period. TST declines to about 10 to 11 hours per day (Sheldon et al., 1999) while 60 minute cycles of REM and NREM sleep are experienced. By 5 years of age, the cycle grows to about 90 minutes and the structure of the sleep cycle begins to change. Unlike infancy in which an equal balance between REM and NREM sleep is seen, NREM sleep begins to outweigh that of REM sleep in respect to TST. Additionally, TST decreases to 8 ½ to 9 hours each day in adolescence (Aldrich, 1999).

During young adulthood, individuals experience sleep cycles of approximately 90 to 110 minutes (Kryger et al., 2000). Approximately 2 to 5% of the overall night of sleep is Stage N1, 45 to 55% is Stage N2, 13 to 23% is Stage N3, and 20 to 25% is Stage R sleep. Wakefulness usually accounts for approximately 5% of the night. While deep sleep

dominates the cycle and short periods of REM are experienced in the first third of the night, REM sleep tends to dominate the cycle and deep sleep diminishes in the last third of the night (Sheldon et al., 1999). A single stage of sleep should never comprise too much of the overall night, though as a person ages s/he will experience a greater overall percentage of light sleep each night. As a result, the body will typically compensate for this increase in light sleep by decreasing the amount of time a person spends in deep sleep, yet virtually maintaining time spent in REM sleep. This demonstrates both the normal effects of the aging process as well as the importance of REM sleep in the human body (Aldrich, 1999).

The process of trading deep sleep for light sleep continues steadily throughout the rest of life. By late adulthood, deep sleep occupies only 5 to 10% of one's sleep. Furthermore, by the age of 60 years, SWS may no longer be present. However, there is some controversy over this finding. According to the accepted criteria as outlined by Rechtschaffen and Kales (1968), delta waves have amplitudes of greater than 75 μv. Due to the decline in the aging brain's electrical activity, SWS in the elderly is no longer this high in amplitude. Consequently, sleep professionals do not currently classify low-amplitude waves that meet the frequency criterion as delta waves. As a result, SWS appears to decline drastically with age although this may not actually be the case (Kryger et al., 2000).

Likewise, TST continues to diminish with age. While time sleeping eventually decreases to about 7 ½ to 8 hours, time spent in bed tends to increase to about 8 ½ hours each day. Although healthy adults may fall asleep without difficulty, wakefulness

throughout the night tends to increase. Not only does the amount of light sleep increase, but brief arousals and awakenings also begin to increase during the night (Aldrich, 1999). Furthermore, there are many other factors that result in sleep fragmentation. Degeneration of the central nervous system, inactivity, napping, psychiatric illness, medical illness and sleep disorders all become more common during the aging process. Two specific conditions that result in poor quality of sleep are benign prostatic hypertrophy and osteoarthritis. The respective symptoms of each include increased need to urinate at night, and increased difficulty in finding a comfortable sleeping position, which causes frequent movement and arousal. As a result, the quality of sleep and the time spent sleeping tends to decline with age (Kryger et al., 2000).

A common misconception is that the need for sleep does not appear to change with age (Kryger et al., 2000). Despite research showing that by combining nighttime sleep with daytime napping, TST holds steady and may even increase slightly with age (Aldrich, 1999; Rumble & Morgan, 1992), most sleep researchers still report a decrease in sleep need in the elderly (Kryger et al., 2000; National Sleep Foundation, 2004). Unfortunately, this is a complex area to study; social factors as well as medical conditions can certainly affect the amount of sleep one gets. Inactivity during retirement age is common; daytime naps may be a result of boredom and idle behavior (Kryger et al., 2000). Also, because the chance of having a medical condition increases with age, so does the possibility of abnormal sleep (American Psychiatric Association, 1994). Ironically, it becomes increasingly difficult to find healthy elderly adults to study.

Consequently, sleep research in this area is scarce, which limits the knowledge and evidence needed to support such claims (National Sleep Foundation, 2004).

Contrary to the decay of deep sleep, REM sleep as a percentage of TST is generally maintained as one ages. REM sleep perpetually accounts for 20 to 25% of TST regardless of the fluctuations in SWS and length of time spent sleeping. This stage of sleep has been associated with high brain metabolic and neuronal activity rates, reduced muscle tone, irregular and relatively automatic respiration, and diminished thermoregulation (Siegel, 2005). Although these characteristics do not seem conducive to development, REM sleep has been found to facilitate the cholinergic activity of proteins involved in repairing tissue injury, inflammation, and infection (Aldrich, 1999). During REM sleep, much of the body experiences muscle atonia, or muscle paralysis, which is assumed to allow time to carry out essential processes such as brain development, cell repair and routine maintenance of the brain. Consequently, the depression of the system is necessary to facilitate physiological processes (Pagel, 2000).

Function of Sleep

Relatively speaking, sleep medicine is a recently developed area of medicine, with documented scientific investigation beginning in 1930 with the first electroencephalogram. However, sleep disorders have probably been around in one form or another ever since human existence. For many centuries, theorists have been debating the causes of sleep along with the psychological implications of sleep. As early as the 5th century BC, a Greek philosopher named Empedocles taught his students that "sleep occurred when the blood cooled and fire was separated from water, air, and earth…"

(Aldrich, 1999, p.4). In the 4th century, Hippocrates argued that a warming of the blood occurs during sleep as blood passes to the inner core of the body; meanwhile Aristotle guessed that sleep was induced by warm vapors passing to the brain after digestion. Over the years, many have theorized on the nature and function of sleep, guessing at the chemical and neural causes of this thing called sleep. Finally in 1875, a British psychologist named Caton discovered brain electricity while studying the brains of a laboratory dogs and rabbits. This discovery led to the 1930 investigation, performed by the German physician Berger, in which human brain activity was recorded on paper. From this and similar experiments, it was determined that sleep was a passive state of wake activity (Aldrich, 1999).

On the other hand, some researchers assert that sleep is essential to learning and memory (Maquet, 2001; Mitru, Millrod, & Mateika, 2002). Bower (2000) recounts an example of learning tasks that require quick, visual processing such as gymnastics or playing an instrument. While performing these tasks, the initial night of sleep after a learning experience proved critical in the memory of the task. Additionally, these findings supported that sleep earlier in the night (SWS) aids in procedural recall while sleep later in the night (REM) strengthens memories reinforced by SWS (Bower, 2000). As Maquet (2001) explains after his investigations, the first night of sleep after learning a new skill is critical.

Furthermore, one important function of REM sleep is the role it plays in memory. Research illustrates that "the amount of REM sleep increases after intense learning experiences, and deprivation of REM sleep in humans impairs recall of the previous

day's events" (Aldrich, 1999, p. 19). REM sleep has been associated with intellectual

functioning, and in cases of organic brain dysfunctions REM sleep declines rapidly

(Kryger et al., 2000). Although many theories about the function of REM sleep have yet

to be proven, most are widely accepted by the scientific community. Scientists are still

unsure of the purpose of dreaming, and yet it is well accepted that the stage of REM sleep

is an intrinsic part of normal sleep. During REM sleep, much of the body experiences

muscle atonia, or muscle paralysis, which is thought to allow time to carry out essential

processes such as brain development, cell repair and routine maintenance of the brain

(Pagel, 2000). Due to the prevalence of REM sleep among mammalian species and the

consistent nature of REM sleep throughout an organism's lifetime, REM sleep is

considered an important physiologic function (Kryger et al., 2000).

Much research proposes that sleep not only aids in learning and memory

functions, but also in creativity and insight. In one study, participants were given math

puzzles to solve. The group that had slept between sessions finished the problems

significantly faster than the other group; this supports the claim that sleep facilitates

learning and memory (Miller, 2005; Wolfson, & Carskadon, 1998). Moreover, this group

was also more likely to find the hidden solutions to the puzzles that required insight. The

implication is that REM sleep also improves one's insight. Theorists suggest that dreams

may advance the thinking skills required for insight due to the elaborate, yet illogical

connections made in dreams (Miller, 2005). Consequently, some research has shown that

subjects do better on cognitive tests requiring flexibility after higher proportions of REM

sleep (Smith & Rose, 1997).

On the other hand, the other stages of sleep are equally important; many essential bodily processes occur in NREM sleep as well. For instance, it is during SWS sleep that an increase in growth hormone release is seen (Laberge, Petit, Simard, Vitaro, Tremblay, & Montplaisir, 2001). This explains the prevalence of deep sleep in youth when growth is rapid and constant, and the depletion of deep sleep throughout adulthood when growth is no longer necessary. NREM sleep also maintains a thermoregulatory function which is absent in REM sleep. Thermoregulation induces a decline in temperature of about 1 to 2 degrees Celsius during sleep to allow for heat dissipation and vasodilation during metabolic processes (Siegel, 2005). A similar physiological function seen in NREM sleep is the regulation of hormones in the immune system. This ability of the body to regulate immune system hormone levels during NREM sleep is a good illustration of how sleep is essential to good health (Aldrich, 1999).

The restorative definition of sleep has long been one of the leading explanations of the purpose of sleep. The hypothesis maintains that sleep is a time in which the restorative processes of utilized human resources are enhanced. This is achieved by an increase in the net rate of protein synthesis (Idzikowski, 1984). Extensive research supports the protein-synthesis hypothesis which is illustrated in a study of monkeys and rats. Maquet (2001) reports a positive correlation between SWS and cerebral protein synthesis in monkeys; in this case, learning is reportedly enriched with sleep. Conversely, in an experiment with rats, a protein synthesis inhibitor is injected during REM sleep and learning is reportedly impaired the next day. When the inhibitor is administered in non-REM sleep, the learning impairment is not observed. These results support the hypothesis

of the induction of chemical processes in sleep which are conducive to learning and essential for growth and development (Maquet, 2001).

Despite the research, sleep is clearly a necessary function that often goes overlooked. With today's advanced technology and 24-hour supermarkets, people are constantly sacrificing sleep for more hours in the day and minimizing the importance of sleep (National Sleep Foundation, 2004). Yet, human growth and development demand proper sleep from infancy through late adulthood. Likewise, continual changes in sleep cycles occur in order to support age-appropriate physiological and mental functioning. As such, infants spend a significant amount of time in REM sleep in order to support brain development (Sheldon et al., 1999). Throughout infancy, childhood, and adolescence individuals spend a substantial amount of time in deep sleep to facilitate growth, learning and memory (Miller, 2005). Although SWS begins to diminish when growth is no longer occurring, consistent REM sleep continues throughout life. This facilitates the consolidation of memories as well as the cell/tissue repair needed throughout the body. With age, there is a steady decline in the need for sleep. Decreasing from 17 hours of sleep as an infant to 7 ½ hours in late adulthood, the human body gradually experiences a lower demand for the biological process of sleep (Aldrich, 1999; Kryger et al., 2000; Sheldon et al., 1999). Regardless, the impact of sleep on learning and memory, brain development, cell/tissue repair, growth, immune system function, and proteins synthesis implies that sleep is involved in nearly every aspect of life (Kryger et al., 2000). Though the definitive function of sleep is still unknown, studies suggest that sleep is not only refreshing, but it is also a vital human function (Kryger et al., 2000).

Sleep Quality

As previously stated, the normal amount of time adults need to sleep is approximately 7.5 hours per night. While some people can feel refreshed after a shorter period of sleep, there are others need even more sleep time to feel the restorative effects of sleep. There are many factors that affect the amount of sleep a person needs including age, genetic disposition, general medical health, medications used, alcohol consumption, psychiatric health, sleep hygiene and/or the presence of a sleep disorder, and many more (Aldrich, 1999).

<center>**Obstructive Sleep apnea**</center>

Obstructive Sleep apnea (OSA) is a disease in which a person suffers from respiratory difficulties while sleeping due to a physical obstruction in one's airway. This disease progresses from snoring to periods of time where one ceases to breathe while sleeping.

Underlying Causes

OSA is a respiratory disorder during sleep that is characterized by the cessation of airflow for periods of time. This may be the most commonly understood sleep disorder as it has become increasingly publicized in the media. However, many of the risk factors for OSA are misunderstood. There is a plethora of structural and nonstructural risk factors for OSA that may predispose individuals to pharyngeal collapse, even in the absence of obesity (Downey, Rowley, Wickramasinghe & Gold, 2016). Examples of structural risk factors related to craniofacial bony anatomy changes include inferior displacement of the hyoid, adenotonsillar hypertrophy, Down syndrome, and high arched palates. Pharyngeal

collapse may also occur due to structural factors related to nasal obstruction such as nasal polyps, deviated septum, the presence of tumors, defects status post trauma and stenosis. Consequently, researchers have shown that differences in head form, as measured by the cranial index, poses a correlation to the severity of sleep apnea, especially in the absence of obesity. This suggests that there may be value in phenotyping individuals in order to identify the presence and severity of OSA (Downey et al., 2016).

On the other hand, there are more commonly identified, nonstructural risk factors that correspond to the presence and severity of OSA. For example, obesity, central fat distribution, male sex, age, postmenopausal state, alcohol and sedative consumption, smoking status, allergen exposure, supine sleep position, and habitual snoring with daytime sleepiness are all nonstructural risk factors that medical practitioners utilize when screening for the presence of OSA. A family history of OSA increases the probability of developing OSA by 2 to 4 times. Similarly, a history of strokes and hypothyroidism has been correlated to an increased likelihood in developing OSA (Downey et al., 2016).

OSA generally results from a collapsing pharyngeal airway due to excessive tissue presence as well as relaxed throat muscles in sleep. As one falls asleep and the muscles begin to relax, the tissue will continue to move closer together until the airway is obstructed. A precursor and sign of OSA is snoring. Snoring is the noise that results from a partial obstruction in the air passages, while OSA is a complete obstruction. Snoring and OSA are usually accompanied by a decrease in blood oxygen levels, frequently seen as low as 70% or lower, due to the decrease in airflow. Furthermore, many patients with

severe OSA wake up gasping for air when the airway collapses completely. This is the body's defense mechanism against death or other medical consequences. Similarly, a symptom of both snoring and OSA is an abundance of neurological arousals. These arousals awaken a patient from continuous sleep progression, and typically prohibit the person from entering or maintaining deep sleep periods in which the throat muscles are more relaxed. Though a person may not physically wake up (or remember waking,) the brain wakes from deep sleep and re-enters light sleep in which the throat muscles are less susceptible to collapsing (Aldrich, 1999).

Consequences

As OSA results in sleep interruptions which simulate sleep deprivation, individuals with OSA generally report symptoms such as daytime sleepiness and fatigue. Often weight gain accompanies these complaints, but clinicians are unsure which issue is the causal factor (Punjabi, 2008). Reports of sleep loss are also correlated with higher levels of depression, anxiety, difficulty concentrating, car accidents and illness (Anderson et al., 1999). Other reported symptoms include snoring, irritability, reduced libido, dry mouth and sore throat, frequent nocturnal awakenings, frequent nighttime urination and heavy nighttime sweating. Difficulty sleeping in the supine position may also be experienced as a results of the increased pressure on the thoracic cavity and throat due to gravity. Morning headaches are also a classic sign and symptom of OSA resulting from the lack of oxygen to bodily tissues including the brain (Weaver et al, 2008).

Recent research shows that OSA may be a leading contributing factor to the development of other medical diseases such as heart disease, lung disease, strokes and

diabetes (Downey et al., 2016). Punjabi (2008) has identified OSA as a contributing factor in the development of comorbidities such as hypertension, coronary heart disease, congestive heart failure, and strokes. Neural, cardiac and pulmonary activity can be greatly affected by the cessation of airflow resulting in low oxygen levels; it is easy to see how heart and lung disease can accumulate over the years (Leung & Bradley, 2001). Furthermore, studies show that stroke patients are four times as likely to have underlying snoring and/or sleep apnea, and patients with angina or myocardial infarctions have a two-fold higher prevalence of respiratory-related sleep disorder (Aldrich, 1999). Endocrine activity is also affected as many patients experience a reduction in growth hormone secretion due to the lack of deep sleep. As a result, insulin levels are affected and contribute to the development of diabetes mellitus or hyperglycemia. Other associated conditions include obesity, hypothyroidism and gastroesophageal reflux disease (GERD). With the increased prevalence of these life-threatening diseases, individuals with OSA are at significantly higher risk for mortality.

Frequently, the diagnosis and subsequent treatment of OSA will improve the affiliated conditions (Carswell et al., 2004; Aldrich, 1999). Cardiovascular function along with metabolic processes quickly improve upon effective treatment of OSA. Unfortunately, 70 to 80 percent of individuals afflicted with this disorder remain undiagnosed and therefore, untreated (Punjabi, 2008). Proper recognition of this disorder is essential in order to ensure appropriate intervention. Therefore, proper screening and treatment are critical.

Diagnosis

Although sleep medicine was not officially introduced to the public for testing until the 1950's, sleep studies (polysomnography) have become almost as common as a CAT scan or an MRI (Hobson and Pace-Schott, 2002). When Kleitman discovered REM sleep (also known as dream sleep) in 1953, scientists began to think of sleep as an active process in which the brain performs necessary functions to prepare for daily life (Campbell, 2000). In the 1960's, scientists uncovered many physiological effects of sleep and began to improve the process of monitoring the body during sleep. Sensors were invented to be placed in strategic locations to identify and measure muscle tone, respiration, cardiac rhythm, and electrical activity in the brain. With these techniques, investigators were able to study subjects with no apparent sleep-wake complaints to define normal sleep. It is imperative that one understands the basic findings of sleep medicine research in order to study sleep development over the human lifespan (Aldrich, 1999).

In the field of sleep medicine, polysomnography (PSG) is considered to be the gold standard in detecting sleep disorders (Boone, 2004; de Souza et al., 2003). These studies allow for a thorough assessment of physiological parameters such as brain waves (EEG), breathing, respiratory effort, muscle movement, heart rate and rhythm and more (Kryger et al., 2000). Normal diagnosis of sleep disorders takes place in a sleep disorders center. Patients must obtain an order from a physician prior to testing for proper patient screening. The testing procedure, also called a polysomnogram, is non-invasive and generally includes superficial application of monitoring devices. Standard procedures

comprise of the use of surface electrodes on the head to monitor neural patterns, a

pressure sensitive device that measures airflow, elastic belts on the torso to monitor

respiratory effort, an SaO_2 probe to measure oxygen saturation levels in the blood, and

electrocardiogram electrodes to monitor cardiac activity. Similarly, other superficial

monitoring devices may be applied as needed for diagnosis, including an End Tidal

CO_2 machine to measure the level of carbon dioxide when exhaling, and leg/arm

sensors to monitor muscle movement during sleep. Then, a typical night in the lab is as

follows:

> The patient arrives an hour before bedtime to learn about the process of the study
>
> itself and to be connected to all sleep diagnostic equipment. The patient is
>
> monitored for a minimum of 6 hours via sleep diagnostic equipment, as well as
>
> audio and visual monitors. The patient must wear all sleep diagnostic equipment
>
> the entire night, and must be seen sleeping in all positions to rule out a positional
>
> sleep disorder (such as snoring while only supine) which is treated with positional
>
> therapy. In the morning the patient is unhooked from all of the monitors and is
>
> free to go until treatment can begin.

If the patient is subsequently diagnosed with a sleep disorder as described in the

characteristics of each disorder discussed, the patient and physician must follow up with

the appropriate course of treatment. Close follow up is essential to make sure the patient

is compliant and treatment is appropriate and effective (Aldrich, 1999).

Polysomnograms are typically performed overnight in a clinical setting and involve numerous wires being attached to the patient. As a result, many patients have difficulty sleeping, especially away from home. In addition, PSGs can be costly and time consuming. Consequently, a more recent alternative to polysomnography includes in-home sleep studies. Not only does the patient sleep in the comfort of his own home, but the test is also very simple with only 2 to 4 connections to the patient. Because of its simplicity, sleep studies are limited in the type of data collected. Instead of a comprehensive diagnostic test, these studies are used as screening tools to rule out sleep disordered breathing. As such, this is a significantly less expensive option when compared to polysomnography.

Calculating the AHI – Current Method

The current method of calculating the AHI has been the accepted method of determining the severity of sleep apnea since the inception of sleep medicine. To calculate the AHI, total the apneas and hypopneas experienced and then divide by the number of hours slept:

$$\frac{\text{apnea} + \text{Hypopneas}}{\text{TST (Hrs)}} = \text{AHI}$$

Example:

100 apneas, 50 Hypopneas, Average event 16 secs, 7.25 Hours of TST

$$\text{AHI} = \frac{(100+50)}{7.25} = 20.7$$

Calculating the AHI – Proposed Methods

   Time Based - The first proposed method of calculating the AHI would include the length of the respiratory events. To calculate the AHI, total the apneas and hypopneas experienced. Multiply by the average length of the events (seconds) divided by 10 seconds, the minimum number of seconds required for an event by the AASM (2017). Divide by the number of hours slept:

$$\frac{\text{apnea} + \text{Hypopneas}}{\text{TST (Hrs)}} \quad \text{x} \quad \frac{\text{Average Length of Event (secs)}}{\text{10 Seconds}} \quad = \quad \text{AHI(t)}$$

  Example:

  100 apneas, 50 Hypopneas, Average event 16 secs, 7.25 Hours of TST

$$\text{AHI} = \frac{(100+50)}{7.25} \text{ x } \frac{16}{10} = 33.1$$

   Weighted Events - The second proposed method of calculating the AHI would include the type of the respiratory events. To calculate the AHI, multiply the number of apneas by 2 and the hypopneas by 1. Total. Divide by the number of hours slept:

$$\frac{(\text{Apneas x 2}) + (\text{Hypopneas x 1})}{\text{TST (Hrs)}} \quad = \quad \text{AHI(w)}$$

  Example:

  100 apneas, 50 Hypopneas, Average event16 secs, 7.25 Hours of TST

$$\text{AHI} = \frac{(100 \text{ x } 2) + (50 \text{ x } 1)}{7.25} = 34.5$$

Combination - The third proposed method of calculating the AHI would include the length and type of the respiratory events. To calculate the AHI, multiply the number of apneas by 2 and the hypopneas by 1. Total. Multiply by the average length of the events (seconds) divided by 10 seconds, the minimum number of seconds required for an event by the AASM (Berry et al., 2017). Divide by the number of hours slept:

$$\frac{(Apneas \times 2) + (Hypopneas \times 1)}{TST \ (Hrs)} \quad x \quad \frac{Average \ Length \ of \ Event \ (secs)}{10 \ Seconds} \quad = \quad AHI(wt)$$

Example:

100 apneas, 50 Hypopneas, Average event 16 secs, 7.25 Hours of TST

$$AHI \ = \ \frac{(100 \times 2) + (50 \times 1)}{7.25} \times \frac{16}{10} \ = \ 55.2$$

Treatment

Methods of treatment for OSA and snoring include the following: positional sleep therapy, smoking cessation, reduction/avoidance of alcohol and/or sedatives, surgical options, CPAP, and dental appliances (Downey et al., 2016). The most common method is Continuous Positive Airway Pressure also known as CPAP therapy. CPAP therapy consists of a mask worn over the nose and/or mouth which is attached to a machine that delivers pressurized air to the patient to hold the airway open during the night. This treatment is very successful in relieving nearly all symptoms of OSA as well as snoring (Carswell et al., 2004).

A commonly prescribed version of CPAP is called automatic CPAP. In this mode of treatment, the machine is able to recognize when the airway is compromised and will

adjust the pressure accordingly. The machine is programmed at a start pressure based on

the patient's comfort. Then, a radio frequency signal is sent through the tubing to the

patient's airway to measure the extent to which the patent's airway is open; the machine

then automatically increases the pressure when the airway is collapsing or decreases the

pressure when the airway is open significantly. Therefore, the pressure may fluctuate

throughout the period of usage; the 95th percentile pressure is calculated by the machine

to represent the most appropriate pressure needed to eliminate apneic events, at least 95

percent of the time. The use of an automatic CPAP machine for the purposes of treating

OSA has become a widely accepted use of technology. Research has confirmed the

accuracy of the ability of an automatic CPAP machine to identify respiratory disturbances

and adjust accordingly (Prasad et al., 2010). Automatic CPAP therapy has been shown to

be more efficacious than standard CPAP when treating OSA due to its ability to fluctuate

and give the patient more pressure only when needed. Furthermore, individuals utilizing

automatic CPAP machines report increased comfort when utilizing an automatic CPAP

machine as compared to a standard CPAP machine (Mulgrew, Cheema, Fleetham, Ryan

& Ayas, 2007).

  For those who cannot tolerate CPAP (or who are unwilling to use the machine for

lifestyle reasons) surgical options are available, but are not considered as reliable.

Surgery may be as simple as an adenoidectomy and tonsillectomy (A&T) if indicated, or

may involve a uvulopalatoplasty (UPPP) which is the removal of the uvula, and portions

of the soft palate and pharyngeal tissue. Less often, craniofacial reconstruction may be

performed to correct the anatomical abnormality that may be causing sleep apnea

(Downey et al., 2016). Some surgeons perform soft palate implants, also known as the Pillar Procedure. In this surgical procedure, three polyester rods are inserted into the soft palate to initiate a slight stiffening to the palate due to an inflammatory response to the rods. Individuals with snoring or mild sleep apnea may experience a reduction of symptoms. For those with a large tongue base, sometimes a hyoid advancement or tongue advancement may expand the airway enough to reduce snoring or apneic events. There are several other surgical options for the treatment of sleep apnea; however, it is important to note that short of a tracheostomy (placing a hollow breathing tube directly into an individual's windpipe to divert the individual's ability to breathe through the nose/mouth), surgery is not considered a long-term solution and is often unsuccessful (Morgan, & Lindman, 2015).

Another option includes dental appliances which attempt to shift the lower jaw forward so as to create a larger opening at the back of the throat where obstruction can occur. However, dental appliances are only effective in patients with mild upper airway obstructions, and will not benefit many moderate to severe OSA patients (Aldrich, 1999). In overweight patients, weight loss is highly recommended to decrease the possibility of excess tissue growth in the throat. Also, some patients with minor snoring or sleep apnea may be given a medication to increase the muscle tone of the throat; however, the patients who have mild snoring and/or sleep apnea rarely complain of other symptoms so medications are rarely prescribed (Carswell et al., 2004; Aldrich, 1999).

Summary

OSA is characterized by the cessation of airflow along with arousals which awaken a patient from continuous sleep progression and typically prohibit the person from entering or maintaining deep sleep periods in which the throat muscles are more relaxed. There is a high prevalence of sleep apnea in today's society, which has been implicated in the frequency of motor vehicle accidents, substandard grades/learning, workplace accidents, mood changes, poor social relations and more (Kim & Young, 2005; Maquet, 2001; Miller, 2005; Punjabi, 2008). Furthermore, medical disorders such as hypertension, stroke, diabetes, arrhythmias, and other heart disease have all been linked to sleep apnea (Park et al., 2001). In order to minimize the medical and social consequences of this disorder, medical practitioners must determine the likelihood that their patients have sleep apnea and then send those with a high probability to specialists for diagnosis and treatment.

While medical practitioners currently rely on screening tools, such as the ESS , to determine which patients should undergo a sleep study (Johns, 1992), the ESS score is not strongly correlated with the presence and severity of sleep apnea which leaves many healthcare practitioners without a useful way of accurately screening for OSA (Guimaraes et al., 2012). Instead, overnight sleep studies are completed to document the presence and/or severity of sleep disorders such as OSA (Boone, 2004; de Souza et al., 2003) by assessing breathing and respiratory effort throughout the night (Kryger et al., 2000). The severity of sleep apnea is defined by counting the total number of apneas plus Hypopneas and dividing by the TST (Berry et al., 2017). This yields the AHI. This

number is often utilized by healthcare practitioners to make decisions about the

individual's current and future healthcare (Dempsey et al., 2010). In addition, health

insurance companies utilize the AHI and ESS to determine coverage for treatment of

sleep apnea and related conditions. Consequently, much rests on the accuracy of this

number.

Once sleep apnea is diagnosed, the most common method of treatment is CPAP.

CPAP therapy consists of a mask worn over the nose and/or mouth which is attached to a

machine that delivers pressurized air to the patient to hold the airway open during the

night, much like an air splint. This treatment is very successful in relieving nearly all

symptoms of OSA as well as snoring (Carswell et al., 2004). With automatic CPAP, the

machine is able to recognize when the airway is compromised and will adjust the

pressure accordingly. Therefore, the pressure may fluctuate throughout the period of

usage; the 95th percentile pressure is calculated to represent the most appropriate pressure

needed to eliminate apneic events, at least 95% of the time (Prasad et al., 2010).

Treatment of OSA is often difficult to predict with the curent method of AHI calculation.

Unfortunately, research has revealed a poor relationship between the AHI and the 95th

PAP pressure (Cancelo et al., 2013).

One goal of the proposed research study is to explore whether or not the

relationships between the ESS, the severity of sleep apnea, and the 95th percentile CPAP

pressure may be lower due to an incorrectly calculated level of apnea severity. In the

current rules for the calculation of the AHI, both apneas and hypopneas are weighed

equally as a respiratory event, and the length of time each event occurs is not a factor in

the calculation (Berry et al., 2017). Consequently, it appears that the sleep apnea diagnosis of one patient with an AHI of 20 would be equivalent to any other patient with an AHI of 20 events per hour of TST. However, when comparing the raw data these patients may not be equally severe. This study explores the impact of the length of respiratory events as well as the classification of the event to determine the correlation of the AHI to the current screening tool for sleep apnea, the ESS, along witih the 95th percentile pressure of the automatic CPAP machine used to treat the individual's sleep apnea.

Chapter 3: Research Method

Introduction

In 2014, the American Academy of Sleep Medicine published statistics on the prevalence of sleep apnea in the United States. Experts believe that this medical disorder afflicts nearly 25 million Americans (New York Cardiovascular Associates, 2013). However, recent researchers have estimated that 80% of cases of sleep apnea are completely undiagnosed (Downey et al., 2016). Furthermore, between 29 and 83% of those diagnosed and prescribed CPAP are not adherent to these recommendations (Sawyer et al., 2011; Weaver & Grunstein, 2008).

A common reason an individual may not be diagnosed is the under-recognition of the disorder by the medical community (Punjabi, 2008). Many practitioners do not recognize the presence of sleep apnea, especially in the absence of physical characteristics such as obesity or an enlarged neck circumference (Strohl, 2016). Consequently, medical practitioners must rely on screening tools to help determine which patients should seek further diagnostic care such as a sleep study. Many healthcare practitioners administer the ESS, an 8-item Likert survey that yields a total score of up to 24 points, to screen individuals for symptoms of daytimes sleepiness (Johns, 1992). An elevated score on this measure (ESS > 10) may result in a healthcare practitioner ordering a formal diagnostic test, such as a sleep study, to determine if sleep apnea (or another sleep disorder) exists. This tool has been widely accepted since its inception and is still recommended to this day (Epstein et al., 2009).

Although multiple studies have shown the validity of the ESS, it is not strongly correlated with the presence and severity of sleep apnea (Guimaraes et al., 2012). Instead, the presence and/or severity of sleep apnea disorder is determined during an overnight diagnostic sleep study. Sleep apnea severity is calculated by counting the total number of apneas plus hypopneas and dividing by the TST (Berry et al., 2017). This yields the AHI.

In the current rules for the calculation of the AHI, both apneas and hypopneas are weighed equally as respiratory events, and the length of time each event occurrs is not a factor in the calculation; thus, a 50-second apnea is seen as equivalent to a 10-second hypopnea (Berry et al., 2017). Consequently, it appears that the sleep apnea diagnosis of one patient with an AHI of 20 would be equivalent to any other patient with an AHI of 20 events per hour of TST. However, when comparing the raw data, the patient with 50-second apneas seems to have a more compromised respiratory system than the patient with 10-second hypopneas. As such, if the two patients had significantly different severity levels of sleep apnea, they may have proportionately different daytime symptoms; thus, one would expect their ESS scores to be different as well. Since the AHI calculation does not consider the length and severity of the respiratory events, it would seem as though the correlation between AHI and ESS is not strong. Regardless, these patients would be considered equally severe according to today's accepted standards, and the ESS would possibly yield a misleading prediction of severity. Perhaps medical practitioners would rely less and less on the ESS and discontinue its use, leaving them without a tool to help establish cause for a sleep study in some patients.

Once diagnosed, an individual is often prescribed CPAP therapy. This form of treatment involves the individual sleeping with a mask over the nose and/or mouth, which is connected to a machine that blows pressurized air to maintain an open airway. Unfortuantely, Sawyer et al. (2011) found that anywhere between 29 and 83% of these individuals are not adherent to CPAP therapy. There are many reasons adherence may be low. One reason cited by CPAP users is that treatment is intrusive; the individual has to learn to sleep with a mask on the face. Second, there may be a social stigma to wearing CPAP and being diagnosed with sleep apnea; many people incorreclty assume that sleep apnea is a disease of the obese and are ashamed of the diagnosis (Roy, 2014). Furthermore, the prescribed pressure setting is a critical factor when considering adherence. If an individual's diagnosed level of sleep apnea severity is not accurate, the prescribed pressure settings may not be appropriate. Some other reasons for non-adherence include, but are limited to, poor user education regarding the disease and treatment, incorrect assumptions about the effectiveness of treatment, and the incorrect settings prescribed by physicians. Specifically, when a CPAP user is prescribed the wrong pressure settings, treatment may prove ineffectual and/or very uncomfortable causing compliance to be low. Consequently, it is important to understand the predictive settings based on sleep apnea severity so that physicians and other practitioners may correctly administer treatment.

A tangential problem involves those patients who may not receive an accurate diagnosis of sleep apnea and are precribed the wrong modality of treatment. In some cases, the individual may or may not qualify for treatment due to current diagnostic

standards or health insurance standards. The lack of or ineffective treatment may have other medical ramifications; thus, accurate diagnosis is critical to one's future heatlh. Under the current standards, sleep apnea severity levels do not account for the length of respiratory events or the type of respiratory events documented. As such, diagnosis may prove to be inaccurate, especially in borderline cases. Furthermore, those using CPAP therapy may or may not be prescribed appropriate pressure settings in the absence of accurate predictive values for physicians to utilize.

Purpose of the Study

The purpose of this study was to quantitatively investigate methods of calculating the severity of sleep apnea, and to determine the relationships between each proposed method and the ESS, the current screening tool used to identify patients who may have sleep apnea, as well as the pressure setting on a CPAP machine, a device used to treat sleep apnea. First, participants underwent an in-lab polysomnogram; during scoring, I documeted all apneas and hypopneas per the guidelines set forth by American Academy of Sleep Medicine (2017). I calculated the AHI according to the currently accepted rules. Then, I calculated the AHI with consideration given to the length of the events experienced. Next, the type of events were weighted and I recalculated the AHI. And finally, I determined the AHI once again with both the length of the events considered along with weight given to the nature of the events experienced. I explored the relationship between the reported score on the ESS and each of these calculations to determine which option might accurately represent the patient's reported symptoms. Lastly, the 95th percentile pressure setting on the participant's automatic CPAP machine

was retrieved by this researcher to investigate the relationship between the pressure and the calculated AHIs.

Research Hypotheses

I designed this study to test the following null and alternative hypotheses associated with each of the research questions.

Research Question #1

Is there a significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods?

Null Hypothesis #1

There is no significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

Alternative Hypothesis #1

There is a significant difference in the relationship between daytime sleepiness, as measured by the ESS, and the severity of sleep apnea, as measured by the AHI when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

Research Question #2

Is there a significant difference between the 95th percentile of CPAP pressure, as

determined by an automatic CPAP machine, and the AHI, when calculated using the

currently accepted formula, a time-based method, a weighted method, or a combination

of time-based and weighted methods?

> *Null Hypothesis #2*
>
> There is no significant difference between the 95th percentile of CPAP pressure,
>
> as determined by an automatic CPAP machine, and the AHI, when calculated
>
> using the currently accepted formula, a time-based method, a weighted method, or
>
> a combination of time-based and weighted methods.
>
> *Alternative Hypothesis #2*
>
> There is a significant difference between the 95th percentile of CPAP pressure, as
>
> determined by an automatic CPAP machine, and the AHI, when calculated using
>
> the currently accepted formula, a time-based method, a weighted method, or a
>
> combination of time-based and weighted methods.

Participants

Using G*Power 3.1.9.2, I performed an a priori power analysis to compute the

required sample size given an alpha of 0.05, power of 0.95, and effect size of 0.25. The

results indicate that a minimum sample size of 70 participants was required to achieve

these parameters (Green, 1991). A sample size of less than this may have been too few to

provide the statistical power to reject the null hypotheses. Consequently, I set the final

sample size for this research study at 75 in order to minimize the bias in a correlational analysis.

Demographics and Selection

I retrieved data from archived records of an independently-owned, private sleep disorders clinic in Illinois. There were approximately 200 participants initially selected from the archived records. Each participant must have received a diagnosis of OSA, pursued automatic CPAP therapy, and met industry standards for usage compliance. However, I excluded some participants from the study if the following criteria were not met.

Because the ESS was validated on a population between the ages of 22 and 71 years old, the sample in this study was within the range of the normative data (Aldrich, 1999). Furthermore, participants may have been of any gender, race, religion, education status, and/or socioeconomic status, which is reflected the normative data. All qualifying test results were used regardless of demographic data to ensure an appropriate representation of the United States adult population.

Potential Risk for Participants

As the data utilized for this study has been previously collected, participants were not subject to any significant risk through participation. All participants were notified about the potential inclusion of data in a future research study; consent was obtained from each participant prior to data collection. See Appendix A for an example of the Informed Consent signed by participants. All private healthcare information has been and will be

protected and remain confidential. Participants have not and will not be subject to any additional risk surrounding this research study.

Exclusions from Participation

Exclusionary factors include protected individuals such as pregnant women, prisoners, and children. In addition, those participants not fluent in English were excluded as consent and survey completion may not have been understood when presented in English. The presence of psychiatric disorders with psychotic features, and other significant psychological disorders that are currently being treated with medication known to affect sleep are also exclusionary factors. Individuals who are actively prescribed sedative medication were also excluded from the trial to eliminate the potential interference from symptoms and sleep disorders. Participants who completed testing for the purposes of employment/insurance requirements were excluded as symptoms of daytime sleepiness may not be properly reported when employment depends on the responses. Similarly, participants with any traumatic disorders were excluded from participation so as to minimize the possibility of misdiagnosis or interference with results. And finally, the AASM (Berry et al., 2017) requires a minimum of 6 hours of recording time in order to consider the diagnostic test to be accurate and comprehensive; results of polysomnograms achieving less than 6 hours of recording were not included.

<div align="center">

Instrumentation

</div>

Demographic information including medical history, medication use, symptoms and complaints, vitals, and other related information were obtained at the time of service;

a review of this information aided in determining the appropriateness of participation to support the accuracy of the findings.

Statistical Software

SPSS statistical software was used to analyze the data. Descriptive statistics were performed on the demographics of the participants. Chi-square tests were completed to ensure that the sample is representative of the normal population. A Repeated Measures ANOVA was used to analyze the AHIs amongst the four methods of AHI calculation to determine if there is statistical significance in the difference between each of the outcomes. Next, a Linear Regression was completed utilizing the same ESS and each of the AHI data to determine which method represents the best relationship between the level of daytime sleepiness and the severity of sleep apnea. A second Repeated Measures ANOVA was used to analyze the CPAP pressure and AHI amongst the four groups of data to determine if there is statistical significance in the difference between each of the outcomes. Finally, a Linear Regression was utilized to analyze which method of AHI calculation represents the strongest relationship between the 95th percentile CPAP pressure and the severity of sleep apnea.

Screening Tool – ESS

Initially created by Murray Johns at the Epworth Hospital in Melbourne, Australia, the ESS is a self-report survey that measures the extent of a person's sleepiness. The ESS is a test that requires subjective responses to physiological circumstances (Kryger et al., 2000). The format of the survey consists of 8 statements to which the participant identifies the likelihood of falling asleep based on his/her typical

sleepiness in that particular situation. The patient's answers are based on a Likert Scale of 0 to 3 to indicate the likelihood that one would fall asleep in a given situation. (A "0" would indicate no chance of dozing, a "1" would indicate a slight chance of dozing, a "2 would represent a moderate chance of dozing, and a "3" would suggest a high chance of dozing in a particular situation.) See Appendix B for the specific list of proposed situations the respondent must consider. The patient's cumulative score gives a total ESS score ranging from 0 to 24 (Johns, 1991).

In 1997, Johns et al. performed an investigation to define normative data for the ESS. This study included the scores of 72 subjects who were selected from a group of 331 people, presumably healthy male and female workers, 22-59 years of age. Each participant completed a detailed sleep questionnaire to rule out the possibility of having a sleep disorder. The criteria for participation were strict to insure the absence of confounding variables such as comorbid medical conditions. Based on the findings of Johns et al., the normal range of ESS scores in adults was determined to be 0 to 10, with a mean of normal scores at 4.6 +/- 2.8 (SD). Variability within this range of scores appears to reflect psychological differences between patients, in the absence of sleep disorders. Most clinicians consider scores greater than 10 to represent excessive daytime sleepiness (EDS) and may be indicative of a sleep disorder (Johns et al., 1997).

However, as stated by Murray Johns (1991), ESS cannot diagnose the presence or severity of a sleep disorder. Instead, the score simply indicates the presence of daytime symptoms. Along with the completion of the ESS, the physician considers other pertinent complaints to determine whether a sleep study is indicated. The ESS is intended to serve

as an aid to physicians when screening patients for sleep studies or other sleep diagnostic tests. The ESS is not a substitute for medical testing of any kind.

Therefore, in an effort to promote his new sleep diagnostic tool, Johns performed validity and reliability investigations to support the ability of the ESS to measure what it purports to measure, as well as demonstrate the internal consistency in ESS scores. The study included 54 patients diagnosed with sleep-disordered breathing, or abnormalities in respiratory function while asleep, as well as 104 medical students to serve as controls. Each participant of the study completed the questionnaire before and after treatment for the sleep-related breathing disorder (Johns, 1991). Polysomnography (PSG), the gold standard of sleep medicine, was performed to diagnose the sleep disorder; and Continuous Positive Airway Pressure (CPAP) was applied to treat the breathing abnormalities (Kryger et al., 2000). Student control participants had a mean score of 7.6, which is well within the normal range of less than 10. On the other hand, the patients diagnosed with a sleep disorder had a mean score of 14.3 prior to treatment and 7.4 after treatment. Multiple studies have been replicated to support the validity of the ESS. For example, Lim and colleagues (2000) performed a similar study with 71 patients suffering from sleep-related breathing problems and/or snoring. In the study, the researchers were able to correctly identify 93% of patients who are chronic snorers. While the study could not statistically differentiate between severity of sleep disturbances *(r = 0.60)*, the overall result is supportive of the validity of the ESS (Lim & Curry, 2000).

Similarly, the reliability of the ESS has been tested and supported. In Johns' investigations, the medical students that served as controls were tested a second time 5

months after the initial study. The paired ESS scores were consistent with each other as shown by Linear Regression $(r = 0.82, p < 0.001)$. In addition, those patients treated for sleep-disordered breathing were tested a third time within 3 to 9 months of treatment and the post-treatment scores were highly correlated $(r = 0.88)$ (Johns, 1992).

Diagnostic Testing - Polysomnography

A norm-referenced, diagnostic medical assessment was administered to each participant to document the presence and severity of sleep apnea (i.e. sleep study or polysomnography). Polysomnography entails a minimum of the following sensors: Electroencephalogram (EEG) leads on the participant's head and face to determine sleep stages and neurological abnormalities; belts across the chest and abdomen to monitor respiratory effort; pressure/heat sensitive nasal cannula to record respiratory flow; surface electrodes on legs and chin to monitor muscle tension, Electromyography (EMG); electrodes on the chest to measure cardiac activity, Electrocardiogram (EKG); and pulse-oximetry, a finger probe to monitor blood-oxygen saturation (SaO_2) as well as heart rate. These sensors detect neurological, respiratory, muscular, or other physiological arousals and disturbances (Berry et al., 2017).

The data retrieved from polysomnography is analyzed to determine the presence of respiratory disturbances such as apneas and hypopneas. In conjunction with the other parameters of sleep, the AHI is calculated to determine the number of times an individual has a respiratory disturbance per hour of TST.

Polysomnography is a well-researched diagnostic procedure and has been clinically accepted by the American Medical Association since the 1970's (Aldrich,

1999). Significant advancement in technology has allowed polysomnography to become less expensive and more common in the medical community. In addition, the rapid advancement in technology has made recording, analyzing, and interpretation of sleep data more accurate and multifaceted (Haba-Rubio & Krieger, 2012). In short, polysomnography has transformed the world of sleep medicine.

AASM Scoring Rules

The American Academy of Sleep Medicine (AASM) is the organization responsible for determining the standards by which all U.S. sleep centers and practitioners follow in the diagnosis, research and treatment of sleep disorders. In 2017, the AASM published the most recent guidelines for classification during polysomnography studies. It includes the rules for identifying events such as respiratory abnormalities, EEG arousals, limb movements, sleep staging, and more. See Appendix C for details on the current analysis of respiratory events and arousals during polysomnography as it pertains to this research study (Berry et al., 2017).

Continuous Positive Airway Pressure – 95th Percentile

One form of treatment for OSA is Continuous Positive Airway Pressure in which the CPAP machine delivers pressurized air to hold the airway open like an air splint. The individual wears a mask that covers the nose and/or mouth; this mask is connected to the machine with a six-foot long tubing to allow the machine a medium through which to deliver the pressurized air. A commonly prescribed version of CPAP is called automatic CPAP. In this mode of treatment, the machine is charged with recognizing when the airway is compromised and adjusting accordingly. The machine is programmed at a start

pressure based on the patient's comfort. Then, a radio frequency signal is sent through the tubing to the patient's airway to measure the extent to which the patent's airway is open (Sawyer et al., 2011). The machine documents when the patient is experiencing a respiratory event, and then it automatically increases the pressure settings until the respiratory event is eliminated. If the radiofrequency signal is delivered faster than anticipated, the machine assumes the airway is open to a significant degree and the pressure decreases. Therefore, the pressure may fluctuate throughout the period of usage. The 95th percentile pressure is then calculated. This number indicates the pressure that the machine is at or below approximately 95% of the time in order to effectively respond to respiratory events and minimize airway narrowing. After continuous usage, data is downloaded to determine compliance and the 95th percentile of CPAP machine pressure. The data may also be downloaded periodically to provide continuity of care for that individual (Weaver & Grunstein, 2008).

The use of an automatic CPAP machine for the purposes of treating OSA has become a widely accepted use of technology. Research has confirmed the accuracy of the ability of an automatic CPAP machine to identify respiratory disturbances and adjust accordingly (Prasad et al., 2010). There is a strong correlation between the AHI's when calculated by manual scoring by a Registered Polysomnographic Technologist (RPSGT) and the automated scoring from an automatic CPAP machine *(r = 0.710, p < 0.001).* Furthermore, the automatic CPAP machine increases the pressure when respiratory events are detected, and decreases the pressure when the airway is determined to be unobstructed. Consequently, the accuracy of the machine when determining the presence

of respiratory disturbances is the key in the machine's ability to auto adjust the pressure appropriately (Ueno, Kasai, Brewer, Takaya, Maeno, Kasagi, Kawana, Ishiwata & Narui, 2010). Automatic CPAP therapy has been shown to be more efficacious than standard CPAP when treating OSA. Furthermore, individuals utilizing automatic CPAP machines report increased comfort when utilizing an automatic CPAP machine as compared to a standard CPAP machine (Mulgrew et al., 2007).

Design of the Study

The structure of the proposed study is a repeated measures research design that uses historical data from diagnostic testing completed post February 2002. Results should reveal a comparative relationship between the variables. The expectation is that the simplicity of the research design and absence of any risk of participation allows the data associated with previous sleep testing to be utilized for manipulation and analysis to study predictive relationships in the proposed methods of calculating sleep apnea severity along with relationship of sleep apnea severity to treatment with CPAP.

Procedures

Participants were identified by this researcher from the historical database of a private sleep disorders center in IL. All potential participants have given permission for pertinent data to be utilized for research purposes by signing an informed consent form prior to the recording of data (See Appendix A). Once participation has been confirmed as acceptable and not excluded, the participant's polysomnogram record was reviewed by this researcher for consistency with the current guidelines for scoring as published by the AASM. The number of apneas and hypopneas were documented along with the average

length of each category of events. In order to document the presence and calculate the

severity of OSA, I calculated the AHI in both the currently accepted manner as well as

the proposed alternatives: time-based, event based, and a combination of both time and

event-based (See Chapter 2: Calculating the AHI for details.) I retrieved the individual

ESS scores and then utilized them in the analysis of all four methods of calculating the

AHI. A download from the participant's CPAP machine was reviewed. Data within one

year of diagnosis is considered with the best 30 days of compliance utilized. The 95th

percentile pressure were retrieved for analysis with regards to all four methods of AHI

calculation.

Analysis

SPSS statistical software was used to analyze the data. Descriptive statistics were

performed on the demographics of the participants. For the analyses to follow, each

research question was considered separately.

First, I used within-subjects repeated measures ANOVA (rANOVA) to analyze

the AHIs as calculated by the proposed methods to determine if there is statistical

significance between each of the outcomes. The severity of departure from sphericity was

calculated using Machly's test to ensure that the variances of differences between the

conditions are equal, and that the statistical power is acceptable. Significance level

(*alpha*) was set at 0.05 to maintain a significance of 95%. If the *p-value* is greater than

0.05, the conclusion was that there the variances of differences are not statistically

significant and that the sphericity condition is met. If the *p-value* is less than 0.05, then

sphericity was violated and the appropriate correction was utilized. As the data being

manipulated were calculated in four proposed methods that are inherently different from one another, violation from sphericity was anticipated. As such, sphericity was not assumed and the conclusion was that there are significant differences between the variances of the differences.

Next, I completed linear regression utilizing the ESS score and each set of AHI data to determine which method represents the most statistically significant relationship between the level of daytime sleepiness and the severity of sleep apnea. The *p-value* was set at less than 0.05 to assess the appropriateness of the null hypothesis. If $p < 0.05$, the null hypothesis should be rejected. Furthermore, the method of AHI calculation with the highest b-coefficient calculated represents the strongest relationship with the ESS.

Finally, I utilized linear regression to analyze which method of AHI calculation represents the strongest relationship between the CPAP pressure and the severity of sleep apnea. The *p-value* was set at less than 0.05 to assess the appropriateness of the null hypothesis. If $p < 0.05$, the null hypothesis was rejected. Furthermore, the method of AHI calculation with the highest b-coefficient calculated represents the strongest relationship with the ESS score.

Independent and Dependent Variables

For the Repeated Measures ANOVA calculations, the independent variable is the method of AHI calculation, and the dependent variables are the AHIs. For the first Linear Regression analysis, the independent variable is the ESS scores while the dependent variables are the four calculated apnea Hypopnea Indices. For the second Linear

Regression analysis, the dependent variable is the CPAP Pressures while the independent variables are the four calculated AHIs.

Ethical Considerations

With the utmost respect for the APA standards for ethical protections in the treatment of human participants in research, I was able to adhere to the ethical guidelines set forth by Walden University and the appropriate Institutional Review Board (IRB). All private healthcare information will remain confidential at all times. Data will be maintained on the hard drive of this investigator's business computer along with an external hard drive for back up purposes for a period of not less than 5 years. The disposition of the data will be subject to the discretion of Walden University.

Chapter 4: Results

Introduction

The purpose of this study was to quantitatively investigate methods of calculating the severity of sleep apnea, and to determine the relationship of each AHI to both the ESS score, used to identify patients who may have sleep apnea, and the 95th percentile of pressure from the patient's automatic CPAP machine. First, I calculated the AHI in the currently accepted format as determined by the American Academy of Sleep Medicine (2015). Then, I calculated the AHI with consideration given to the length of the events experienced. Next, the type of events were weighted and the AHI was recalculated. And finally, the AHI was determined once again with both the length of the events considered along with weight given to the nature of the events experienced. I explored the correlation between the reported score on the ESS and each of these options to determine which option might accurately represent the patient's reported symptoms. Furthermore, I conducted a statistical analysis of the relationship between the 95th percentile of the automatic CPAP machine and the four AHI calculations.

This chapter commences with a description of the manner in which data were collected along with a synopsis of the participants' relevant demographic characteristics. A discussion of the descriptive statistics follows, along with the statistical results. The chapter concludes with a summary of the findings. The IRB approval number for this study is 10-23-17-0025870.

Participant Selection and Demographics

The participants of this study were selected from archived records from an independently-owned, private sleep disorders clinic in Illinois. This facility has demonstrated that all policies and procedures followed those recommended by the American Academy of Sleep Medicine (2017), the authority on sleep disorders testing and treatment. I examined the historical records of all individuals who had completed diagnostic polysomnography for the evaluation of a sleep disorder in the last 10 years. A database of individuals utilizing automatic CPAP therapy was made available to me in order to narrow down the participant selection. All individuals who had received a diagnosis of OSA, were prescribed automatic CPAP therapy, and pursued this treatment were considered for participation. I then thoroughly examined the patient's electronic medical record to document the age, gender, race, mental health history, medication usage, reason for testing, and compliance with treatment. An excel spreadsheet was completed to track this information.

I initially selected 193 participants from the archived records; however, 118 participants were excluded from participation due to the presence of exclusionary mental health disorders, age outside the stated range, non-compliance with PAP therapy, or missing data from the sleep study results. There were 75 participants who met all criteria for inclusion in the statistical analysis. Ultimately, each participant included in the data had previously received a diagnosis of OSA, was prescribed treatment with automatic CPAP therapy, pursued treatment with the automatic CPAP therapy machine, and met industry standards for usage compliance. Furthermore, no included participants reported a

history of traumatic or psychotic mental health disorders, use of sedative or stimulant

medications, or employment-related reasons for testing.

Research Questions and Hypotheses

The proposed research was designed to test the following null and alternative

hypotheses which have been derived from the review of the existing literature in the area

of sleep, sleep apnea, and subjective sleepiness:

Research Question #1

Is there a significant difference in the relationship between daytime sleepiness, as

measured by the ESS, and the severity of sleep apnea, as measured by the AHI when

calculated using the currently accepted formula, a time-based method, a weighted

method, or a combination of time-based and weighted methods?

Null Hypothesis #1

There is no significant difference in the relationship between daytime sleepiness,

as measured by the ESS, and the severity of sleep apnea, as measured by the AHI

when calculated using the currently accepted formula, a time-based method, a

weighted method, or a combination of time-based and weighted methods.

Alternative Hypothesis #1

There is a significant difference in the relationship between daytime sleepiness, as

measured by the ESS, and the severity of sleep apnea, as measured by the AHI

when calculated using the currently accepted formula, a time-based method, a

weighted method, or a combination of time-based and weighted methods.

Research Question #2

Is there a significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods?

Null Hypothesis #2

There is no significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

Alternative Hypothesis #2

There is a significant difference between the 95th percentile of CPAP pressure, as determined by an automatic CPAP machine, and the AHI, when calculated using the currently accepted formula, a time-based method, a weighted method, or a combination of time-based and weighted methods.

Demographics Analysis

I performed descriptive statistics on participant demographics. A summary of the age and gender of the participants is provided in Table 1. Of the 75 participants, the average age was 48 years with a standard deviation of 9.8 years; the gender ratio was nearly equal between males (1) and females (2) with a ratio equal to 1.40.

Table 1

Demographics (N = 75)

Variable	*n*	%
Sex		
Male	45	60
Female	30	40
Age (years)		
Mean	48	
Minimum	22	
Maximum	64	

Findings and Implications

In the following subsections, I first discuss the results of the repeated measures ANOVA. I then present the linear regression results, organized by research question. A separate analysis was completed for each research question.

Repeated Measures ANOVA

I performed repeated measures ANOVA to determine if there were significant differences in the severity level of sleep apnea, as evidenced by the AHI, when calculated by four separate methods:

1. AHI: The currently accepted format of summing the total number of apneas (A) and hypopneas (H) and then dividing by the TST.

2. AHI (t): Multiplying the apneas and hypopneas by the length of time (t) each event lasted and then dividing by 10 seconds, the current minimum amount of time a respiratory event must last, and then proceeding to divide by the TST.

3. AHI (w): Weighting apneas twice as heavily as the hypopneas, totaling the respiratory events and then dividing by the TST.

4. AHI (tw): A combination of the proposed timed and weighted calculations in which both scenarios are applied.

In Table 2, I present findings from multivariate tests. A power of 1.000 is a strong indicator of a significant effect. The relatively low value of the Wilks' Lambda (0.278) indicated statistically significant differences in the means of the four AHI calculations. Furthermore, the combination of a relatively low Wilks' lambda and a low significance level implies that there were statistically significant differences in the four AHI calculations, $F(3,72) = 62$, $p = 0.000$. I thus rejected the null hypothesis that the four calculations would not be statistically different from one another.

Table 2

Multivariate Tests

Effect	Value	F	Hypothesis df	Error df	Sig	Partial eta squared
Wilks' lambda	.278	62.322	3.000	72.000	.000	.722

Next, the sphericity of the rANOVA was computed and analyzed to determine if the variances of the differences between each of the four AHI calculations were equal. The assumption of sphericity was violated as assessed by Mauchly's test for sphericity ($W = 0.010$); however, I anticipated the potential for a violation of sphericity due to the nature of the experiment. Although this research design was within-subjects, the data were manipulated in a purposefully different manner with each new calculation of the AHI. Therefore, a significant difference in the variances of the AHIs is possible

depending on the average length of respiratory event as well as the type of respiratory events experienced.

Table 3

Mauchly's Test of Sphericity

Within subjects effect	*Mauchly's W*	*Approx chi square*	*df*	*Sig.*	*Greenhouse-Geisser*
AHIs_DV	.010	337.835	5	.000	.427

Regardless, I used the Greenhouse-Geisser correction to correct for the lack of sphericity. The *F* statistic *(F* = 110.559) remains unchanged, but the degrees of freedom was adjusted from 3 to 1.280. The Greenhouse-Geisser test statistic ($\varepsilon = 0.427$) provides a measure of departure from sphericity. Consequently, analysis of the significance revealed a $p < 0.001$, which implied that the results were statistically significant.

Table 4

Tests of Within-Subjects Effects

Source	*Type III sum of squares*	*df*	*Mean square*	*F*	*Sig.*	*Partial eta squared*
AHIs_DV Greenhouse-Geisser	295199.848	1.280	230693.001	110.559	.000	.599

Due to the violation of the sphericity assumption, a post-hoc analysis was completed using a pairwise comparison to establish within-subjects differences. A statistically significant difference was observed between each comparison of the four groups *(p* < .001). Therefore, Linear Regression was pursued to analyze the relationship of the dependent variables to each of the AHI calculations.

Prior to conducting a regression analysis, this researcher explored the assumptions for Linear Regression to ensure they were all met. First, sample size was verified using G Power statistical software to determine the total number of participants required for a medium effect size ($f^2 = 0.25$), *power* = 0.95 and *alpha* = 0.05. Results indicated that this linear multiple regression with four independent variables requires a minimum of 70 participants; therefore, a total of 75 participants meets this criterion (Lee & Wang, 2003). Next, independent variables cannot be multicollinear and must correlate to the dependent variables. To verify linearity, a scatterplot was graphed using all dependent and independent variables. This analysis revealed linear relationships between all of the variables. Consequently, the linearity assumption for regression analyses was met and could be performed with confidence.

To check for normal distribution of variables, I generated descriptive statistics using a histogram and normality plots (Young, 1993). The skewness and kurtosis were examined for each of the AHI calculated values. In the current method of calculation, the skewness was 0.413 and kurtosis was -0.598 with *z-values* of 0.114 and -1.091 respectively. In the timed method, skewness 0.359 and kurtosis was -0.730 with *z-values* of 1.296 and -1.332 respectively. In the weighted method, the skewness was 0.797 and the kurtosis was -0.259 with *z-values* of 2.877 and -0.473 respectively. In the combination method, the skewness was 0.787 and the kurtosis was -0.117 with *z-values* of 2.841 and -0.214 respectively. For ESS scores, skewness was 1.778 and kurtosis was 7.171 with *z-values* of 6.419 and 13.086 respectively. And finally, 95th Percentile CPAP

Pressures had a skewness of 3.344 and a kurtosis of 14.223 with *z-values* 12.072 and

25.954 respectively.

Table 5

Descriptive Statistics for all Variables

Variable	*Skewness*	*z-value*	*Kurtosis*	*z-value*
AHI (Current)	0.413	0.114	-0.598	-1.091
AHI (Timed)	0.359	1.296	-0.730	-1.332
AHI (Weighted)	0.797	2.877	-0.259	-0.473
AHI (Combo)	0.787	2.841	-0.117	-0.214
ESS	1.778	6.419	7.717	13.086
95th Percentile CPAP Pressure	3.344	12.072	14.223	25.954

The Kolmogorov-Smirnov results were then examined and reported: the Epworth

Sleepiness Score, the current AHI, and the AHI (timed) were all found to have normal

distribution with *p-value*s greater than or equal to 0.05; however, 95th Percentile CPAP

pressures, AHI (Weighted) and AHI (Timed & Weighted) had *p* values less than or equal

to 0.05 which indicates non-normality. Further analysis of this result demonstrates that

the non-normality of the data is likely due to the presence of outliers along with extreme

skewness and kurtosis of the data sets. However, the participants in this study do not

necessarily represent the average population. In fact, all participants have been diagnosed

with OSA and have been prescribed CPAP therapy as a treatment which inherently

indicates some degree of skewness in the severity of sleep apnea; therefore, the lack of

normality is an acceptable deviation. Visual inspection of the histograms generated

support the claim that the data is skewed, but otherwise normally distributed.

Furthermore, rANOVA is robust to normality and provides accurate results despite non-normality, especially with sample sizes greater than 30 (Lee & Wang, 2003).

Table 6

Tests of Normality

Variable	*Statistic*	*df*	*Sig.*
AHI (Current)	.090	75	.200
AHI (Timed)	.082	75	.200
AHI (Weighted)	.122	75	.008
AHI (Combo)	.123	75	.007
ESS	.083	75	.200
95th Percentile CPAP Pressure	.255	75	.000

Linear Regressions - Research Question #1

A Linear Regression was run utilizing the ESS and each set of AHI data to determine which method represents the best relationship between the level of daytime sleepiness and the severity of sleep apnea. A regression equation was found for each method of calculating the AHI.

For the current AHI method, regression yielded the equation F (1,73) = 0.069 with an R^2 of 0.020. Participants predicted AHI is equal to 42.210 + 0.151 where the ESS score is an ordinal measure. The AHI is increased 0.151 for each point increase on the ESS. However, the ESS was not a significant predictor of the AHI in the current method of calculation *(p = 0.793)*.

For the timed AHI method, regression yielded the equation F (1,73) = 0.320 with

an R^2 of 0.004. Participants predicted AHI is equal to 88.717 + 0.707 where the ESS

score is an ordinal measure. The AHI is increased 0.707 for each point increase on the

ESS. However, the ESS was not a significant predictor of the AHI in the timed method of

calculation *(p = 0.573)*.

For the weighted AHI method, regression yielded the equation F (1,73) = 0.001

with an R^2 of 0.000. Participants predicted AHI is equal to 56.439 - 0.020 where the ESS

score is an ordinal measure. The AHI is decreased 0.020 for each point increase on the

ESS. However, the ESS was not a significant predictor of the AHI in the weighted

method of calculation *(p = 0.982)*.

For the combo AHI method, regression yielded the equation F (1,73) = 0.095 with

an R^2 of 0.001. Participants predicted AHI is equal to 117.226 + 0.579 where the ESS

score is an ordinal measure. The AHI is increased 0.579 for each point increase on the

ESS. However, the ESS was not a significant predictor of the AHI in the combo method

of calculation *(p = 0.759)*.

Table 7

Linear Regression – Research Question #1

Variable	*df*	*F*	*R^2*	*Constant (y-intercept)*	*Predictive Factor (slope)*	*p-value*
AHI (Current)	73	0.069	0.020	42.210	0.151	0.793
AHI (Timed)	73	0.320	0.004	88.717	0.707	0.573
AHI (Weighted)	73	0.001	0.000	56.439	-0.020	0.982
AHI (Combo)	73	0.095	0.001	117.226	0.579	0.759

Linear Regressions - Research Question #2

Finally, Linear Regression was utilized to analyze which method of AHI calculation represents the strongest relationship between the 95th percentile CPAP pressure and the severity of sleep apnea.

For the current AHI method, regression yielded the equation F (1,73) = 0.02 with an R^2 of 0.000. Participants predicted PAP Pressure is equal to 12.079 – 0.004 (AHI) where the AHI score is an ordinal measure. The PAP Pressure is decreased 0.004 for each point increase of the AHI. However, the AHI in the current method of calculation was not a significant predictor of the CPAP Pressure *(p = 0.872)*.

For the timed AHI method, regression yielded the equation F (1,73) = 0.167 with an R^2 of 0.002. Participants predicted PAP Pressure is equal to 11.439 + 0.005 (AHI) where the AHI score is an ordinal measure. The PAP Pressure is increased 0.005 for each point increase of the AHI. However, the AHI in the timed method of calculation was not a significant predictor of the CPAP Pressure *(p = 0.684)*.

For the weighted AHI method, regression yielded the equation F (1,73) = 0.101 with an R^2 of 0.001. Participants predicted PAP Pressure is equal to 11.594 + 0.005 (AHI) where the AHI score is an ordinal measure. The PAP Pressure is increased 0.005 for each point increase of the AHI. However, the AHI in the weighted method of calculation was not a significant predictor of the CPAP Pressure *(p = 0.752)*.

For the combo AHI method, regression yielded the equation F (1,73) = 0.866 with an R^2 of 0.012. Participants predicted PAP Pressure is equal to 11.008 + 0.007 (AHI)

where the AHI score is an ordinal measure. The PAP Pressure is increased 0.007 for each

point increase of the AHI. However, the AHI in the combo method of calculation was not

a significant predictor of the CPAP Pressure *(p = 0.355)*.

Table 8

Linear Regression – Research Question #2

Variable	*df*	*F*	*R²*	*Constant (y-intercept)*	*Predictive Factor (slope)*	*p-value*
AHI (Current)	73	0.02	0.000	12.079	0.004	0.872
AHI (Timed)	73	0.167	0.002	11.439	0.005	0.684
AHI (Weighted)	73	0.101	0.001	11.594	0.005	0.752
AHI (Combo)	73	0.866	0.012	11.008	0.007	0.355

Summary

The present study employed a quantitative design with a biopsychosocial model

of application. Procedures involved analyzing historical data of participants who

underwent an overnight polysomnogram and were diagnosed with OSA. Each of the

participants then began treatment on automatic CPAP therapy, a device that uses

pressurized air to maintain the airway opening. An analysis of the relationship between

the symptoms reported prior to diagnostic testing and the severity of sleep apnea was

performed. Subsequently, an analysis was conducted on the relationship between the

severity of the sleep apnea and the amount of CPAP pressure required to treat the sleep

apnea.

Results of the rANOVA indicate a significant difference in the calculations of the

AHI; conversely, sphericity was violated due to the skewness and kurtotic tendencies of

the data. Although the variances of the differences between the related groups of data is not equal, this violation of sphericity is acceptable due to the vast differences in how the data are being calculated. Therefore, a visual inspection of the data was performed which revealed skewness, but otherwise seemingly normal results.

Regression analyses revealed neither a significant relationship between the ESS and the four AHI calculations nor a relationship between the AHIs and the 95th Percentile CPAP Pressure. On the other hand, there was stronger correlation between the ESS and timed method of calculating AHI *(p = 0.573)* as compared to the current method *(p = 0.793)*, the weighted method *(p = 0.982)*, or the combo method *(p = 0.759)*. Similarly, there was stronger correlation between the CPAP Pressure and combo method of calculating AHI *(p = 0.355)* as compared to the current method *(p = 0.872)*, the weighted method *(p = 0.752)*, or the timed method *(p = 0.684)*.

Although the outcomes of the statistical analyses demonstrate a non-significant predictive relationship between the ESS and the AHI, results did trend in a positive predicted direction in all cases. Furthermore, despite the non-significant predictive relationship between the AHI and the CPAP Pressure, results did trend in the predicted direction in the case of the timed, weighted and combination methods of calculating the AHI.

Chapter 5: Discussion, Conclusions, and Recommendations

Overview

The high prevalence of sleep apnea today is alarming, with an estimated 4% of men and 2% of women affected. Despite the increase in public awareness, researchers believed that only 10 to 20% of those affected actually seek diagnosis and treatment (American Sleep apnea Association, 2018). Untreated individuals have greater risk of motor vehicle or other accidents, substandard grades/learning, poor social relations, cardiac disease, stroke, depression, diabetes, and more (American Sleep apnea Association, 2018; Kim & Young, 2005; Maquet, 2001; Miller, 2005; Park et al., 2001; Punjabi, 2008).

Medical practitioners are charged with identifying patients who may have sleep apnea and then requesting diagnostic testing and treatment, if necessary. Historically, the correlation between a common screening tool, the ESS, and the presence/severity of sleep apnea, as defined by the AHI, has been inconsistent (Guimaraes et al., 2012). Similarly, past research has shown a poor correlation between the AHI and the prescribed treatment method, CPAP therapy, as defined by 95th percentile CPAP pressure (Cancelo et al., 2013). Those in the medical field are constantly searching for more precise screening tools along with more accurate predictive CPAP pressures in order to be more effective in diagnosing and treating their patients (Park et al., 2001).

One of the factors surrounding the relationship between the current methods of screening and treatment of sleep apnea is the way in which the severity of level of sleep apnea is calculated. Currently, all abnormal respiratory events experinced during sleep

are considered equal despite the length of the event as well as the type of event that has occurred. For example, if an individual stops breathing 20 times per hour for an average of 45 seconds per event, this would be undifferentiated from another individual with shallow breathing 20 times per hour for an average of 12 seconds per event. The question remains: should the severity levels of each individual be considered equivalent?

The purpose of this study was to quantitatively investigate alternative methods of calculating the severity of sleep apnea and to determine the relationship of each AHI to the ESS score, the current screening tool used to identify patients who may have sleep apnea, as well as the 95th percentile of pressure from the patient's automatic CPAP machine. The correlation between the reported score on the ESS and each of four proposed methods of calulating the presence and severity of sleep apnea, the AHI, was explored to determine which method most accurately represents the patient's reported symptoms. Furthermore, I investigated a statistical analysis of the relationship between the 95th percentile of the automatic CPAP machine and the four AHI calculations as well.

Interpretation of Findings

In this research study, multivariate statistical tests showed a significant difference bewteen the AHI values as caluated by the four separate methods. However, linear regression results did not support a strong correlation between either the ESS and the AHI calculations or the 95th percentile pressure and the AHI calculations. Still, the time method of calculating the AHI was most strongly correlated with the ESS; likewise, the combination method of calculating the AHI was most strongly correlated with the 95th percentile CPAP pressure.

Although the outcomes of the statistical analyses demonstrate a non-significant predictive relationship between the ESS and the AHI, results did trend in a positive predicted direction in all four cases. This would generally indicate that as the ESS score increases, so does the AHI. The strongest correlation was seen in the regression analysis of the time-based AHI calculation and the ESS; the time-based method $(p = 0.573)$ is statistically more significant than the current method of calculating the AHI $(p = 0.793)$ as well as the weighted method $(p = 0.982)$ and the combination method $(p = 0.759)$. A stronger relationship between the individual's symptoms and diagnosis may imply that the time-based method of calculating the AHI is more accurate at depicting the individual's level of sleep apnea. However, just as the previous research indicated on the relationship between the ESS score and AHI, the relationship is still not strong. As a result, the ESS cannot be used to predict the presence and/or severity of sleep apnea in any of the proposed methods of calculation. Consequently, the ESS may be one tool in the arsenal of medical practitioners use to treat their patients, but it certainly should not be the only screening instrument in the case of sleep apnea.

Furthermore, despite the non-significant predictive relationship between the AHI and the CPAP pressure, results did trend in the predicted direction in the case of the timed, weighted, and combination methods of calculating the AHI. This would generally indicate that as the AHI increases, so does the CPAP pressure. The strongest correlation was seen in the regression analysis of the combination method of the AHI calculation and the 95th percentile CPAP pressure; the combination method $(p = 0.335)$ is statistically more significant than the current method of calculating the AHI $(p = 0.872)$ as well as the

weighted method *(p = 0.752)* and the time method *(p = 0.684)*. A stronger relationship

between the individual's diagnosis and treatment may imply that the combination method

of calculating the AHI is more accurate at predicting the individual's treatment needs.

However, just as the previous research had indicated regarding the relationship between

the CPAP pressure and AHI, the relationship is still not statistically significant. As a

result, none of the proposed methods of calculating the AHI can be used to predict the

prescribed CPAP pressure. Consequently, the proposed methods of calculating the AHI

may need adjustment in order to more accurately predict treatment. Conversely, perhaps a

study of the proposed AHI calculations could be done on a larger scale to minimize the

outliers that could potentially skew the data. However, it is possible that there simply will

never be a correlation between the severity of one's sleep apnea and the most appropriate

CPAP pressure. Regardless, the current method of calculating the AHI is certainly not the

most accurate of the four proposed methods.

Implications for Social Change

This study indicates that the current method of calculating the severity of sleep

apnea does not have a strong correlation between an individual's symptoms or treatment.

Due to a high prevalence of this disorder along with widespread complications of the

disorder (such as other medical problems, motor vehicle accidents, learning and memory

problems, and more), proper screening, diagnosis, and treatment is crucial to a healthy

society.

Serving as liaisons between sleep disorders centers and patients, healthcare

practitioners are expected to recognize the possibility of a sleep disorder during a

patient's physical exam. However, the current screening tool is not as useful as expected; the ESS is not highly correlated with the presence of a sleep disorder, especially sleep apnea (Guimaraes et al., 2012). Without a useful screening tool, healthcare practitioners may not be able to assess the likelihood of a sleep disorder and therefore may not be able to intervene in those without obvious signs and symptoms. By examining the way medical practitioners screen for sleep disorders and finding stronger correlations between screening tools and diagnoses, healthcare practitioners would be able to screen for sleep disorders more accurately and efficiently, and more patients with the disorder would be recognized and treated. In essence, these healthcare practitioners would be able to provide patients with better overall medical care along with improved quality of life.

Furthermore, proper screening of individuals with a potential sleep disorder would result in earlier detection and earlier treatment of sleep apnea. Not only would this improve individual health, but it would also contribute to a reduction in healthcare costs. By identifying and treating sleep apnea early, other consequences of untreated sleep apnea, such as hypertension, cardiac problems, strokes, diabetes, depression, motor vehicle accidents, and so on, may be prevented or minimized. However, screening is not the ultimate solution. Once diagnosed, accurate treatment is required in order to bring about all of the positive benefits discussed here. If the severity level of sleep apnea could accurately predict treatment protocol, individuals would receive the proper treatment in a timelier manner and would also require fewer visits to their healthcare practitioners. Comorbidities may be prevented or at least minimized. In addition, this could bring about other social benefits such as a reduction in automobile or work-related accidents due to

sleepiness, improvement in academic or workplace performance, and an improvement in social interactions between all of us. Consequently, more efficient screening and treatment for sleep apnea could result in a massive long-term cost reduction along with a happier, healthier society.

Limitations and Recommendations for Future Studies

One of the limitations of this study involved the use of historical data. Because I retrieved the data from archived records, none of the individuals I used in this research could be medically screened in a universal fashion to ensure that the individuals did not have conditions that affect the results. Since patients do not always report all medications and medical history accurately, the results of testing and treatment could have been affected; furthermore, patients may have undiagnosed conditions that could also affect results from one day to the next. Additionally, every patient experiences symptoms differently, so reported symptoms could vary from day to day. The patients available to me may have been different in health, medication use, and so on, which may have affected the results. Unforeseen comorbid conditions, a patient's diet the day of the overnight sleep study, the previous few nights of sleep prior to diagnostic testing, and/or other factors may have ultimately altered the results obtained in this small scale research study. Sleep study results could be significantly worse on one day versus another. And finally, the subjective technician influence when identifying respiratory events is always a factor in the calculation of one's AHI.

A new research design may be able to control for some of these factors if planned prior to the study. A proposed study would include participants who may be identically

screened for current health. Current vitals, blood tests, medication list, and other medical records could be obtained for each participant to identify other conditions that may influence the reporting of symptoms, the severity of sleep apnea, and the use of CPAP therapy. This could include categorizing participants by a primary health concern, or by simply eliminating participants with any major medical concerns.

Because of weak correlation between the responses on a subjective screening tool, the ESS, and the AHI, an objective measure of symptoms might be considered in future research to see if there is a stronger correlation. For example, hypertension and sleep apnea are highly correlated (Leung & Bradley, 2001; Punjabi, 2008). Obtaining consistent blood pressure checks on participants before during and after testing may provide a measure of a participant's objective symptoms rather than subjective symptoms. As blood pressure checks are commonplace for most physician visits, a potential correlation between blood pressure and the severity or treatment of sleep apnea could reveal a more accurate screening measure for healthcare practitioners. Furthermore, many other medical disorders have been correlated with the presence of sleep apnea; there may be a correlation between sleep apnea severity and/or treatment based on the presence of other medical disorders as well.

In addition, future research may consider the use of home sleep tests with automatic respiratory scoring instead of in-lab polysomnography. Home sleep testing has continually improved in accuracy over the years and is more widely accepted now than ever. The use of a respectable device that has proven accuracy in respiratory event detection would eliminate the subjectivity of scoring results by a technician. Results may

be more consistent when utilizing the exact same algorithm of scoring programmed by a computer rather than a human. The use of home sleep testing is also significantly less expensive than in-lab testing, thereby reducing the costs of research. Furthermore, home sleep testing is portable and may be mailed to potential participants. The portability factor along with reduced costs would allow for a larger, more diverse pool of participants to minimize the presence of individual deviation form normalcy.

Another factor that could affect the correlation between AHI and CPAP pressure involves the types of respiratory events used in the AHI calculation. Currently, only apneas and hypopneas are included in the AHI calculation. However, there are other respiratory events defined by the American Academy of Sleep Medicine (AASM, 2012) such as RERAs, or respiratory event related arousals. The overall respiratory disturbance index (RDI) may have a different relationship to the diagnosis and treatment of sleep apnea. Future studies may consider the inclusion of all respiratory events acknowledged by the AASM.

Conclusions

Sleep has become increasingly recognized as a crucial vital sign (Grandner & Malhotra, 2015). Not only have we learned the basics about sleep cycles, rhythms, and sleep disorders, but we have also discovered the impact sleep has on many physical and psychological functions including immune system functioning; muscle and tissue repair; learning and memory; growth and development; and so much more (Kryger et al., 2000). There is a high prevalence of sleep disorders in American society, which is often

correlated with medical comorbidities and social consequences. Sleep apnea, the cessation of airflow during sleep, is one of the most common sleep disorders today. This study explores the method of calculating the severity of sleep apnea (AHI) and the relationship to a screening tool (ESS) and treatment method (CPAP 95th Percentile Pressure) we use today.

The statistical analyses in this research study indicate a significant difference in the calculations of the AHI; however, results do not support a definitive correlation between the methods of calculating the AHI and the ESS score or the 95th Percentile CPAP pressure. On the other hand, when analyzing the relationships between the AHIs and the ESS score, the strongest relationship was seen in the time-based AHI calculation and the ESS score. When analyzing the relationships between the AHIs and the 95th Percentile CPAP Pressure, the strongest correlation was seen in the combo method and the 95th percentile CPAP pressure. The informed conclusion would be that the current method of calculating the severity level of sleep apnea should be altered to provide more accurate relationships between the screening tools that represent an individual's symptoms along with the treatment method used today.

Although the outcomes of this research demonstrate a non-significant predictive relationship between the ESS and the AHI as well as the AHI and 95th Percentile CPAP pressure, this research has added value to field of sleep medicine. Stronger relationships between the proposed AHI calculations and diagnostic/treatment tools indicates the need for a significant change in this industry. The current method of AHI calculation is evidently not the most effective way to classify one's sleep apnea. A comprehensive

change to the rules should commence as soon as possible in order to provide more

effective screening and treatment of sleep apnea in today's society. However, future

research is required to identify the most accurate way to proceed.

References

Antic, N. A., Catcheside, P., Buchan, C., Hensley, M., Naughton, M. T., Rowland, S., & McEvoy, R. D. (2011). The effect of CPAP in normalizing daytime sleepiness, quality of life, and neurocognitive function in patients with moderate to severe OSA. *Sleep, 34,* 111-119. pmid: 21203366

Ayas, N. T., Patel, S. R., Malhotra, A., Schulzer, M., Malhotra, M., Jung, D., Fleetham, J., & White, D. P. (2004). Auto-titrating versus standard continuous positive airway pressure for the treatment of OSA: results of a meta-analysis. *Sleep*: 27, 249-253. pmid: 15124718

Borrell-Carrio, F., Suchman, A. L,. & Epstein, R.M. (2004). The biopsychosocial model 25 years later: principles, practice and scientific inquiry. *Annals of Family Medicine*, *2*(6), 576-82. doi: 10.1370/afm.245

Cancelo, L., Hernandez, V., Bravo, D., Martinez, C., Egea, C., & Duran, J. (2013). CPAP titration: Correlation between 2 different equations and autopap. *Sleep Medicine*, *14*(1), 87-90.

Dempsey, J. A., Veasey, S. C., Morgan, B. J., & O'Donnell, C.P. (2010). Pathophysiology of sleep apnea. *Physiology Review*, *90*(2), 797-8. pmid: 20086074

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, *196*(4286), 129-136. doi: 10.1126/science.847460

Epstein, L. J., Kristo, D., Strollo Jr., P. J., Friedman, N., Malhotra, A., Patil, S. P., Ramar, K., Rogers, R., Schwab, R. J., Weaver, E. M., & Weinstein, M. D. (2009).

Clinical guideline for the evaluation, management and long-term care of OSA in adults. *Journal of Clinical Sleep Medicine*, 5, 263-276. PMID: 19960649

Gay, P., Weaver, T., Loube, D., & Iber, C. (2006). Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. *Sleep*, *29*, 381-401. pmid: 16553025

Guimares, C., Martins, M.V., Vaz Rodrigues, L., Teixeira, F., & Moutinho Dos Santos, J. (2012). Epworth Sleepiness Scale in obstructive sleep apnea syndrome – an underestimated subjective scale. *National Institutes of Health: 18*(6): 267-71. pmid: 22743061

Idzikowski, C. (1984). Sleep and memory. *British Journal of Psychology*, *75*(4), 439-449. doi: 10.1111/j.2044-8295.1984.tb01914.x

Johns, M. (1991). A new method for measuring daytime sleepiness: The Epwortth Sleepiness Scale. *Sleep, 14,* 540-545. pmid: 1798888

Johns, M. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep, 15*(4), 376-381. pmid: 1519015

Kim, H. & Young, T. (2005). Subjective daytime sleepiness: Dimensions and correlates in the general population. *Sleep, 28*(5), 625-634. pmid: 16171277

Laberge, L., Petit, D., Simard, C., Vitaro, F., Tremblay, R. E., & Montplaisir, J. (2001). Development of sleep patterns in early adolescence. *Journal of Sleep and Respiratory Medicine, 10*(1), 59-67. doi: 10.1046/j.1365-2869.2001.00242.x

Leung, R.S. & Bradley, T.D. (2001). Sleep apnea and cardiovascular disease. *American Journal of Respiratory Critical Care Medicine*, *164*(12), 2147-65. doi: 10.1164/ajrccm.164.12.2107045

Lim, P. & Curry, A. (2000). The role of history, ESS score and body mass index in identifying non-apnoeic snorers. *Clinical Otolaryngology, 25,* 244-248. doi: 10.1046/j.1365-2273.2000.00351.x

Mauchly, J. W. (1940). Significance test for sphericity of a normal n-variate distribution. *The Annals of Mathematical Statistics*, *11,* 204-209. pmid: 2235878

Mitru, G., Millrod, D. L., & Mateika, J. H. (2002). The impact of sleep on learning and behavior in adolescents. *Teachers College Record, 104*(4), 704-26. pmid: 258443682

Mulgrew, A. T., Cheema, R., Fleetham, J., Ryan, C. F., & Ayas, N. T. (2007). Efficacy and patient satisfaction with auto adjusting CPAP with variable expiratory pressure vs standard CPAP: A two-night randomized crossover trial. *Sleep & Breathing, 11*(1), 31-37. pmid: 17053928

Oksenberg, A., Arons, E., & Froom, P. (2006). Does the severity of obstructive sleep apnea predict patients requiring high continuous positive airway pressure? *The Laryngoscope, 116*(6), 951-955. doi: 10.1097/01.MLG.0000215833.68519.7B

Osman, E., Osbourne, J., Hill, P., & Lee, B. (1999). The Epworth Sleepiness Scale: Can it be used for sleep apnea screening among snorers? *Clinical Otolaryngology 24,* 239-241. doi: 10.1046/j.1365-2273.1999.00256.x

Park, E., Nasser, R. L., & Mallari, P. (2001). Heart failure assessment with impedance respiration sensor. *Europace*, *6*(24-27), 561-565. pmid: 25917747

Prasad, B., Carley, D. W., & Herdegen, J. J. (2010). Continuous positive airway pressure device-based automated detection of obstructive sleep apnea compared to standard laboratory polysomnography. *Sleep & Breathing, 14,* 101-7. doi: 10.1164/rccm.201307-1282ST

Punjabi, N. (2008). The epidemiology of adult obstructive sleep apnea. *Proceedings of the American Thoracic Society, 5*(2), 136-143. doi: 10.1513/pats.200709-155MG

Robert, C., Wilson, C. S., Gaudy, J., & Arreto, C. (2006). A year in review: Bibliometirc glance at sleep research literature in medicine and biology. *Sleep and Biological Rhythms*, *4*, 160-170. doi: 10.1111/j.1479-8425.2006.00211.x

Rosenthal, L., Gerhardstein, R., Lumley, A., Guido, P., Day, R., Syron, M.L., & Roth T. (2000). CPAP therapy in patients with mild OSA: implementation and treatment outcome. *Sleep Medicine*: *1*, 215. pmid: 10828432

Sawyer, A.M., Gooneratne, N.S., Marcus, C.L., Ofer, D., Richards, K.C., & Weaver, T.E. (2011). A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. *Sleep Medicine Rev*iew, *15*(6), 343-56. doi: 10.1016/j.smrv.2011.01.003

Sheldon, S.H., Riter, S., & Detrojan, R. (1999). Atlas of sleep medicine in infants and children. *Psychosomatic Research*: *49*(5), 160-4.

Tryon, W.W. (1996). Nocturnal activity and sleep assessment. *Clinical Psychology Review, 16*(3), 197-213. doi: 10.1016/0272-7358(95)00059-3

Ueno, K., Kasai, T., Brewer, G., Takaya, H., Maeno, K., Kasagi, S., Kawana, F., Ishiwata, S., & Narui K. (2010). Evaluation of the apnea hypopnea index determined by the S8 auto-CPAP, a continuous positive airway pressure device, in patients with obstructive sleep apnea-hypopnea syndrome. *Journal of Clinical Sleep Medicine: 1*0;6 (2), 146-151. doi: 10.1007/s11325-012-0672-8

U.S. Department of Health and Human Services. (2003). *2003 National Sleep Disorders Research Plan*. Bethesda, MD: National Heart, Lung, and Blood Institutes of Health.

Weaver, E. (2005). Self-identified sleep apnea is associated with subjective sleepiness. *Sleep. 28*, 620, A208.

Weaver, E. & Grunstein, R. R. (2008). Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *American Thoracic Society. 5*(2), 173. doi: 10.1513/pats.200708-119MG

Weaver, T.E., Maislin, G., Dinges, D.F., Bloxham, T., George, C.F., Greenberg, H., Kader, G., Mahowald, M., Younger, J., & Pack, A.I. (2007). Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. *Sleep*: *30*, 711. pmid: 17580592

Young K. D. S. (1993). Bayesian diagnostics for checking assumptions of normality. *Journal of Statistical Computation and Simulation*, *47*(3–4), 167–180. doi: 10.1080/00949659308811528

Nonperiodical (Book):

Berry, R.B., Brooks, R., Gamaldo, C.E., Harding, S.M., Lloyd, R.M., Quan, S.F., Troester, M.M., & Vaughn, B.V.; for the American Academy of Sleep Medicine. (2017). The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Darien, IL: AASM.

Haba-Rubio, E. & Krieger, A. (2012). Chapter 2: Evaluation Instruments for Sleep Disorders: A Brief History of Polysomnography and Sleep Medicine. *Introduction to modern sleep technology*, (pp. 296). New York, NY: Springer Science.

Halligan, P.W. & Aylward, M. (2006). *The power of belief: Psychosocial influence on illness, disability and medicine*. Oxford University Press, UK.

Lee, E. & Wang, J. (2003). *Statistical Methods for Survival Data Analysis*, 3rd edition. Wiley-Interscience.

Internet-based (Websites):

American Academy of Sleep Medicine. (2017). Scoring manual. Retrieved from https://aasm.org/clinical-resources/scoring-manual/ on December 1, 2017.

American Sleep Apnea Association. (2018). Sleep apnea information for clinicians. Retrieved from https://www.sleepapnea.org/learn/sleep-apnea-information-clinicians/

Downey, R., Rowley, J.A., Wickramasinghe, H. & Gold, P.M. (2016). Obstructive Sleep

Apnea: practice essentials, background, pathophysiology. Medscape: Retrieved

from www.emedicine.medscape.com on August 14, 2016.

Morgan, C.E. & Lindman, J.P. (2015). Surgical approach to snoring and sleep apnea.

Medscape: Retrieved from www.emedicine.medscape.com on March 5, 2017.

Appendix A: Epworth Sleepiness Scale

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. Use the following scale to choose the most appropriate number for each situation:

0 = no chance of dozing

1 = slight chance of dozing

2 = moderate chance of dozing

3 = high chance of dozing

TYPE OF SITUATION	CHANCE OF DOZING
Sitting and reading	
Watching TV	
Sitting inactive in a public place (i.e. a theater or meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon (if circumstances permit)	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in traffic	
TOTAL	

Appendix B: AASM Scoring Manual Guidelines

The following includes the rules and definitions for scoring respiratory events:

MEASURING EVENT DURATION

1. For scoring either an apnea or a hypopnea, the event duration is measured from the nadir preceding the first breath that is clearly reduced to the beginning of the first breath that approximates the baseline breathing amplitude.

2. For apnea duration, the oronasal thermal sensor signal (diagnostic study) or CPAP device flow signal (CPAP titration study) should be used to determine the event duration. For hypopnea event duration, the nasal pressure signal (diagnostic study) or CPAP device flow signal (CPAP titration study) should be utilized. When the diagnostic study sensors fail or are inaccurate, alternative sensors may be used.

3. When baseline breathing amplitude cannot be easily determined (and when underlying breathing variability is large), events can also be terminated when either there is a clear and sustained increase in breathing amplitude, or in the case where a desaturation has occurred, there is event-associated resaturation of at least 2%.

SCORING OF APNEAS

1. Score a respiratory event as an apnea when BOTH of the following criteria are met:
    a. There is a drop in the peak signal excursion by ≥90% of pre-event baseline using an oronasal thermal sensor (diagnostic study), CPAP device flow (titration study) or an alternative apnea sensor (diagnostic study).
    b. The duration of the ≥90% drop in sensor signal is ≥10 seconds.

2. Score an apnea as <u>obstructive</u> if it meets apnea criteria and is associated with continued or increased inspiratory effort throughout the entire period of absent airflow.

3. Score an apnea as <u>central</u> if it meets apnea criteria and is associated with absent inspiratory effort throughout the entire period of absent airflow.

4. Score an apnea as <u>mixed</u> if it meets apnea criteria and is associated with absent inspiratory effort in the initial portion of the event, followed by resumption of inspiratory effort in the second portion of the event.

SCORING OF HYPOPNEAS
Scoring hypopneas as central or obstructive events is optional. Choose 1A <u>or</u> 1B when scoring hypopneas.

1A. Score a respiratory event as a hypopnea if ALL of the following criteria are met:
    a. The peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure (diagnostic study), CPAP device flow (titration study), or an alternative hypopnea sensor (diagnostic study).
    b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
    c. There is a ≥3% oxygen desaturation from pre-event baseline or the event is associated with an arousal.

1B. Score a respiratory event as a hypopnea if ALL of the following criteria are met:
    a. The peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure (diagnostic study), CPAP device flow (titration study), or an alternative hypopnea sensor (diagnostic study).
    b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
    c. There is a ≥4% oxygen desaturation from pre-event baseline.

2. If electing to score obstructive hypopneas, score a hypopnea as <u>obstructive</u> if ANY of the following criteria are met:
    a. There is snoring during the event.
    b. There is increased inspiratory flattening of the nasal pressure or CPAP device flow signal compared to baseline breathing.
    c. There is an associated thoracoabdominal paradox that occurs during the event but not during pre-event breathing.

3. If electing to score central hypopneas, score a hypopnea as <u>central</u> if NONE of the following criteria are met:
    a. There is snoring during the event.
    b. There is increased inspiratory flattening of the nasal pressure or CPAP device flow signal compared to baseline breathing.
    c. There is an associated thoracoabdominal paradox that occurs during the event but not during pre-event breathing (Berry et al., 2017, Section VIII, Part 1).